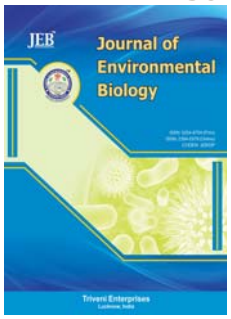
Review Article

Journal Home page : www.jeb.co.in ★ E-mail : editor@jeb.co.in

Journal of Environmental Biology

DOI : http://doi.org/10.22438/jeb/38/6/MS-248

ISSN: 0254-8704 (Print)
ISSN: 2394-0379 (Online)
CODEN: JEBIDP



Alternaria blight of rapeseed-mustard–A Review

Authors Info

H.K. Singh*[1], R.B. Singh[1], P. Kumar[1], M. Singh[1], J. Kumar Yadav[1], P.K. Singh[2], M. P. Chauhan[2], R.C. Shakywar[3], K.N. Maurya[2], B. Shainy Priyanka[1], T. Srivastava[2], S.K. Yadav[1] and M.K. Maurya[1]

[1]Department of Plant Pathology, Narendra Deva University of Agriculture and Technology, Faizabad-224 229, India

[2]Department of Genetics and Plant Breeding, Narendra Deva University of Agriculture and Technology, Faizabad-224 229, India

[3]College of Horticulture and Forestry, Central Agricultural University, Pasighat-791102, India

*Corresponding Author Email : hksndu@gmail.com

Key words

Alternaria blight
Necrotrophs
Rapeseed mustard
Seed shrivelling

Publication Info

Paper received : 21.01.2016
Revised received : 14.01.2017
Accepted : 25.03.2017

Abstract

This review on Alternaria blight caused by *Alternaria brassicae, A. brassicicola and A. raphani* singly or by mixed infection is one of the most widespread and destructive disease of oilseed brassicas in all the continents. These pathogens are necrotrophs. The disease appears as black spot but later on enlarge and develops into prominent round spots with concentric rings. Many spots coalesce to form large patches showing blight and cause defoliation in severe cases. The spots on the mid-ribs of the leaves are linear and sunken. Circular to linear lesions also develop on stem and pods, which elongate at later stages. Infected pods produce small, discoloured and shrivelled seeds. This disease causes substantial yield losses as a result of several factors including reduced photosynthetic potential, early defoliation, flower bud abortion, premature ripening, siliquae dehiscence, seed shrivelling and reduced seed size, impairs seed colour and reduced oil content. This paper reviews the research on the development of Alternaria blight, describes the pathogens of Alternaria blight on rapeseed-mustard, which includes geographical distribution, economic importance, symptoms, habitat and host range, pathogen taxonomy, survival, pathogenicity and disease development, epidemiology, host resistance, breeding for disease resistance, genotypic stock, and management.

Introduction

Oilseed brassicas, also known as rapeseed-mustard *(Brassica spp.)*, is the second largest oilseed crop that contribute 32 per cent of the total oilseed production in India. Out of 75.55 m tones of rapeseed-mustard in the world from over 30.51 m ha area, India shares 7.80 m tones of rapeseed-mustard production from the area of 6.50 m ha with productivity of 1208 kgha^{-1}. (Anonymous, 2013). Among the oilseed brassicas, yellow *sarson (Brassica rapa* var. yellow *sarson),* brown *sarson (B. campestris* var. brown *sarson),* toria *(B. campestris* var. *toria),* Indian mustard *(B. juncea),* Karan rai *(B. carinata)* and oilseed rape *(B. napus)* are grown for edible oil, where as black mustard *(B. nigra)* is used as condiment and for pickle making, food preserving and spices to improve flavour, also as fodder for live-stocks. The leaves of young plants are used in the human diet as green vegetable. Mustard seed and oil has multiple uses in health care system. It improves the body complexion because of its antifungal property. It is used as a very good massage oil, which brings vitality and strength to the body and improves the circulatory system and cures bodyache. It also kills various microbes and thus, keeps skin infections away. Oral doses of oil help in strengthening the teeth and cure various mouth related diseases. It helps in healing wounds by stopping the pus formation and in curing various skin disorders by removing unwanted fluids from the body (Kumar and Chauhan, 2005). Erucic acid and glucosinolate are the two major deterrents of oil and seed meal in oilseed brassica, respectively (Singh *et al.,* 2013[c]). The oilseed brassicas usually contain 38-57% of erucic acid, 4.7-13% linolenic acid and 27% of oleic and linoleic acid, which are of high nutritive value required for human health (Singh *et al.,* 2011[a]; Singh *et al.,* 2012).

Huge volume of edible oil is still to be imported to meet the ever increasing domestic need, which can easily be met by bridging the wide gap existing between the crop potential and

the realized yield at the farmer's fields. A major contributory factor to this gap is its unchallenged exposure to a number of biotic, mesobiotic and abiotic stresses. Among the biotic stresses, Alternaria blight caused by *Alternaria brassicae, A. brassicicola and A. raphani* is one of the most widespread and destructive disease of oilseed brassicas species in all the continents. This disease is also called the black spot (Louvet, 1958) or grey spot (McDonald, 1959) based on the symptoms produced on the host is reported from across the world. Alternaria blight causing pathogens can attack all the aerial parts of plant and can cause huge losses in yield. This article reviews the work done on the different aspects of Alternaria blight disease in oilseed brassicas.

Geographical distribution : Three species of *Alternaria,* viz.*, A. brassicae* (Berk.) Sacc., *A. brassicicola* (Schw.) Wilts. and *A. raphani* Groves and Skolko have been found to affect the rapeseed and mustard crop quite commonly throughout the world. *A. brassicae* is more destructive via greater tissue damage and occurs frequently over the other species. In India, its first authentic observation was in 1901 on *Sarson (Brassica campestris* var. *sarson)* crop raised at Tirhoot near Pusa, Bihar (Butler, 1918). It is reported to occur in Bangaladesh, Canada, China, England, France, Germany, Holland, India, Japan, Nepal, Poland, Spain, Sri Lanka, Sweden and Trinidad (Howlider *et al*. 1985, Kolte, 1985; Verma and Saharan, 1993, Singh and Singh, 2002; Shrestha *et al.* 2005). The disease has great economic significance in Canada also. The spots caused by *A. brassicae* and *A. brassicicola* may occur together on the same plant. *A. brassicicola* causing the disease in rapeseed in the Prairie Provinces is of later origin in the country (Petrie, 1974). *A. brassicae* and *A. raphani* being most common pathogen, however, the disease caused by *A. brassicicola* is more in the Kiel-Kitzeberg area of Germany (Domsch, 1957).

Economic importance : The disease plays havoc with the crop by reducing its photosynthetic area, growth and seed yield. Besides quantitative loss in yield, the quality of the seeds, *i.e.,* its size, colour and germinability and oil yielding capacity, are also affected adversely due to the disease (McDonald, 1959; Bandhopadhyay *et al.,*1974; Chahal and Kang, 1979a; Randhawa and Aulakh, 1981). The extent of reduction in yield depends upon severity of disease, host species, variety, time of initial infection and state of predisposing factors. In India, the commercial cultivars of *B. juncea* are comparatively more tolerant to *A. brassicae,* than the cultivars of *B. campestris* var. brown *sarson, B. campestris* var. *toria* and *B. campestris* var. yellow *sarson* (Butler, 1918; Hussain and Thakur, 1963). The reduction in yield due to the disease varies at different locations and situations. In northern parts of India, it ranges between 10 to 70%, the maximum loss being in *B. campestris* var. yellow *sarson* or *B. campestris* var. brown *sarson*. (Singh and Bhowmik, 1985; Chahal and Sekhon, 1980; Kadian and Saharan, 1984; Kaushik *et al.,* 1984; Kolte, 1985; Kolte *et al*., 1987; Shivanna and Sawhney, 1993; Kumar, 1997; Kolte, 2002; Meena *et al.,* 2002; Gupta *et al.,* 2003; Chattopadhyay, 2008). In mid-eastern India, the avoidable yield losses due to this disease were reported from 20% to 36% in different *Brassica* spp. depending upon the severity (Singh and Singh, 2005a; Kumar and Singh, 2012; Singh *et al.,* 2013a; Kumar *et al*., 2014)

McDonald (1959) from Canada reported a yield loss of about 20% in 1955 and 1956 crop season due to *A. brassicae* infection. The combined effect of *A. brassicae* and *A. raphani* under greenhouse conditions resulted in 70 and 42% loss of seed yield in *B. campestris* and *B. napus,* respectively. *A. brassicae* alone causes 63% loss to *B. campestris* and 42% to *B. napus* and *A. raphani.* The average yield losses in Nepal due to Alternaria blight has ranged between 32 to 57 per cent (Shrestha *et al*., 2005). Infected seeds show poor germinability and loss in oil and protein content (Vasudeva, 1958; Nijhawan and Hussain, 1964; Bandhopadhyay *et al.,* 1974; Degenhardt *et al.,* 1974; Chahal and Kang, 1979c; Singh and Bhajan, 2004). The extent of reduction in oil content varies with the crop species and variety involved. Reduction is higher in rapeseed than in Indian mustard, varying between 4.7 to 36%, and 2 to 29%, respectively (Ansari *et al*., 1988a; Kaushik *et al*., 1984; Kumar, 1997).

Symptoms : Alternaria blight attacks all the green aerial parts of the plant reducing its photosynthetic area and vigour. The disease, in northern parts of India is usually seen during mid December to early January as chlorotic specks, later turning into minute dark-brown or black spots on lower leaves of young plants. On older leaves, the spots turn into circular, dark-brown, sunken necrotic lesions surrounded by light yellow halo and bear conidiophores and conidia in concentric rings, at the grayish-white centre, giving them a target board effect. Under congenial weather conditions, lesions enlarge and coalesce fast, resulting in mild to severe defoliation due to the production of senescence promoting compounds (Dahiya *et al.,* 1988; Dahiya and Tiwari, 1991; Saharan and Mehta, 2002). Gradually, the disease progresses to the upper leaves, stems and pods. The spots on young stems and green pods appear as black specks. On stem, they turn into dark-brown elongated lesions with pointed ends, while on pod they become round to elliptical and sunken, rarely encircling the stem or pod. Lesions are more on the surface of the stem or pod facing the air currents. In advanced stage, the center of lesions gets laden with dark spore mass to give it grayish white hue. Intermittent late winter rains, generally coinciding with pod maturation, accelerate pod infection contributing to higher reduction in seed yield (Dey, 1948; Stankova, 1972; Chahal and Kang, 1979[b]). Infected pods ripe pre-maturely, shrink and shatter or disintegrate easily. Seeds, particularly those directly under pod lesions show dark-brown spots on seed coat, become under sized, deformed, discoloured and infected. Such seeds, when peeled, show dark-brown speaks on their cotyledons and have low percentage of oil content (Meena *et al.,* 2010).

Habitat and Host range

Alternaria species are either parasites on living plants or saprophytes on organic substrates. The host range of pathogenic *Alternaria* is very broad. Mostly *A. brassicae* and *brassicola* have been found to infect number of crucifers, viz. *Brassica rapa* var.

Table 1: Comparative morphological characteristics of *A. brassicae, A. brassicicola and A. raphani*

Fungal structures	*A. brassicae*	*A. brassicicola*	*A. raphani*
Mycelium	Septate, brown to brownish-gray	Septate, grayish black to olive gray	Cottony whitish to greenish gray, or dark olive
Conidiophores	Dark, septate, arise in fascicles, measuring 14-74 µ x 4-8 µ	Olivaceous, septate, branched, measuring 35-45 µ x 5-8 µ	Septate, olive-brown, simple or branched, measuring 29-160 µ x 4-8 µ
Conidia	Brownish black, obclavate, muriform with long beak, longer and wider with more septations than that of *A. brassicicola and A. raphani,* produced singly or sparingly in chains of 2 to 3.	Dark, cylindrical to oblong, muriform without beak, few septations and smaller than those *of A. brassicae and A. raphani,* produced in long chains of 8 to 10 spores.	Olive-brown to dark, obclavate, muriform with poorly developed or no beak, wider than those of *A. brassicae,* less uniform in shape than either of the other two species, more or less pointed at each end, appear singly or in chains of up to 6 spores.
Spore body length (µ)	96-114	45-55	45-58
Spore beak length (µ)	45-65	None	10-25
Spore body width (µ)	17-24	11-16	13-21
Overall spore length (µ)	148-184	45-55	60-83
Transverse septation	10-11	5-8	6-9
Longitudinal septation	0-6	0-4	3-6
Rate of growth and sporulation on media	Rudimentary growth, grow slowly	Produce well-developed black sooty colony with distinct zonations, grow faster, sporulates abundantly	Cottony mycelia colony distinguishes this from other species, less abundant sporulation.
Formation of chlamydospores in culture or on host	Formed less frequently in culture	Not known	Usually olive brown chlamydospores are formed in culture as on the partially decayed affected plant parts

Source: (Meena *et al.,* 2010)

yellow *sarson*, *B. campestris* var. brown *sarson, B. campestris* var. *toria* and *B. juncea, B. nigra, B. alba, B.carinata, B. napus, B. chinensis, B. perkinensis, B.tournefortii, B. rugosa, B. oleracea* var. *capitata B. oleracea* var. *botrytis, Raphanus sativus* and *Eruca sativa* under field as well as glasshouse conditions. In addition, it has been observed on weed host like *Camelina sativa, Crambe abyssinica, Crambe maritime, Anagalis arvensis* (hirankhuri), *Convolvulus arvensis* and *Chenopodium album* (bathua) usually found in *Brassica* field (Saharan and Kaidan 1983a; Tripathi and Kaushik, 1984; Verma and Saharan, 1994; Mehta *et al.,* 2002; Saharan and Mehta, 2002). These hosts can play a major role in disease perpetuation.

Pathogen

Taxonomy : As early as in 1836, Berkeley identified the fungus causing blight on Brassicaceae as *Macrosporium brassicae* Berk., which was later renamed as *Alternaria brassicae* (Berk.) Sacc. by Saccardo (1886). According to Elliott (1971), *Alternaria* contains forty four species. Additional species groups are *A. arborescens, A. brassicicola, A. porri* and *A. radicina* groups (Simmons, 1995; Roberts *et al.,* 2000; Pryor and Gilbertson, 2000, 2002).

The three *Alternaria* species infecting rapeseed-mustard can be readily distinguished by microscopic observation (Table 1). Three distinct *A. brassicae* isolates, A (highly virulent), C (moderately virulent) and D (avirulent) are prevalent in India (Vishwanath and Kolte, 1997). Variation among the Indian isolates of *A. brassicae* has also been noted (Meena *et al*., 2005; Patni *et al.,* 2006; Kaur *et al.* 2007; Singh *et al.,* 2007[a] ; Kumar *et al.,* 2007; Singh *et al.,* 2009; Goyal *et al.,* 2011; Singh and Singh, 2014; Pramila *et al.,* 2014; Singh *et al.,* 2014[a]). Khan *et al.* (2007[a]) and Singh *et al.* (2015[a]) have also reported variation in conidial length, breadth and septation in ten isolates collected from different agro-climatic zone of India. They have also studied the physiological and pathogenic variability of all those isolates and grouped them into three categories (Singh *et al*., 2015[b]). Studies on pathogenic variability have to be the foundation for development of pre-breeding populations as strategic defence mechanism. Formation of chlamydospores is reported in *A. brassicae* and *A. raphani* both, while microsclerotia are found to be produced by former only based on the descriptions given by Kolte, 1985; Verma and Saharan, 1994 and Meena *et al.,* 2010.

Variation at DNA level among *A. brassicae* (Berk.) Sacc., *A. brassicicola* (Schwein) Wiltshire, *A. raphani* Groves and Skolko and *A. alternata* (Fr.) Keissel have been established by restriction fragment length polymorphism (RFLP) (Jasalavich *et al.,* 1995). Hong *et al.* (1996) by studying 53 strains of *A. brassicae* collected from different regions in China showed differences in virulence on 4 groups of Chinese cabbage with varying levels of resistance. The main focus is on genes being expressed during Alternaria infection of *Brassica* Cramer and Lawrence, (2004) and also using suppression subtractive hybridization between RNA isolates from spores of *A. brassicicola* in water and on the leaf surface of an ecotype of *Arabidopsis thalianas*. They cloned and sequenced cDNA clones that were differentially expressed. One gene, expressed only during infection, was identified. Labuda *et al.* (2008) separated *Alternaria jesenskae* from the other related large-spores and filament-beaked *Alternaria* species on the basis of sequences of ITS1 and ITS2 region, as well as by its distinctive morphology.

Survival : The pathogen perpetuates through seeds, plant debris, soil and weed hosts plant (Chahal, 1981; Saharan *et al*., 1982; Tripathi and Kaushik, 1984; Kolte, 1987; Ansari *et al.,* 1989a; Mehta *et al.,* 2002). Seed borne inoculum does not cause infection in north Indian plains in the coming season due to high temperatures prevailing during summer in storage (Chahal, 1981; Kolte, 1985; Singh, 2009). Kumar *et al.* (1994) also reported that the pathogen survives for more than six month at lower temperature as compared to room temperature. *A. brassicae* survives in the leaf debris buried at 7.5 cm deep in the field in the form of macrosclerotia and chlamydospores (Tsuneda and Skoropad, 1977; Tripathi and Kaushik, 1984; Saharan and Metha, 2002). Air currents, rain splashes and driving rains disperse the inoculum in the field. The primary infection is noticed on the cotyledonary leaves. In temperate climate countries, seeds from affected plants also act as a primary source for initial infection (Humpherson-Jones and Maude, 1983). The foliar lesions give rise to sporulation, thus acting as sources of secondary infection of this polycyclic disease (Kolte, 1985). The infection occurs through the stomata and under favourable climatic conditions, new lesions arise within 4-6 days and produce spores. The pathogen infects all the green tissues and even reaches the seeds through pod infection (Kavita *et al.,* 2013).

Pathogenicity and disease development : Conidia of *Alternaria* species germinate readily in the presence of moisture by giving rise to a germ tube from any cell. Sometimes all cells of a spore germinate, giving rise to several germ tubes. *A. brassicae* penetrates leaf only through stomata, whereas *A. brassicicola* penetrates leaf tissue directly and through stomata. Direct penetration is known in the case of *A. raphani.* New spots develop about 3 to 4 days after inoculation. *Alternaria brassicae* enters the leaves through stomatal opening, the newly formed spots bearing spores 3-4 days after infection (Butler, 1918). *Alternaria brassicicola,* on the other hand, enters the host directly by forming appresoria (Hung and Chung, 1993).

A. brassicae produces cellulase enzyme (Nehemiah and Deshpande, 1976; 1977) and toxins (Husain and Thakur, 1966; Degenhardt *et al.,* 1975; Durbin and Uchytil, 1977) which assist in the infection and disease development process. However, the exact role of this enzyme or toxin in pathogenesis is not known.

Alternaria brassicae has a multitoxin system and produces at least three phytotoxins. One phytotoxin is a cyclodepsipeptide, destruxin B, having molecular formula $C_{30}H_{51}N_5O_7$ and a molecular weight of 593 (Ayer and Pena-Rodriguez, 1987; Bians and Tewari, 1987). Indication about the possible role of a toxin produced by *A. brassicae* inciting leaf blight of rapeseed and mustard came from Hussain and Thakur (1966) when they reported that the culture filtrates of the pathogen induce severe wilting besides causing water soaked spots in yellow mustard cuttings within 12h. The culture filtrate increased the permeability of host cell and loss of electrolytes (Dubey *et al*. 1980). According to the semi-purified preparation of culture filtrates contains two groups of non-specific toxin which cause disease symptoms in leaves. Later, a purified preparation of the toxin was obtained which tested host-specific and was found identical to cyclodepsipeptide, destruxin B (Ayer and Pena-Rodriguez, 1987; Bains and Tiwari, 1987), reported earlier from the culture filtrates of *A. brassicae* (Pena-Rodriguez, 1985). Both fungus and toxin cause symptoms of different severities on different *Brassicas*, ranging from severe chlorosis and necrosis to almost no visible chlorosis. The order of sensitivity of different *Brassicas* to phytotoxin is similar to the order of susceptibility to *A. brassicae* (Bains and Tiwari, 1987).

The phytotoxin destruxin B of *A. brassicae* was not found host-specific on 30 different plant species tested. It caused necrotic and chlorotic symptoms both on host and non-host plants. But there were significant differences between taxonomic plant groups in their sensitivity to destruxin *B. Brassica* spp. were most sensitive to the toxin, and sensitivity decreased as relatedness of plant groups became more distant. No genotype within the *Brassica* species has been found specifically sensitive to destruxin B suggesting it to be host-selective nature. It is considered to be a virulent factor that contributes to the aggressiveness of the pathogen by conditioning the host tissue, and thereby determining the susceptibility of the host (Buchwaldt and Green, 1992). Two other destruxins viz., homodestruxin B (Ayer and Pena-Rodriguez, 1987) and destruxin B_2 (Buchwaldt and Jensen, 1991) elaborated by *A. brassicae*, are also phytotoxic to leaves of oilseeds rape. Like destruxin B, homodestruxin B also causes symptoms of different severities on leaves of various non-host plant species, and therefore is non-host specific in nature (Bains *et al.* 1993). A plant growth regulator antagonistic to dextruxin B is found in *B. napus* leaves infected with *A. brassicae* (Agrwal *et al*., 1994).

Epidemiology : The disease initiates with the primary infection on cotyledonary and lower leaves moist with dew, during early hours of the day, which in turn, act as the source of inoculum for secondary

infection. The relative humidity during the period of crop growth ranges between 46-96% during day and 73-92% during night (Wadhwani and Dudeja, 1982). Sporulation in *A. brassicae* and *A. brassicicola* on naturally infected leaf discs of oilseed rape requires RH ≥ 91.5% and 87%, respectively. The optimum temperature required for the germination of conidia in artificial medium is 20-23°C for *A. brassicae* (Ansari *et al*. 1988) and 22-32°C for *A. brassicicola,* (Sarkar and Sengupta, 1978). Generally for *A. brassicicola* optimum growth required is about 4°C higher temperature than *A. brassicae* (Hung and Chung, 1993). Both *A. brassicae* and *A. brassicicola* require free water coupled with optimum temperature of 15°C and 25°C, respectively for infecting the crop. A minimum duration of 16 hrs is required for initiation of infection and 48-72 hrs for optimum disease development (Humpherson -Jones and Maude, 1983).

The optimum temperature for sporulation is 18-24°C for *A. brassicae* and 20-30°C for *A. brassicicola* at which both the pathogens produce spores in 12-14 hrs. Above 24°C, sporulation in *A. brassicae* is inhibited (Singh, 2009). Interrupting a 16 hr wet period at 20°C with a dry period of 2 hr at 70 to 80% relative humidity did not affect sporulation in either fungus, but a dry interruption of 3 to 4 hrs relative humidity inhibits sporulaion in both. Exposure of both fungi to alternating wet (18 hrs at 100% RH, 20°C) and dry periods (6 to 30 hrs at 55-65% RH, 20°C) does not affect the concentration of spores produced during wet period. Sporulation periods are not affected either by the host type or the age of the host tissue (Humpherson-Jones and Phelps, 1989). Alternate light and darkness is better for growth than continuous light or darkness (Ansari *et al.,* 1989).

Susceptibility of oilseed *Brassicas* to *A. brassicae* increases with the age of the plant, prevailing temperature and duration of host surface wetness (Chahal, 1986a; Mridha and Wheeler, 1993; Hong and Fitt, 1995). The disease intensity increases with the age of plant starting from 21 to 71days after germination (Sinha *et al.,* 1992). Maximum leaf disease severity was seen during rosette and flowering stage of the crop (Awasthi and Kolte, 1994). On older leaves of oilseed rape (*B. napus*), infection was optimum at 25°C and on pods maximum infection was observed at 20°C. The duration of minimum-wetness period required for infection of leaves and pods is longer at lower temperature than at higher temperature. A minimum wetness period of 4 hrs was needed for infection by *A. brassicae* at 18°C; an increase in the duration of wetness up to 12 hrs increased disease severity. On leaves, dry periods interrupting with wet periods limited the lesion development as compared to initial wet periods, but on pods some further infections developed when pods were re-wetted (Mridha and Wheeler, 1993). In rapeseed and mustard, a 16-24 hrs period of leaf wetness is necessary at critical temperature of 25°C for high infection (Kadian and Saharan, 1984). Chattopadhyay *et al.*, (2005) noted a positive correlation between the severity of Alternaria blight on leaf and a maximum and minimum daily temperature of 18 to 27°C and 8-12°C along with more than >92% and > 40% morning and afternoon relative humidity, respectively, in preceding week. Disease severity on pods was favoured by maximum daily temperature of 20-30°C, daily mean temperature of 14°C, morning RH >90%, daily mean RH >70%, >9 hrs of sunshine and >10 hrs of leaf wetness. Mehta *et al.* (2005) reported a maximum temperature of 20-25°C, minimum temperature 15°C, morning RH >90% and evening >70% as conducive to the Alternaria blight development. Sangeetha and Siddaramaiah (2007) reported positive correlation between disease development and maximum temperature at Bangalore (Karnataka). In their study, a maximum temperature of 26-29°C and average relative humidity of >65% favoured the disease development. Bal and Kumar (2014) reported that the Alternaria blight was favoured by mean temperature ranging between 13.5 to 19.3°C along with an average RH of more than 70% in Punjab.

Disease incidence is also influenced by the concentration of inoculums. The incidence of the disease on pods of spring rape, *B. napus* cv. Starlight increased as the inoculum concentration was increased from 80-104 spores ml^{-1}, further increase in inoculum concentration increased only the severity of disease but not its incidence (per cent of pod diseased). The incubation period decreased as temperature increased from 6-20°C, wetness period from 2-12 hrs, inoculum concentration from 80 to 2x103 spores/ml and leaf age from 4-10 days. The incubation period usually decreased sharply with increased lesion density (Hong and Fitt, 1996).

Foggy weather generally favoured the severe Alternaria blight attacks in India (Butler, 1918). There is a positive correlation between weather factors and disease progress. Severe blight is associated with low temperature (minimum 2-12°C, maximum 16-26°C), high RH (80-96%), average rain fall of 30 mm and wind velocity of 2-6 $kmhrs^{-1}$ (Sinha *et al.,* 1992; Dang *et al.,* 1995; Gadre *et al.,* 2002;). According to Chahal and Kang (1979[b]) and Chahal (1982), average temperature of 18°C, prevalence of frequent rains, atmospheric humidity of 80% or above, stormy weather coupled with high wind velocity during flowering and pod formation stage led to the development of the disease in epiphytotic form at Ludhiana, Punjab. Gupta *et al*. (2003) correlated the weather factor and Alternaria blight development and reported that 85 day-old-plant showed highest disease severity with negative correlation with maximum and minimum temperature and positive correlation with relative humidity. Yadav and Brar (2003) observed increased susceptibility to the disease during rosette to flowering stage of the crop when relative humidity ranges 81-94% with total rainy days of 4-11 days. Kumar *et al.* (2014) reported maximum disease development in the first week of February in case of yellow *sarson* at Faizabad, which was favoured by mean min. /max. temperature (8.6-9.90°C/22-25°C) and min. /max. relative humidity (50-70% /85-95%), respectively. They reported significant positive correlation of disease intensity, with min. /max. temperature and min. /max. relative humidity in case of most of the cultivars tested.

Host resistance : The existence of wide inter and intra-specific variation in reaction to the disease is a common feature. Lesion

type and sporulation capacity seem to indicate the levels of resistance in *Brassica* spp. (Saharan and Kadian, 1983[b]; Bhowmik and Munde, 1987; Kolte, 1987; Banasal *et al.* 1990; Sharma and Singh, 1992[a]). Based on these characteristics, Bhowmik and Munde (1987) defined four broad reaction types in *Brassica* spp. towards *A. brassicae.* Their general rank in order of most to least resistance is: *B. carinata* and *B. napus* > *B. tournefortii, B. nigra* and *B. fructiculosa* > *B. campestris* (vars. Brown *sarson, Toria* and Yellow *sarson*). According to Sharma and Singh (1992[a]), out of *B. juncea, B. carinata, B. napus, B. rapa* and *B. hirta,* the latter has high degree of resistance as evident from small spot size and minimum sporulation on both the surfaces of spot and absence of yellow halo, intra-vertical differences in both lesion size and sporulation are also commonly discernable.

Plants more distantly related to cruciferous species are often resistant to Alternaria blight (Tewari *et al.* 1987; Conn *et al*., 1988). *Sinapis alba (B. alba),* accessions of *Eruca sativa, Camelina sativa, Capsella bursa-pastoris* and *Neslia peniculata* possess high degree of resistance to *A. brassicae. Eruca sativa* exhibits a hyper type of reaction against infection by *A. brassicae* (Bhander and Maini, 1965; Grontofit, 1986; Conn *et al.,* 1988; Downey and Rimmer, 1993; Tewari and Conn, 1993). Both *C. sativa* and *C. bursa-pastoris* produce phytoalexins following inoculation with conidial suspension of the pathogen (Conn *et al.,* 1988). *Camelina sativa* produces phytoalexin even when very few conidia are deposited on leaves. The phytoalexin accumulates within leaf areas under and in conidial droplets. The rapid rate of phytoalexin accumulation shortly after inoculation is considered responsible for inhibition of fungal growth on the leaf surfaces (Jejelowo *et al*., 1991). Two thizoyl substituted indole phytoalexins; camalexin (C11N8N2S) and 6-methoxycamalexin ($C_{12}H_{10}N_2SO$) are produced in leaves of *C. sativa* following elicitation by the pathogen (Browne *et al.,* 1991).

Capsella bursa-pastoris elicits camalexin, 6-methyloxycamalexin and N-methylcamalexin ($C_{12}H_{10}N_2S$) upon infection by the pathogen (Jimenez *et al.,* 1997). Similarly, *A. brassicae* or its phytotoxin, destruxin B may induce production of phytoalexin, "sinalexin" in *S. alba* (*B. alba*) for resistance to the pathogen (Pedras and Smith, 1997). It is believed that qualitative and/or quantitative differences in elicitation of phytoalexin may account for differences in resistance observed in plants (Conn *et al.,* 1988).

Resistance to *A. brassicae* in crucifers is layered and multi-component (Tewari, 1991). *Alternaria brassicae* shows no difference in its interaction with resistant and susceptible hosts at the early stages of infection. The histology of *A. brassicae* infection in leaves of susceptible *B. campestris* cv. Candle and moderately susceptible *B. napus* cv. Alex is similar; the pathogen becomes sub-cuticular after direct penetration and then colonizes in the epidermal and mesophyll cells indicating that differential susceptibility of cultivars does not reside in the early stages of host-pathogen interaction (Tewari, 1986). The percentage conidial germination does not vary significantly for any Alternaria pathogens of *Brassica* species between the host and non-host plants.

The pathogens are affected by interactions occurring at the plant surface and inhibition of conidial germination is not a feature of resistance in oilseed brassicae (McRoberts and Lennard, 1991). The presence of epicuticular wax in some cultivars of *B. napus* and *B. oleracea* var. *alboglabra* confers a physical type of resistance to *A. brassicae* (Tewari and Skoropad, 1976; Munde and Bhowmik, 1985). Wax layer being hydrophobic, may affect conidial germination by impeding movement of foliar exudates to surface, by preventing water droplets to stay on the leaf surface and by providing a sort of mechanical barrier to the invading pathogen. (Skoropad and Tewari, 1977; Munde and Bhowmik, 1985; Berry and Lennard, 1988; Conn and Tewari, 1989).

Young *Brassica* plants show a high degree of resistance to *A. brassicae* but they become susceptible with age (Chahal, 1986a; Sarkar and Sengupta, 1978). Quantitative measurements of necrosis in leaf and pod tissue of oilseed rape after inoculation with *A. brassicae* and *A. brassicicola* showed that active defence mechanisms are involved in young tissue and susceptibility of plants to infection increases greatly by senescence and physiological stress. This was also evident from the fact that field application of BAS 11104 W, a growth regulator of the triozole group, retarded the aging of rape tissue and delayed infection by *Alternaria* spp. (Kohel and Hoffman, 1989). The changes in glucosinolate content in leaves of oilseed rape following infection by *A. brassicae* may restrict the spread of existing infection or inhibit subsequent attempted infections, especially in younger leaves. It has been observed that *B. campestris* (*B. rapa*) seedlings release alkenyl iso-thiocyanates for catabolism of glucosinolates during infection, which is a pre-requisite for their involvement in resistance to the invading pathogen (Doughty *et al.,* 1991; 1996).

Resistance to *A. brassicae* in some *Brassica* genotypes has been associated with quantitative changes in certain biochemical compound following infection. According to Munde (1983) one *B. napus* strain resistant to blight, recorded higher total phenols, polyphenol and peroxidase (BO-54 strain), following inoculation with *A. brassicae.* The resistant strain showed higher accumulation of anthocyanins of the halo area surrounding the disease lesion and had scanty sporulation. Similar changes in the levels of total phenols, sugars, nitrogen and ascorbic acids etc., in rapeseed and mustard cultivars following infection with *A. brassicae* have been also correlated with their resistance or susceptibility to the disease by other workers (Chattopadhyay, 1989; Begum *et al.,* 1993; Gupta *et al.,* 1984; 1995).

The *A. brassicae* tolerant *B. juncea* cultivars RC-781, PHR-1 and KRV-tall have coloured leaves. The cv. Pusa Bold with normal leaves is susceptible to *A. brassicae.* In F_1 populations derived from non-coloured (cv. Pusa Bold) x coloured (cv. RC 781) parents, only the coloured individuals showed more

tolerance to infection (Bhowmik, 1985). This may be due to higher levels of total phenols, glucosinolates and sugars in leaves of RC 781 compared to that in susceptible cultivar, like Varuna (Gupta *et al.,* 1998; Garg *et al.,* 1999).

Resistance to *A. brassicae* in cv. RC 781 is governed by a single dominant gene (Tripathi *et al.,* 1980). Saharan and Kadian (1983[b]) reported high horizontal resistance in cultivars Tower and RC-781. Sharma and Singh (1992[b]) transferred resistance to *A. brassicae* in *B. juncea* through inter-specific hybridization and ovary culture technique on modified MS-medium. Hybrids of *B. juncea x B. hirta, B. juncea x B. napus* and *B. rapa* showed the resistant reaction to *A. brassicae* at the level of their respective male parents indicating the unique role of resistant parent in resistant breeding. Inter-specific transfer of *S. alba* resistance into *B. napus* was effected by manual pollination using reciprocal crosses and by somatic hybridization from protoplast fusion. Subsequently, embryo rescue and cytogentic analysis led to the generation of normal *B. napus* plants with resistance to *A. brassicae* similar to that of *S. alba* (Chevre *et al.,* 1991). Katiyar and Chamola (1994), on the other hand, transferred *A. brassicae* resistance from a *B. carinata* accession to the susceptible *B. juncea* cv. Varuna by interspecific hybridization and continuous selection. This led to the isolation of a fully fertile and productive genotype which was superior in resistance to all other commercial mustard varieties tested under natural incidence of *A. brassicae* in field. The feasibility of transferring disease resistance genes of wild *B. maurorum* Dur. to the cultivated *B. juncea* plants was reported by Bijral *et al*. (1995).

Cytoplasm also plays a role in conferring resistance to *A. brassicae* in *Brassica* spp. Alloplasmic lines of *B. juncea* having cytoplasm of either *B. napus* or *B. carinata* are comparatively more resistant to leaf blight under field conditions than euplasmic lines. On the other hand, *B. juncea* lines with cytoplasm of *B. rapa* (*B. campestris*) are more susceptible (Banga *et al.,* 1984). Synthetic *B. napus* developed by chromosome duplication of *B. campestris* x *B. oleracea* displayed resistance to both Alternaria and White rust disease (Prakash and Raut, 1983[a, b]). Mutants of *B. juncea* induced by exposing the cvs. Varuna, PR 5 and RS 3 to 60 kr gamma rays were resistant to both *A. brassicae* and *A. brassicicola* (Verma and Rai, 1980). Similarly, mutant recovered from M_2 populations of *B. juncea* cvs. Varuna and Kranti following mutations were resistant to Alternaria. However, in the absence of major genes for resistance to *A. brassicae* in crucifers, it will be prudent to concentrate dispersed minor genes techniques like diallel selective mating system besides attempting to incorporate non-host type of resistance in cultivated oilseed *Brassicas* (AICRP R-M, 1996). The latter is a difficult task but the use of conventional breeding methods, mutation; polyploidy and bio-techniques/genetic engineering may provide the desired result.

Screening of germplasm for resistance : Appropriate method of disease assessment is a prerequisite for identification of resistance to Alternaria blight in rapeseed-mustard. The disease appears on leaves and siliquae both, which is responsible for reduction of yield. Therefore, disease is assesed on leaves and siliquae separately by observations using disease rating scale.

Disease rating scale : Screening of genotypes/ germplasm lines for resistance to *A. brassicae* and other species is usually done using disease rating scales under natural field infection/and or artificial inoculation under favorable disease condition. For that Conn *et al.* (1990) developed an assessment key to asses the severity of (0 to 5 point scale) Alternaria blight in rapeseed and mustard. Presently, modified scale of Conn *et al.* (1990) is being used as 0 to 9 point scale by the scientists working under All India Coordinated Research Project on Rapeseed-Mustard (AICRP R-M, 2015) system in the country, which is described as 0=No lesion (Immune); 1= Non sporulating pinpoint size or small brown necrotic spots, less than 5% leaf area covered by the lesions (Highly resistant); 3= small, slightly sporulating larger brown necrotic spot, about 1-2 mm in diameter with a distinct margin or yellow halo, 5-10% leaf area covered by lesions (Resistant); 5= moderate sporulation, non-coalescing larger brown spots, about 2-4mm in diameter with a distinct margin or yellow halo, 11-25% leaf area covered by the lesions (Moderately resistant); 7=moderately sporulating, coalescing, larger brown spots about 4-5 mm in diameter, 26-50% leaf area covered by the lesions (Susceptible); 9 = profusely sporulating, rapidly coalescing, brown to black spots measuring more than 6mm in diameter without margins covering more than 50% leaf area (Highly susceptible). Different screening techniques are employed to produce maximum disease severity on *Brassica* species or cultivars under most favourable conditions using suitable disease rating scales to identify sources of resistance to *A. brassicae* are as follows :

Screening at the cotyledonary stage : Seeds are sown in small pots or trays filled with compost-mixed garden soil. The cotyledons of young seedling are pin-pricked with sterilized needle or pin on the seventh or eighth day of sowing. Using a 10 microlitre pipette, a 10 µl drop of inoculums of *A. brassicae* ($5x10^4$ conidia ml^{-1}), prepared from a freshly isolated culture of the pathogen, is placed over wounded cotyledons. Inoculated seedlings are transferred to growth chambers at 18°C, 80% relative humidity and 14 hrs light, and are rated for disease development after 2-4 days. Seedling reaction highly correlates with adult plant reaction. The technique is very useful for rapid screening of large populations at early stage without sacrificing the test plants (McNabb *et al.,* 1993; Bhowmik, 2003).

Screening with detached leaves : Fully expanded fourth leaf from 3-week-old plants of the test cultivars are placed on a small tray lined with moist paper towel. A small hole of about 3 mm diameter is made on either side of the mid-rib of each leaf with a sterile needle. A drop of conidial suspension made from young culture of *A. brassicae* in Rose Bengal (0.4 mgl^{-1}), is placed over each wound. The tray is sealed and then incubated at room temperature under continuous light for lesion development. Observations on lesion diameter and halo are taken on the fourth day following inoculation (Bansal *et al.,* 1990; Gugel *et al.,* 1990; Bhowmik, 2003).

***In-vitro* selection of pollen grains :** The effect of toxin destruxin B of *A. brassicae* is tested on *in-vitro* pollen germination and pollen-tube growth of *Brassica* species to ascertain their relative sensitivity to the toxin. The effects of toxin are also seen on leaves of the host species, and the degree of sensitivity of leaves of different species is comparable to that of their pollen grains. The results on the response of pollen grains and leaves to the toxin agree with the degree of susceptibility/resistance of these species to *A. brassicae* determined earlier in the field. The technique offers a simple and effective method of application of selection pressure to eliminate pollen grains susceptible to the toxin for effecting fertilization (Shivanna and Sawhney, 1993; Bhowmik, 2003).

Screening of young plants : Test plants are raised in small pots. A hole of about 4 mm diameter is made on either side of the mid-rib of the second leaf of each plant. Small agar disc containing *A. brassicae* is placed with the infected side against the upper side of the leaf over one hole, the other hole serves as control. The inoculated plants are transferred into moist chamber maintaining high humidity for symptom development. Disease reaction is rated by the size of the lesion developed (Grontoft and O'Connor, 1990; Bhowmik, 2003).

Another efficient method of evaluation is to raise plants in 25 cm pots, four in each and five pots for every test cultivar. Sixty-day-old plants are inoculated uniformly with equal amounts of inoculums (10 ml) of *A. brassicae* and then incubated in moist chamber for 48 hrs. The minimum and maximum temperature and relative humidity during the experimental period are maintained between 9-16°C and 80-100%, respectively. Data on the nature and size of lesions developed on the lower 4th to 6th leaves, and their sporulation are taken to assess the reaction of cultivars (Bhowmik and Munde, 1987; Bhowmik, 2003).

Screening of adult plants : Germplasm lines are exposed to heavy disease pressure under natural condition in the field. Sowing is usually done towards the third week of October under north India conditions in 3-5 m long rows, 30-40 cm apart with 10-15 cm space between plants depending upon the growth habit of the cultivar. A highly susceptible cultivar such as Yellow *sarson* cv. YST-151 (*Brassica compestris* var. Yellow *sarson*) is grown at regular intervals between the test cultivars and around the experimental area to serve as infector. The disease, which normally appears on the lower leaves towards the first of January, develops in severe epiphytotic form by mid-February, when the reaction of cultivars is rated (Bhowmik and Munde, 1987). To ensure high disease development, screening nursery is inoculated once or twice from a 10 day-old-cutture of *A. brassicae* grown in nutrient medium or from washings of *Alternaria* infected leaves (Tripathi *et al.,* 1980; Bhowmik, 2003).

Management

Alternaria blight of *Brassicas* can be managed by following a variety of practices delineated below :

Use of genotypes : Improved commercial varieties of rapeseed-mustard in India are resistant to this disease and genotypes reported highly tolerant to *A. brassicae* from other species under artificial conditions are: *Brassica (Sinapis) alba; B. hirta; B. napus* lines Lethbridge, Westar, WRG-1, Midas, HNS-3, UP 75-BN-5403, Target-84, Exotic-131 and *B. napus* (str. art.); *B. carinata* lines PC-3 and -5, BCON-1, CE-8 and -9, and PPSC-1; *B. tournefortii; B. nigra; B. oleracea var. alboglabra; B. fructiculosa; B. juncea* lines BC-115, CSR-448, DIRA-251, PHR-2, Zem-1 , RC-781, RH-8312, RS-104, RSK-21, RW-5453, B-2, K-41732; and *B. campestris* lines Tobin and PYS-3 (Hussain and Thakur, 1963; Rai *et al.,* 1976; Tripathi *et al.,* 1980; Munde and Bhowmik, 1985; Bhowmik and Munde, 1987; AICRP (R&M). 1990; 1996; Sharma and Singh, 1992[a]).

The cultivars Gulivar, Tower, GSL-1 and EC-126743 of *B. napus;* PC-5 and HC-1 of *B. carinata;* TM-7, TMV-2, YRT-3, KRV-Tall, PHR-1, Vardan, Saurabh, RC-1401, CSR-43, -142, -142-2, -343, -622 and -741, RH-8113 and -8114 and B-108(3) of *B. juncea;* and Torch, T-8 and -22, DYS-1 and Jata sarson of *B. campestris* have consistently been tolerant to *A. brassicae* under field conditions over years across the country (Kolte, 1987; Saharan and Chand, 1988; Dikshit and Srivastava, 1991; Kumar and Chauhan, 2005; Mehta, 2014). The different genotypes of *Brassica* spp. namely BAUSM-92-1-1, RC-376, PBC-9221, EC-399299, JMM-99-01, PBN-2002, PBN-2001, RH-9912, JMM-992, PBC-2002-2, NPC-14, PBC-2004-1, NPC-15, GSL-1, HNS-004, ONK-1, NUDB-26-11, OCN-13, CAN-133, RGN-55, NRCR-837, NPN-1, PBC-2002, PHR-2, EC-399299 remained consistently field tolerant in mid-eastern of India (Singh and Singh, 2005a; Singh *et al*., 2006; Singh *et al*., 2007; Singh *et al.,* 2008; Kumar and Singh, 2008; Kumar *et al.,* 2009; 2014; Singh *et al.,*2010; Singh *et al.,* 2015c; AICRP R-M, 2016).

The *B. napus* cvs. Midas and Tower in Canada (Tewari and Skoropad, 1976), cvs. Norde and Vestal in Poland (Rozej, 1974); *B. juncea* cv. Picra, *B. carinata* cv. Awassa, *B. nigra* cv. Junius and the *Sinapis alba* cv. Emergo in France (Brun *et al*. 1989) have been reported resistant. In China, the *S. alba* cv. J-10 has been reported to be immune to *A. brassicae* (Gong *et al.,* 1994).

Cultural : The field and its neighbourhood should be cleared of weeds before taking up sowing operations. Sound and bold seeds of recommended cultivars that are free from discoloured ones should only be sown. Usually, rapeseed and mustard sown by mid-October escapes the severe attack of Alternaria blight in north India (Singh *et al.,* 1998; Yadav *et al*., 2002; Prasad *et al*., 2003; Kumar and Kumar, 2006; Singh and Singh, 2006; Singh *et al.,* 2014[b]; Singh, 2016). In West Bengal, delay in sowing of Indian mustard by one month, beyond 22nd October, increased the incidence of *Alternaria brassicae* and *A. brassicicola* on leaves by 37 % and pods by 31% (Dasgupta, 1991). Most of the scientist have reported higher Alternaria blight severity on leaves in late sown (Nov. and Dec. sown) crops, but reverse in case of pod blight (Meena *et al.,* 2002, 2011a; Yadav, 2004; Singh *et al.,* 2014b), while Srivastava *et al.* (2005) reported relatively higher

Alternaria blight severity in early sown crop (Sept. last week to Oct. first week) than normal (15 to 30 Oct.) and late (Nov. 15 to Dec. 10) sown crop.

Use of recommended seed rate for sowing is also essential, as crop density per unit area directly affects the disease incidence and yield. The effect of plant density on disease intensity and crop yield showed that an increase in plant population through reduction in both inter-and-intra row distances, increases the disease intensity, reduces the per plant yield but increases the total per plot yield because of higher number of plants per unit area. Sowing of yellow sarson cv. YS-8 at 30 x 10 cm inter- and-intra row spacing gave the highest per plot yield over other spacing combinations, although this resulted in lower per plant yield (Munde, 1983).

High fertilizer doses, especially of nitrogen, increase the severity of Alternaria blight in oilseed rape (Stankova, 1972; Kumar and Kumar, 2006) but Meena *et al.* (2002) reported decrease in severity with increased the level of nitrogen. In both pot and field experiments at Pantnagar, Uttarakhand, rape plants (*B. campestris*) fertilized with potash (40 kg K_2O ha^{-1}) were resistant to lodging and produced seeds with low infection, increased weight and germinability. Application of nitrogen and potash (90 kg Nha^{-1} + 40 kg K_2O ha^{-1}) decreased the disease severity and consistently increased the seed yield 68% more than those of control and other treatments (Sharma and Kolte, 1994; Godika *et al*., 2001). Significant reduction in disease severity and highest yield of mustard were obtained under field conditions at Faizabad, UP when NPK were used at 100:40:40 $kgha^{-1}$ along with three sprays of Indofil M-45 (Singh and Chauhan, 1997). In general, blanced fertilizer dose (N: P: K: 80:40:40) coupled with prophylactic sprays of fungicide (Mancozeb) provide maximum increase in yield. Recommended dose of NPK (90:60:40 Kg ha^{-1}) along with 40 Kg sulphur ha^{-1} gave maximum control of Alternaria blight (Kumar and Kumar, 2006). Soil application of minerals like sulphur, borax, potash and zinc were found effective in management of Alternaria blight of mustard at different places (Meena *et al*., 2011; Singh *et al.,*2014b).

Chemical : With the steady supply of an array of highly effective and newer broad spectrum fungicides over the past decades, the use of chemicals has become the most important component of disease management strategy in rapeseed and mustard.

Seed treatments with brassicol or captafol @ 4 g (Chahal and Sekhon, 1980; Chahal, 1982; Saini, 1982), iprodione @ 1.25g (Stovold *et al.* 1987) and carbendazim @ 2.5 g or mancozeb @ 2 gkg^{-1} of seed eradicate the seed borne infection of *A. brassicae,* though 24 hr storage period after their treatment is needed (Kumar and Singh, 1986). Seed treatment ensures emergence of healthy seedlings in temperate countries such as in UK (Evans *et al.,* 1984), but under our sub-tropical climate the seed born inoculums of *A. brassicae* gets eliminated during storage in summer months, thus avoiding need of seed treatment.

Spray application of copper fungicides reduces the incidence of Alternaria blight of rapeseed and mustard. A minimum of four sprays with 0.5% bordaux mixture is required to reduce the disease incidence and increase the crop yield significantly. Losses in seed yield were negligible when the disease intensity on pods was reduced to 10.7% with six sprays of Bordaux mixture (Chahal and Kang, 1979[a]). Copper oxychloride, cupravit, blitox or fytolan are also effective but phytotoxic (Singh and Bhowmik, 1985; Howlider *et al.* 1985; Shivpuri *et al*. 1988; Mridula *et al.* 1994). Chahal (1986[b]) reported the best cost/benefit ratio with copper oxychloride sprays, four times starting when the crop was 75 days old.

Among the more popular chemicals, spraying with difolatan (captafol); dithane-M 45, dithane- Z 78 and cuman L (dithiocarbamates); bavistin (carbendazim); panolil (guazatin); baycor (bittertanol); syllit (dodecyl guanidine acetate); daconil (chlorothalonil); wettable sulphur; dueter (fentin hydroxxide); fentin acetate; Topsin-M (thiophanate-methyl) and TPTH (triphenyl tin-hydroxide) @ 0.2-0.25% provide effective control of the disease and result in significant increase in crop yield (Chahal and Sekhon, 1980; Kaushik *et al.,* 1983; Singh and Bhowmik, 1985; Chahal, 1986b; Kolte *et al.,* 1987; Tripathi *et al.,* 1987; Dasgupta, 1991; Chattopadhyay and Bagchi, 1994; Mridula *et al.,* 1994; Godika *et al.,* 2001; Singh and Singh, 2005b; Singh and Singh 2006; Khan *et al.*, 2007b; Singh *et al.,* 2013a, b; Srivastava *et al.,* 2014; Singh *et al*.,2014b; Singh *et al.,* 2015c).

In a comparative study on the persistence and efficacy of fungicides against *A. brassicae* on Indian mustard cv. Pusa Bold during 1978-80 crop seasons, Singh and Bhowmik (1985) found that fungicides which are more toxic are also more persistent on the host surface and that the rate of decline in their toxicity is more rapid in the beginning than later. In their trial, out of eight fungicides tested, difolatan was the most persistent and effective in both reducing the disease intensity and increasing seed yield of plants. Dithane-M 45 was the next best. High efficacy of these two chemicals has also been confirmed from other field trials (Kaushik *et al.,* 1983; Gupta *et al.,* 1985; Chattopadhyay and Bagchi, 1994).

Iprodione is also highly effective. In a field trial during 1986-87 at Jaipur, India the efficacy of iprodione (rovral) against *A. brassicae* was compared with five other fungicides on Indian mustard cv. Varuna. All the chemicals controlled the disease but iprodione (0.2%) was the best, though captafol, mancozeb (diathane-M 45), indofil M-45 (mancozeb + thiophenate methyl) and ridomil MZ (mancozeb + metalaxyl) were also highly effective. Iprodione caused minimum defoliation (Kumar, 1996; Shivpuri *et al.,* 1988) and increased the crop yield up to 20.2 and 10.0% as compare to check. Seed oil content in iprodione treated crop was 39.37% as compared to 36.75% in check crop (Brar and Chahal, 1993). In spray trials at different locations in the country during 1994 to 96 seasons against Alternaria blight, iprodione (rovral 50 WP) @ 0.2% was 15 to 60% more effective over mancozeb and resulted in corresponding yield increase of 6 to 35% (Chattopadhyay and Bhunia, 2003, Murkherjee *et al.* 2003;

Singh and Bhajan, 2004 and Singh and Singh, 2005[b]). The amount of residues of iprodione in edible commodity (seeds) was less than the recommended MRL (0.5 mgkg^{-1}) safety level [AICRP (R&M), 1995, 1996; Chatterjee *et al.,* 1997]. Singh and Maheshwari (2003) reported Cantaf as most effective fungicide for the control of disease on leaves on pod, while Singh and Singh (2007[a]) found Mancozeb treatment was best against Alternaria blight of *Brassica compestris* var. yellow *sarson*. Iprobenfos @ 0.1% and Propiconazole @0.05-0.1% were also found effective against Alternaria blight in case of Indian mustard under field condition at Kumarganj, Faizabad (Kumar *et al.,* 2009)

Spray operations with fungicides should be taken up with the first appearance of disease spots and then repeated two to three times at 10 to 15 days intervals depending upon the crop variety, its growth stage and the severity of infection (Chahal, 1986[b]; Chattopadhyay and Bagchi, 1994; Gupta *et al.,* 1985; Kushik *et al.,* 1984; Tripathi *et al.,* 1987). Post-flowering application of fungicides on pods is most important. It protects them from infection, increases seed yield and improves germination of harvested seeds (Chahal and Kang, 1979[a]; Humpherson-Jones and Maude, 1982; Ogilvy, 1984). Some resistance inducing chemicals viz., Benzoic acid, Napthelic acid, acetic acid, Salicylic acid, Phosphoric acid, Isonicotinic acid were identified effective for the management of this disease under field condition (Atwal and Sangha, 2004; Sanjula and Sohal, 2010; Singh *et al.,* 2014c).

Integrated control of Alternaria blight and aphid (*Lipaphis erysimi*), the most serious insect pest of rapeseed and mustard, was achieved by treating the crop with fungicide (diathane M-45, difoltan, deconil 2787 and blitox) and insecticide (metasystox, rogor and dimecrone) mixture. Three application of dithane M-45 (0.2%) + metasystox (0.03%) mixture on Indian mustard cv. Pusa bold starting from 15th January at fortnightly intervals gave a low intensity of both disease and aphid infestation and the highest seed yield amongst the various spray treatments (Munde, 1983; Munde and Bhowmik, 1984; Tripathi *et al.,* 1987).

Biological : Indiscriminate use of pesticide has lead to the problems like pest resistance, pest resurgence and environment pollution. Till date, chemical management was the only option against the problem. However, some reports indicate possibility of biological management of the disease. *Streptomyces arabicus* is a natural enemy of *A. brassicae.* Fistupyrone, metabolites of *Streptomyces* sp. strain TP-A0569 inhibit spore germination and infection of *A. brassicicola* (Aremu *et al.*, 2003). The effect is specific for *A. brassicicola* other *Alternaria* spp. is not effective. Phyllosphere residents *Aureobasidium pullulans* and *Epicoccum nigrum* reduced the infection by *A. brassicicola,* especially when they were inoculated 14 hr before the pathogen (Pace and Campbell, 1974). Spray of soil isolates of *Trichoderma viride and Pseudomonas fluorescencs* at 45 and 75 days after sowing could manage Alternaria blight of Indian mustard (*Brassica juncea*) as effectively as mancozeb (Meena *et al.,* 2004, 2008, 2011; Patni *et al.,* 2005;). Botanicals viz., bulb extract of *Allium sativum* has been reported to effectively manage Alternaria blight of Indian mustard (Meena *et al*., 2004; Patni and Kolte, 2006; Nigam *et al*., 2011; Singh, and Singh, 2016). Application of aqueous solution of garlic bulb either alone or in combination with *Trichoderma* spp. and fungicides like mancozeb was found effective in controlling the disease severity at different places (Meena *et al.,* 2004, Singh and Singh, 2007; Singh *et al.,* 2008; Meena *et al.,* 2008; Singh *et al.,* 2013a,b). Foliar spray of aqueous bulb extract of *Allium sativum* (garlic) and *Eucalyptus globosus* (Eucalyptus) have been reported to effectively manage the Alternaria blight on leaves and pods and could be eco-friendly substitute for chemical fungicide mancozeb in the management of mustard diseases (Kumar *et al.,* 2006; Meena *et al.,* 2008, 2011; Yadav, 2009)

Community health : To make the process of disease management more efficient, economical and eco-friendly, crop protection programme planning by the community of farmers should be done collectively. If some of the farmers are following long term rotations, while their neighbours grow rapeseed-mustard every year, their efforts will yield poor results as their rapeseed-mustard will have easy availability of primary inoculum from the neighbouring fields.

Future thrusts : The following issues need to be effectively explore further against Alternaria blight in rapeseed-mustard : To establish relationship between pathogenic and molecular variability among *Alternaria* isolates apart from relation with morphological and cultural data for the same; Molecular diagnostics of *Alternaria* taxonomy; Through differential hosts sequential data, type of cultures need to study; Alternate host of *Alternaria* species and sub species infecting other host plant besides *Brassica* species need to be investigated; Detailed study is required for various *Alternaria* spp. complexes affecting various vegetables/*Brassica* species; Cryvo-preservation/liophilisation of various *Alternaria* cultures and its maintenance; Epidemiology based eco-friendly and sociologically acceptable management of disease; To find out resistant doners for resistant to different races of *Alternaria* species causing blight disease in rapeseed-mustard.

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