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REVIEW ARTICLE

Epidemiology and prediction models for themanagement of rapeseed–mustard diseases: current status and future needs

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Abstract

The productivity of rapeseed-mustard in India is quite low as compared to the world scenario. It is mainly due to important diseases, Alternaria blight, white rust, downy mildew, powdery mildew, and white or Sclerotinia rot. Knowledge of epidemiologyand forecasting provide the basic information to developefficient and workable plant disease control models. The various weather variableslike temperature (T), relative humidity (RH), rainfall, wind velocity, and direction, leaf wetness duration, and solar radiation influence differentparameters of infection process, and disease development. Interaction between these weather variables and disease development pave the way for the development of the prediction models. Prediction models developed for the management of important diseases of rapeseed-mustard revealed that Alternaria blight is favoured by Tmax of 20–25 °C, Tmin of 15 °C, RHmor > 90% and RHeve > 50% where as white rust influencedby > 15 °C and RH > 65% with intermittent rains. Similarly, for downy mildew, Temprange of 15–20 °C with high RH was considered optimal for its progress. Leaf wetness duration of 4–6 h at 20 °C and 6–8 h at 15 °C is essential to initiate the downy mildew infection. Stag-head due to mixed infection of downy mildew and white rust is favoured by a Temp 20 °C with high RH and reduced period of sunshine (2–6 h/day) with rainfall up to 161 mm. Powdery mildew development is favoured by Temprange of 16–28 °C, mean RH < 60% and dry weather during February–March. The Sclerotinia stemrot progression is favoured by high RH (> 80%), Tmax up to 25 °C and Tmin of 5–12 °C. Often prediction models developed at one location may not fit atother locations. It indicates that data needs to be generated for a longer period and the model be tested atMultilocation. The disease-forecasting models must be developed by taking into account the crop variety, the prevalence of a particular pathotype and the microclimatic factors.

Keywords Epidemiology · Prediction models · Rapeseed-mustard diseases · *Alternaria* · *Albugo* · *Hyaloperonospora* · *Erysiphe* · *Sclerotinia*

Introduction

The severe attack of many diseases not only deteriorates the quality of the seed but reduces the oil content considerably in different oil yielding *Brassica* crops. More than 30 diseases are known to occur on *Brassica* crops in India. Amongst these, Alternaria blight (*Alternariabrassicae* (Berk.) Sacc.), white rust (*Albugo candida* (Pers.) ex. Lev.), downy mildew (*Hyaloperonosporaparasitica*(Pers. ex Fr.) Fr.), powdery mildew (*Erysiphecruciferarum*Opiz ex. Junell), and white or Sclerotinia stem rot (*Sclerotiniasclerotiorum*(Lib.) deBary)are considered economically important.Most of the commercially grown varieties are susceptible or moderately susceptible to these diseases and chemical sprays are only means to manage them. However, studies have been conducted to develop a suitable prediction models for adopting timely protection measures. In plant pathology, to study disease progress over time, where time (*t*) is modeled as a continuous variable rather than as a discrete variable. Many different population growth models have been used for modeling disease progress curves. Five common growth curve models such as Exponential, Monomolecular, Logistic, Gompertz, and Weibull Modelsare being used for the development of prediction model along with their assumptions. For the illustration of some of these models in R, (Table 1) which compared different models for disease progress, based on nonlinear

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regression analysis. The initial disease incidence is $y_0 = 1/(13*45) = 0.0017$ (another exp.) s $y_0 = 1/(15*67) = 0.001$ are given in Table 1.

A prediction model is a simplificationof reality and attempts to summarize the mainprocesses, to put forward hypotheses as well as to verify theircoherence and consequences. It also represents a trial todetermine the minimal hypotheses which would allowminimal mathematical representation of real processes.Epidemiological models can be classified in severalways. Kranz and Royle (1978)classified them into three types–descriptive, predictiveand conceptual—according to their mainobjective. Descriptive models provide hypotheses orgeneralize experimental results, but they do not usuallyreveal the mechanisms underlying the processes.Predictive models, which are also descriptive, allowthe prediction of the occurrence and the severity ofepidemics. Both descriptive and predictive models usemathematical tools, such as simple or complex functions, regression and differential equations, or simpledecision models. The conceptual models, also knownas explanatory or analytical models, allow the identificationof problems by distinguishing cause fromeffect and quantify the effects of specific events onepidemic development. They are constructed as representationsof underlying biological and ecologicalprocesses. These models may eventually lead to thedevelopment of complex simulation models. It shouldbe pointed out that models can be disease-specific, butcan also be very general. A descriptive model is oftenconcerned with understanding and predicting developmentof specific diseases, and thus is generally used forassisting growers in making tactical decisions in managingdiseases. A conceptual model is often concernedwith the theoretical understanding of generic featuresof epidemic development and thus is used more formaking policy and strategic decisions.The advances in computer tools have made mathematical/statistical modeling more accessible and have led to the development of more complex modelsfor many diseases.

The importanceof the interactions between pathogen and hostpopulation dynamics has long been underestimated and is now require due attention. The various observations required to develop a prediction models has been discussed as under:

Inoculum: Pathogen inoculum is of prime concern; its source, densityand type will greatly influence the design of theforecasting scheme.The population canbe described by the proportion or absolute quantityof individuals at each stage, i.e. age structure of the population. For modeling purposes, pathogen may consist ofthree components: (1) spores, each potentially capableof infection, (2) mycelia, and (3) resting structure. Passage from one stage to the next can be veryfast, usually depending on environmental conditions. Inoculum may also be simply divided into primary andsecondary; which is very usefulin modeling soil-borne pathogens. Theimportance of the life-cycle stages in the control of fungaldynamics, by adapting a patch-occupancy model. Fungal populations are difficult to study under fieldconditions because individual mycelia and spores cannotgenerally be easily quantified. Theassessment of the amount of the pathogen on and in theleaves is difficult. Visual assessment of the percentage leaf area coveredby lesions can be rapid, but is quite subjective, andhence, may not be a reliable assessment of inoculum. Numbers of coloniesor pustules can be counted accurately on a small scale, but this is also not practical on a large scale. A pathogen population is often measuredindirectly as disease incidence or severity.

Inoculum dispersal: Inoculum dispersal fulfils essentially three functions: (1) population survival, (2) colonization of new habitatsand (3) reproduction. Spore dispersalcomprises three phases; liberation, which can be passiveor active, transport, anddeposition. The dispersal scale dependson inoculum properties as well as the transport vector, and may range from a few meters through rain splash, to 100–10,000 m for airborne spores. Often, spore dispersal can bedescribed by either an exponential or a power function.Ferrandino (1993) derived a functionto account for loss of spores both by escape from thecanopy and by deposition. The model generated diseasegradients that became shallower as the epidemics progressed.Such a pattern has been observed in diseasedcrops. Theefficiency of spore dispersal affects the density of newinfections. Gourbiereet al. (1999) considered dispersal as the main parameterwhich determines the number of newly colonized units in their model, which has been further extended to simulate the distribution and frequency of new infections along weather gradients.

Table 1 The comparison of different model curves for disease progression based on nonlinear regression analysis

Growth Curve Models			
Model	Differential Equation Form	Integrated Form	Linearized Form
Exponential	$\log y = \log y_0 + rt$	$y = y_0 \exp(rt)$	$\log y = \log y_0 + rt$
Monomolecular	$\ln\{1/(1-y)\} = \ln\{1/(1-y_0)\} + rt$	$y = 1-(1-y_0)\exp(-rt)$	$\ln\{1/(1-y)\} = \ln\{1/(1-y_0)\} + rt$
Logistic	$y = 1/[1 + \{-\ln y_0/(1-y_0) + rt\}]$	$y = 1/[1 + \exp\{-\ln y_0/(1-y_0) + rt\}]$	$\ln(y/(1-y) = \ln\{y_0/(1-y_0) + rt\}$
Gompertz	$dy/dt = ry \ln(1/y) = ry(-\ln y)$	$y = \exp(\ln y_0 \exp(-rt))$	$-\ln(-\ln y) = -\ln(-\ln y_0) + rt$
Weibull	$dy/dt = c/b\{(t-a)/b\}^{(c-1)\exp[-\{(t-a)/\mathrm{b}\}c]}$	$y = 1-\exp[-\{(t-a)/b\}^\wedge c]$	$\ln[\ln\{1/(1-y)\}] = -c\ln b + \ln(t-a)$

Latent and infectious periods: Latent period is the intervalbetween the onset of spore germination and the appearanceof the next spore generation. The rate of epidemicdevelopment is largely influenced by the length oflatent period, which determines the number of potentialinfection cycles that can be completed during agrowing season. Theshorter the latent period, the more reproduction cycles, the pathogen can have in a season mostly in polycyclic diseases. The monocyclic diseases have only onereproductive cycle throughout a single season.Latent periods have beenreported to depend on inoculum and lesion density, but are mainly influencedby temperature and humidity. They mayalso vary with the level of host susceptibility and withhost growth stages, features that emphasize the importanceof studying both pathogen and host dynamics.Another key factor influencing the development of anepidemic is the length of the infectious period as this determines the quantity ofspores that a single colony is likely to produce duringits lifetime.

Pathogen dynamics and regulation: The role of predation andparasitism in pathogen regulation might be greater thanpreviously recognized.Another natural regulation comes from pathogenpopulation competition. The aspect of microbial communityinteraction (symbiosis or competition) is, ingeneral, poorly understood for fungal pathogens; itis now becoming gradually more important with thepresent moves to more integrated disease managementstrategies.

Host dynamics

The epidemic modeling has emphasized pathogen activity, ignoring effects of the host onpathogen development. Particular interests are changesin susceptibility, and the contribution of resistance, e.g., to the length of the latent period. Another reason why host dynamicsshould be included in epidemiological models arisesfrom the fact that pathogen population dynamics arelinked to host dynamics, and pathogens may affectgrowth and reproduction of their hosts. Simple models can be developed tocapture the essential features of host–pathogen interactions, though more complex models are usuallynecessary.

Host susceptibility and resistance: Among the host factors which need to be taken intoaccount are the levels of intrinsic host resistanceand age-related resistance associated with specifichost tissues. Some cultivars displayincreased tolerance or partial resistance. The nature of host resistancewill affect the rate of disease development and musttherefore be taken into consideration in modeling. Theoretical models have beendeveloped on the effects of cultivar mixtures or cropheterogeneity on epidemic development based onthe gene-for-gene relationship. Host resistanceand/or pathogen infectivity/aggressiveness mayalso depend on the age of host tissues.

Multiple hosts and crop rotation: Paramount among farm practices is crop rotation, which has conventionally been adopted to reduce thecarry-over of pathogens from one crop to another and one season to another as well. However, crop rotationsare likely to favour certain pathogens whenmultiple hosts are available. Croprotation emphases the importance of the infection time length, and as consequence, the survival of inoculum.

Environmental factors

It is generally agreed that the environment is the drivingforce in the development of epidemics. This includesmajor climatic variables, such as temperatureand humidity. Wind and rain are essential for pathogendispersal; rain provides free water on host surfacesfor most pathogens to infect and sporulate and sunprovides favourable temperatures for disease development. The duration of each event as wellas its timing is also important. Moisture, particularly the duration of wetness, isthe dominant factor for most pathogens. Free water or near saturation moistureon the host surface is essential for germination andpenetration of the host for many pathogens. Thus, a single parameter indicating water availabilityis used in several forecasting systems. Predictionof actual wetness duration is preferable to prediction ofoccurrence because majority of pathogens cause more damageas the duration of wetness increases.

The role of temperature has been studied for manypathosystems, mostly for its influence on initial germination, infection, and the length of incubation, latentand infectious periods. The relationships between disease development andenvironmental factors are the key component and often the only component of disease forecasting systems.Both past and future weather forecasts can be usedin these systems for predicting epidemic development.

Mathematical representation ofepidemic development

Overall, modeling can be divided into three steps: model development, model analysis and hypothesistesting. When developing a model, biological characteristicsof the pathosystem are expressed as mathematicalrelationships. In model analysis, epidemicdynamics are investigated in relation to the parametersof interest (or variables, which may be formulatedas functions of external factors such as rain andtemperature). Finally in hypothesis testing, the resultsfrom model analysis are used to test or verify whether orunder what conditions the hypothesis (i.e. the specifiedproblem/question) is valid. Mathematical expression ofbiological features of the system is critical, since it will, more or less, determine the techniques to be used inmodel analysis and hypothesis testing.

Disease progression curves

It is well known that polycyclicdiseases could be described by logistic modelsand monocyclic diseases by monomolecular models.Growth models (monomolecular, Gompertz and logisticmodels) provide a range of curves that are oftensimilar to disease progress curves. These non-linearcurves can be easily fitted to experimental data byany standard statistical package. The important parameters in these models arethe initial amount of disease, the apparent rate ofdisease increase and the level of maximum disease.These parameters can be estimated separately for eachindividual treatment and their relationships with treatmentor environmental factors can then be investigated.Alternatively, the mathematical relationships betweenthe model parameters and treatment/environmental factorscan be incorporated into the growth curve modelsand fitted to the observed data directly. More complexmodel fitting procedures are required for the latterapproach.Most analyses of disease progress data rely on timeas the independent factor. This may not be appropriatewhen data are collected in different years, seasons, locations, etc. A measure of heat-sum or degree-daysprovides an alternative method. This assumes that temperatureis the most important factor driving growthrate of the host, the pathogen and the disease. Many othermodifications to standard growth models are possibleto take into account the temporal variabilityof host susceptibility to pathogens.

Area under disease progress curve (AUDPC)

Not all disease progress curves are well or easilydescribed by a growth curve model. Alternativemethods to quantify epidemic development includethe area under the disease progress curve (AUDPC). Vander Plank (1963) related area under the stem rustprogress curve to the yield loss in wheat. The resulting AUDPC values can be used as ameasure of epidemic development and used in furtheranalysis and hypothesis testing, such as regression andin variance analyses.

Linked differential equations

One of most commonly used mathematical techniquesin modeling epidemics is the linked differential equation (LDE), which is usually used to investigatetheoretical questions concerning the dynamicsof plant disease in relation to host, environment andhuman interventions. Vander Plank (1963) demonstratedhow analytical models written as differentialequations could be integrated and used to quantify thevarious parameters associated with disease progress. The LDE models are of the susceptible, exposed, infectiousand removed (SEIR) type, which is the standardmodeling approach in human disease epidemiology,and is also widely used in plant disease epidemiology.

In this approach, the host population is usuallydivided into several non-overlapping categories, suchas healthy susceptible, latently infected, infectious andremoved (post-infectious). When an individual plantbecomes infected, the pathogen moves through thelatent stage to become infectious at a rate whichis the inverse of mean latent period. Infected plantslose infectiousness and proceed into the removed orpost-infectious stage at a rate which is the inverse ofmean infectious period. Plant populations may be constant, but may also assume increaseor decrease to model host growth or senescence processes.Depending on the hypothesis to be tested, thenumber of these categories used varies greatly. When modeling the effect of induced resistance, an extra category, healthy resistant, may also berequired.Linked differential equation models are specified foreach defined plant category, written generically as

$$\frac{dp}{dt} = B(P) - D(P),$$

where *B(P)* and *D(P)* are functions describing theincrease and decrease of the host population of category*P*. *B(P)* and *D(P)* are jointly determined by hostgrowth functions, pathogen attributes, pathogen transmission/dispersal characteristics and disease management.

Linked differential equation models are usually evaluatedanalytically to determine the key dynamic featuresof the system, and then numerically to explorethe dynamics in the important conditions identified.

Computer simulation

Many computer simulation models have been developedin the past decades. Computer simulation isin general a natural extension of LDE modeling.In computer simulation, model parameters in LDEare often assumed to be functions of external factorssuch as temperature and humidity. These functionscan either be of simple linear type or complexnonlinear type. Computer simulation can be used tostudy both theoretical and applied problems. Usinga stochastic simulation model, the relationships ofspatio-temporal statistics with underlying biological, physical and biological factors have been successfullystudied. Oneof the earliest spatio-temporal simulation models wasEPIMUL (Kampmeijer and Zadoks 1974), which laidthe foundations for further developments. However, no such computer simulation models in the management of rapeseed mustard have been developed.

The model thus developed could explain realistic disease intensity using independent natural weather factors. These models need to be tested at larger location for its validity, accuracy and effectiveness. The information available on epidemiology, disease cycle, disease predictionmodels and disease management of fivemajor diseases viz., Alternaria blight, white rust, downy mildew, powdery mildew and stem rotof rapeseed-mustard isbeing summarized**.**

Alternaria blight

Alternaria blight of rape-seed is one of the most common, and destructive disease worldwide.The seed production isgreatly reduced by the attack of this disease, which invade siliquae and penetrate the seeds besides damaging the assimilatory tissues of the leaves and stem. Under severe infection results in shriveling of seed, reduction in quantity of oil content changes in chemical composition of seed including protein, total carbohydrates and ash. In India, yield losses of 35–45%in case of yellow sarson, 25–45% in brown sarson, and 17–48% in rayahave been reported (Saharan et al. 2005). It is believed that*Alternaria*survives through seed, plant debris, soil and weed hosts. However, it has been reported that*Alternaria* does not survive through seed to cause infection in the nextseason due to high storagetemperature during summer months in north India. Nevertheless, the possibility of its survival through seed on hills cannot be ruled out. Mehta et al. (2002a) reported that diseased debris placed in deep freezer conditions (− 10 °C) were able to cause 100% infection in the next crop season when mixed in the soil as compared to the debris placed in the field and laboratory conditions.

Epidemiology

The primary infection occurs on the cotyledonary leaves forming the source of secondary infection for the entire crop. For infection, a minimum of 4 h of leaf wetness is required. Increased leaf wetness duration at 25 °C increased infection and spread of the disease rapidly. Under favourable temperature conditions and presence of dew, the spores infect other parts of the plant as well. The infection occurs through the stomata and under favourable climatic conditions the new lesions arise within 4-6d bearing spores. The pathogen penetrates the tissues of the pods and infects the seed.The congenial factors for *Alternaria* spores germination has been reported as darkness or low light intensity (< 1000 lx), 25 °C temperature and more than 90% RH.

The favourable environmental factors for disease progression under field conditions have been reported as Temp. 12–25 °C, RH > 70% with intermittent winter rains or irrigation,wind velocity around 2–5 km/h, closer plant spacing (30 × 15 cm) and high doses of nitrogen (80 kg/ha). The Tmax of 26–29 °C with RHAv > 65% favour the disease development (Sangeetha and Siddaramaiah 2007).The raya crop sown in the last week of October recorded 52% disease while that sown in the third week of November had only 15.5% disease. *Alternaria* spores were trapped in a 7-day volumetric spore traps about 10–11 days before the disease appearance and their concentration increased and reached maximum in March. The spore was trapped maximum during 10.00 am to 2.00 pm(46% of total spores) and minimum during 10.00 pm to 6.00 am, thereafter, its concentration decreased and decline sharply after 2.00 pm (Singh 2005). A prediction equation developed on the basis of spore trapped as:

$$\begin{aligned}\text{ALTn} =&21.50 + 1.164\text{ALTc} + 0.128\text{ALTp} - 0.165(\text{Tmax})\\&- 0.154(\text{Tmin.}) - 0.203(\text{RHmor}) - 0.095(\text{RHeven}),\end{aligned}$$

where ALTn = Expected Alternaria blight; ALTc = Disease intensity in current week; ALTp = Disease intensity of previous week. The R^2 value recorded was 0.84.

Meena et al. (2011) reported that disease severity increased with delay in date of sowing. The A value AUDPCand ‘r’ value (apparent infection rate) were more in the variety ‘Varuna’ with the delayedsowing. The severity of Alternaria blight was significantly lower in October sown crop. The spread of the disease was more in broadcasting method as compared to line sowing (45 cm). The disease intensity also decreased when K (40 kg/ha) along with recommended dose of fertilizers was applied. Chattopadhyay et al. (2005) analyzed the data for Alternaria blight progression and development from eight locations using cv ‘Varuna’ sown on 10 dates at weekly intervals. The results revealed that first appearance of disease on leaves occurred between 42 and 139 days after sowing (DAS). The disease thenappeared on pods between 67 and 142 DAS, being highest at 99 DAS. Severity of Alternaria blighton leaves was positively correlated to a daily Tmax of 18–27 °C, daily Tmin of 8–12 °C, daily Tmean of > 10 °C, RHmor > 92%, RHeven > 40% and RHmean of > 70% in the preceding week. Disease severity on podswas favoured by a daily Tmax of 20–30 °C, daily Tmean of > 14 °C, RHmor > 90%, daily RHmeanof > 70%, sunshine > 9 h and leaf wetness > 10 h. It was concluded that temperature and RH conditions favourable to disease development recorded in the field matched withlaboratory findings. Regional and cultivar-specific models could predict the crop age at which Alternariablight first appeared on leaves and pods, the highest blight severity on leaves and podsat least one week ahead of first appearanceof the disease. The prediction models for all eight locations were developed. In addition to weather factors, the role of varieties in disease development has also been reported. The rate of disease development was faster on the varieties belonging to *B. juncea* (RH-30, RH-8113, RH-8695, RH-8546) and *B. campestris* (YSPb-24, BSH-1, Candle, Shiva) compared to *B. carinata* (HC-2, HC-9001), *B. napus* (GSH-1) and *B. alba* (Mehta et al. 2008a; Saharan et al. 2016).

Disease prediction models

The different models viz., Gompertz, Logistic, Monomolecular and Exponential have been used for the development of prediction models for the Alternaria blight. Dang et al. (2006) developed prediction equation for the development of Alternaria blight using Gompertz model and two factors can be explained by the following equation

$$\mathrm{DI} = \mathrm{Exp}^{[\log \mathrm{A}\ \mathrm{Log}\ (-\mathrm{B}*\ \mathrm{Time})]} + \mathrm{C} * \ \text{Sowing day} + \mathrm{D} * \ \text{Factor 1},$$

where DI = Disease Intensity; (A and B are the two parameters of the Gompertz model and C and D are the coefficient of the sowing day and factor 1). All the varieties are based on different genetic makeup, which reacted differentially to the natural inoculum and factor 1 may be interpreted as weather index and factor 2 as contrast between the heating factor and moisture factor. The best-fitted models worked out for each variety were as:

$$\mathrm{RH}-30:\ \mathrm{Y} = \mathrm{E}^{[\mathrm{Log}\ (0.620)*\ \log\ (4.998*\ \mathrm{Time})]} - 0.890 * \ \text{sowing Day} + 0.238\ \text{factor 1};\ \left(\mathrm{R}^2 = 0.389\right),$$

$$\mathrm{RH}-8113:\ \mathrm{Y} = \mathrm{E}^{[\mathrm{Log}\ (2.547)*\ \log\ (1.072*\ \mathrm{Time})]} - 0.561 * \ \text{sowing Day} + 0.086\ \text{factor 1};\ \left(\mathrm{R}^2 = 0.482\right),$$

$$\mathrm{YSPb}-24:\ \mathrm{Y} = \mathrm{E}^{[\mathrm{Log}\ (2.199)*\ \log\ (4.827*\ \mathrm{Time})]} - 1.868 * \ \text{sowing Day} + 0.105\ \text{factor 1};\ \left(\mathrm{R}^2 = 0.539\right),$$

$$\mathrm{BSH}-1:\ \mathrm{Y} = \mathrm{E}^{[\mathrm{Log}\ (3.008)*\ \log\ (1.335*\ \mathrm{Time})]} - 1.260 * \ \text{sowing Day} + 0.166\ \text{factor 1};\ (\mathrm{R2} = 0.482),$$

where Y = Percent disease intensity.

The above equations revealed that for a given cultivar, if the sowing time is known and weather parameters at particular time are known, disease incidence can be predicted using the above models. Mehta et al. (2008a) developed a prediction model for adopting better disease management practices where four varieties each of *B. juncea* (RH-30, RH-8113, RH-8695, RH-8546), *B. campestris* (YSPb-24, BSH-1, Candle, Shiva), two of *B. carinata* (HC-2, HC-9001) one each of *B. napus* (GSH-1) and *B. alba* (local) were monitored for the development and progression of Alternaria blight. Each cultivar was inoculated artificially with *A.brassicae* spores when the crop was about 2-month-old. Studies conducted revealed that Temp and RH had prominent role in disease development in addition to varietal behaviour. The prediction equations so developed wereat par with observed values. Regression equations for the development of Alternaria blight on different varieties of rapeseed and mustard are:

$$B.\,juncea(\text{var. RH}-30)\ \mathrm{Y}_1 = -4.3635 + 0.3488\ \mathrm{X}_1\left(\mathrm{R}^2 = 0.66\right),$$
$$\mathrm{Y}_1 = 4.4927 + 0.2755\ \mathrm{X}_1 - 0.0789\ \mathrm{X}_3\left(\mathrm{R}^2 = 0.71\right),$$

$$B.\,campestris(\text{var. YSPb}-24)\ \mathrm{Y}_1 = -4.2310 + 0.3420\ \mathrm{X}_1\left(\mathrm{R}^2 = 0.68\right),$$
$$\mathrm{Y}_1 = 4.7131 + 0.2680\ \mathrm{X}_1 - 0.0797\ \mathrm{X}_3\left(\mathrm{R}^2 = 0.73\right),$$

$$B.\,carinata(\text{var.HC}-1)\ \mathrm{Y}_1 = -4.8061 + 0.3584\ \mathrm{X}_1\left(\mathrm{R}^2 = 0.64\right),$$
$$\mathrm{Y}_1 = 6.4244 + 0.2654\ \mathrm{X}_1 - 0.1001\ \mathrm{X}_3\left(\mathrm{R}^2 = 0.71\right),$$

where X_1 = Tmax; X_3 = RHmor.

Sangwan et al (2000) demonstrated that Gompertz model can be effectively used for prediction of Alternaria blight with two factors A and B drawn from the analysis of weather parameters and disease progress.

Factor A: 0.091 × Tmax + 0.887 × Tmin + 0.036 × RH mor + 0.808 × RHeve-0.644 × Sunshine h.

Factor B: 0.317 × Tmax + 0.317 × Tmin + 0.933 × RH mor + 0.347 × RHeve-0.618 × Sunshine h.

These two factors (A and B) explained 60.3 and 24.5% of total variation, respectively and together explained 85% of total variation among the weather variables. The model for each group is as follows:

$$\text{Disease} = \text{Exp.}\left[\log\ (\mathrm{a})\ \mathrm{X} - \ \log\ (\mathrm{b})\ \times\ \text{Time}\right] + \mathrm{c}\ \times\ \text{Factors}\ (1) + \mathrm{d}\ \times\ \text{Factors}\ (2),$$

where a & b are constants of Gompertz model.

The equation for each genotype of *Brassicas* was developed as follows:

B. juncea

Exp. [log (2.915) × log (−0.430 × Time] − 0.154 × Factors (1) + 0.204 × Factors (2),

$R^2 = 0.931$ (observed vs predicted).

B. campestris

Exp. [log (2.722) × log (0.470 x Time] − 0.203 × Factors (1) + 0.167 × Factors (2)

$R^2 = 0.974$ (observed vs predicted).

B. carinata

Exp. [log (2.717) × log (0.479 × Time] − 0.202 × Factors (1) + 0.168 × Factors (2)

$R^2 = 0.982$ (observed vs predicted).

B. napus

Exp. [log (2.908) × log (0.424 × Time] − 0.185 × Factors (1) + 0.170 × Factors (2)

$R^2 = 0.975$ (observed vs predicted).

B. alba

Exp. [log (4.312) × log (0.270 x Time] − 0.284 × Factors (1) + 0.212 × Factors (2)

$R^2 = 0.970$ (observed vs predicted).

B. oleracea

Exp. [log (2.823) × log (0.455 × Time] − 0.196 × Factors (1) + 0.192 × Factors (2)

$R^2 = 0.984$ (observed vs predicted).

Experiments were conducted at two locations for development of prediction models for two varieties of different *Brassica* genotypes viz., *B.juncea* (RH-30) and *B. campestris* var. yellow sarson (YSPb-24). These were monitored for the progression of Alternaria blight—disease progression was faster on YSPb-24 compared to RH-30. Maximum lesion size 6.31 mm was recorded on YSPb-24 whereas it was 4.21 mm on variety RH-30. The favourable weather conditions for the progression of the disease were observed to be at T (max) of 20 °C with RH > 90%. The stepwise regression analysis revealed that the T (max) and RH (mor) played significant and positive roles in disease progression. The R^2 value recorded was > 0.9 in all the cases, which showed that weather variables played major role in disease progression in addition to the varietal factors.The prediction equations developed for leaves and pods for a variety for two locations were as follows (Mehta et al. 2002b; Saharan et al. 2016). Similar equations were developed for other varieties as well.

On Leaves

$$\mathrm{RH}-30\,(\mathrm{location}-1)\ \mathrm{Y} = 1.984 + .196\ \mathrm{X}_1 + .951\ \mathrm{X}_5\left(\mathrm{R}^2 = 0.92\right)$$

$$\mathrm{RH}-30\,(\mathrm{location}-2)\mathrm{Y} = 2.156 + .235\ \mathrm{X}_1 - 5.49 + 10^{-2}\mathrm{X}_4\left(\mathrm{R}^2 = 0.87\right),$$

On Pods

$$\mathrm{RH}-30\,(\mathrm{location}-1)\mathrm{Y} = -0.471 + 5.802 + 10^{-2}\mathrm{X}_1\left(\mathrm{R}^2 = 0.84\right)$$

$$\mathrm{RH}-30\,(\mathrm{location}-2)\mathrm{Y} = -0.317 + 1.543 + 10^{-2}\mathrm{X}_4\left(\mathrm{R}^2 = 0.97\right),$$

where X_1 = Tmax; X_2 = Tmin: X_4 = RHeve.; X_5 = Wind speed.

Jha et al. (2013) recorded that Tmax positively correlated with disease index. Tmax of 23.2 °C, RHmax of 80% and RHmin of 66% with correlation co-efficient (r) = 0.73for Tminand r = 0.51 of RHmin favoured the disease development. The regression equation developed for leaves as:Y1 = − 47.388 + 5.114 Tmin-2.371 Tmax + 1.4s92 RHmin with $R^2 = 0.7376$ whereas for siliquaY2 = 31.524 + 4.225 Tmin-1.883 Tmax. with $R^2 = 0.69203$.

White rust

White rust affects a number of *Brassica*plants of economic importance but its incidence and damage is more in mustard (*B. juncea*) and rapeseed. With increase in area under mustard cultivation, the intensity of white rust has increased throughout the *Brassica* growing areas in India. Foliar infection is damages the crop to some extent but floral infection may cause complete loss to the crop. Both phases of the disease viz., local and leaf phase and systemic or floral infection can cause yield losses from 23 to 54.5%.The mixed infection of white rust and downy mildew in the inflorescence has become very common. This type of infection reduces pod formation by 37–47% with grain yield reduction of 17–32% under Punjab conditions.

The pathogen perpetuates through the oospores formed in the hypertrophied tissues lying in the soil in diseased plant debris or moving with diseased debris along with the seeds as contaminant. The oospores can survive in diseased host tissues under dry storage conditions for more than 21 years. Oospores germinate at suitable temperature (10–20 °C) and RH (> 70%) and cause primary infection in the host leaves after penetrating directly or through stomata or natural openings. The secondary spread takes place through sporangia and zoospores formed in the diseased pustules. The sporangia readily carried away by air currents after breaking open the mature pustules. Moisture on the host surface is essential for germination and infection through sporangia and zoospores. Oospores are formed in the hypertrophied tissues (leaves, stems, inflorescence, pods, and roots) of infected plants. Over summered oospores in infected plant debris in soil function as the source of primary inoculum.

Epidemiology

The optimum temperature for sporangial germination is about 10 °C at which the rate of germination and the number of zoospores formed are maximum, and no germination occurs above 25 °C. For the germination of oospores, 10–20 °C has been reported to be most favorable. Oospores buried at the depth of 7.5 cm in soil cause early infection when frequent irrigation is given to maintain soil moisture. Deep (15 cm), and shallow (on soil surface) placement of oospores delays infection in the host plants. Date of sowing has great bearing on floral infection of white rust. The disease intensity increaseswith delayed sowing. When mustard sowing was delayed from first week of October to third week of November, the disease intensity increased from 4.6 to 68.5%and the November sown crop had less stag heads as compared to October sown crop (Kaur et al. 2006). Anand et al. (2009) also observed that white rust intensity increased with delayed sowing. It was also reported thatT-mean of 13-22°C with RH > 60% were mostcongenial for the formation of maximum stag heads (Kaur et al. 2006). Epidemic development of the disease under fieldconditions occurred when temperature around 12 °C, RH > 70% (mostly between 60 and 80%), wind velocity from 2.7 to 3.4 km/h. and winter rains found as most congenial. The mixed infection of white rust and downy mildew was favoured by temperature between 13.8 and 14.8 °C and rainfall > 151.9 mm. The incubation period of the pathogen in *B. juncea* susceptible cultivars was 6–7 days. The severity of stag head formation due to white rust and downy mildew was reported to be favoured by 2–6 h of sunshine per day concomitant with Tminof 6–10 °C, Tmax of 21–25 °C and rainfall of 161 mm;temp > 15 °C,RHav > 65% and intermittent rains were conducive on susceptible cultivars. Early planting from 15th September to October escapedstag head formation and gave higher yield. Hypertrophy in the plant has been reported to be directly correlated with the amount of oospores formation. The sporangia survived up to 30 °C when detached from leaf tissues and at 32 °C when attached with leaf tissues. Sporangia germinate at 6–22 °C, withmaximum germination occurring at12-14°C, dark conditions and 7 h of incubation period. A quadratic equation $Y = -\ 103.13 + 26.99–1.01x^2$ has been proposed to determine the percent sporangial germination (Y) at any known temperature (X). Anand et al. (2009) reported thatTmax and Tmin had significant and negative correlation with disease intensity whereas RH had significant and positive correlation with disease intensity.

Disease prediction models

Mehta and Saharan (1998) developed the prediction models for RH-30 variety as under:

$$\text{White rust } Y = 56.119 + 1.017X_1 - 4.086X_2 + 8.702X_3 - 2.015X_4 + 2.723X_5 - 0.890X_6\left(R^2 = 0.66\right),$$

$$\text{Stag head incidence } Y = -0.261 + 0.048X_1 - 0.323X_2 + 1.460X_3 - 0.202X_4 + 0.342.X_5 - 0.123X_6\left(R^2 = 0.26\right),$$

$$\text{Stag headlength } Y = -0.784 + 0.079X_1 - 0.271X_2 + 1.078X_3 - 0.172X_4 + 0.257X_5 - 0.094X_6R^2 = 0.28).$$

White rust severity on leaves was positively correlated to > 40% afternoon (minimum) RH (R^2: 0.92), > 97% morning (maximum) RH (R^2:0.89), > 72% daily mean RH (R^2: 0.8), > 10 °C daily mean temperature (R^2: 0.79) and 16–24 °C maximum daily temperature (R^2: 0.83). Stagheads formation was significantly and positively correlated to 20–29°Cmaximum daily temperature (R^2: 0.81) and further aided by > 12 °C minimum dailytemperature (R^2: 0.84), > 97% morning (maximum) RH (R^2: 0.89) and > 72% dailymean RH (R^2: 0.85). Empirically, a look at weather data available from theautomatic weather station indicated > 10 h of leaf wetness during the preceding 3 days and also favoured the progress of rust severity on leaves and formation ofstagheads.Crop age (d.a.s.) at first appearance of the rust on the crop (Y1), crop age (d.a.s.)at peak severity of the rust (Y2) and highest disease severity (Y3) were related withweather variables in different weeks including pre-sowing week and the interactionswere found significant. Regional and cultivar specific models devised using data predicted the crop age at which white rust first appears onthe leaves, crop age at highest rust severity on leaves and the peakrust severity on leaves, number of stagheads. Most of the models saw entry of variable maximum temperaturewith minimum temperature, morning RH, afternoon RH and sunshine hours alsogetting entered in some cases. Proper monitoring of disease progress duringrecording of observations in experiments can enable devise models for providingaccurate forecasts a few weeks after sowing, about crop age at first appearance, cropage at highest severity and highest level of disease severity. The predictions werepossible at least 1 week ahead of first appearance of the disease on the crop, thusallowing growers to undertake timely sprays. The disease was never found to appearbefore 36 d.a.s. or beginning of the sixth week after sowing, while the prediction for crop age at first appearance of rust on leaves was possible for most of the locations inthe beginning of the fifth week (29d.a.s.) (Chattopadhyay et al. 2011).

Downy mildew

The downy mildew disease is caused by *Peronospora parasitica* (Pers. ex Fr.) Fr. (Syn. *Hyaloperonospora parasitica*). In the family crucifereae, about 50 genera and more than 100 different species have been observed to be susceptible to infection by the downy mildew pathogen. Downy mildew alone or in combination with white rust is responsible for causing severe losses in yield of several temperate and tropical brassicaceous crops particularly rapeseed and mustard. Yield losses due to downy mildew infection alone are very difficult to estimate, since in most cases, it is always associated with white rust. Leaf infection is more serious in young plants and may cause seedling death but does not progress much after the plants have grown up. However, systemic infection causes malformation of stem and inflorescence resulting in heavy losses in the yield. There may be seedling death up to 75% when infection occursatthe cotyledonary.

The pathogen perennates in the soil through oospores that are formed in abundance in the malformed tissues of the infected plants. Seeds may be contaminated with plant trash containing oospores during threshing operation. Infection originated when such seeds are sown after getting suitable temperature and relative humidity. Oospores formed in malformed and senesced host tissues constitute an important means of survival of *H. parasitica* over periods of unfavourable conditions. It is also known to survive through mycelium and conidia. In radish and rapeseed-mustard, there is abundant production of oospores in infected leaf tissues, on the seed surface and pericarp and embryo of seeds. However, in rapeseed and mustard, seed transmission is low and may be non-systemic, ranging from 0.4 to 0.9% in the seedlings grown from infected seeds (Saharan et al. 2005; 2017).

Epidemiology

In epidemics, the pathogen population starts a low level of initial inoculum which then increases exponentially through successive cycles on the host during the growing season. The relationship of host–pathogen-interaction in case of downy mildew of crucifers is a complex phenomenon, which determines the rate of diseases development. *Peronospora* produces both sexual (oospores) and asexual (sporangia) spores, which are helpful in survivaland dissemination of the pathogen. The rate of spore germination and host penetration is affected by temperature variations. At 15 °C conidia germinate in 4–6 h, appressoria form in 12 h and penetration occurs in 18–24 h. It has been observed that a temperature of 15 °C seems to be the most favourable for epidemic development as this favours slower growth of both host and pathogen resulting in less drastic damage and hence more profuse disease development. Floral infection increases in the late sown crops. The disease has been observed to be favoured by damp and cool environmental conditions. More infections occurredat low temperature (8–16 °C), moist weather and low light intensity. Temperature and rainfall have a great impact on the appearance of the disease. Its severity was 26% at 14.3 °C and 151.90 mm rainfall but declined to 0.52% at 17 °C and low rainfall of 50–80 mm in a crop season in Punjab. Haustoria development took place atoptimum temp of 20–25 °C. Downy mildew developed very fast at 24 °C. Sporangia exposed to air remainedinfectious for six weeks but direct sun light may kill them within 5–6 h. RH above 70% helped in rapid development of the disease. In a subsequent study, 15–20 °C was the best temperature for infection and development of downy mildew. At this temperature regime, infection occurredwithin 24 h of inoculation. The infection frequency reduced at 25 °C while no infection observed at 30 °C. Leaf wetness duration of 4–6 h at 20 °C and for 6–8 h at 15 °C has been reported to be essential for severe infection and disease development on mustard. The infection frequency and disease development increased significantly with the increase in duration of leaf wetness (Mehta et al. 1995). In India, infection of mustard foliage starts by the end of October (cotyledon stage) and progressedup to November. The crop planted after mid November may not contract downy mildew. However, downy mildew growth as a mixed infection with white rust on floral parts can be seen up to March (Mehta and Saharan 1998).

Disease prediction models

Kolte et al. (1986) developed prediction equations for the stag head severity in relation to planting dates and associated weather factors as under:

i. Stag head incidence (%)$Y = 16.925 + 0.019X_1 - 0.132X_2 - 0.086X_3 + 0.158X_4 + 0.030X_5 - 1.469X_6.(R^2\text{-}0.68)$

ii. Stag head Severity (%) $Y = 86.169 - 1.241X_1 - 0.129X_2 - 0.503X_3 + 0.054X_4 + 0.472X_5 - 2.125X_6.(R^2\text{-}0.62)$

Where X_1 = mean max. Temp; X_2 = mean min. temp; X_3 = mean RH; X_4 = total rain fall (mm)X_5 = total rainy days X_6 = mean bright sunshine period (h/day).

Mehta and Saharan (1998) developed the prediction models for the progression of downy mildew of rapeseed-mustard as under.

A. Leaf Infection:$Y = -32.7 + 0.09\,X_1 + 0.31\,X_2 + 1.31\,X_3 + 0.12\,X_4 + 0.22\,X_5 - 0.03\,X_6(R^2 = 0.36)$.
B. Stag head- i. Incidence:$Y = -18.6 - 2.8\,X_1 + 2.5\,X_2 + 4.5\,X_3 + 0.6\,X_4 - 0.2\,X_5 + 1.0\,X_6\left(R^2 = 0.23\right)$.

ii. Length:$Y = -17.4 - 1.5\,X_1 + 1.5\,X_2 + 2.6\,X_3 + 0.4\,X_4 - 0.1\,X_5 - 1.0\,X_6(R^2 = 0.26)$

Where X_1 = Tmax. X_2 = Tmin. X_3 = Sunshine, X_4 = RHmor, X_5 = RHeve, X_6 = RF.

Powdery mildew

Three species of *Erysiphe*, i.e., *E.polygoni, E. communis* and *E. cruciferarum* caused powdery mildew in rapeseed–mustard. The disease does not cause much damage except during epidemic outbreak on late sown crop when especially when it appears at early stage of the crop growth. The pods heavily covered with powdery mass remained empty or produced few seeds at base with twisted sterile tips. It has been observed that average crop losses due to this disease varies form 19–29.5% depending upon the variety used (Mehta et al. 2008b).

The off-season host plants of *Brassica* species and other weed may carry the fungal mycelium and conidia was source of primary inoculum. The pathogen produced abundant number of cleistothecia on diseased tissues at the maturity stage of the crop. Long distance dissemination of the pathogen took place through wind currents under low humid conditions. Conidia fallen on the host germinate, grow and spread in the form of mycelium, later producing conidiophores and conidia in the form of white mildew growth (Saharan et al. 2005).

Epidemiology

The optimum temperature for the germination of conidia, germ-tube growth and appressorium formation is 20–25 °C. Conidia could not germinate below 15 °C and above 30 °C. Maximum conidia germinate at 40–50% RH. To initiate the spore germination, at-least 30% RH is essential and there is no conidial germination above 60% RH. Conidial germination is not influenced by light and darkness. For onset and epidemic development of disease under field conditions, moderate temperature, low humidity, minimum rainfall or dry season during the months of February and March are more favourable in Haryana. Mean temp between 16 and 28 °C, mean RH below 60% and low or no rainfall are the most congenial weather factors for the development of the disease under field conditions. It has been observed that maximum cleistothecial formation is favoured by alternating low and moderate temperature. Heavy sporulation took place with low nutrition of the host, low relative humidity, dry soil and aging of the host. Dange et al. (2003) reported that early planted crop i.e. in the month of October, resultedin less severity as compared to late sown conditions.

There are number of environmental factors which are very crucial to influence the powdery mildew development of crucifers in to epidemic form after host–pathogen interaction. These factors determine the progress of powdery mildew on host plants with their influence, and effects on interacting partners, host, and pathogen. To cause the infection in susceptible host after landing of pathogen conidia on host surface their germination and formation of appressoria is maximum between 15 and 20 °C temperatures. Their germination is greatly reduced at > 30 °C temperature. Infection, and disease development is faster with the influence of mean temperature (16–22 °C), minimum temperature (> 7 °C), maximum temperature (25–28 °C), relative humidity (27–65%), sunshine hours (> 9 h/day), wind velocity (2 km/h), and ageing of the host plants. Infection rate is positively favoured by host age, and ambient temperature. Infection rate increases with ageing host tissues. There is no infection on younger than 37 days host, and freshly emerging new leaves. Disease develops at fast rate if host and pathogen interact coinciding with favourable host age, plant growth stages, and environmental factors. Stem infection is maximum with the increase in length of time they are exposed to the pathogen, and maturity level of the host. Symptoms are visible at anamorph state or asexual stage with the development of pathogens mycelium, conidiophores, and conidia on host surface. Date of crop planting has significant bearing on disease epidemiology under late sown conditions coinciding with congenial and critical factors at 40–120 days after sowing. Teleomorph or sexual stage appears in the form of dark brown spherical bodies of cleistothecia or chasmothecia embedded in powdery mass of host leaf, stem, and pods at maturity stage of crop when temperature is 11–27 °C (19 °C), alternate moderate temperature, heavy sporulation, low host nutrition, low relative humidity, dry soil, and ageing host tissues. Host resistance, and progression of disease is measured using parameters like AUDPC, disease intensityincubation period, latent period, infection rate, number of colony/leaf, number of conidia/ microscopic field (sporulation rate), and R^2 values (Saharan et al. 2019).

Disease prediction models

Singh et al. (2008) revealed that powdery mildew progression was maximum during mid of Marchat Tmax 32.5 °C, Tmin 12.7 °C, RHmor 49.5% and RHeve 38.5% prevailed. The disease intensity and AUDPC increased from 48 to 74% and 326–440, respectively, with delayed sowing. The apparent infection rate (r) was also higher during mid of March sowing. The regression equation developed for each variety in relation to date of sowing was observed as mentioned below.

Ist date of sowing (Nov 15)		
RH-30	$Y = -19.20 + 0.73X_1 - 0.08X_7$	$R^2 = 0.94$
RH-8113	$Y = -19.47 + 0.74X_1 - 0.09X_7$	$R^2 = 0.92$
RH-9304	$Y = -20.38 + 0.77X_1 - 0.09X_7$	$R^2 = 0.94$
RH-9801	$Y = -20.19 + 0.76X_1 - 0.08X_7$	$R^2 = 0.93$
IInd date of sowing (Nov22)		
RH-30	$Y = -19.90 + 0.75X_1 - 0.10X_7$	$R^2 = 0.94$
RH-8113	$Y = -20.96 + 0.80X_1 - 0.09X_7$	$R^2 = 0.90$
RH-9304	$Y = -19.18 + 0.73X_1 - 0.11X_7$	$R^2 = 0.94$
RH-9801	$Y = -21.79 + 0.83X_1 - 0.10X_7$	$R^2 = 0.91$
IIIrd date of sowing (Dec 2)		
RH-30	$Y = 11.34 + 0.40X_1 - 2.26X_8$	$R^2 = 0.92$
RH-8113	$Y = 6.46 + 0.48X_1 - 2.01X_8$	$R^2 = 0.94$
RH-9304	$Y = 6.74 + 0.47X_1 - 2.03X_8$	$R^2 = 0.94$
RH-9801	$Y = 10.65 + 0.41X_1 - 2.20X_8$	$R^2 = 0.92$

Where X_1=Tmax.);X_2=Tmin.); X_3=RHmor); X_4=RHeve; X_5=Ave Evaporation (mor); X_6=AveEvaporation (eve); X_7=Wind Speed; X_8=Sunshineh (Singh et al.2008).

The prediction models for the different varieties belonging to various genotypes also revealed that environmental factors contributed more than 80% in disease development. The correlation matrix in relation to weather variables revealed that Tmax and RHmor had positive and significant role in disease development (Mehta et al. 2009). The regression equations developed are as follows:

RH-30	$Y = -19.38 + 0.74X_1 - 0.10X_7$	$R^2 = 0.91$
RH-8812	$Y = -11.71 + 0.48X_1 - 0.08X_7$	$R^2 = 0.83$
RH-9304	$Y = -14.12 + 0.57X_1 - 0.10X_7$	$R^2 = 0.81$
RH-9801	$Y = -12.74 + 0.52X_1 - 0.11X_7$	$R^2 = 0.83$
RH-9901	$Y = -15.14 + 0.60X_1 - 0.08X_7$	$R^2 = 0.86$
RC-781	$Y = -11.34 + 0.47X_1 - 0.10X_7$	$R^2 = 0.81$
Purple Mutant	$Y = -9.67 + 0.40X_1 - 0.07X_7$	$R^2 = 0.86$
GSH-1	$Y = -0.80 + 0.08X_1 - 0.01X_7$	$R^2 = 0.47$

Where X_1 = Tmax; X_7 = Wind speed.

The models developed indices were used in developing forecast models through regression approach. The form of the model was developed as per formula of Agrawal and Mehta (2007) as mentioned below:

$$Y = a_0 + \sum_{i=1}^{P} \sum_{j=0}^{1} \mathrm{aij\ Zif} + \sum_{i\#1}^{P} \sum_{j=0}^{1} \mathrm{biij\ Zii\ j} + \varepsilon,$$

where Y is variable to forecast; a_0, a_{ij}, b $_{ii`j}$ are constants; Ɛ is error term, and other symbols have same meaning as explained earlier. Stepwise regression technique was used for selecting important variables to be included in the model.

Multilayer perception (MLP), and Radial basis function (RBF) architecture based neural network models (NNM) were developedwith different hidden layers, and different number of neurons in a hidden layer with hyperbolic function as an activation function with varying learning rates, and RBF architecture, were obtained, and best architecture was selected having lowest Mean Absolute Percentage Error (MAPE).

The forecasting performance of various Artificial Neural Network (ANN) models, and regression models was judged by Mean Absolute Percentage Error (MAPE).

$$\mathrm{MAPE} = 1/n \sum \frac{Yt - Ft}{Yt} \times 100,$$

where Yt is actual observation, Ftis the forecast from model and n is the total number of test data point. Weather Indices (WI) based regression models were developed for various characters, and models have been validated using data on subsequent years not included in developing the models. The analysis has been done by using SAS (Statistical Analysis

System) Version 9.2 software package available at Indian Agricultural Statistics Research Institute, New Delhi. Neural network models using MLP architecture with different hidden layers (one and two), and different number of neurons (4, 5 and 6) in a hidden layer with hyperbolic function as an activation function with varying learning rates (from 0.3 to 0.8), and RBF architecture, were obtained, and best architecture was selected having lowest Mean Absolute Percentage Error (MAPE). The analysis has been done by using Statistical Neural Networks Version 6.1 available at Indian Agricultural Statistics Research Institute, New Delhi. The Mean Absolute Percent Error (MAPE) for different characters of powdery mildew in mustard crop in two varieties for various developed models which reveals that the neural network models using MLP have lowest MAPE as compared to other developed models in most of the cases. The ANN model has non-linear pattern recognition capability which is valuable for modeling and forecasting complex non-linear problems in practice. Kumar et al. (2013) found that neural network model using multilayer perception (MLP) architecture is better than RBF and weather indices based regression models in terms of MAPE. Therefore, reliable forewarning for maximum severity of disease, crop age at first appearance of disease, crop age at peak severity of disease in two different varieties of mustard crop for powdery mildew is possible well in advance (Kumar et al. 2013; Saharan et al. 2019).

Powdery mildew prediction model based on crop age, and weather variables have been devised by Desai et al. (2004). Crop age at first appearance of the mildew on the crop (Y_x), crop age at highest severity of the mildew (Y_y), and peak disease severity (Y_z) were related with weather variables in different weeks including pre-sowing week, and the interactions were found significant. The regional and cultivar specific models devised using data of initial 4 years thereby could predict the crop age at which powdery mildew first appears on the crop, crop age at highest mildew severity, and the peak disease severity. The predictions were possible at least 3 weeks ahead of first appearance of the disease on the crop, thus allowing growers to undertake timely fungicidal sprays. The disease was never found to appear before 50 d.a.s or eighth week after sowing, while the prediction for crop age at first appearance of mildew was possible for both the locations in the beginning of fifth week (Desai et al. 2004).

Sclerotinia stem rot or white stem rot

This disease is most frequently found in regions tending to be cool and moist but it has been reported to occur in semi-arid regions where conditions would seem unfavourable for disease development. Yield losses due to Sclerotinia stem rot in susceptible crops may be as high as 100%. The loss estimates have been made as high as 28% in individual rapeseed fields in Alberta, Canada. The yield losseshave been reported to be 11.1 to 14.9% in Saskatchewan, Canada (Saharan and Mehta 2008). The disease is gaining importance particularly in raya growing areas since it lead to complete crop failure with up to 80% incidence in some parts of the Punjab and Haryana.

Sclerotia may survive for 3–5 years in soil assuring pathogen availability when a host crop is planted. When conditions become favourable, these sclerotia germinate to form either a mycelium or apothecia. Large quantities of ascospores forcibly get discharged into the air and carried by air currents for distances ranging from a few centimeters to several kilometers. Once a blossom is colonized, the mycelium remainsviable for more than a month. On contact with susceptible healthy host tissue, the ascosporic mycelium produces an appressorium andpenetration occursby mechanical rupture of host cuticleand entry may be through the natural openings. After entering the host plant, the fungus grows through the host tissues causing cell to die in advance of the invading hyphae.

Secondary infection results from infected area but no secondary infection propagules produced. The mycelium producessclerotia externally on affected plant parts and/or internally in stem pith. During harvesting and threshing operations, these sclerotia remain on the fields with the crop debris. Some sclerotia get buried in the soil by subsequent tillage operations. The sclerotia survive in the soil and in the plant debris to complete the life cycle.

The sclerotia have been found to remain viable and virulent for up to 7 years. However, viability of the sclerotia depends on the type of sclerotia and several other environmental factors.It has been reported that moist sclerotia die rapidly, whereas the dry ones remain viable at 3 °C for 480 days and at 8 °C upto 300 days. Sclerotia do not survive for more than 2 years or more at 20 °C or over 14 months at 25 °C, 10–14 months at 30 °C and 3–4 months at 35 °C. The pathogen has been reported to survive in the form of ascospores to some extent when favourable temperature and RH prevail under field as well as greenhouse conditions. Dry ascospores survive for a longer period, therefore ascospores act as source of inoculum in some specific situations (Saharan and Mehta 2008).

Epidemiology

Species of *Sclerotinia* can function as either soil borne or air borne pathogen. Infection of above ground plant parts results from ascosporic inoculum whereas soil borne infection may result from either ascospores or sclerotia. Below ground infection, however, resultsfrom mycelial germination of soil borne sclerotia. Continuous moisture for about 10 days is required for apothecial development and even a slight moisture tension prevent apothecial formation. Apothecia of *S. sclerotiorum*are produced at an optimum temperature of 15 °C and ascospores survive at a wide range of conditions but high temperature and humidity reducethe viability. No apothecial initials are produced at either 30 or 5 °C. Approximately 48–72 h of continuous leaf wetness is required for infection by ascospores. The infection by *S. sclerotiorum* on yellow sarson and in *B. campestris* var. toria got aggravated by low temperature, heavy rainfall and close spacing in Uttar Pradesh and Bihar (Saxena and Rai 1988). The study conducted in Canada revealed that increase in seeding rate increased the disease intensity. The lodging of plant also increased when seeding rate exceeded 6.7 kg/ha. This is due to fact that higher seeding rates modify the microenvironment and increase the potential of lodging and may be responsible for plant-to-plant spread of the disease.Temperature of 6–10 °C during March and April and high soil moisture until the apothecia has developed, with subsequent changing weather favours infection. Ascospores released and petal fall should occurat the same time (Kruger 1980). The pattern of petal fall and petal deposit on leaves suggests that the crop is most vulnerable to infection towards the end of flowering about 25 days after the beginning of flowering in the UK (Mc Cartney et al. 2001a, b). The role of extrinsically produced ascospores in causing disease in rapeseed fields may therefore be of considerable importance (Saharan and Mehta 2008).

Accordingly, ascospore concentrations above the crop canopy and on plant surfaces might reflect the disease potential in a crop better than the density of apothecia in the field. Gugel and Morrall (1986) demonstrated a significant positive relationship between petal infestation at early bloom and disease incidence. Infested petals and disease incidence regularly found when apothecia were absent, thereby demonstrating the rate of extrinsically produced ascospores in the infestation of crops. Flowers of rapeseed fromthe time expand and retained on their petals on an average for 6 days. During this period, thepetals"insitu" get contaminated by ascospores of *Sclerotinia.* Infection takes place preferentially on senescent petals because young petals are resistant to a certain extent. The senescent petals have been reported to be most easily colonized and do provide the ascospores with a source of carbon, which permits their germination. The hyphae, which, develop subsequently, play a very important role in the initiation of infection. Dead petals often stick to leaves and this allows the disease to become established.While studying clonal dispersal and spatial mixing of *S. sclerotiorum* isolates fromrape fields in Canada, it was observed thatspatial mixing of ascospore inoculum fromresident or immigrant sources took place (Kohliet al. 1995).

Disease prediction models

Forecasting systems have been developed for white stem rot of mustard that use petal testing, a checklist or other tools based on environmental conditions. While no forecast system is 100% accurate they do provide practical direction in making a decision whether a fungicide application is warranted.Many factors influence a forecasting system and its ability to predict the actual incidence of disease. Most predictive models evaluate several environmental and crop variables such as: i. field cropping history ii. field disease history iii. apothecia and ascospore presence, iv. Rainfall v. soilmoisture vi. weather forecast vii. canopy density. Other important variables factors affecting the incidence of the disease include:changing inoculum levels during flowering, heat units, daily and weather related inoculum fluctuations, light penetration, leaf area index, crop height, and leaf wetness.

Field and nearby field cropping and disease history are an indirect means of measuring the potential for presence of spores. While apothecia produced from sclerotia within the field are considered the main source of spores, spores produced in nearby fields and blown into the crop are important in disease development. Studies have shown that most ascospores are deposited within 100 m of the source, and therefore adjacent fields can be an important source of inoculum. Fields with sclerotia at distances of greater than 500 m to 2 km would likely represent a minimal source of inoculum, as most ascospores would have been deposited within 100–400 m of the apothecia from which they were produced. The presence of apothecia is a good indicator of the potential for spore production.

New tools that can test for the presence of ascospores are becoming available. Mustard petal tests that look for the presence of *S. sclerotiorum* DNA are commercially available, and ascospore samplers too can be installed in fields that capture ascospores, which sent to a laboratory, can determine the amount of inoculum in the field. Measures of ascospores with these technologies will help in determining if the risk of the pathogen is significant for a field. But it is weather conditions that will determine the risk of the disease be significant.Rainfall and soil moisture are necessary for sclerotia germination, spore production, and spore germination and growth. Ideal mustard growing weather is also ideal for Sclerotinia. Soil moisture indicates sclerotia germination and, therefore, the potential for ascospore production rather than infection and disease development. Frequently water from heavy dews dripping off the plant is enough moisture for sclerotia germination.Weather forecasts can improve the reliability of the disease forecast because sudden weather changes can cause infestations to occur unexpectedly or high-risk fields may show limited disease development.

Hot, dry weather can greatly reduce the risk of Sclerotinia infection.

The possibility of forecasting stem rot of rapeseed based on petal infestation (PI) with the pathogen was first suggested by Gugel and Morrall (1986) and later refined by Turkington et al. (1991a). A strong relationship between disease incidence and percentage plant infection (PI) at early bloom stage has been established. Sampling at 5–6 sites per crop and plating 40 petals per site is enough to estimate percentage PI with standard error of about 5% in most fields. Forecasting is done by collecting petals in the afternoon and should wait several hours after rainfall as precaution against slight under estimation of PI values. Canopy density affects stem rot. By altering the microclimate in the crop, the relationship between inoculum and disease incidence is also affected, andmore disease occurred per unit of PI in dense crops (Turkington et al. 1991b). According to Bom and Boland (2000), the model that include petal infestation and soil moisture predicts more fields correctly than the model using petal infestation alone, but the accuracy of both affected by the timing of soil moisture measurements in relation to petal infestation and threshold values in discriminating categories of soil moisture and petal infestation. Twengstrom et al. (1998a, b) suggested a forecasting system of *Sclerotinia* stem rot in spring sown oilseed rape. The disease forecasts are based on the following information: (a) accumulated number of germinated sclerotia in depots, including the number of sclerotiawith active apothecia (turgid, light brown); (b) frequency of apotheciumoccurrence in rape fields selected at random and in fieldswith previous attack of *S. sclerotiorum*; (c) growth stage of the crop compared with the development of the fungus; (d)rainfall (and temperature) at localities with depots of sclerotia; and (e) weather prognosis for 5 days at the time of the forecast.

High apothecial development only takes place after a rainfall of a minimum of 30 mm within a period of 7–14 days. On the other hand, this precipitation does not necessarilycause a high germination because of evaporation or an unfavourable microclimate. To cause any serious damage, the germination of the sclerotia must have started 7–14 days before initial flowering. Apothecia formed after this time will come toolate to do any damage.Preliminary experience seems to indicate that there is a risk of attacks when the accumulated number of germinating sclerotia in the depots is over 30%at the time of the forecast. Besides this, the majority must have active apothecia. Ghasolia and Shivpuri (2005) observed that Sclerotia at upper surface of soil produced more apothecia. The prediction model developed for white stem rot as under:

$$Y = -19.14 - 4.59\,X_1 + 2.53\,X_2 + 1.02\,X_3 - 0.19\,X_4 - 0.00\,X_5 + 0.63\,X_6,$$

where X_1 = Tmax.; X_2 = Tmin.; X_3 = RHmor; X_4 = RHeve; X_5 = Sunshine h; X_6 = RF.

Aghajani et al. (2010) from Iran reported that Gompertz model with a mean $R^2$of 94.69 was selected as most appropriate model for determining Sclerotinia stem rot progress in the field.Sclerotinia incidence can vary greatly among fields and years, making scheduled routine spraying of fungicides unprofitable. However, when Sclerotinia risk is high, preventative fungicide applications can effectively lower disease severity and improve yield.

Conclusion and future strategies

- Alternaria blight, white rust, downy mildew, powdery mildew and white stem rot are major five diseases occurring in rapeseed-mustard, and are responsible for huge losses both quantitatively and qualitatively.
- Cultural practices viz., field sanitation, clean cultivation, timely sowing, adjusting the date of sowing keeping in view occurrence of the disease in the particular area, destruction of disease debris after the harvesting, deep summer ploughing during May–June, use of proper dose of fertilizers, timely and proper irrigation and crop rotation at least for three years with non–cruciferous crops play significant role in minimizing and reducing the primary inoculum, thus cause reduction in the secondary inoculum.
- Deployment of resistant varietieswherever available has amajor role in mitigating the losses due to diseases.
- Disease prediction models wherever developed can be of great help in the timely deployment of disease management practices.
- Use of fungicides, bioagents, botanicals, organic amendments etc. as worked out for various diseases needs to be adopted atrecommended doses will prove useful for containing the disease losses. Their interaction with various forecasting models needs to undertaken.
- Analysis of host–pathogen-environment interaction for developing disease forecasting models. The role of individual dependent and independent factors needs to be study in details.
- Development of simple, functional, reliable, easy to understand by the farmers and effective disease forecasting models.
- Use of Information Technology (IT) to manage, storage, processing, analysis and presentation of data.
- Identification of slow white rusting, slow mildewing, slow blightening, disease tolerant and partial resistance genes.

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