



Alternaria-Brassica pathosystem: development and perspective

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Abstract

A sudden outbreak of a plant disease might result in significant economic losses. Understanding the disease triangle, disease epidemics, and forecasting are all crucial in dealing with pathogens. The Genus *Alternaria* is a notorious fungus having ~275 species, responsible for enormous economic losses varying from 10% to 70% in crucifers which are considered as major edible oil crops all over the world. The *Alternaria* blight/ black spot disease is the major bottleneck in the global production of commercial oilseed Brassicas. Among *Alternaria* species, *A. brassicae*, *A. brassicicola*, *A. raphani*, and *A. alternata* are well-documented for infecting several members of the Brassicaceae family. Recent advanced taxonomic investigations have yielded a wealth of data that may be used to outline the evolutionary lineages within *Alternaria* and related genera. In addition, some R genes/QTLs such as *chitinase*, *endochitinase 'ech42'*, *glucanase*, *PmAMP1*, *AbVF19*, *Amr1* etc. from host crop and non-hosts have also been identified through molecular markers targeting *A. brassicae* and *A. brassicicola*. Though the prediction models are available, but their practical utility is still limited for effective forewarning. Another problem is the lack of robust data on the potentiality of indigenous as well as exotic genetic resources with a resistance to *Alternaria* blight making the exploitation of non-host resistance (NHR) mechanisms unfeasible in protecting Brassica crops. The availability of resistant sources is not yet reported in the U triangle species. Thus, the focus of this review is to identify gaps and bottlenecks in our understanding of the *Alternaria-Brassica* pathosystem in multiple dimensions which could aptly incorporate in holistic management approach with a sustainable and profitable solution to deal with the black spot disease of crucifers.

Keywords: *Alternaria* spp., black spot, resistance, race, R genes, QTLs

Introduction

Cultivated oilseed *Brassica* crops are challenged by numerous biotic stresses causing considerable economic losses of average 19.9 percent influenced by environmental factors all over the world. Out of 44 pathogens known to infect crucifers, 16 pathogens are considered as the major ones based on their geographical distribution, host range, losses, and resources deployment to manage them. *Alternaria* blight is one of the serious threats for the oilseed Brassica in temperate regions. The genus *Alternaria* is a universal fungus that includes saprophytic, endophytic, and plant pathogenic species. Various species of the *Alternaria* genus grow and multiply on plant parts, agricultural products, animals, and soil. Species of *Alternaria* genus are recognized as serious fungi, responsible for huge losses in various crops. *Alternaria* has been reported to have more than 4000 *Alternaria*-host associations according to the United

States Department of Agriculture Fungal Host Index and ranks 10th among nearly 2000 fungal genera based on the entire host records. Numerous *Alternaria* species have been illustrated through their taxonomic and morphological characteristics, pathogenic nature, and their distribution (Simmons 1967, 2002, 2007). Due to the considerable harmful effects of *Alternaria* spp. on plants and their surroundings, accurate and speedy detection of *Alternaria* species by agriculturists, researchers, and medical mycologists is required. Similarly, a wide understanding of host-pathogen system as well as the resistant plants and their mechanism of resistance is important to tackle this pathogen and avoid heavy losses (Woudenberg *et al.*, 2013). In this review, the taxonomy, origin, distribution and economic impact of *Alternaria* species, which is one of the most important pathogen of the crucifers will be discussed in details. Moreover, different aspects of the related host-pathogen system

including the genetics and molecular mechanism have also been described to understand the current advancement in research towards this direction. This will help researchers to identify the lack in the field and employ novel techniques to control its spread and infection. The review also describes in detail the different disease prediction models that can be used to forecast the different

aspects of disease spread. Furthermore, different aspects of host resistance against the disease and management of the disease has also been discussed in this review to address the current problem of global food security and the threat posed by the attack of this pathogen to the cultivation of Brassica plants (Fig. 1).

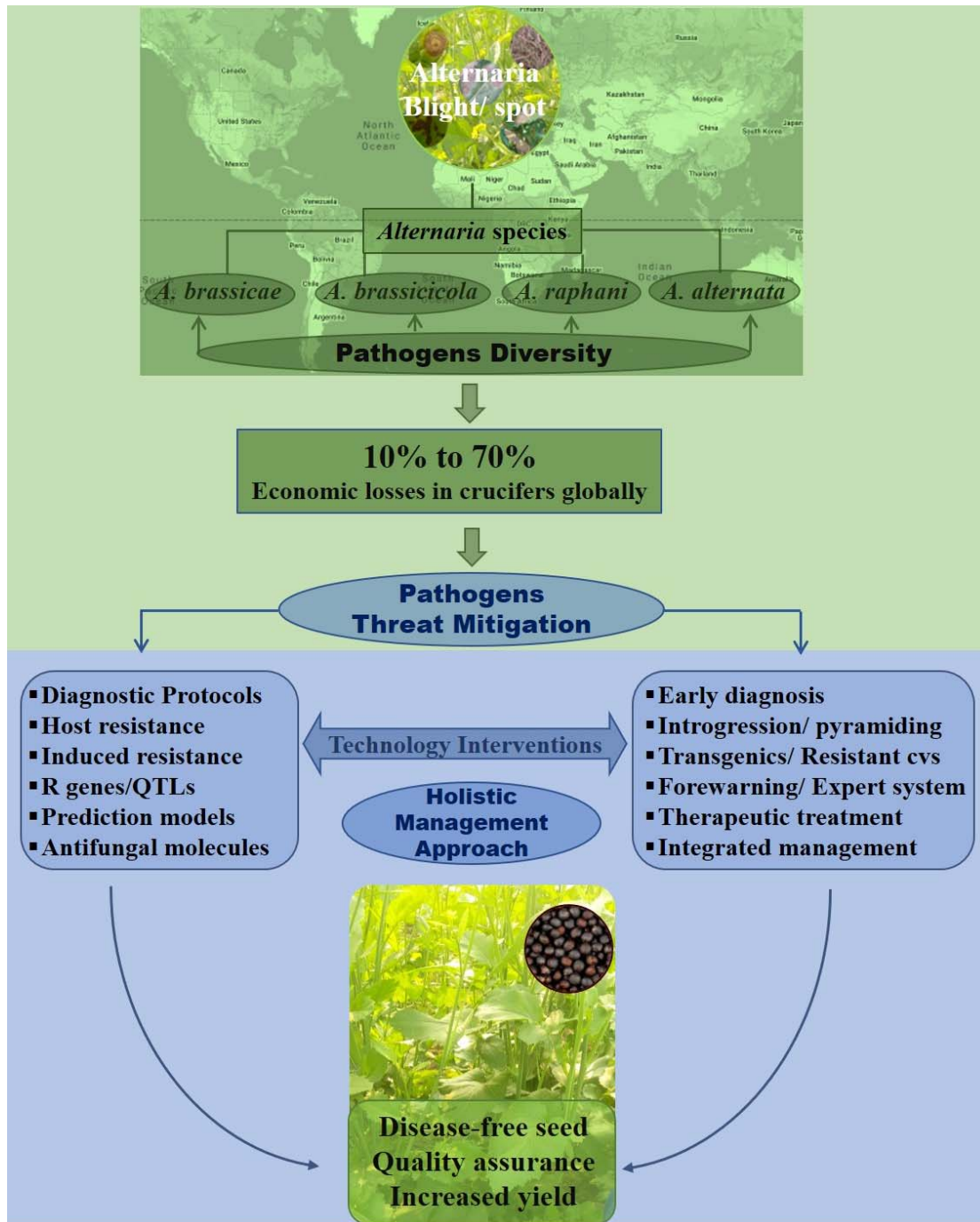


Fig. 1: Alternaria Pathogen threat mitigation through holistic management approach

Taxonomy

Alternaria was first illustrated by Nees (1816) and established as *A. tenuis* species. *Alternaria* genus produces peculiar dark colored, phaeo-dictyosporous chains that have a beak of tapering apical cells. Although some *Alternaria* species emerge to have a sexual stage to complete their life cycle, the majority of them does not possess sexuality. Von Keissler (1912) synonymies both *Torula alternata* and *A. tenuis* with *Alternaria alternata*, being indistinctness in Nees's description of *A. tenuis*. The outcome of life span research on *Alternaria* taxonomy by Simmons (2007) summarized and documented 275 *Alternaria* species based on morphological characteristics. One species of *Alternaria* was moved to the genus *Prathoda* whereas *Alternariaster*, *Chalastospora*, and *Teretispora* were removed from the genus. The molecular analysis found multiple non-monophyletic genera in *Alternaria* genus, and *Alternaria* species clades, which were inconsistent in associating species-group based on morphological characteristics (Pryor *et al.*, 2009; Wang *et al.*, 2011; Lawrence *et al.*, 2012). The *A. alternata*, *A. brassicicola*, *A. infectoria*, *A. porri*, and *A. radicina* species-group were strongly supported by these findings and two new species groups, *A. sonchi* (Hong *et al.*, 2005) and *A. alternantherae* (Lawrence *et al.*, 2012) and three new genera, *Crivellia* (Inderbitzin *et al.*, 2006), *Undifilum* (Pryor *et al.*, 2009), and *Sinomyces* (Wang *et al.*, 2011), were illustrated. Most recent molecular studies by Lawrence *et al.* (2013) recognized *A. panax* and *A. gypsophilae* as the two novel species-groups of *Alternaria*. Further, the findings raised eight species-groups to sections within *Alternaria*. *A. infectoria* species-group could not obtain the grade of a section through the eight asexual phylogenetic lineages in sexual phylogenetic *Alternaria*. The *Alternaria* complex presently comprises *Alternaria*, *Chalastospora*, *Crivellia*, *Embellisia*, *Nimbya*, *Stemphylium*, *Ulocladium*, *Undifilum*, and *Sinomyces* (Simmons, 2007).

The Brassicas

The *Brassica* belongs to the family *Brassicaceae* which comprises 3709 species in 338 distinct genera. The member of the family includes various economically important crops contributing to high-quality edible and industrial oils, vegetables, and weeds (Warwick *et al.*, 2006). The area, production, and yield during 2018–2019, globally, were estimated to be 36.59 Mha, 72.37 Mt, and 1980 kg/ha, respectively for rapeseed-mustard. India contributed 19.8 and 9.8%, area and production, respectively, and holds third position globally (USDA, 2020). The major oilseeds and rapeseed-yielding species belong to the genus *Brassica*. These species include *B.*

napus L., *B. rapa* L. (AA, 2n = 20), and *B. juncea* (L.) Czern. & Coss. (AABB, 2n = 36), which are commonly known as leaf mustard, turnip rape, and rape respectively. Other common names for *B. napus* include rapeseed, oil rape, colza, oilseed rape, swede rape, and Argentine rape. Similarly for *B. rapa*, Polish rape, oil turnip, and rapeseed for *B. juncea*, rapeseed, Indian mustard, oriental mustard, and brown mustard are the popular common names. The common names of the three species are generally fallen under different groups in different countries. In India, under rapeseed, turnip rape and mustard are included, whereas in North America and Europe, under rapeseed, rape and turnip rape are included. China cultivates all three species but winter-grown rape is the chief source of rapeseed. Besides these three rapeseed crops, Brassicaceae includes *B. rapa* L. var. Toria, *B. rapa* L. var. Brown, *B. rapa* L. var. Yellow Sarson, *B. nigra* (L.) Koch, *B. hirta* Moench (= *Sinapis alba* L.), *B. carinata*, A. Braun, *B. tournefortii* Gouan, *Eruca sativa* Mill. (= *E. vasicaria* ssp. *sativa* (Mill.) Thell.), *Camelina sativa* Crantz., *Crambe abyssinica* Hochst. ex O.E. Schulz, and *C. hispanica* L.

Occurrence and distribution

Alternaria brassicae and *A. brassicicola* have been found on Brassicaceae hosts throughout the world (Meena *et al.*, 2010; Saharan *et al.*, 2016; Akhtar *et al.*, 2017a). On oil-yielding brassicas, *Alternaria brassicae* and *A. brassicicola* are the most harmful, and both are frequent on vegetable crucifers. According to CAB International (2007), Black spot, caused by these two fungi, is a major disease in the Netherlands and other European countries, which causes yield losses of up to 75% in different crops. While doing quarantine testing during the import of Brassica germplasm into India from more than 20 countries, Akhtar *et al.* (2017a) reported that *A. brassicicola* was more common as compared to *A. brassicae* in seeds, whereas, *A. brassicae* was observed only in the material from nine countries. This is also evident from several reports that yield loss in rape due to *A. brassicicola* ranged up to 50% in Germany (Mac Kinnon *et al.*, 1999), and the seed yield loss of 59% was determined in cabbage in Bangladesh due to *A. brassicicola* (Hossain and Mian, 2005).

Alternaria raphani has been found in Canada, Denmark, Egypt, Greece, India, Iran, Japan, the Netherlands, and the United States on diverse brassicaceous hosts (Ellis, 1971). It is most frequent on radish, but can also be seen on other Brassica plants and crops that produce oil (Verma and Saharan, 1994; Akhtar *et al.*, 2017a). *A. raphani* was detected in 80% of a seed lot of *R. sativus* and a large proportion of diseased seedlings died before emergence

due to this fungus (Vannacci and Pecchia, 1988). Rude *et al.* (1999) intercepted *A. raphani* in seed samples of *Brassica rapa* from Saskatchewan and Alberta provinces of Canada with a significant reduction in seed germination. Akhtar *et al.* (2017a) also intercepted *A. raphani* on *B. rapa* from Australia and *B. carinata* from Germany.

While *Alternaria alternata* is a widespread saprophyte that is found on a variety of plants and other substrata, it is regarded as a poor pathogen among the *Alternaria* species that is identified to infect Brassicaceous hosts. It appears on a wide range of Brassicaceous plants, including oil-producing crops such as rapeseed-mustard, Crambe, and Brassica vegetables (Ellis, 1971; Verma and Saharan, 1994).

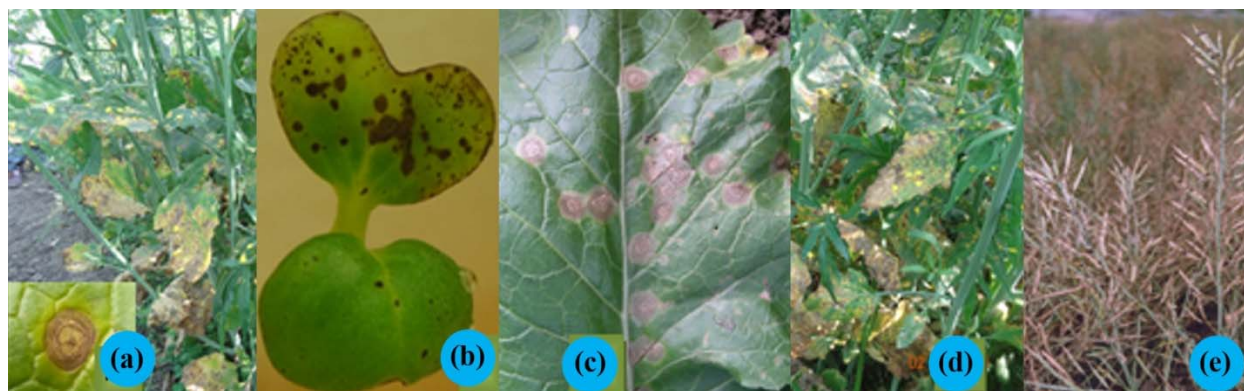


Fig 2: *Alternaria* blight disease lesion (a) with concentric rings, blight on (b) cotyledon, (c) leaf, (d) whole plant, and (e) silique of *Brassica juncea* (Photo: Dr. P.D. Meena)

There may be differences in color, size, shape, formation of concentric rings, and yellow-halo nearby the lesions depending on the diverse agro-ecological locations, host genotypes, soil nutritional status, and involved pathotypes. Expanding necrotic lesions reduce photosynthetic capability, hasten leaf aging, and cause leaf death which may further lead to the collapse and death of plants under high pathogen inoculum stress. Moreover, the effects on the cell membrane, chloroplast, and mitochondria have been seen in rapeseed (Verma and Saharan, 1994; Tewari, 1991a; Saharan *et al.*, 2016).

Oilseed Brassica yield losses due to *Alternaria* blight range from 10% to 70%, depending on the period of infection, post-infection environmental conditions, and the adopted management measures (Saharan *et al.*, 2016). *Alternaria* blight causes a 32-57 percent decline in seed output and a 4.2-4.5 percent fall in oil content in mustard in Nepal (Shrestha *et al.*, 2005). The maximum test weight seed loss (50.0%) in cauliflower from Uttarakhand due to

The disease and economic impact

Among the key diseases of *Brassica* crops, the black spot disease or *Alternaria* blight is the major hindrance in global production. Incidence of *Alternaria* in the early stage of the crop may lead to seedling decay, as symptoms start arising on the cotyledons leading to a poor crop stand, whereas infection in the later growth stage of plants on leaves, leaf petioles, stems, inflorescences, siliquae, and seeds drastically reduce quality and final yield of the crop (Akhtar *et al.* 2017a). Symptoms in the form of lesions are nearly the same in every infected host species (Meena *et al.* 2010; Meena *et al.* 2016a). Dark brown spots/lesions with a characteristic concentric edge, often with a yellowish chlorotic halo, occur on older leaves, stems, and siliques as the disease progresses (Figure 2).

Alternaria blight (*A. brassicae* and/ or *A. brassicicola*) was reported by Prasad and Vishunavat (2006). Kumar (1997) reported yield loss of 27.5, 25.0, and 20.3 percent due to *Alternaria* blight in *B. rapa* var. *yellow sarson*, *B. rapa* var. *brown sarson* and *B. juncea*, respectively from Himachal Pradesh, India. The disease during the extreme stage affects the seed quality where the seeds are deformed with patches on the seed coat which impact the marketing of the seed as well.

The disease is generally assessed using descriptive keys, standard area diagrams, incidence-severity relationships, inoculum disease intensity relationships, infection type, and host reaction as resistant, tolerant, and susceptible, and disease stress tolerance attributes (Saharan *et al.*, 2016).

The Pathogens

Four *Alternaria* species have been mainly found to be harmful to cruciferous crops: *Alternaria brassicae* (Berk.)

Sacc., *A. brassicicola* (Schwein.) Wiltsh., *A. raphani* Groves and Skolko, and *A. alternata* (Fr.) Keissl. These are known to cause the Black spot disease of *Brassicaceae* crops. These four *Alternaria* species, responsible for disease transmission to oilseed Brassicas, cruciferous crops, wild cruciferous hosts, and weeds, have a wide range of hosts. Recently, morphologically and phylogenetically identified 10 species of genus

Alternaria namely, *A. alternata*, *A. arborescens*, *A. brassicae*, *A. ethzedia*, *A. hordeicola*, *A. infectoria*, *A. japonica*, *A. malvae*, *A. metachromatica*, and *A. tenuissima* were confirmed pathogenic on *B. napus* cv. Thunder TT and *B. juncea* cv. Dune as well (Al-Lami *et al.*, 2019). The identification of the majority of *Alternaria* species infecting rapeseed-mustard crops is based on morphological and physiological parameters (Table 1).

Table 1 Description of morphological characters, colony growth and sporulation pattern of *Alternaria* spp. from rapeseed-mustard (Source: Blagojevic *et al.*, 2020)

Key character	<i>A. brassicae</i>	<i>A. brassicicola</i>	<i>A. japonica</i>	<i>A. alternata</i>
Mycelial texture and shape	Cottony, circular	Cottony, circular	Cottony, circular	Aerial or cottony, circular
Colony colour	White and brownish	Brown black	Grey greenish	Grey, brown, and green
Colony margin	Cream and pale	Yellowish or cream	Various	Various
Colony growth (mm/day)	3.3 ± 0.7	5.9 ± 0.5	4.0 ± 0.5	10.0 ± 1.1
Conidiophores (µm)	125.3 ± 19.4	112.7 ± 45.6	105.1 ± 44.4	72.9 ± 23.2
Conidial shape	Obclavate mostly	Ellipsoid, ovoid, or obclavate	Ellipsoid, ovoid, or obclavate	Obclavate to long ellipsoid
Conidial length (µm)	152.2 ± 32.7	36.5 ± 8.7	66.9 ± 9.9	36.9 ± 5.8
Conidial width (µm)	13.1 ± 1.7	9.0 ± 2.1	10.4 ± 4.0	9.1 ± 3.2
Beak length (µm)	62.3 ± 12.4	2.4 ± 0.5	3.0 ± 1.3	4.6 ± 1.6
Number of transversal septa (min.-max.)	3–15	1–8	1–8	1–8
Number of longitudinal septa	2-5	0-5	2-7	0-6

Many liquid and solid media for *in vitro* culture and sporulation of *Alternaria* spp. have been documented (Verma and Saharan, 1994; Meena *et al.*, 2012). *A. brassicae*, *A. brassicicola*, and *A. raphani* grow well in most of the carbon sources (Taber *et al.*, 1968). For improved sporulation, the ideal temperatures and relative humidity for *A. brassicae*, *A. brassicicola*, and *A. raphani* are 20-25°C and 95-100 percent, respectively (Ansari *et al.*, 1989; Taber *et al.*, 1968; Changsri and Weber, 1960, 1963). These three species require a pH range of 6-8 for growth and sporulation. Alternating light and darkness promote abundant development and sporulation in *A. brassicae* (Ansari *et al.*, 1989; Verma and Saharan, 1994; Taber, 1964); however, continuous light inhibits sporulation (Ansari *et al.*, 1989; Verma and Saharan, 1994; Taber, 1964; Singh and Suhag, 1983). Infected seeds, diseased plant debris, pathogen propagules deep in the soil, and other crucifers/weed hosts in a given agro-ecosystem are all the ways for the pathogen to survive and propagate (Chupp and Sherf 1960; Dixon 1981; Verma and Saharan 1994; Meena *et al.* 2016b). All the four *Alternaria* species are found on seeds of crucifers (Figure 3) and such seeds have been found to carry the inoculum

with a high rate of transmission under favorable temperature. (Atkinson, 1950; Vannacci and Pecchia, 1988; Sivapalan and Browning, 1992; Kubota *et al.*, 2006; Shrestha *et al.*, 2000). Infections on host plants begin with infections on infected seed, spores produced on agricultural leftovers, infections on cruciferous hosts, weeds, or potentially micro-sclerotia, and the chlamydospores produced on infected debris. Conidia are prevalent during wet weather, and rain splash and wind distribute them locally. Lesions grow and produce wind-borne spores in favorable weather circumstances, which can spread the infection to other plants on the same or nearby plants. When conditions are favorable for infecting seed and other portions of plants, which constitute the pathogen's source of survival, the cycle continues throughout the season. (Saharan, 1992; Verma and Saharan, 1994; Mehta *et al.*, 2005). Akhtar *et al.* (2017b) documented the survival of *A. brassicicola* in cryo-preserved seeds of *B. juncea* for more than 14 years.

Depending on the *Alternaria* spp. and the interaction between host-pathogen, the intensity of the disease can differ, this is generally regulated by the virulent genes.

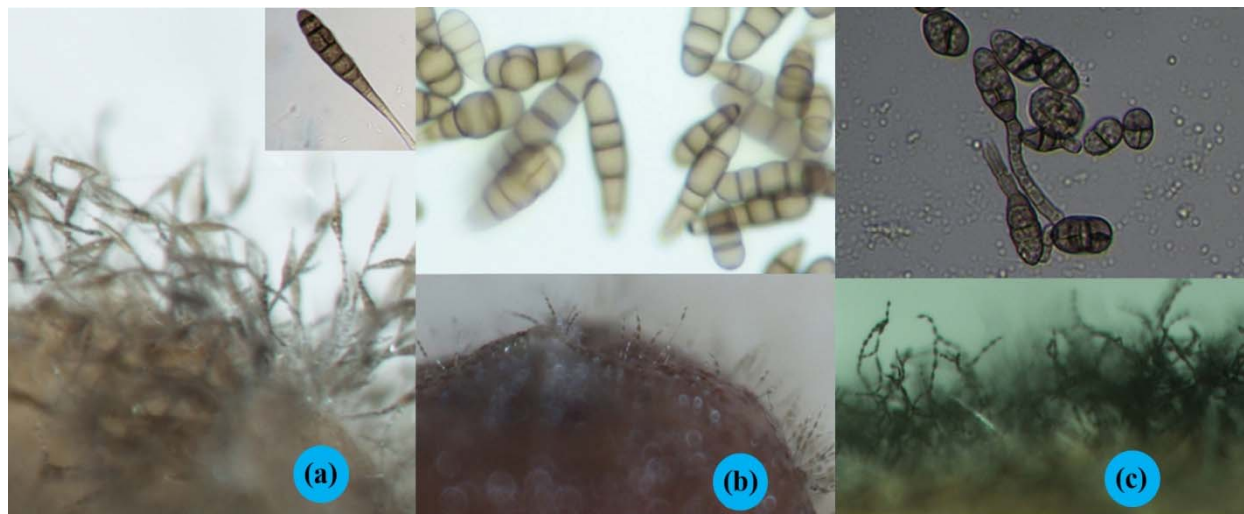


Fig. 3: Growth and conidial morphology of (a) *Alternaria brassicae*; (b) *A. brassicicola*; (c) *A. alternata*. (Photo: Jameel Akhtar)

The pathogenicity-related genes can be identified, cloned, and sequenced for *Alternaria* species that infect crucifers which would help to clarify various questions about their relationship with crucifer hosts. (Cramer *et al.*, 2006; Jasalavich *et al.*, 1995; Kim *et al.*, 2007). In *A. brassicicola*, pathogenicity factors and the transcription factor *Amr1* have been discovered (Mamgain *et al.*, 2013; Cho *et al.*, 2012). A study has reported that cell wall integrity, conidial survival, and pathogenicity of aged *A. brassicicola* spores are all dependent on a non-ribosomal peptide synthase gene (*AbNPS2*) (Kim *et al.*, 2007). Generally, for a functional genomics study, *A. brassicicola* is regarded as the species of choice because over 100 genes have been functionally evaluated using various strategies such as gene knockdown and overexpression (Oide *et al.*, 2006; Cho *et al.*, 2006, 2007, 2009; Kim *et al.*, 2007; Mamgain *et al.*, 2013).

Pathogenesis

On cruciferous hosts, the infection mechanism and pathology of four *Alternaria* species have been extensively researched. The role of external and internal variables, as well as enzymes, toxins, and genes driving pathogenesis, has been determined. The *A. brassicicola* genes *AbVF19* and *Amr1* makes this pathogen an effective and successful crucifer facultative parasite (Verma and Saharan, 1994; Cho *et al.*, 2007, 2009, 2012; Giri *et al.*, 2013; Mamgain *et al.*, 2013). The presence of brown-colored dead cells at the infection site indicates a hypersensitive host response to pathogen invasion, which inhibits pathogen proliferation. There is still a need for more information on the *Alternaria-Brassica* interaction to establish accurate and repeatable screening

approaches as well as properly defined pathogenic variability (Meena *et al.*, 2015).

A variety of toxins and metabolites are produced by *Alternaria* species, including terpenoids, pyranones, steroids, and nitrogen-containing compounds (s). During *Alternaria*-crucifer interactions, many biochemical changes occur in both the host and the pathogen. Different types of primary and secondary metabolites are produced as a result of these metabolic changes, which influence the host defense system and pathogen virulence. *A. brassicicola* produces compounds like antitumor depudecin, antibiotic complex brassicocolin and phytotoxic brassicenes. *Alternaria* species produce both host-specific and non-host-specific toxins, which aid pathogenicity and help the pathogen become successful. The role of toxins in the infection process, as well as their biosynthesis, method of action, chemical structure, role in host defense, and accumulation, have been thoroughly discussed (Verma and Saharan, 1994; Lou *et al.*, 2013; Pedras *et al.*, 1998, 2001, 2002, 2009; Thomma, 2003; Marmath *et al.*, 2013a; Meena *et al.*, 2014).

Pathogen diversity

Despite its reputation as an imperfect fungus, *Alternaria* exhibits genetic variability within species, which could be attributed to mutation, somatic hybridization, heterokaryosis, uniform host selection, significant dispersal, or the presence of a cryptic sexual stage. In four species of *Alternaria* that cause blight and black spot disease in crucifers, pathogenic diversity has been identified in the form of pathotypes or variations. Pathogenic variability in *Alternaria* species is driven by

several variables such as pathological, symptomatological, morphological, cultural, nutritional, biochemical, genetical, molecular, proteome level, thermo-sensitivity, and fungicidal sensitivity, among others. Primary interpretation on dissimilarity in cultural characteristics and pathogenesis of different isolates of *Alternaria* were made by Stoll (1952), in *A. brassicicola* - vegetables by Van Schreven (1953), in *A. brassicae*- Brassica, and by Atkinson (1953) in *A. raphani* – radish host-pathosystem. Based on the dissimilarity in their physiological and pathological characteristics, three strains of *A. alternata* have been isolated from *Crambe*, strain B is the most pathogenic, strain A is moderately pathogenic, and strain C is the least harmful. Similarly, Atkinson (1953) had divided *A. raphani* into two races: “Wild Type” and “Variant Type” based on the pathological and physiological differences. Three isolates of *A. brassicae*, A, C, and D have been identified that differ in appearance, growth, sporulation, and culture properties, as well as virulence in *B. carinata* (Vishwanath and Kolte, 1997). Stoll (1952) classified three pathotypes of *A. brassicae* that infect cauliflower siliquae as very aggressive, less aggressive, and non-pathogenic. Gupta *et al.* (2004) discovered four pathotypes of *A. brassicae* from *B. juncea* and named them Bj-4, Bj-5, Bj-6, and Bj-7 based on host differences and symptomatological differences. Mehta *et al.* (2003) found the pathotypes DLK, RSR-1, and GDP of *A. brassicae*. Brassica seeds, cotyledons, and leaves all showed a positive reaction when categorizing *A. brassicae* isolates as virulent or avirulent, according to Meena *et al.* (2017). Such research could be utilized to produce and evaluate resistant brassica germplasm, particularly in case of populations of *A. brassicae* that are more virulent.

Some researchers merely reported the number of isolates with differentials rather than pathotypes (Kumar *et al.*, 2003; Mehta *et al.*, 2003; Sangwan and Mehta, 2007). Other characteristics have been utilized to identify pathogenic variability in *Alternaria* species that infect crucifers in the absence of host differentials. Symptomatology (Gupta *et al.*, 2004; Goyal *et al.*, 2013), morphology (Vishwanath and Kolte, 1997), genetics (Sharma *et al.*, 2013; Pramila *et al.*, 2014), and proteome level are among the criteria that have been linked to pathotype virulence. The aggressiveness of pathogens reveals a wide range of tolerance to Brassica species, including *A. brassicae*. (Meena *et al.*, 2012). Using two marker systems, universal rice primers (URP) and inter simple sequence repeat (ISSR), the molecular characterization of 38 *A. alternata* isolates obtained from indigenous and alien sources indicated variations based on the geographical origin of diverse isolates. This was

the first research to employ URP-PCR in conjunction with ISSR-PCR to characterize genetic diversity in *A. alternata*, and was sensitive and trustworthy (Kandan *et al.*, 2014).

Epidemiology and Forewarning

The primary infection of *Alternaria* takes place on the cotyledonary leaves, which serve as a secondary infection source for the entire crop. For infection, >4 hours of leaf wetness at 25°C results in enhanced infection and disease transmission. The spores infect other sections of the plant when temperature conditions are favourable and if dew is present. Under ideal climatic circumstances, the infection spreads through the stomata, and new lesions appear within 4-6 days. The pathogen affects the seed by penetrating the silique’s tissues. Darkness or low light intensity of 1000 lux, temperature of 25°C, and relative humidity of >90 percent have been described as favourable conditions for *Alternaria* conidial germination (Shrestha *et al.*, 2005).

Temperatures of 12–25°C, relative humidity >70% with occasional winter rains or irrigations, wind speeds of 2–5 km/h, closer plant spacing (30–15 cm), and high nitrogen dosages (80 kg/ha) have been described as favorable environmental factors for disease growth under natural conditions (Sangeetha and Siddaramaiah, 2007). The penultimate week of October’s raya crop had 52 percent disease, but the third week of November’s harvest had only 15.5 percent disease. *Alternaria* spores were trapped in 7-day volumetric spore traps for around 10-11 days prior to the onset of the disease, and their concentration rose until it peaked in March. The spores were trapped at its peak between 10 a.m. and 2 p.m. (46 percent of total spores) and were lowest between 10 p.m. and 6 a.m., following which its concentration dropped dramatically after 2 p.m. (Singh 2005).

According to Meena *et al.* (2011), disease severity rose when the planting date was postponed. With the delayed sowing, the A value AUDPC and the ‘r’ value (apparent infection rate) were higher in the cv. ‘Varuna.’ *Alternaria* blight severity was substantially lower in the October planted crop. In comparison to line sowing, the disease spread faster using the broadcasting method (45 cm). When K (40 kg/ha) was administered along with the recommended fertilizer amount, the disease intensity fell as well (Gupta *et al.*, 2018). Chattopadhyay *et al.* (2005) used cv. ‘Varuna’ seeded on 10 dates at weekly intervals to assess data for *Alternaria* blight progression and development from eight locations. The earliest development of disease on leaves occurred between 42 and 139 days after sowing (DAS), according to the findings. The disease then developed on pods with 67 to

142 DAS, with a peak at 99 DAS. Severity of *Alternaria* blight on leaves was positively correlated to a daily T_{max} of 18-27°C, daily T_{min} of 8-12°C, daily T_{mean} of >10°C, RH_{mor} >92%, RH_{even} >40% and RH_{mean} of >70% in the preceding week. Disease severity on pods was favoured by a daily T_{max} of 20-30°C, daily T_{mean} of >14°C, RH_{mor} >90%, daily RH_{mean} of >70%, sunshine >9 h and leaf wetness >10 h. Temperature and relative humidity (RH) factors favourable to disease development were found to match laboratory findings. At least one week before the disease first emerged on leaves and pods, regional and cultivar-specific models could forecast the crop age at which *Alternaria* blight first appeared on leaves and pods, as well as the peak blight severity on leaves and pods (Chattopadhyay *et al.*, 2005). The role of variations in disease development has also been highlighted, in addition to weather considerations. In comparison to *B. carinata* (HC-2, HC-9001), *B. napus* (GSH-1) and *B. alba*, the rate of disease development was faster on *B. juncea* (RH-30, RH-8113, RH-8695, RH-8546) and *B. campestris* (YSPb-24, BSH-1, Candle, Shiva) types (Mehta *et al.*, 2008a; Saharan *et al.*, 2016).

Disease prediction models

The Gompertz, Logistic, Monomolecular, and Exponential models have all been utilized to construct prediction models for the *Alternaria* blight (Table 2). Dang *et al.* (2006) used the Gompertz model to establish a prediction equation for the development of *Alternaria* blight, which explained two elements, $DI = \text{Exp}^{[\log A \text{ Log} (-B * \text{Time})]} + C * \text{Sowing Day} + D * \text{Factor 1}$, where DI stands for 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 kinds (with varied genetic composition) that reacted differently to the natural inoculum and factor 1 can be regarded as the weather index, while factor 2 can be interpreted as the contrast between the heating factor and the moisture factor (Dang *et al.*, 2006).

Sangwan *et al.* (2000) showed that the Gompertz model may be used to predict *Alternaria* blight using two components A and B derived from weather conditions and disease progression analyses.

Where, Factor A = $0.091 \times T_{max} + 0.887 \times T_{min} + 0.036 \times RH_{mor} + 0.808 \times RH_{eve} - 0.644 \times \text{Sunshine h}$, and Factor B = $0.317 \times T_{max} + 0.317 \times T_{min} + 0.933 \times RH_{mor} + 0.347 \times RH_{eve} - 0.618 \times \text{Sunshine h}$. These two components (A and B) explained 60.3 and 24.5 per cent of total variation in the weather variables, respectively, and jointly explained 85 per cent of total variation.

The most favourable weather conditions for disease progression were found to be at T_{max} of 20°C and RH_{mor} >90%. The T_{max} and RH_{mor} performed substantial and favourable effects in disease development, according to the stepwise regression analysis. In all cases, the R^2 value was >0.9, indicating that meteorological variables, in addition to varietal characteristics, played a significant impact in disease progression. The following were the prediction equations established for leaves and pods for a variety of locations: (Mehta *et al.*, 2002b; Saharan *et al.*, 2016).

Jha *et al.* (2013) reported that T_{max} is found to be positively linked with disease index and demonstrated that T_{max} (23.2°C), RH_{max} (80%), and RH_{min} (66%), with correlation coefficients (r) of 0.73 for T_{min} and 0.51 for RH_{min} favoured disease development. The regression equation developed for leaves as $Y1 = -47.388 + 5.114 T_{min} - 2.371 T_{max} + 1.492 RH_{min}$ with $R^2 = 0.7376$, whereas for siliqua $Y2 = 31.524 + 4.225 T_{min} - 1.883 T_{max}$ with $R^2 = 0.69203$.

Other researchers reported that black spot of crucifers develops in epidemic form when temperatures range from 18 to 25°C, relative humidity is above 90%, wind speeds are between 2 and 5 km h⁻¹, with intermittent showers (Ansari *et al.*, 1988; Saharan, 1991; Verma and Saharan, 1994). The severity of disease in rapeseed-mustard was significantly exacerbated by closer plant-to-plant spacing, high nitrogen dosages, and frequent irrigation (Saharan, 1991; Stankova, 1972; Verma and Saharan, 1994). The leaf wetness period, minimum, maximum temperature, and relative humidity, date of sowing, crop age, variety, and species of Brassica crops cultivated under various agro-ecological circumstances were all taken into account when developing disease forecasting models (Verma and Saharan, 1994; Dang *et al.*, 2006; Magarey *et al.*, 2005; Mehta *et al.*, 2002b, 2008b; Kumar *et al.*, 2013; Mahapatra and Das, 2014; Mehta, 2014). Disease severity increases with the extended sowing time (Meena *et al.*, 2011). With the delayed sowing, the A value AUDPC (area under disease progress curve) and the 'r' value (apparent infection rate) were higher in the variety 'Varuna.' The disease usually occurs 40-45 days after sowing, with the most essential stages for disease commencement being 45 days and 75 days for disease peak (Meena *et al.*, 2004).

Models based on weekly meteorological data from the week of sowing to the sixth week of crop growth can be utilized to provide reliable *Alternaria* blight forewarning. As a result, credible forewarning for *Alternaria* blight in different varieties of crop for crop age at first appearance of disease, crop age peak severity of disease, and maximum severity of disease in different types of crops is possible

Table 2: Development of different prediction models for the Alternaria blight of rapeseed-mustard

Species	Cultivar	Model	Prediction equation	Reference
<i>Brassica juncea</i>	—	Based on spore trapped data	$ALTn = 21.5 + 1.164ALTe + 0.128ALTp - 0.165(T_{max} - T_{min}) - 0.154(T_{min} - T_{max}) - 0.203(RH_{min}) - 0.095$	Singh, 2005
	RH-30	Gompertz model	$Y = E^{[\log(0.620)^* \log(4.998 * Time)]} - 0.890 * \text{sowing Day} + 0.238 \text{ factor 1} \dots \dots \dots (R^2 = 0.389)$	Dang <i>et al.</i> , 2006
	RH-8113	—	$Y = E^{[\log(2.547)^* \log(1.072 * Time)]} - 0.561 * \text{sowing Day} + 0.086 \text{ factor 1} \dots \dots \dots (R^2 = 0.482)$	Mehta <i>et al.</i> , 2008
	RH-30	—	$Y_1 = -4.3635 + 0.3488 X_1 \dots \dots \dots (R^2 = 0.66) Y_1 = -4.4927 + 0.2755 X_1 - 0.0789 X_3 \dots \dots (R^2 = 0.71)$	Sangwan <i>et al.</i> , 2000
	—	Gompertz model	Exp. $[\log(2.915) \times \log(-0.430 \times \text{Time}) - 0.154 \times \text{Factors (1)} + 0.204 \times \text{Factors (2)}]$ $\dots R^2 = 0.931$	Jha <i>et al.</i> , 2013
<i>B. napus</i>	—	—	$Y1 (\text{Leaf}) = -47.388 + 5.114 T_{min} - 2.371 T_{max} + 1.492 RH_{min} \dots \dots R^2 = 0.7376 Y2 (\text{Silqua}) = 31.524 + 4.225 T_{min} - 1.883 T_{max}$, with $R^2 = 0.69203$	Sangwan <i>et al.</i> , 2000
	—	Gompertz model	Exp. $[\log(2.908) \times \log(0.424 \times \text{Time}) - 0.185 \times \text{Factors (1)} + 0.170 \times \text{Factors (2)}]$ $\dots R^2 = 0.975$	Dang <i>et al.</i> , 2006
	YSPb-24	Gompertz model	$Y = E^{[\log(2.199)^* \log(4.827 * Time)]} - 1.868 * \text{sowing Day} + 0.105 \text{ factor 1}; (R^2 = 0.539)$	Mehta <i>et al.</i> , 2008
<i>B. rapa subsp. yellow sarson</i>	YSPb-24	Gompertz model	$Y_1 = -4.2310 + 0.3420 X_1 \dots \dots \dots (R^2 = 0.68) Y_1 = 4.7131 + 0.2680 X_1 - 0.0797 X_3$ $\dots (R^2 = 0.73)$	Sangwan <i>et al.</i> , 2000
	—	Gompertz model	Exp. $[\log(2.722) \times \log(0.470 \times \text{Time}) - 0.203 \times \text{Factors (1)} + 0.167 \times \text{Factors (2)}]$ $\dots R^2 = 0.974$	Dang <i>et al.</i> , 2006
<i>B. rapa subsp. brown sarson</i>	BSH-1	Gompertz model	$Y = E^{[\log(3.008)^* \log(1.335 * Time)]} - 1.260 * \text{sowing Day} + 0.166 \text{ factor 1}; (R^2 = 0.482)$	Mehta <i>et al.</i> , 2008
<i>B. carinata</i>	HC-1	—	$Y_1 = -4.8061 + 0.3584 X_1 \dots \dots \dots (R^2 = 0.64) Y_1 = 6.4244 + 0.2654 X_1 - 0.1001 X_3$ $\dots \dots (R^2 = 0.71)$	Sangwan <i>et al.</i> , 2000
	—	Gompertz model	Exp. $[\log(2.717) \times \log(0.479 \times \text{Time}) - 0.202 \times \text{Factors (1)} + 0.168 \times \text{Factors (2)}]$ $\dots R^2 = 0.982$	Sangwan <i>et al.</i> , 2000
<i>B. alba</i>	—	Gompertz model	Exp. $[\log(4.312) \times \log(0.270 \times \text{Time}) - 0.284 \times \text{Factors (1)} + 0.212 \times \text{Factors (2)}]$ $\dots R^2 = 0.970$	Sangwan <i>et al.</i> , 2000
<i>B. oleracea</i>	—	Gompertz model	Exp. $[\log(2.823) \times \log(0.455 \times \text{Time}) - 0.196 \times \text{Factors (1)} + 0.192 \times \text{Factors (2)}]$ $\dots R^2 = 0.984$	Sangwan <i>et al.</i> , 2000

far in advance. (Kumar *et al.*, 2013). Between 67 and 142 DAS, the disease first showed on pods, peaking at 99 DAS. A maximum daily temperature of 18-27°C, a minimum daily temperature of 8-12°C, a daily mean temperature of >10°C, >92 per cent morning relative humidity (RH), >40 per cent after noon RH, and a mean RH of >70 per cent in the preceding week were all positively correlated with the severity of *Alternaria* blight disease on leaves. A maximum temperature of 20-30°C, a daily mean temperature of >14°C, a morning RH of >90%, a daily mean RH of >70%, >9 h of sunshine, and >10 h of leaf wetness all favoured disease severity on pods. (Chattopadhyay *et al.*, 2005).

Such variety-specific models could accurately forecast the crop age when AB first emerged on leaves and pods, the highest blight severity on leaves and pods, and the crop age when blight severity was highest on leaves and pods at least one week before the disease manifested on the crop. This will allow for prompt and effective fungicidal spraying.

In comparison to diseased debris placed in the field and laboratory conditions, Mehta *et al.* (2002a) found that infected debris stored in a deep freezer (10°C) was able to generate 100% infection in the following season when mixed in the soil. The higher survival of *A. brassicae* during the investigation clearly demonstrated that the primary infection in Indian mustard resulted from inoculum that survived in infected plant debris and seeds that were over-summered (April to June). According to the findings, direct sunshine on the soil surface in unploughed fields destroyed the pathogen on contaminated plant waste (Meena *et al.*, 2016b). *B. juncea* (RH-30, RH-8113, RH-8695, RH-8546), *B. campestris* (YSPb-24, BSH-1, Candle, Shiva), two *B. carinata* (HC-2, HC-9001), and one each of *B. napus* (GSH-1) and *B. alba* (local) were observed for the development and progression of *Alternaria* blight by Mehta *et al.* (2008b).

Genetics of host-pathogen interaction

Resistance to *A. brassicae* is said to be governed by a single dominant gene in *Brassica juncea* cultivar RC 781 (Tripathi *et al.*, 1980). Even with *Brassica alba* cultivar Emergo significant resistance to *A. brassicae* has been documented (Brun *et al.*, 1987a, b; Brun *et al.*, 1989). Rajarammohan (2017) reported six QTLs related to *Alternaria* resistance in the mapping population of *Arabidopsis* species which revealed the involvement of multiple loci in resistance confirming the quantitative nature of host responses. Epicuticular wax concentration is greater in intra-specific crosses between *B. napus* and *B. juncea* (Singh *et al.*, 1999). When studying the various mechanisms of horizontal resistance (HR) genotypes

against *A. brassicae*, large differences in the number of lesions, size of lesions, latent duration, sporulation capability, and infection rate were observed in *B. napus* cultivar Tower and *B. juncea* cultivar RC-781 (Saharan and Kadian, 1983). Infection was delayed up to 25 days in *Brassica juncea* cultivar Tower, with just a few small lesions (0.95). The latent durations of cultivars Tower and RC-781 are 18 and 12 days, respectively, as compared to 3 days in cv. Prakash. Prakash had a higher rate of conidial production (265 conidia per lesion) than Tower (92 conidia per lesion) (Saharan, 1991, 1992). Kolte (1987) proposed that the size of lesions and the number of sporulation could be used as primary criteria for determining the level of tolerance or resistance to *Alternaria* blight in Brassica species.

Inheritance of resistance

Krishnia *et al.* (2000) examined the genetics of *Alternaria* blight resistance in inter- and intra-specific crosses of *B. juncea* and *B. carinata*. The population of six generations viz., P₁, P₂, F₁, F₂, BC₁ and BC₂ of six crosses viz., Pusa Basant x EC322092, Kranti x EC322092, Varuna x EC322092, RH 30 x EC322093, RH 30 x HC-1, and Varuna X PCC-2, were evaluated for the inheritance of resistance to *A. brassicae*. The F₂ populations of all six crosses were more susceptible to *A. brassicae* than their F₁ counterparts. Backcross progenies tend to diverge from their recurrent parents' performance. In all of the crosses, the X² values in the joint scaling test were highly significant, showing that the simple additive dominance model was insufficient. The six-parameter model demonstrated significant additive gene effects in all six crosses, but only in the three crosses Varuna x EC322092, RH 30 x EC322092, and Varuna x PCC-2 showed dominant gene effects. In all crosses, however, the bulk of additive gene effects outweighed dominant gene effects. Except for Pusa Basant x EC322092 and Kranti x EC322092, non-allelic interaction and additive x additive I were significant in all crosses, but additive x dominance epistasis was significant only in Pusa Basant x EC322092, Kranti x EC322092, and RH 30 x HC-1 crosses. Significant dominance x dominance (I) type epistasis was seen in the crosses Kranti x EC322092, Varuna x EC322092, and RH 30 x EC322093. Based on these findings, it is recommended that breeding procedures that take advantage of both types of gene effects to be used. As a result, genotypes resistant to *Alternaria* blight can be developed by reciprocal recurrent selection or diallel selective matings (Saharan and Krishnia, 2001).

In each cross, the progenies of R x R cross families have a lower percent disease intensity (PDI) of *Alternaria* blight at the leaf stage than R self and R open families,

demonstrating that inter-mating between resistant plants aids in improving the level of resistance. Multiple disease resistance has been improved with the frequency of favorable genes increasing in the population, increasing the likelihood of gaining multiple disease resistance in other crops as well (Saharan and Krishnia, 2001). When additive or additive x additive forms of genetic variants are combined with repulsion phase linkage between genes, the efficacy of bi-parental approaches can be enhanced (Singh *et al.*, 1986). Singh and Singh (1989) advocated for selecting resistance to *Alternaria* blight in inter-mated populations rather than F₂, F₃, and F₄ populations, because generations of inter-varietal hybrids would avoid the harmful effects of linkages and linkage disequilibrium, and shuffle the desirable genes in one recombinant population (Comstock and Robinson, 1952; Matzinger and Cockerham, 1963; Gates *et al.*, 1957).

The significant 'j' type epistasis was observed in crosses, Pusa Basant x EC322092, Kranti x EC322092 and RH 30 x HC-1 whereas 'I' type epistasis was significant in crosses, Kranti x EC322092, Varuna x EC322092 and RH 30 x EC322093. As a result, rather than F₂, advanced segregating generation should be used to select resistant plants. As a result, crosses with a primarily significant additive gene effect and additive x additive types of epistasis must be used to improve through simple selections. In most of these crosses, progenies of R x S cross families had the greatest GCV, PCV, h², and GG. In vulnerable plant progenies, the value of heritability and genetic gain is lower than in resistant progenies. A high order correlation in *Alternaria* blight resistance at both the leaf and siliquae phases is indicated by a nearly same trend in all crossings for *Alternaria* blight index at the siliquae phase. (Saharan and Krishnia, 2001).

Since polygenes rule resistance to *Alternaria* black spot, breeding for resistance should include pyramiding of minor genes to give additive/polygene resistance. The transfer of *Alternaria* resistance genes into commercial crucifer cultivars is thought to be dependent on the accumulation of horizontal resistance genes in general (Sharma *et al.*, 2002). As a result, it is critical to identify different sources of horizontal resistance across Brassica plants and then combine them to improve protection against *Alternaria*. Strong cross-incompatibility, a polygenic background of resistance (additive and dominant gene interactions), and differences in ploidy (number of chromosomes) between *Brassicaceae* species make it difficult to transfer *Alternaria* resistance from wild species to cultivated forms (Nowicki *et al.*, 2012).

Host Resistance

Many different Brassica species have been found as sources of resistance to *Alternaria* species, but only a few have been used to generate resistant cultivars (Verma and Saharan, 1994; Sharma *et al.*, 2002). The improvement in the accumulation of resistance to numerous diseases would be aided by a strong and positive association in the enhanced degree of resistance to different pathogens documented in *Brassica* species (Kumar and Saharan, 2002). Despite significant obstacles in the development of resistant cultivars, several methods/techniques have been used to include desired features in cruciferous crops against black spot disease using both conventional and biotechnology approaches. (Nowicki *et al.*, 2012; Aneja and Agnihotri, 2013). The exploitation of NHR mechanisms and their applications, as well as the resistance sources present in host germplasm, could be a strategy for safeguarding Brassica crops from *Alternaria* blight. The findings could be applied to a broader range of plant pathogens. (Fatima *et al.*, 2019). *Brassica alba*, *Camelina sativa*, *Eruca sativa*, *Capsella bursa-pastoris*, and *Neslia paniculata* were among the wild crucifers that exhibited resilience (Tewari and Conn, 1993). Sharma *et al.* (2002) evaluated cultivated and wild allies species belonging to nine genera and reported that eight species (*Brassica desnottesii*, *Camelina sativa*, *Coincya pseuderucastrum*, *Diplotaxis berthautii*, *D. catholica*, *D. cretacea*, *D. eruroides*, and *Erucastrum gallicum*) were found completely resistant, whereas others were classified as moderately resistant, susceptible or highly susceptible. Since resistance is unavailable within the cultivated species, so these resistant wild species could be used as donor parents for introgression of resistance to *Alternaria* blight disease in Indian mustard. Kumari *et al.* (2020) introgressed the gene(s) imparting resistance/tolerance against *Alternaria* blight in *B. juncea* from *Sinapis alba* through somatic hybridization followed by further backcrossing. Various factors contribute to crucifer host resistance to *Alternaria* species. Resistance inheritance in inter- and intra-specific crosses of *B. juncea* and *B. carinata* are governed by adaptive genes, dominant genes, additive x additive type epistatic genes, additive x dominance, and dominance x dominance type non-allelic interaction of genes (Singh and Singh 1989; Krishnia *et al.*, 2000b). Inter-mating between resistance plants aids in the development of resistance to *A. brassicae* through the pyramiding of resistance genes in oilseed Brassicas genotypes (Saharan and Kadian, 1983b; Saharan and Krishnia, 2001). Disease tolerance traits have been reported in Brassica genotypes Rajat, Kranti, RH-781, and RL-1359 (Gupta *et al.*, 2002). PR-8988 and PR-9024 genotypes have a higher level of partial resistance or

slow blighting (Kumar and Kolte, 2001). Based on biochemical analysis, real-time PCR, and cluster analysis, the *B. juncea* mutant DRMR-M-172 was shown to be tolerant to *Alternaria* blight, while the DRMR-M-178, DRMR-M-167, and DRMR-M-177 mutants appeared to be tolerant and may be employed in the breeding programs (Meena *et al.*, 2020).

Horizontal resistance

Genetic resistance in plant can be classified two components, i.e., horizontal and vertical. Resistance that effectively provides protection against certain races of a pathogen is called vertical or specific resistance, whereas resistance against all races of a pathogen is called horizontal or general resistance. Out of the two, horizontal resistance is assumed to be polygenic and 'durable' such that the targeted pathogens cannot overcome it. It has been hypothesized, that the cumulation of horizontal genes could possibly favor the introduction of *Alternaria* resistance genes in the commercial varieties of crucifers (Sharma *et al.*, 2002). Hence, it is crucial to identify different sources of horizontal resistance and subsequently combine them to increase durable *Alternaria* resistance among Brassica plants. Kolte (1987), suggested that lesion size and volume of sporulation are also crucial determinants in determining resistance to *A. brassicae*. In genotypes, PR-8988 and PR-9024, *A. brassicae* produced significantly reduced number of spots (4.36-15.89), smaller size of spots (2.12-6.17 mm), lower sporulation ($0.30-1.84 \times 10^3$ conidia), lower disease index (36.51-42.2%), reduced apparent infection rate ($r=0.047-0.080$), and lesser values of AUDPC (45.35-126.70) on leaf and pod, respectively along with reduced leaf defoliation (38.40-44.40%) in comparison to on national susceptible genotype Varuna. As a result, these genotypes exhibited greater partial resistance or slowed blighting. Therefore, the two genotypes could possibly serve as the source for horizontal resistance.

Transfer of horizontal resistance against *Alternaria* can be a bit tricky because of polygenic background of the resistance (additive and dominant gene interactions), strong cross-incompatibility, and differences in ploidy between members of Brassicaceae family. Also application of advanced *in vitro* techniques like, somatic hybridization, embryo and ovary rescue, or protoplast fusion makes the process much more complex.

Structural barriers

In *Brassica* species, epicuticular wax (cvs. Candle, Tobin, Altex, Midas, Tower), a small number of stomatal apertures (cvs. Tower, RC-781), and a low number and narrow stomatal aperture (cvs. Tower, RC-781) confer

resistance to *Alternaria* infection (Saharan and Kadian, 1983b; Conn *et al.*, 1984; Tewari, 1991b). The species *B. napus*, *B. carinata* and *B. alba* are relatively less sensitive to *Alternaria* blight as they have more epicuticular wax than *B. rapa* and *B. juncea* (Conn *et al.*, 1984; Tewari, 1986). Intolerant mustard genotypes had increased levels of phenolic chemicals, polyphenol oxidase activation, and catalase activity (Gupta *et al.*, 1990). Plant breeding has been credited with a variety of high-yielding and stress-tolerant rapeseed-mustard cultivars, for example, high quantities of epicuticular wax have been observed in the progeny of interspecific crosses between *B. napus* and *B. juncea* as a result these progenies are less sensitive to *Alternaria* blight (Singh *et al.*, 1999). However, where no donor germplasm source is available, genetic engineering-mediated interferences are required in rapeseed-mustard crop improvement, notably for features like *Alternaria* blight resistance.

Biochemical barriers

After being exposed to *Alternaria*, phytoalexins were elicited and accumulated in crucifers, and their function in disease resistance was established (Verma and Saharan, 1994). *A. brassicae* has a calcium sequestration property that can be utilized to improve rapeseed resistance to this disease by applying calcium compounds to the soil or to the leaves. (Tewari, 1991a, b). Resistance to *Alternaria* blight in mustard has been found to be associated with the development of secondary metabolites like glucosinolates and phytoalexins (Verma and Saharan, 1994; Atwal *et al.*, 2003; Mathpal *et al.*, 2011; Sharma *et al.*, 2010; Doughty *et al.*, 1996; Jung *et al.*, 2002). Several types of biochemical changes which include the phenolic level were found to be important in the investigation of resistance mechanisms (Arora and Wagle, 1985; Meena *et al.*, 2008). For the proper management of *Alternaria* blight in mustard, Mangain *et al.* (2019) discovered the possible function of chemical elicitors, primarily salicylic acid, in inducing systemic resistance.

Molecular basis of host resistance

On the molecular basis, plant resistance is generally achieved either via interfering with the pathogen-derived effectors or elicitation of defense response. In recent years, great progress has been made related to the molecular basis underlying host-pathogen interactions. Resistance in *Arabidopsis thaliana* has been widely studied and is thought to be mediated by GLIP1 and ethylene signaling. (Oh *et al.*, 2005). Chitinase modifying proteins (cmeps) are common proteases released by the fungal pathogen to inhibit the activity of chitinase produced by the host in response to pathogen attack. In

crucifers, it has been observed that fungalsin (cmp belonging to the fungalsin class of protease) activity is inhibited, therefore plant resistance to numerous fungal diseases can emerge in cruciferous hosts (Naumann and Wicklow, 2013). *B. juncea* transformed with chitinase gene tagged with over expressing promoters 35S CaMV produced defense response by destroying the cell walls of invading fungi in plants (Mondal *et al.*, 2003). The defense mechanisms of *B. juncea* against *Alternaria* pathogenesis are aided by increased levels of PAL, PPO, and peroxidase (Parihar *et al.*, 2012). Treatment with α -aminobutyric acid results in an appropriate equilibrium of oxidants and antioxidants, allowing *B. carinata* to display resistance against *A. brassicae* by preventing pathogen entry during the early phases of colonization (Chavan *et al.*, 2013). Zeatin, a cytokinin, boosts plant immunity by increasing MAPK-4 levels and counteracting the effects of *A. brassicae* (Marmath *et al.*, 2013b). Transgenic expression of hevein, the rubber tree lectin, in *B. juncea* cv. RLM-198 confers defense against *A. brassicae* (Kanrar *et al.*, 2002), α -amino-butyric acid pre- treatment of *B. juncea* plants induces *A. brassicae* resistance mediated through an enhanced

expression of pathogenesis related protein genes, independent of SA and JA accumulation (Kamble and Bhargava, 2007). Brassica homologs of the hypersensitive response gene (hsr 203J) play an essential role in differential defensive response against *A. brassicaceae* (Mishra *et al.*, 2010). The genome of *A. brassicae* has been assembled in a highly contiguous manner and sequenced using Nanopore MinION sequencing with an N50 of 2.98 Mb, resulting in nine complete chromosome-level sequences. A new study has added to the existing genomic resources for the *Alternaria* genus, allowing for more research into the mechanisms driving the pathogenicity of this important necrotrophic pathogen. (Rajarammohan, 2019).

B. napus genome has been effectively integrated with the cDNA expressing AMP1, which is an antimicrobial peptide isolated from *Pinus monticola* with a high cysteine content. (Verma *et al.*, 2012). The in-planta expression gives higher protection to *B. napus* against *A. brassicae*. In transgenic *B. juncea*, a combined expression of barley class II chitinase and Type I ribosome-inactivating protein resulted in resistance

Table 3: Resistance genes/ QTLs identified against *Alternaria brassicae* and *A. brassicicola* causing Alternaria blight.

Gene (s)	Host species	Pathogen	Reference
<i>RtAbeCvG2-1</i> , <i>RtAbeCZ5-1</i>	<i>Arabidopsis thaliana</i>	<i>A. brassicae</i>	Rajarammohan <i>et al.</i> (2017)
Hevein (chitin binding lectin protein)	<i>B. juncea</i> cv. RLM 198	<i>A. brassicae</i>	Kanrar <i>et al.</i> (2002)
Chitinase	<i>B. juncea</i>	<i>A. brassicae</i>	Mondal <i>et al.</i> (2003), Bashir <i>et al.</i> (2015), Munir <i>et al.</i> (2016), Rawat <i>et al.</i> (2017)
Osmotin	<i>B. juncea</i>	<i>A. brassicae</i>	Taj <i>et al.</i> (2004)
Osmotin-ferritin	<i>B. juncea</i> cv. Pusa Jaikisan	<i>A. brassicae</i>	Nirupa <i>et al.</i> (2007)
<i>MSRA1</i>	<i>B. juncea</i> cv. Varuna	<i>A. brassicae</i>	Rustagi <i>et al.</i> (2014)
lectin	<i>B. juncea</i> cv. Varuna	<i>A. brassicae</i>	Kumar <i>et al.</i> (2015)
<i>NPR1</i>	<i>B. juncea</i> cv. Varuna	<i>A. brassicae</i>	Ali <i>et al.</i> (2017)
<i>MPK3</i>	<i>B. juncea</i>	<i>A. brassicae</i>	Tasleem <i>et al.</i> (2017)
<i>BAR</i> and <i>neo</i>	<i>B. napus</i>	<i>A. brassicae</i>	De Block <i>et al.</i> (1989)
Class II chitinase (AAA56786) gene and type I ribosome inactivating protein (RIP; AAA32951)	<i>Hordeum vulgare</i>	<i>A. brassicae</i>	Chikara <i>et al.</i> (2012)
<i>PmAMP1</i>	<i>Pinus monticola</i>	<i>A. brassicae</i>	Verma <i>et al.</i> (2012)
Glucanase	<i>Solanum lycopersicum</i>	<i>A. brassicae</i>	Mondal <i>et al.</i> (2007)
<i>NHL10</i> , <i>HCHIB</i> and <i>XLG2</i>	<i>A. thaliana</i>	<i>A. brassicicola</i>	Pathak <i>et al.</i> (2020)
<i>PR-1</i> , <i>PR-2</i> , <i>PR-3</i> , <i>NPR-1</i> , <i>PDF1.2</i>	<i>B. juncea</i> and <i>Synapsis alba</i>	<i>A. brassicicola</i>	Nayanakantha <i>et al.</i> (2016)
<i>PR-1</i>	<i>S. alba</i>	<i>A. brassicicola</i>	Mazumder <i>et al.</i> (2013)
endochitinase gene 'ech42'	<i>Trichoderma virens</i>	<i>A. brassicae</i> and <i>A. brassicicola</i>	Kamble <i>et al.</i> (2016)

against *A. brassicae* (Chhikara *et al.*, 2012). Exposure to the brassicaceous defensive metabolites camalexin and allyl-isothiocyanate elicited transcriptional responses in *A. brassicicola* (Sellam *et al.*, 2007).

Certain effector proteins have been proven to play a part in studies, but the majority has yet to be defined, and their molecular targets and functions remain unclear. In the proposed gene-for-gene model, the majority of the effectors/avirulence genes have yet to be found. Mondal *et al.* (2003, 2007) identified antifungal genes for *A. brassicae* resistance. *Brassica* genes such as class II chitinase (AAA56786), type I ribosome-inactivating protein (RIP; AAA32951), and tomato glucanase provide better resistance to *Brassica* lines against *A. brassicae* (Table 3).

Disease tolerance

The disease stress tolerance index (DSTI) was discovered to be a useful selection criterion for evaluating the disease stress tolerance and yield potential of mustard genotypes. The genotypes Rajat, Kranti and RH-781 under normal sown, and Rajat, RL-1359 and Kranti under late sown conditions, performed with uniform superiority under both non-disease and disease-stress environments (Saharan *et al.*, 2015). Yield potential in a controlled environment (Y_p) was found to be significantly and positively linked with yield in a disease-stressed environment (Y_s). Under normal sown and non-disease stress conditions (Y_p), potential yield, mean productivity (MP), disease tolerance (TOL), geometric mean productivity (GMP), and disease stress tolerance index (DSTI) all demonstrated a significant positive relationship with yield under disease stress conditions (Y_s) (Gupta *et al.*, 2002).

Disease Management

To control *Alternaria* epidemics of Brassica crops, it is advised to select tolerant/resistant cultivars, use chemicals/bioagents at the appropriate time with adequate foliage coverage, use of clean, bold, healthy, and treated seeds of recommended cultivars, long crop rotation (3-4 years), sanitation, weed control, shallow (2 cm depth) planting at the recommended time, use of balanced nutrients (80 kg/ha N, 40 kg/ha P, 40 kg/ha K), proper plant density (45 x 20 cm), drainage in the field, and plant debris management, and educate farmers about the necessity of these techniques. (Gupta *et al.*, 2018). According to Meena *et al.* (2015), soil application of nutrients such as Potassium (K) + Zinc Sulphate ($ZnSO_4$) + Copper Sulphate ($CuSO_4$) + Sulphur + foliar spray (FS) of Mancozeb + carbendazim fungicides and lower leaf

removal at 40 days after sowing + FS of Ridomil, reduced the maximum *Alternaria* blight severity and resulted in higher seed yields.

Managing seed-borne inoculum using a hot water treatment at 50°C for 20 minutes was found to be highly efficient without impacting seed germination (Randhawa and Aulakh, 1984). To manage *Alternaria* seed-borne disease, a variety of pesticides and bioagents have been suggested (Verma and Saharan, 1994; Vannacci and Harman, 1987; Latif *et al.*, 2006). *In-vitro* and *in-vivo* testing of a wide range of molecules against *Alternaria* species has revealed that they are very successful in managing the disease in the field and boosting yield. The number of sprays, optimum dosages, optimal crop growth stage, spraying time, compatibility with insecticides, residual toxicity, persistent nature, spray intervals, and cost-benefit ratio of the most efficient fungicides have all been determined (Verma and Saharan, 1994; Singh and Singh, 2005; Meena *et al.*, 2004; Khan *et al.*, 2007; Mondal *et al.*, 2007; Saharan, 1991, 1992; Marshall and Haris, 1984; Bonin and Fratzczak, 1987; Brazauskiene and Peteraitiene, 2004; Davies *et al.*, 1986). The indiscriminate use of excessive dosages of fungicides to a crop's pollen biology can have a negative impact on seed output (Williams *et al.*, 1987; Jain *et al.*, 2000). Iprodione, procymidone, and fludioxonil, among other fungicides, have exhibited resistance to isolates of *A. brassicicola*, which may alter their efficacy in managing the disease in the field. (Huang and Levy, 1995; Iacomi-Vasilescu *et al.*, 2004). For the management of *Alternaria* infections of crucifers, several plant extracts and biocontrol agents exhibit efficacy comparable to fungicides, as well as superior yields (Meena *et al.*, 2008, 2013). In a few selected host-pathogen systems, antagonistic bio-control mechanisms have been investigated. Biocontrol agents such as *Trichoderma harzianum*, *Pseudomonas fluorescens*, and *Bacillus subtilis* cause a variety of biochemical alterations in *B. juncea* to trigger plant defense response against pathogens (Verma and Saharan, 1994; White *et al.*, 1990; Tsuneda and Skoropad, 1980; Danielsson *et al.*, 2006; Sharma *et al.*, 2010). The use of resistant cultivars is the simplest, most cost-effective, environmentally beneficial, and safest method of plant disease management. However, efforts are being done around the world to create resistant crucifer cultivars to combat black spot disease (Verma and Saharan, 1994). Incorporation of all plant disease management approaches including cultural, chemical, biological, host resistance; biotechnological/genetic engineering is the best way to deal with black spot disease of crucifers (Verma and Saharan, 1994; Saharan and Mehta, 2002; Kolte, 2005; Mehta, 2014)

Conclusion and Future Perspectives

Intensive crop cultivation has resulted in the reproduction, build-up, and dissemination of *Alternaria* species on crucifers in the locations where these crops are produced with the lack of host resistance. The primary causes of the surge in the inoculum of pathogenic *Alternaria* species are due to the lack of genetic sources of resistance, cultivation of high yielding susceptible cultivars with high plant density, irrigation along with high nitrogenous fertilizer dosages, adjacent areas under monoculture, staggered sowing dates, poor weed management, and poor plant protection strategies. According to the information presented in this review on *Alternaria* diseases of crucifers, some gaps and bottlenecks still exist in our comprehensive understanding of various dimensions of the *Alternaria-Brassica* pathosystem (Figure 4) and should be resolved on a priority basis in areas of crucifer production.

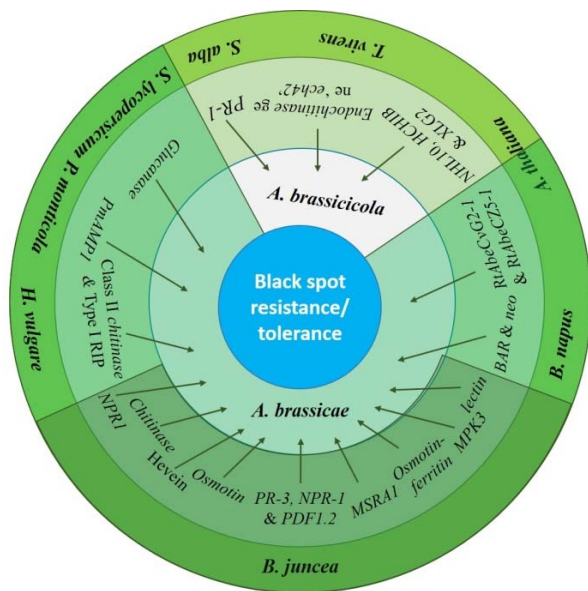


Fig. 4: Scheme showing genes/ QTLs from different sources and their mode of actions conferring black spot resistance/ tolerance for the sustainability in edible oilseed crops

The factors that influence disease onset and progression are not completely understood. A better understanding of numerous epidemiological elements will aid in the development of methods to slow the spread of these dangerous diseases. By computing disease progression at regular intervals, multi-location trials with staggered planting dates could be useful in assessing disease development in connection to the environment. These

efforts could be used to investigate how different “Disease Prediction Models” (geo-phytopathological, bioclimatic, simulation system analysis etc.) can help design an effective disease control plan. Identification and standardization of host differentials is a necessary precondition for collecting useful data on races and pathotypes. This is one of the high-priority areas of investigation. Uniform designation and nomenclature of pathotypes with international acceptance is an urgent need. In *Alternaria* spp. virulent and avirulent gene(s), as well as QTLs/genes governing resistance in the host, may also be identified. A very little information is available on genetic sources of resistance, nature, mechanism, and inheritance of resistance. Although the key genes for resistance to *Alternaria* diseases are unknown, the possibility of minor genes/horizontal resistance, tolerance, and gradual blighting should be explored. To transfer the resistant genes from distantly related natural sources, the feasibility of induced resistance as well as genetic engineering procedures can be used. Identification and sequencing of resistant genes along with identification of suitable gene combinations may be explored and taken up. Hybrids and GM cultivars with multiple disease resistance have to be explored for the sustainability of edible oil in India (Figure 4). Characterization of secondary metabolite biosynthetic genes, as well as signal transduction and its role in pathogenicity and fungal development, as well as disruption of Aso 1, a gene required for anastomosis and required for *Alternaria* to become pathogenic, are some of the important and useful areas that must be investigated further. Because there is a lot of genetic heterogeneity in related and distantly related crucifers all over the world, exploitation of morphological, structural, and biochemical foundations of resistance requires in-depth research.

Comparative research on all aspects of host-parasite interaction in crucifers with all four *Alternaria* species should yield some useful insights for disease management. Some relevant information on chemical control of several *Alternaria* diseases is available; however, much more is needed to build a feasible disease control strategy in the field. Efforts should also be made to find low-cost, high-effective compounds that can provide cost-effective disease control. It is necessary to identify the active ingredient in plant extracts. Insecticides should be included in integrated pest and disease management systems because the role of insects such as aphids (viral transmission) and flea beetles (transmission of *A. brassicicola* to cabbage) are duly important. Microbial antagonists should be investigated for the treatment of *Alternaria* infections in rapeseed-mustard. Seed

treatments using *Trichoderma*, *Gliocladium*, *Penicillium*, and *Streptomyces* species should be explored further in the field to evaluate their efficacy and cost-effectiveness. Similarly, management of this pathogen by foliar applications of *Streptomyces rochei*, *S. higroscopicus*, *S. arabicus*, and *Nectria inventa* should also be explored under varied field conditions.

More R-gene cloning work in *Brassica* will help us to better understand the plant defense system, and provide the information to exploit novel R-genes for breeding objectives in a much effective and quick manner. Brassica genome sequencing will lead to fresh discoveries and understandings about the genetic links between and within Brassicaceae members. Although hybridization between divergent groups in the Brassicaceae family, which contains a large number of R-genes, is already possible, particularly in India, this is an area with a lot of future potentials, as these methodologies become more widely adopted and rates of success with wide-hybridization events are quite imperative. However, the resistance resources in *Brassica* species are limited and the species

in the crop wild relatives of *Brassica*, such as *B. incana*, *Sinapis alba*, *Diplotaxis erucooides*, *D. catholica*, *B. cretica* (C genome), and *B. fruticulosa* (B genome), as well as its close relatives could be used to facilitate resistance gene exchanges in the breeding programs. The development of introgression lines from the wild and related species to the cultivated species is one of the strategies to transfer the resistance gene(s) for the Alternaria blight which can be speeded up by using the molecular biology tools and techniques for developing the resistant cultivars. Within the *Brassica* genus, embryo rescue, reciprocal crossing, and marker-assisted selection (MAS) is commonly used for interspecies crossing. To transfer and use the resistance contained in the A and B genomes, interspecies hybridization must be explored. The new resistant germplasm created in such investigations will be useful in future *Brassica* crop breeding improvement projects. MAS allows us to stack R alleles for multiple diseases and create multi-resistant cultivars utilizing a large number of genetic markers. The breeding circle has been substantially shortened along with the use of MAS in conjunction with other techniques such as hybridization and microspore

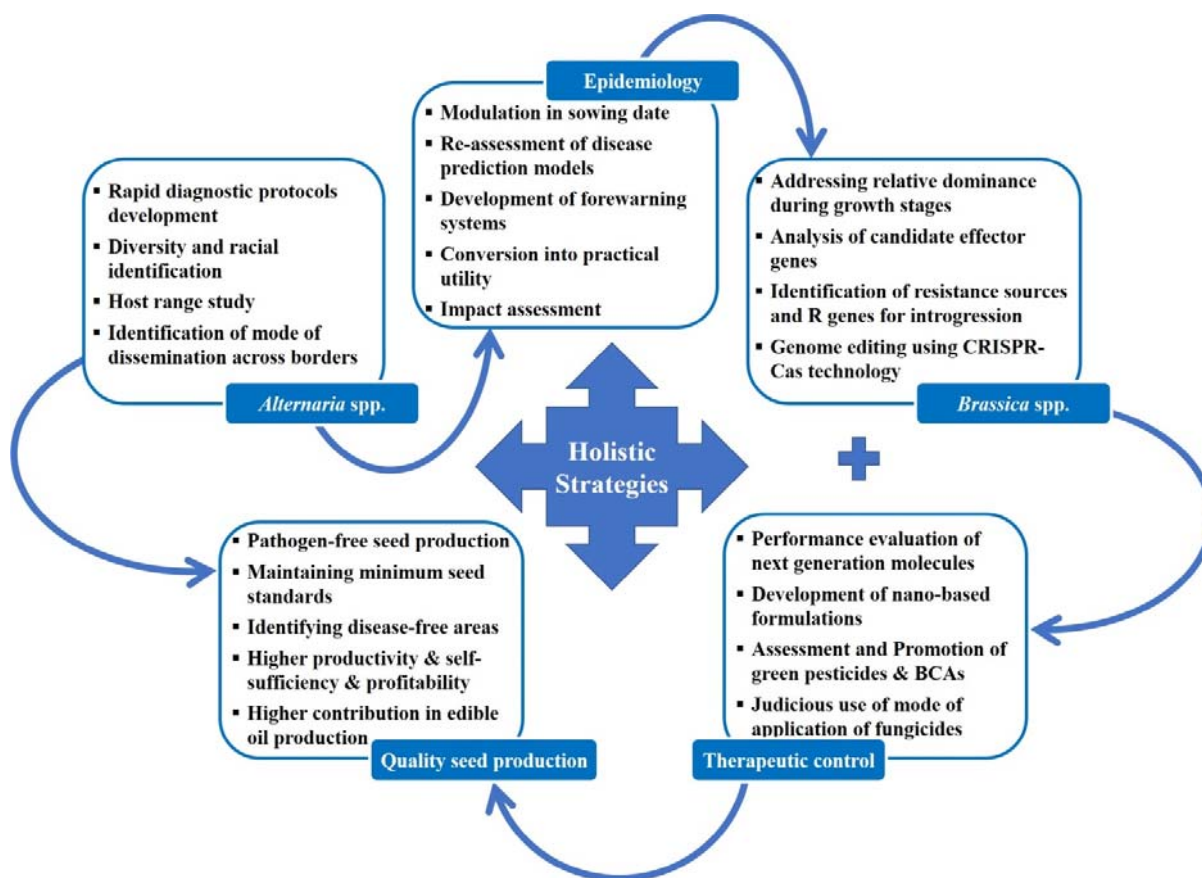


Fig. 5: A holistic approach to the management of Brassica against Alternaria blight disease

culture. For the development of resistant cultivars, the collaboration between plant breeders, molecular biologists and plant pathologists is highly essential.

In dealing with biological systems, not one strategy or approach could be both effective and cost-efficient. To effectively manage the disease, all strategies of plant disease management, such as diagnostics, forewarning, chemical/biological/cultural control methods, host resistance, and genetic engineering techniques must be combined in a unified manner to bring holistic management of this disease for the farming community (Figure 5).

Moreover, the latest molecular and omics methods, such as transcriptomics, proteomics, and metabolomics, open up new avenues for mining genes in the resistance-regulating network, which could be used either directly in resistance breeding or indirectly in pre-breeding studies to better understand Brassica-pathogen interactions. In addition, the sequenced *Brassica* accessions do not contain all R-genes due to variations between individuals, whereas, the establishment of the pan-genomics could facilitate gene mining from a wider platform. The identified genetic resources of *Brassica* and its wild relatives can be exploited to the fullest potential to develop a mapping population to map genomic regions conferring the resistance to *Alternaria* blight in crucifers.

The reported genes/QTLs can be validated in the identified accessions/cultivars. Further, the fine mapping would help in the cloning of genes that would help in understanding the molecular mechanism of resistance. The identified genes/QTLs can be introgressed in the susceptible but agronomically superior varieties through marker-assisted selection. There is no doubt about the pivotal role of host resistance in disease management, however, the sustainability in Brassica cultivation with higher productivity profitability and production of quality seed could be achieved through combining all the disease management strategies in a holistic way.

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References

Akhtar J, Singh B, Kandan A, Kumar P, Maurya AK, Dubey SC. 2017a. Interception of pathogens during quarantine processing: an effort towards safe import of oilseed and vegetable *Brassicaceae* germplasm in India. *J Oilseed Brassica* **8**:126-130

Akhtar J, Singh B, Kandan A, Kumar P, Maurya AK, Chand D, Gupta V, Dubey SC. 2017b. Survival of *A.*

brassicicola in cryo-preserved *Brassica* spp. seeds. *Indian Phytopathol* **70**:256-257

- Ali S, Mir ZA, Tyagi A, Mehari H, Meena RP, Bhat JA, Yadav P, Papalou P, Rawat S, Grover A. 2017. Overexpression of *NPR1* in *B. juncea* confers broad spectrum resistance to fungal pathogens. *Front Plant Sci* **8**:1693.
- Al-Lami HFD, You MP, Barbetti MJ. 2019. Incidence, pathogenicity and diversity of *Alternaria* spp. associated with *Alternaria* leaf spot of canola (*B. napus*) in Australia. *Plant Pathol* **68**:492-503.
- Aneja JK, Agnihotri A. 2013. *Alternaria* blight of oilseed brassicas: epidemiology and disease control strategies with special reference to use of biotechnological approaches for attaining host resistance. *J Oilseed Brassica* **4**:1-10.
- Ansari NA, Khan MW, Muheet A. 1988. Effect of temperature and relative humidity on spore germination of *A. brassicae* and development of *Alternaria* blight on oilseed crucifers. *Rev Trop Plant Pathol* **5**:79-84.
- Ansari NA, Khan MW, Muheet A. 1989. Effect of some factors on growth and sporulation of *A. brassicae* causing *Alternaria* blight of rapeseed and mustard. *Acta Bot Indica* **17**:49-53.
- Atkinson RG. 1950. Studies on the parasitism and variation of *A. raphani*. *Can J Res Sec C* **28**:288-317.
- Atkinson RG. 1953. Survival and pathogenicity of *A. raphani* after five years in dried soil cultures. *Can J Bot* **31**:542-547.
- Atwal AK, Ramandeep, Munshi SK, Mann APS. 2003. Biochemical changes in relation to *Alternaria* leaf blight in Indian mustard. *Plant Dis Res* **19**:57-59.
- Bains PS, Tewari JP. 1989. Bioassay and mode of action of an *A. brassicae* toxin. *Can J Plant Pathol* **11**:184-189.
- Bashir A, Ambreen, Khan MS, Haider A, Khan I. 2015. *Agrobacterium* mediated transformation of *B. juncea* (L.) Czern. & Coss. with chitinase gene conferring resistance against fungal infections. *Pak J Bot* **47**:211-216.
- Blagojevic JD, Vukojevic JB, Ivanovic ZS (2020) Occurrence and characterization of *Alternaria* species associated with leaf spot disease in rapeseed in Serbia. *Plant Pathol* **69**:883-900.
- Bonin K, Fratzcak E. 1987. Control of fungal diseases in winter oil seed rape. Proc. 7th Intl Rapeseed Congress, Poznan, Poland, pp 1281-1287.
- Brazauskienė I, Petraitiene E. 2004. Effects of fungicide

- application timing on the incidence and severity of Alternaria blight (*A. brassicae*) and on the productivity of spring oilseed rape (*B. napus* L. ssp. *oleifera annua* Metzg.). *Agron Res* **2**:121-133.
- Brazauskiene I, Petraitiene E. 2006. The occurrence of Alternaria blight (*Alternaria* spp.) and Phoma stem canker (*Phoma lingam*) on oilseed rape in central Lithuania and pathogenic fungi on harvested seed. *J Plant Prot Res* **46**:295-311
- Brun H, Plessis J, Renard M. 1987a. Resistance of some crucifers to *A. brassicae* (Berk.) Sacc., pp 1222-1227. Proc. 7th Intl Rapeseed Congress, Poznan, Poland.
- Brun H, Renard M, Plessis J. 1987b. Resistance of some crucifers to *A. brassicae* (Berk.) Sacc. Proc. 7th Intl Rapeseed Congress, Poznan, Poland, pp 247
- Brun H, Renard M, Tribodet M, Plessis I, Tanguy X. 1989. Contribution to the genetic control of rapeseed diseases. *Phytoma* **404**:36-37; 40-41
- Buchwaldt L, Green H. 1992. Phytotoxicity of destruxin B and its possible role in the pathogenesis of *A. brassicae*. *Plant Pathol* **41**:55-63.
- CAB International (2007) Crop Protection Compendium, 2007th Edn. Centre for Agriculture and Bioscience International. Wallingford, Oxon, UK
- Changri W, Weber GF. 1960. Studies of *Alternaria* spp. pathogenic on Cruciferae. *Phytopathol* **50**:631.
- Changri W, Weber GF. 1963. Three *Alternaria* species pathogenic on certain cultivated crucifers. *Phytopathol* **53**:643-648.
- Chattopadhyay C, Agrawal R, Kumar A, Bhar LM, Meena PD, Meena RL, Khan SA, Chattopadhyay AK, Awasthi RP, Singh SN, Chakravarthy NVK, Kumar A, Singh RB, Bhunia CK. 2005. Epidemiology and forecasting of Alternaria blight of oilseed Brassica in India - a case study. *J Plant Dis Prot* **112**:351-365.
- Chavan V, Bhargava S, Kamble A. 2013. Temporal modulation of oxidant and antioxidative responses in *B. carinata* during α -aminobutyric acid-induced resistance against *A. brassicae*. *Physiol Mol Plant Pathol* **83**:35-39.
- Chen LY, Price TV, Silvapulle MJ. 2005. Dark leaf spot (*A. brassicicola*) on Chinese cabbage: spatial patterns. *Aust J Agric Res* **56**:699-714.
- Chhikara S, Chaudhury D, Dhankher OP, Jaiwal PK. 2012. Combined expression of a barley class II chitinase and type I ribosome inactivating protein in transgenic *B. juncea* provides protection against *A. brassicae*. *Plant Cell Tissue Organ Cult* **108**:83-89.
- Cho Y, Cramer R, Kwang-Hyung K, Davis J, Mitchell T, Figuli P, Pryor B, Lemasters E, Lawrence CB. 2007. The *Fus3/Kss1* MAP kinase homolog *Amk1* regulates the expression of hydrolytic enzyme genes in the fungus *A. brassicicola*. *Fungal Genet Biol* **44**:543-553
- Cho Y, Davis JW, Kim KH, Wang J, Sun QH, Cramer RAJ, Lawrence CB. 2006. A high throughput targeted gene disruption method for *A. brassicicola* functional genomics using linear minimal element (LME) constructs. *Mol Plant Microbe Interact* **19**:7-15.
- Cho Y, Kim KH, La Rota M, Scott D, Santopietro G, Callihan M, Mitchell TK. 2009. Identification of virulence factors by high throughput targeted gene deletion of regulatory genes in *A. brassicicola*. *Mol Microbiol* **72**:1316-1333.
- Cho Y, Srivastava A, Ohm RA, Lawrence CB, Wang KH, Grigoriev IV, Marahatta SP. 2012. Transcription Factor Amr1 induces melanin biosynthesis and suppresses virulence in *A. brassicicola*. *PLoS Pathogens* **8**: e1002974.
- Chupp C, Sherf AF. 1960. Crucifer diseases. In: Vegetable diseases and their control. Ronald Press Co, New York, pp 237-288
- Conn KL, Tewari JP, Awasthi RP. 1990. A disease assessment key for Alternaria black spot in rapeseed and mustard. *Can Plant Dis Surv* **70**:19-22.
- Conn KL, Tewari JP, Hadziyev D. 1984. The role of epicuticular wax in canola in resistance to *Alternaria brassicae*. *Phytopathol* **74**:851.
- Cramer RA, La Rota CM, Cho Y, Thon M, Craven KD, Knudson DL, Mitchell TK, Lawrence CB. 2006. Bioinformatic analysis of expressed sequence tags derived from a compatible *A. brassicicola*-*B. oleracea* interaction. *Mol Plant Pathol* **7**:113-124.
- Dang JK, Sagwan MS, Mehta N, Sharma OP, Dhandapani A. 2006. Development of prediction model for Alternaria blight caused by *A. brassicae* of rapeseed and mustard. *Plant Dis Res* **21**:199-201.
- Danielsson J, Reva O, Meijer J. 2006. Protection of oilseed rape (*B. napus*) towards fungal pathogens by strains of plant-associated *Bacillus amyloliquifaciens*. *Microb Ecol* **54**:134-140.
- Davies JML, Ann DM, Dobson SC, Jones OW, Gladders P, Melville SC, Mc Pherson GM, Popham MD, Price RJ, Wafford JD. 1986. Fungicide spray timing on brussels sprouts. *Asp Appl Biol* **12**:21-28.
- De Block M, De Block D, Tenning P. 1989. Transformation of *B. napus* and *B. oleracea* using *Agrobacterium tumefaciens* and the expression of the bar and neo genes in the transgenic plants. *Plant Physiol* **91**:694-701.

- Dixon GR. 1981. Pathogens of Crucifer Crops. In: Vegetable crop diseases. AVI Publ Co Inc, Westport, Connecticut, pp 119-123
- Domsch KH. 1957. The blackening of rape and cabbage siliquae. *Z Pflanzenkr Pflanzen schutz* **64**:65-79.
- Doughty KJ, Blight MM, Bock CH, Fieldsend JK, Pickett JA. 1996. Release of alkenyl isothiocyanates and other volatiles from *B. rapa* seedlings during infection by *A. brassicae*. *Phytochem* **43**:371-374
- Dueck J, Degenhardt K. 1975. Effect of leaf age and inoculum concentration on reaction of oilseed *Brassica* spp. to *Alternaria brassicae*. *Proc Amer Phytopathol Soc* **2**:59.
- Elliott JA. 1917. Taxonomic characters of the genera *Alternaria* and *Macrosporium*. *Am J Bot* **4**:439-476.
- Ellis MB. 1971. Dematiaceous hyphomycetes. Commonwealth Mycological Institute, Kew, Surrey, England, pp 464-497
- Fatima U, Bhorali P, Borah S, Kumar MS. 2019. Perspectives on the utilization of resistance mechanisms from host and non-host plants for durable protection of *Brassica* crops against *Alternaria* blight. *Peer J* **7**:e7486
- Fontem DA, Berger RD, Weingartner DP, Bartz JA. 1991. Progress and spread of dark leaf spot in cabbage. *Plant Dis* **75**:269-274.
- Giri P, Taj G, Kumar A. 2013. Comparison of artificial inoculation methods for studying pathogenesis of *A. brassicae* (Berk.) Sacc. on *B. juncea* (L.) Czern. (Indian mustard). *Afr J Biotechnol* **12**:2422-2426.
- Gorshkov AK. 1976. Infectious damping-off of cabbage seedlings under glass. *Trudy Vses s-kh Inst Zaoch obrazovaniya* **117**:25-29.
- Goyal P, Chattopadhyay C, Mathur AP, Kumar A, Meena PD, Datta S, Iquebal MA. 2013. Pathogenic and molecular variability among *Brassica* isolates of *A. brassicae* from India. *Ann Plant Protect Sci* **21**:349-359.
- Gupta AK, Rishiraj Kumari K, Singh SP, Solanki IS. 2018. Management of major diseases in Indian mustard through balanced fertilization, cultural practices and fungicides in calcareous soils. *Proc Natl Acad Sci India Sect B Biol Sci* **88**:229-239
- Gupta K, Saharan GS, Singh D. 2002. Effective selection criteria to assess disease tolerance in Indian mustard [*Brassica juncea* (L) Czern and Coss.]. *J Mycol Plant Pathol* **32**:72-80.
- Gupta K, Saharan GS, Mehta N, Sangwan MS. 2004. Identification of pathotypes of *A. brassicae* from Indian mustard [*B. juncea* (L) Czern & Coss.]. *J Mycol Plant Pathol* **34**:15-19.
- Gupta SK, Gupta PP, Yadav TP, Kaushik CD. 1990. Metabolic changes in mustard due to *Alternaria* leaf blight. *Indian Phytopathol* **43**:64-69
- Gupta V, Chawla HKL, Dhawan K, Mehta N. 1998. Alterations in total phenols and glucosinolates in *B. juncea* leaves during interaction with *A. brassicae*. *Cruciferae Newsletter* **20**:83-85.
- Hong SG, Cramer RA, Lawrence CB, Pryor BM. 2005. Alt a1 allergen homologs from *Alternaria* and related taxa: analysis of phylogenetic content and secondary structure. *Fungal Genet Biol* **42**:119-129.
- Horsfall JG, Barratt RW. 1945. An improved grading system for measuring plant disease. *Phytopathol* **35**:655.
- Hossain MS, Mian IH. 2005. Effect of planting date on *Alternaria* blight and seed yield of cabbage. *Bangladesh J Plant Pathol* **21**:33-37.
- Huang R, Levy Y. 1995. Characterization of iprodione-resistant isolates of *A. brassicicola*. *Plant Dis* **79**:828-33.
- Iacomi-Vasilescu B, Avenot H, Bataille-Simoneau N, Laurent E, Guenard M, Simoneau P. 2004. In vitro fungicide sensitivity of *Alternaria* species pathogenic to crucifers and identification of *A. brassicicola* field isolates highly resistant to both dicarboximides and phenylpyrroles. *Crop Prot* **23**:481-488.
- Inderbitzin P, Shoemaker RA, O'Neill NR, Turgeon BG, Berbee ML. 2006. Systematics and mating systems of two fungal pathogens of opium poppy: the heterothallic *Crivellia papaveracea* with a *Brachycladium penicillatum* asexual state and a homothallic species with a *Brachycladium papaveris* asexual state. *Can J Bot* **84**:1304-1326.
- Jain AK, Briegleb BP, Minschwaner K, Wuebbles DJ. 2000. Radiative forcings and global warming potentials of thirty-nine greenhouse gases. *J Geophys Res* **105**:773-790.
- James WC. 1974. Assessment of plant diseases and losses. *Annu Rev Phytopathol* **12**:27-48.
- Jasalavich CA, Morales VM, Pelcher LE, Seguin-Swartz G. 1995. Comparison of nuclear ribosomal DNA sequences from *Alternaria* species pathogenic to crucifers. *Mycol Res* **99**:604-614.
- Jha P, Kumar M, Meena PD, Lal HC. 2013. Dynamics and management of *Alternaria* blight disease of Indian mustard (*B. juncea*) in relation to weather parameters. *J Oilseed Brassica* **4**:66-74.
- Jung DS, Na YJ, Ryu KH. 2002. Phylogenetic analysis of *A. brassicicola* producing bioactive metabolites. *J Microbiol* **40**:289-294.

- Kadian AK, Saharan GS (1983) Symptomatology, host range and assessment of yield losses due to *A. brassicae* infection in rapeseed and mustard. *Indian J Mycol Plant Pathol* **13**:319-323.
- Kadian AK, Saharan GS. 1984. Studies on spore germination and infection of *A. brassicae* of rapeseed and mustard. *J Oilseeds Res* **1**:183-188.
- Kamble A, Bhargava S. 2007. α -Aminobutyric Acid-induced Resistance in Brassica juncea against the Necrotrophic Pathogen *A. brassicae*. *J Phytopathol* **155**:152-158.
- Kamble S, Mukherjee PK, Eapen S. 2016. Expression of an endochitinase gene from *Trichoderma virens* confers enhanced tolerance to Alternaria blight in transgenic *B. juncea* (L) Czern & Coss lines. *Physiol Mol Biol Plants* **22**:69-76.
- Kandan A, Akhtar J, Singh B, Dev U, Goley R, Chand D, Roy A, Rajkumar S, Agarwal PC. 2014. Genetic diversity analysis of exotic and indigenous *A. alternata* isolates infecting different crops using URP and ISSR markers. *Indian J Plant Prot* **42**:229-236.
- Kanrar S, Venkateswari JC, Kirti PB, Chopra VL. 2002. Transgenic expression of hevein, the rubber tree lectin, in Indian mustard confers protection against *A. brassicae*. *Plant Sci* **162**:441-448.
- Khan MM, Khan RU, Mohiddin FA. 2007. *In vitro* studies on the variation of different temperature and culture media on the growth of *A. brassicae* (Berk) Sacc infecting rapeseed-mustard. *Ann Plant Protect Sci* **15**:526-527.
- Kim KH, Cho Y, La Rota M, Cramer R, Lawrence C. 2007. Functional analysis of the *A. brassicicola* non-ribosomal peptide synthetase gene AbNPS2 reveals a role in conidial cell wall construction. *Mol Plant Pathol* **8**:23-39.
- Kolte SJ. 1985. Diseases of annual edible oilseed crops, Vol II. CRC Press, Inc Boca Raton, Florida, USA.
- Kolte SJ. 1987. Important diseases of rapeseed and mustard in India: Present research progress and future research needs. Proc IDRC, Canada, 3rd Oil Crops Network Workshop, Addis Ababa, Ethiopia, pp 91-106.
- Kolte SJ. 2005. Tackling fungal diseases of oilseed Brassicas in India. *Brassica* **7**:7-13.
- Krishnia SK, Saharan GS, Singh D. 2000a. Genetics of Alternaria blight resistance in inter and intraspecific crosses of *B. juncea* and *B. carinata*. *Ann Biol* **16**:212-216.
- Krishnia SK, Saharan GS, Singh D. 2000b. Genetic variation for multiple disease resistance in the families of interspecific cross of *B. juncea* x *B. carinata*. *Cruciferae Newsletter* **22**:51-53.
- Kubota M, Abiko K, Yanagisawa Y, Nishi K. 2006. Frequency of *A. brassicicola* in commercial cabbage seeds in Japan. *J Gen Plant Pathol* **72**:197-204.
- Kumar A. 1997. Assessment and economics of avoidable yield losses due to Alternaria blight in oilseed Brassicas. *Plant Dis Res* **12**:152-156.
- Kumar A, Agrawal R, Chattopadhyay C. 2013. Weather based forecast models for diseases in mustard crop. *Mausam* **64**:663-670.
- Kumar B, Kolte SJ. 2001. Progression of Alternaria blight of mustard in relation to components of resistance. *Indian Phytopathol* **54**:329-331.
- Kumar D, Shekhar S, Bisht S, Kumar V, Varma A. 2015. Ectopic overexpression of lectin in transgenic *B. juncea* plants exhibit resistance to fungal phytopathogen and showed alleviation to salt and drought stress. *J Bioeng Biomed Sci* **5**:147.
- Kumar S, Saharan GS. 2002. Sources of multiple disease resistance in Brassica spp. *J Mycol Plant Pathol* **32**:184-188.
- Kumari P, Singh KP, Bisht D. 2020. Somatic hybrids of *Sinapis alba* + *B. juncea*: study of backcross progenies for morphological variations, chromosome constitution and reaction to *A. brassicae*. *Euphytica* **216**:93.
- Kumar S, Sangwan MS, Mehta N, Kumar R. 2003. Pathogenic diversity in isolates of *A. brassicae* infecting rapeseed and mustard. *J Mycol Plant Pathol* **33**:59-64.
- Latif MA, Abu Kaoser MS, Khan MAI, Rahman H, Hossain MA. 2006. Efficacy of some plant extracts in controlling seed-borne fungal infections of mustard. *Bangladesh J Microbiol* **23**:168-170.
- Lawrence DP, Gannibal PB, Peever TL, Pryor BM. 2013. The sections of *Alternaria*: Formalizing species-groups concepts. *Mycologia* **105**:530-546
- Lawrence DP, Park MS, Pryor BM. 2012. *Nimbya* and *Embellisia* revisited, with nov comb for *A. celosiae* and *A. perpunctulata*. *Mycol Prog* **11**:799-815.
- Lou J, Fu L, Peng Y, Zhou L. 2013. Metabolites from *Alternaria* fungi and their bioactivities. *Molecules* **18**:5891-5935.
- Magarey RD, Sutton TB, Thayer CL. 2005. A simple generic infection model for foliar fungal plant pathogens. *Phytopathol* **95**:92-100.

- Mahapatra S, Das S. 2014. Effect of meteorological factors on progression of *Alternaria* leaf blight of mustard and comparison of logistic and gompertz growth models in predicting disease severity. *Indian Phytopathol* **67**:155-158.
- Mamgain A, Biswas MK, Dey N. 2019. Potential role of chemical elicitors in induced systemic resistance for the effective management of *Alternaria* blight in mustard. *J Pharmacogn Phytochem* **8**:2246-2250.
- Mamgain A, Roychowdhury R, Jagatpati T. 2013. *Alternaria* pathogenicity and its strategic controls. *Res J Biol* **1**:1-9.
- Marmath KK, Giri P, Sharma S, Taj G, Kumar A. 2013a. *In-silico* Interaction Studies of *A. brassicae* Toxin Destruxin B and Potential Partners of MAPK4 Cascade. *Int J Agric Environ Biotechnol* **6**:203-210.
- Marmath KK, Giri P, Taj G, Pandey D, Kumar A. 2013b. Effect of zeatin on the infection process and expression of MAPK-4 during pathogenesis of *A. brassicae* in non-host and host *Brassica* plants. *Afr J Biotechnol* **12**:2164-2174.
- Marshall J, Harris RI. 1984. Broad spectrum disease control in oilseed rape with prochloraz. Proceedings of the British Crop Protection Conference, Pest and Diseases, pp 729-734
- Mathpal P, Punetha H, Tewari AK, Agrawal S. 2011. Biochemical defense mechanism in rapeseed-mustard genotypes against *Alternaria* blight disease. *J Oilseed Brassica* **2**:87-94.
- Mayee CD, Datar VV. 1986. Host range and disease assessment scales. In: *Phytopathometry*. Marathwada Agricultural University, Prabhani, India, pp 110-111
- Mazumder M, Das S, Saha U, Chatterjee M, Bannerjee K, Basu D. 2013. Salicylic acid-mediated establishment of the compatibility between *A. brassicicola* and *B. juncea* is mitigated by abscisic acid in *Sinapis alba*. *Plant Physiol Biochem* **70**:43-51.
- Meah MB, Hau B, Siddiqua MK. 2002. Relationships between disease parameters of *Alternaria* blight (*A. brassicae*) and yield of mustard. *J Plant Dis Prot* **109**:243-251.
- Meena PD, Rani A, Meena MC, Sharma P, Kandpal B, Sing D. 2015. Role of nutrients and lower leaf removal against *Alternaria* blight in Indian mustard (*B. juncea* L). *Plant Pathol J* **14**:92-96.
- Meena PD, Rani A, Meena R, Sharma P, Gupta R, Chowdappa P. 2012. Aggressiveness, diversity and distribution *A. brassicae* isolates infecting oilseed Brassica in India. *Afr J Microbiol Res* **6**:5249-5258.
- Meena PD, Awasthi RP, Chattopadhyay C, Kolte SJ, Kumar A. 2010. *Alternaria* blight: a chronic disease in rapeseed-mustard. *J Oilseed Brassica* **1**:1-11.
- Meena PD, Chattopadhyay C, Meena RL. 2008. Eco-friendly management of *Alternaria* blight in *Brassica juncea*. *Indian Phytopathol* **61**:65-69
- Meena PD, Chattopadhyay C, Meena SS, Kumar A. 2011. Area under disease progress curve and apparent infection rate of *Alternaria* blight disease of Indian mustard (*B. juncea*) at different plant age. *Arch Phytopathol Plant Prot* **44**:684-693.
- Meena PD, Gour RB, Gupta JC, Singh HK, Awasthi RP, Netam RS, Godika S, Sandhu PS, Prasad R, Rathi AS, Rai D, Thomas L, Patel GA, Chattopadhyay C. 2013. Non-chemical agents provide tenable, eco-friendly alternatives for the management of the major diseases devastating Indian mustard (*B. juncea*) in India. *Crop Prot* **53**:169-174.
- Meena PD, Jambhulkar SJ, Gupta R, Meena HS, Singh D. 2016a. Rapid screening technique for *Alternaria* blight resistance in Indian mustard (*B. juncea* L) using cotyledonary leaf method. *J Plant Pathol* **98**:463-469.
- Meena PD, Bala M, Sharma P, Singh D. 2014. Interaction and tolerance to *A. brassicae* in Indian mustard (*B. juncea*) genotypes. *J Oilseeds Res* **31**:136-139.
- Meena PD, Meena RL, Chattopadhyay C, Kumar A. 2004. Identification of critical stage for disease development and biocontrol of *Alternaria* blight of Indian mustard (*B. juncea*). *J Phytopathol* **152**:204-209.
- Meena PD, Gupta R, Rani A, Sharma P, Singh D. 2016b. Effect of summer temperatures on survival of *A. brassicae* in infected Indian mustard debris and thermal death point variations amongst geographical isolates. *J Oilseed Brassica* **7**:45-51.
- Meena PD, Gupta R, Meena HS, Sharma P, Jambhulkar S. 2017. Pathogenic variability within Indian *A. brassicae* isolates using seed, cotyledon and leaf of *Brassica* species. *J Phytopathol* **165**:238-248.
- Meena PD, Kumar SMS, Meena HS, Jambhulkar S, Taj G, Pathak D, Srivastava S, Gupta R, Singh D, Gurung B, Rai PK. 2020. Confirmation of induced tolerance to *Alternaria* blight disease in Indian mustard (*B. juncea* L). *Appl Biochem Biotechnol* **192**:965-978.
- Mehta N, Sangwan MS, Srivastava MP. 2002a. Survival of *A. brassicae* causing *Alternaria* blight in rapeseed-mustard. *J Mycol Plant Pathol* **32**:64-67.

- Mehta N, Sangwan MS, Kumar R, Niwas R. 2008a. Progression of *Alternaria* blight on different varieties of rapeseed-mustard in relation to weather parameters. *Plant Dis Res* **23**:28-33.
- Mehta N, Sangwan MS, Saharan GS. 2005. Fungal diseases of rapeseed mustard. In *Diseases of oilseed crops* ed. Sangwan MS, Saharan GS, Mehta N. Indus Publishing Company, New Delhi, pp 15-86
- Mehta N, Sangwan MS, Srivastava MP, Niwas R. 2002b. Relationship between weather variables and *Alternaria* blight development in rapeseed and mustard. *J Mycol Plant Pathol* **32**:368-369.
- Mehta N. 2014. Epidemiology and forecasting for the management of rapeseed-mustard diseases. *J Mycol Plant Pathol* **44**:131-147.
- Mehta N, Sangwan MS, Srivastava MP. 2003. Morphological and pathological variations in rapeseed and mustard isolates of *A. brassicae*. *Indian Phytopathol* **56**:188-190.
- Mishra A, Pandey D, Goel A, Kumar A. 2010. Molecular cloning and *in-silico* analysis of functional homologues of hypersensitive response gene(s) induced during pathogenesis of *Alternaria* blight in two genotypes of *Brassica*. *J Proteom Bioinform* **3**:244-248.
- Mondal KK, Bhattacharya RC, Koundal KR, Chatterjee SC. 2007. Transgenic Indian mustard (*Brassica juncea*) expressing tomato glucanase leads to arrested growth of *A. brassicae*. *Plant Cell Rep* **26**:247-252.
- Mondal KK, Chatterjee SC, Viswakarma N, Bhattacharya RC, Grover A. 2003. Chitinase-mediated inhibitory activity of *Brassica* transgenic on growth of *A. brassicae*. *Curr Microbiol* **47**:171-173.
- Mridha MAU. 1983. Virulence of different isolates of *A. brassicae* on winter oilseed rape cultivars. 6th International Rapeseed Conference, Paris, France, pp 1025-1029.
- Munir I, Hussan W, Kazi M, Farhatullah A, Iqbal A, Munir R. 2016. Production of transgenic *B. juncea* with the synthetic chitinase gene (NIC) Conferring resistance to *A. brassicicola*. *Pak J Bot* **48**:2063-2070.
- Naumann TA, Wicklow DT. 2013. Chitinase modifying proteins from phylogenetically distinct lineages of *Brassica* pathogens. *Physiol Mol Plant Pathol* **82**:1-9.
- Nees von ECG. 1816. Das System der Pilze und Schwamme Stahelschen Buchhandlung. Wiirzburg, Germany, pp 329.
- Nirupa N, Prasad MNV, Jami SK, Kirti PB. 2007. Optimization of *Agrobacterium*-mediated overexpression of osmotin-ferritin genes in *Brassica juncea*. *Transgenic Plant J* **1**:384-392.
- Nowicki M, Nowakowska M, Niezgodna A, Kozik EU. 2012. *Alternaria* black spot of crucifers: Symptoms, importance of disease, and perspectives of resistance breeding. *Veg Crop Res Bull* **76**:5-19.
- Oh IS, Park AR, Bae MS, Kwon SJ, Kim YS, Lee JE, Kang NY, Lee S, Cheong H, Park OK. 2005. Secretome analysis reveals an *Arabidopsis* lipase involved in defense against *A. brassicicola*. *The Plant Cell* **17**:2832-2847.
- Oide S, Moeder W, Krasnoff S, Gibson D, Haas H, Yoshioka K, Gillian TB. 2006. NPS6, encoding a non-ribosomal peptide synthetase involved in siderophore-mediated iron metabolism, is a conserved virulence determinant of plant pathogenic ascomycetes. *Plant Cell* **18**:2836-2853.
- Parihar PS, Prakash O, Punetha H. 2012. Changes in metabolites of *B. juncea* (Indian mustard) during progressive infection of *A. brassicae*. *Nature and Sci* **10**:39-42.
- Pathak RK, Baunthiyal M, Pandey D, Kumar A. 2020. Computational analysis of microarray data of *Arabidopsis thaliana* challenged with *A. brassicicola* for identification of key genes in *Brassica*. *J Genet Eng Biotechnol* **18**:1-20.
- Pedras MSC, Kahn AQ, Taylor JL. 1998. The phytoalexin camalexin is not metabolized by *Phoma lingam*, *A. brassicae*, or phytopathogenic bacteria. *Plant Sci* **139**:1-8.
- Pedras MSC, Zaharia LI, Ward DE. 2002. The destruxins: synthesis, biosynthesis, biotransformation, and biological activity. *Phytochem* **59**:579-596.
- Pedras MSC, Chumala PB, Jin W, Islam MS, Hauck DW. 2009. The phytopathogenic fungus *A. brassicicola*: phytotoxin production and phytoalexin elicitation. *Phytochem* **70**:394-402.
- Pedras MSC, Zaharia IL, Gai Y, Zhou Y, Ward DE. 2001. In planta sequential hydroxylation and glycosylation of a fungal phytotoxin: Avoiding cell death and overcoming the fungal invader. *Proc Nat Acad Sci USA* **98**:747-752.
- Peruch LAM, Michereff SJ. 2007. Saprophytic survival of *A. brassicicola* and management of broccoli leaf debris. *Ciencia Rural* **37**:13-18.
- Pramila GP, Tasleem M, Taj G, Mal R, Kumar A. 2014. Morphological, cultural, pathogenic and molecular variability amongst Indian mustard isolates of *A. brassicae* in Uttarakhand. *Afr J Biotechnol* **13**:441-448.

- Prasad L, Vishunavat K. 2006. Assessment of yield loss in cauliflower seed crop due to *Alternaria* blight. *Indian Phytopathol* **59**: 185-189.
- Prasad R, Saxena D, Chandra S. 2003. Yield losses by *Alternaria* blight in promising genotypes of Indian mustard. *Indian Phytopathol* **56**:205-206
- Pryor BM, Creamer R, Shoemaker RA, Mc Lain-Romero J, Hambleton S. 2009. *Undifilum*, a new genus for endophytic *Embellisia oxytropis* and parasitic *Helminthosporium bornmuelleri* on legumes. *Botany* **87**:178–194.
- Rajarammohan S, Pental D, Kaur J. 2019. Near-complete genome assembly of *A. brassicae*-A necrotrophic pathogen of Brassica crops. *Mol Plant-Microbe Interact* **32**:928–930.
- Randhawa HS, Aulakh KS. 1984. Efficacy of hot water treatment to control seed-borne fungi of raya (*B. juncea*). *Indian J Plant Pathol* **2**:73-76.
- Rawat S, Ali S, Mittra B, Grover A. 2017. Expression analysis of chitinase upon challenge inoculation to *Alternaria* wounding and defense inducers in *B. juncea*. *Biotechnol Rep* **13**:72-79.
- Redman CE, King EP, Brown JF. 1967. Tables for converting Barratt-Horsfall Rating scores to estimate mean percentages. Eli Lilly Co, Indianapolis, pp 100.
- Rotem J. 1994. The Genus *Alternaria*: Biology, Epidemiology and Pathogenicity. APS Press, St Paul, MN, USA.
- Runa F, Park M, Pryor B. 2009. *Ulocladium* systematics revisited: phylogeny and taxonomic status. *Mycol Prog* **8**:35–47.
- Rustagi A, Kumar D, Shekhar S, Yusuf MA, Misra S, Sarin NB. 2014. Transgenic *B. juncea* plants expressing MsrA1, a synthetic cationic antimicrobial peptide, exhibit resistance to fungal phytopathogens. *Mol Biotechnol* **56**:535–545.
- Saharan GS. 1991. Assessment of losses, epidemiology and management of black spot disease of rapeseed-mustard. GCIRC 8th Int Rapeseed Congr, Saskatoon, Canada, 84 p.
- Saharan GS. 1992. Disease resistance. In: *Breeding oilseed Brassicas* ed. Labana KS, Banga SS, Banga SK. Narosa Publishing House, New Delhi, pp 181-200.
- Saharan GS, Chand JN. 1988. Diseases of rapeseed and mustard. In *Diseases of oilseed crops (in Hindi)*. Directorate Publication, Haryana Agric Univ Press, Hisar, India, pp 84-91
- Saharan GS, Kadian AK. 1983a. Physiologic specialization in *A. brassicae*. *Cruciferae NewsLetter* **8**:32-33.
- Saharan GS, Kadian AK. 1983b. Analysis of components of horizontal resistance in rapeseed and mustard cultivars against *A. brassicae*. *Indian Phytopathol* **36**:503-507.
- Saharan GS, Krishnia SK. 2001. Multiple disease resistance in rapeseed and mustard. In: *Role of resistance in intensive agriculture*, ed. Nagarajan S, Singh. DP. Kalyani Publications, New Delhi
- Saharan GS, Mehta N. 2002. Fungal diseases of rapeseed - mustard. In *Diseases of field crops*, ed. Gupta VK and Paul YS. Indus Publishing Company, New Delhi, pp 193-201
- Saharan GS, Mehta N, Meena PD. 2016. *Alternaria* blight of crucifers: Biology, Ecology and Disease Management. Springer Verlag, Singapore, 326 p
- Sangeetha CG, Siddaramaiah AL. 2007. Epidemiological studies of white rust, downy mildew and *Alternaria* blight of Indian mustard [*B. juncea* (Linn) Czern & Coss]. *Afr J Agric Res* **2**:305-308.
- Sangwan MS, Mehta N. 2007. Pathogenic variability in isolates of *A. brassicae* (Berk) Sacc from different agro-climatic zones of India. *Plant Dis Res* **22**:101–107.
- Sangwan MS, Mehta N, Sharma OP, Dhandapani A. 2000. Role of Gompertz model in selection of *Brassica* group for resistance against *Alternaria* blight under epidemic areas. *Indian Phytopathol* **53**:287-289.
- Seem RC. 1984. Disease incidence and severity relationships. *Annu Rev Phytopathol* **22**:133-150.
- Seidle E, Rude S, Petrie A. 1995. The effect of *Alternaria* black spot of canola on seed quality and seed yield and studies on disease control. Agriculture and Agri-Food, Saskatoon, Canada, 41 p
- Sellam A, Dongo A, Guillemette T, Hudhomme P, Simoneau P. 2007. Transcriptional responses to exposure to the brassicaceous defence metabolites camalexin and allyl-isothiocyanate in the necrotrophic fungus *A. brassicicola*. *Mol Plant Pathol* **8**:195–208.
- Sharma G, Kumar VD, Haque A, Bhat SR, Prakash S, Chopra VL. 2002. *Brassica* coenospecies: a rich reservoir for genetic resistance to leaf spot caused by *A. brassicae*. *Euphytica* **125**:411–417.
- Sharma M, Deep S, Bhati DS, Chowdappa P, Selvamani R, Sharma P. 2013. Morphological, cultural, pathogenic and molecular studies of *A. brassicae* infecting cauliflower and mustard in India. *Afr J Microbiol Res* **7**:3351-3363.
- Sharma N, Rahman MH, Liang Y, Kav NNV. 2010. Cytokinin inhibits the growth of *Leptosphaeria maculans* and *A. brassicae*. *Can J Plant Pathol* **32**:306-314.

- Shrestha SK, Mathur SB, Munk L. 2000. Transmission of *A. brassicae* in seeds of rapeseed and mustard, its location in seeds and control. *Seed Sci Technol J* **28**:75-84.
- Shrestha SK, Munk L, Mathur SB. 2005. Role of weather on Alternaria leaf blight disease and its effect on yield and yield component of mustard. *Nepal Agric Res J* **6**:62-72.
- Simmons EG. 1967. Typification of *Alternaria*, *Stemphylium* and *Ulocladium*. *Mycologia* **59**:67-92
- Simmons EG. 2002. *Alternaria* themes and variations (305–309). *Mycotaxon* **83**:127–145
- Simmons EG. 2007. *Alternaria*: an identification manual, CBS Biodiversity Series No 6. Utrecht. The Netherlands, pp 775
- Singh H. 2005. Diurnal pattern of aerospore of *Sclerospora graminicola* and *A. brassicae* in relation to disease development. Ph D Thesis, pp 111+xvii, Department of Plant Pathology, CCS HAU, Hisar
- Singh DN, Singh NK, Srivastava S. 1999. Biochemical and morphological characters in relation to Alternaria blight resistance in rapeseed-mustard. *Annu Agric Res* **20**:472-477
- Singh H, Singh D. 1989. Studies on genetic control of resistance to *A. brassicae* in Indian mustard. Proceedings of XII Eucarpia Congress, Gottingen, Germany, pp 11-13
- Singh K, Suhag LS. 1983. Some pathological studies on *A. alternata* causing leaf and pod blight of radish in Haryana. *Indian Phytopathol* **36**:174-176.
- Singh RB, Singh RN. 2005. Fungicide management of foliar disease of mustard in mid-eastern India. *Indian Phytopathol* **58**:51-56.
- Sivapalan A, Browning JW. 1992. Incidence of *A. brassicicola* (Schw) Wiltsh on *B. oleracea* seeds. *Aust J Exp Agric* **32**:535–537.
- Smith IM, Dunez J, Phillips DH, Lelliott RA, Archer SA. 1988. European Handbook of Plant Diseases. Blackwell Science Publications, Oxford, London, pp 583.
- Stankova J. 1972. Varietal variability of winter rape in its susceptibility to dark leaf spot and the factors influencing the development of the disease. *Rostlinna Vyroba* **18**:625-630.
- Stoll K. 1948. On the Alternaria blackening of Brassicas. *Nachr Bl Dtsch PflSch Dienst, Berl NF* **2**:174-178.
- Stoll K. 1952. The origin, injurious effect and control of Brassica blackening. *NachrBl Dtsch Pflsch Dienst Berl NF* **6**:81-85
- Taber RA, Vanterpool TC, Williard AT. 1968. A comparative nutritional study of *Alternaria raphani*, *A brassicae* and *A brassicicola* with special reference to *A raphani*. *Phytopathol* **58**: 609-616.
- Taber RA. 1964. A study of pathogenicity and *in vitro* growth of *Alternaria* spp. isolated from *Brassica* spp. with special reference to *A. raphani* from rape. M.Sc. Thesis, University of Saskatchewan, Saskatoon, Canada.
- Taj G, Kumar A, Bansal KC, Garg GK. 2004. Introgression of osmotin gene for creation of resistance against Alternaria blight by perturbation of cell cycle machinery. *Indian J Biotechnol* **3**:291-298.
- Tasleem M, Baunthiyal M, Kumar A, Taj G. 2017. Determination of antioxidant activity in overexpressed MPK3 transgenic *B. juncea* for induction of defense against Alternaria blight disease. *J Pharmacogn Phytochem* **6**:2579-2582.
- Tewari JP. 1991a. Structural and biochemical bases of the blackspot disease of crucifers. *Adv Struct Biol* **1**:325-349.
- Tewari JP. 1991b. Current understanding of resistance to *A. brassicae* in crucifers. GCIRC 8th Int Rapeseed Congr, Saskatoon, Canada, pp 84
- Tewari JP, Conn KL. 1993. Reaction of some wild crucifers to *Alternaria brassicae*. *Bull OILB SROP* **16**:53-58.
- Thomma BPHJ. 2003. Pathogen profile- *Alternaria* spp: from general saprophyte to specific parasite. *Mol Plant Pathol* **4**:225–236.
- Tripathi NN, Kaushik CD, Yadava TP, Yadav AK. 1980. Alternaria leaf spot resistance in raya. *HAU J Res* **10**:166-168.
- Tsuneda A, Skoropad WP. 1980. Interactions between *Nectria inventa*, a destructive mycoparasite, and fourteen fungi associated with rapeseed. *Trans Brit Mycol Soc* **74**:501-507.
- USDA. 2020. Foreign Agriculture Service. United States Department of Agriculture. <https://www.fas.usda.gov/regions/india>. Accessed on 30 August 2021.
- Van Schreven DA. 1953. *Alternaria*, *Stemphylium* en *Botrytis* a antasting bij Koolzaad (*B. napus*). *Tijdschr Plantenziekt* **59**:105-136.
- Vannacci G, Harman GE. 1987. Biocontrol of seed-borne *Alternaria raphani* and *A. brassicicola*. *Can J Microbiol* **33**:850-856.
- Vannacci G, Pecchia S. 1988. Location and transmission of seed-borne *A. raphani* Groves and Skolko in *Raphanus sativus* L: a case study. *Archivfur Phytopathologie und Pflanzenschutz* **24**:305-315.
- Verma SS, Yajima WR, Rahman MH, Shah S, Liu JJ, Ekramodoullah AK, Kav NN. 2012. A cysteine-rich antimicrobial peptide from *Pinus monticola*

- (PmAMP1) confers resistance to multiple fungal pathogens in canola (*B. napus*). *Plant Mol Biol* **79**:61-74.
- Vishwanath, K, Kolte SJ. 1997. Variability in *Alternaria brassicae*: Response to host genotypes, toxin production and fungicides. *Indian Phytopathol* **50**:373-38.
- Von Keissler K. 1912. Zur Kenntnis Pilzflora Krains. *Beihefte zum Botanischen Zentralblatt* **29**:434.
- Wang Y, Geng Y, Ma J, Wang Q, Zhang XG. 2011. *Sinomyces*: a new genus of anamorphic *Pleosporaceae*. *Fungal Biol* **115**:188–19.
- Warwick SI, Francis A, Al-Shehbaz IA. 2006. Brassicaceae: species checklist and database on CD-Rom. *Plant Syst Evol* **259**:249–258.
- White JG, Linfield CA, Lahdenpera ML, Uoti J. 1990. Mycostop a novel biofungicide based on *Streptomyces griseoviridis*. Proc of British Crop Prot Confr, Pests and Diseases, Brighton, pp 221-226.
- Williams J, Pink DAC. 1987. Development of an *in vitro* screen in brussels sprout for resistance to *A. brassicae*. *Cruciferae NewsLetter* **12**:91.
- Woudenberg JHC, Groenewald JZ, Binder M, Crous PW. 2013. *Alternaria* redefined. *Stud Mycol* **75**:171–212.