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***Review Paper***

# MINOR PATHOGENS: A WORLDWIDE CHALLENGE TO CULTIVATION OF CRUCIFERS

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## ABSTRACT

**The *Brassicas* encounter several well known pathogens like *Albugo, Alternaria, Hyaloperonospora, Erysiphe, Leptosphaeria, Plasmodiophora,* and *Sclerotinia* causing White rust, Alternaria blight, Downy mildew, Powdery mildew, Black leg or phoma root rot and white stem rot, respectively. In addition to above, several minor pathogens such as anthracnose [*Colletotrichum higginsianum* Sacc. *C. capsici*], light leaf spot [*Pyrenopeziza brassicae*], stem and root rot [*Rhizoctonia solani* Kühn], wilt [*Verticillium longisporum* (ex. *V. dahliae* var. *longisporum* Stark; comb. nov. Karapapa)], Fusarium wilt [*Fusarium oxysporum* f. sp. *conglutinans*; *F. equisiti*], black rot [*Xanthomonas campestris* pv. *campestris* (Pam.) Dowson (Xcc)], and mosaic (*Turnip mosaic virus*: TuMV) are now becoming most widespread and devastating diseases worldwide. Under changing climate situations, the disease scenario is shifting and the minor pathogens are becoming major pathogens and causing considerable yield losses, which range from 10-80 per cent. The information available on various minor pathogens on their host range, distribution, yield losses, symptomatology, epidemiology and effective disease management is presented in this review for the benefit of both researchers and growers of cruciferous crops.**

**Keywords:** Anthracnose, Brassica, Climate change, Fusarium, Plant disease



Brassicaceae (Syn. Crucifererae), the family currently comprising 338 genera (includes 3740 species with 75 accepted species) is one of the ten most economically important plant families widely used as edible oil vegetables. Diseases caused by various pathogens remain barriers to achieve higher productivity of oilseed *Brassica*. Brassicaceous crops are continuously challenged by key diseases including Sclerotinia rot (*Sclerotinia sclerotiorum* (Lib.) de Bary), White rust (*Albugo candida* (Lev.) Kuntze), Downy mildew (*Hyaloperonospora parasitica* Constant. (Pers.: Fr) Fr.), Alternaria blight (*Alternaria brassicae* (Berk.) Sacc./*A. brassicicola* (Schwein.) Wiltshire), Powdery mildew (*Erysiphe cruciferarum* Opiz. L. Junell.), Clubroot (*Plasmodiophora brassicae* Woronin), and Blackleg (*Leptosphaeria maculans* (Desm.) Ces.), responsible for causing considerable losses as influenced by environmental factors all over the world. These crops are grown in tropical as well as in temperate zones and prefer cool moist weather during growing period and dry weather during harvesting. The top five major countries viz., Canada, China, India, France, and Germany are rapeseed-mustard producing countries worldwide. Eight species and sub-species of oilseed *Brassicas* are cultivated in India under the name rapeseed-mustard comprising Indian mustard (*Brassica juncea* (L.) Czern & Coss.; 2n=36; AABB)), Toria (*B. rapa* L. var. Toria), Yellow Sarson (*B. rapa* L. var. Yellow Sarson), Brown Sarson (*B. rapa* L. var. Brown Sarson), Gobhi Sarson (*B. napus* L.; 2n=38: AACC), Karan rai (*B. carinata* A. Braun; 2n=34: BBCC), Black mustard (*B. nigra*; 2n=16: BB) and Taramira (*Eruca sativa/ vesicaria* Mill.) which are also cultivated in about 53 countries. Based on cytogenetic studies, there are three digenomic species, *B. carinata* (2n=34: BBCC), *B. juncea* (2n=36; AABB) and *B. napus* (2n=38: AACC), have originated through crosses between any two of the three basic species *B. nigra* (2n=16: BB), *B. oleracea* (2n=18: CC) and *B. rapa* (2n=20: AA) (Kumar *et al.,* 2015). Rapeseed-mustard production trends all over the world showed the substantial growth in production of 59.8 mt from 32.1 mha area with 1865 kg $ha^{-1}$ productivity during 2009-10 which enhanced to 72.8 mt from 36.5 mha with 1993 kg $ha^{-1}$ during 2017-18 (Kumar *et al.,* 2019).

Under natural conditions, cruciferous plants remain confronted by 167 biotic (44 pathogen, 87 pests, and 36 weeds) and 19 abiotic stresses. Out of 44 pathogens known to infect crucifers, 16 pathogens have received attention for research activities on rapeseed–mustard. However, about 13 pathogens which have so far been considered as minor ones, are now gaining importance in current crucifers' cultivation scenario. In this review, we have compiled the scientific information available worldwide on these minor pathogens causing diseases in crucifers on the various aspects like economic importance, symptomatology, host range and distribution, epidemiology, pathogenic diversity and

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disease management ,etc. as per their availability in respective diseases, since very meagre work has been reported in literature.

Diseases caused by various minor pathogens also remain an impediment to attain superior productivity of *Brassica* crops. The physical losses arise from the loss of production arising from the incidence of disease. The financial loss is on account of both reduced production and lower value of the produce due to reduction in quality of the produce. A proper prioritization of plant diseases in relation to the magnitude of the economic losses is utmost necessary to make economically effective, feasible mitigation over large areas in rapeseed-mustard. The losses caused by various minor pathogens in rapeseed-mustard are presented in Table 1

## 1. *Colletotrichum higginsianum* Sacc. (Anthracnose)

The hemibiotrophic ascomycete fungus *Colletotrichum higginsianum* Sacc. belongs to the family Glomerellaceae. It causes anthracnose disease on many plants in the Brassicaceae family. *Colletotrichum* spp. cause anthracnose diseases that typically appear as small or large dark-colored spots or slightly sunken lesions on the foliage, stems, or fruit of a wide range of important crop and ornamental plants (Agrios, 1988). The pathogen is responsible for typical sunken symptoms on leaves, stems and petioles of rapeseed-mustard. In natural environments, microorganisms normally produce toxic chemicals to compete with other organisms and are capable of tolerating toxicity of these natural chemicals.

### *Economic importance*

*Colletotrichum higginsianum* has been reported to cause typical anthracnose lesions on the leaves, petioles, and stems of turnip, mustard, and Chinese cabbage (Higgins 1917). In South China, this fungus usually causes typical water-soaked lesions on leaves of Chinese cabbage (*B. parachinensis*), leading to 30–40% yield loss yearly (Yang *et al.,* 2008).

To invade host tissue, conidia first attach to plant surfaces and germinate to form melanized appressoria. After that, *C. higginsianum* penetrates the plant cell with high turgor pressure generated in the melanized appressorium, and then large bulbous biotrophic



**Table 1. Pathogens of minor diseases caused yield losses in crucifers**

| Sr. No. | Disease | Causal organism | Yield losses (%) | References |
|---|---|---|---|---|
| 1 | Anthracnose | *Colletotrichum higginsianum / C. capsici* | 30-40 | Yang *et al.* (2008) |
| 2 | Light leaf spot | *Pyrenopeziza brassicae* | 9–15 | Teng *et al.* (1984) |
| 3 | Damping-off of seedling, leaf blight, root and collar rot of mature plant | *Rhizoctonia solani* Kuhn | 30 | Tahvonen *et al.* (1984) |
| 4 | Verticillium wilt | *Verticillium longisporum* | 10-50 | Dunker *et al.* (2008) |
| 5 | Fusarium wilt | *Fusarium oxysporum* f. sp. *conglutinans / Fusarium equiseti* (Corda) Sacc. | 30 | Lange *et al.* (2007) |
| 6 | White leaf spot | *Neopseudocercosporella capsellae* (Ellis & Everhart) Videira & Crous | 30 | Penaud (1987) |
| 7 | Stem Blight | *Nigrospora oryzae* (Berk. & Broome) Petch | NA | Sharma *et al.* (2013) |
| 8 | Root rot | *Sclerotium rolfsii* Sacc. | 80 | Meena *et al.* (2013) |
| 9 | Black rot | *Xanthomonas campestris* pv. *campestris* (Pam.) Dowson (Xcc) | 50-65 | Williams (1980); Dhar and Singh (2014) |
| 10 | Bacterial root rot | *Erwinia carotovora* pv. *carotovora* (Jones) Bergy | 87-96 | Sobti (1983) |
| 11 | Mosaic | *Turnip mosaic virus* (TuMV) | 30-90 | Wei *et al.* (1960); Shattuck and Stobbs (1987); Hardwick *et al.* (1994); Spence *et al.* (2007) |
| 12. | Phyllody | *Phytoplasmas* | NA | |
| 13. | Aster yellows | *Phytoplasmas* | NA | |

NA- No information available

hyphae form in the first infected cell. Finally, the fungus differentiates secondary hyphae to kill host tissues (Narusaka *et al.,* 2004).

The pathogen uses a two-stage hemibiotrophic infection process, initially growing biotrophically inside a living host cell before switching to a destructive and invasive necrotrophic phase. The first step in the establishment of infection is the adhesion of spore to the surface of the plant. After connection to the plant surface, conidia germinate to form dome-shaped, melanized appressoria that pierce the cuticle and cell wall directly, by means of a narrow penetration peg. Swollen primary hyphae then invade living epidermal cells and invaginate the host plasma membrane, like the haustoria of obligate biotrophs. Both the biotrophic phase and the specialized primary hyphae of *C. higginsianum* are entirely restricted to the first infected epidermal cell, in contrast to other hemibiotrophic *Colletotrichum* species (O'Connell *et al.,* 2004). Thereafter, the fungus switches to a necrotrophic mode of growth, associated with production of thin secondary hyphae which ramify within and between host cells and kill host cells ahead of infection. The asexual stage is completed by the production of acervuli on the surface of the dead host tissue. Acervuli consist of a mass of short conidiophores interspersed with long hair-like setae, which have darkly melanized cell walls like the appressoria.

## Host range

The disease has been reported on a wide range of cruciferous plants, including agronomically important species, such as members of genera *Brassica* and *Raphanus* as well as the model plant *Arabidopsis thaliana* (Narusaka *et al.,* 2004; O'Connell *et al.,* 2004), on Arugula (*Eruca sativa*) in Florida (Patel *et al.,*2014), on *Boehmeria nivea* (Wang *et al.,* 2011) and *Rumex acetosa* (Zhang *et al.,* 2018) in China. *Colletotrichum higginsianum*, with straight and long conidia, has been considered as the causal agent of anthracnose of crucifers including Chinese flowering cabbage (Scheffer ,1950; Horie *et al.,* 1988; Zhang *et al.,* 1998, 2000; Zhou *et al.,* 2002; Damm *et al.,* 2014). Mahmodi *et al.* (2013) reported that *C. capsici* also could cause anthracnose on Pak Choi (*B. chinensis* Kitam), another member of Cruciferae family. The *Arabidopsis-Colletotrichum* pathosystem provides an integrated system, with extensive information on the host plant and availability of genomes for both partners, to illustrate many of the important concepts governing fungal–plant interactions, and to serve as an excellent starting point for broad perspectives into issues in plant pathology (Yan *et al.,* 2018).

## 2. *Pyrenopeziza brassicae* (Light leaf spot)

Light leaf spot (LLS) disease of Brassicas, caused by the hemibiotrophic fungal pathogen *Pyrenopeziza brassicae* (anamorph *Cylindrosporium concentricum*), is known as one of the most damaging diseases of winter oilseed rape (*B. napus*) in Northern Europe (Boys *et al.,* 2007). In Ireland, *P. brassicae* was observed for the first time during the 1964-1965 season, causing light leaf spot disease on broccoli, cabbage and Brussels sprouts (Staunton, 1967). The pathogen produces large apothecia (1–2 mm in diameter) which take at least 3 weeks to develop on leaf petioles after infected leaves die, but small apothecia (50–200 um) may form on leaf lamellae after about 15 days. Ascospores can be released for up to 5 days after rain. It has been observed that apothecia can continue to release ascospores for up to 3 weeks even when they are subjected to wet and dry cycles.

Several severe epidemics have been reported in winter oilseed rape in the UK since the first major epidemic recorded in 1974 (Simons and Skidmore, 1988). In France, the disease was first reported in 1978 and there were severe epidemics in the 1980s and 2000s (Pilet *et al.,* 1998; Karolewski *et al.,* 2006). In Germany, occurrence of light leaf spot was widespread in the late 1980s (Pilet *et al.,* 1998). Light leaf spot also occurs in Poland, with severe damage during mild winters (Karolewski, 1999; Koike *et al.,* 2007). *Pyrenopeziza brassicae* is also prevalent on *Brassicas* in the wet, cool climate of New Zealand, with severe outbreaks of light leaf spot reported on vegetable Brassicas (Cheah *et al.,* 1980; Vegetables New Zealand 2016).

## Economic importance

It has been estimated that pre-harvest pathogens cause yield losses of 9–15% in crop production each year. The losses can be a much greater in percentage of seed yield for certain crops (Teng *et al.,* 1984; Oerke, 2006).

## Host range

Light leaf spot also occurs on different types of *B. oleracea* and other related *Brassica* species or subspecies. These include Brussels sprouts (*B. oleracea* var. *gemmifera*), cabbage (*B. oleracea* var. *capitata*), cauliflower (*B. oleracea* var. *botrytis*), broccoli (*B. oleracea* var. *italica*), kale (*B. oleracea* var. *acephala*), turnip (*B. rapa* ssp. *rapa*), swede (*B. rapa* ssp. *rapifera*), Chinese cabbage (*B. rapa* ssp. *pekinensis*) and black mustard (*B. nigra*) (Maddock and Ingram, 1981; Simons and Skidmore, 1988; Boys, 2009; Karolewski, 2010). Spread of light leaf spot between different *Brassica* host species has been suggested (Wafford *et al*., 1986) but

there has been little work on this disease.

### *Epidemiology*

Light leaf spot epidemics are usually initiated by airborne ascospores of *P. brassicae*, which are forcibly released from apothecia (cup-shaped fruiting bodies) produced on diseased plant stem, pod or leaf debris (Gilles *et al.,* 2001a, b). Ascospores produced on crop debris at other times may be important in initiating epidemics on crops of vegetable Brassicas or secondary epidemics on oilseed rape (McCartney and Lacey, 1990; Gilles *et al.,* 2000, 2001b; Karolewski *et al.,* 2012). These airborne ascospores may be taken into consideration as a means of forecasting risk of severe light leaf spot epidemics. Light leaf spot epidemics are favoured by wet weather, which encourages production and dispersal of conidia.

## 3. *Rhizoctonia solani* Kuhn (Damping off, collar rot)

*Rhizoctonia solani* Kühn (teleomorph = *Thanatephorus cucumeris* Donk) is a ubiquitous soil-borne plant pathogenic fungus belongs to the order Agonomycetales, family Agonomycetaceae (*Mycelia sterilia*) in which fungi lack even the perfect state and reproduce by the fragmentation of the mycelium. It is plurivorous fungus causing charcoal rot, damping off of various crop plants. *Rhizoctonia solani* is associated with stem and root rot diseases in dicotyledonous crop species belonging to Brassicaceae (Gugel *et al.,* 1987; Sneh *et al.,* 1991; Tewoldemedhin *et al.*, 2006). Colonies on potato dextrose agar (PDA) were light brown to brown, with concentric zones of oppressed and aerial mycelium. Sclerotia were produced in various sizes and numbers, and were distributed in the zones of aerial mycelia. Infection cushions are formed on both susceptible and resistant hypocotyls, but are more frequent and form earlier on susceptible than on resistant hypocotyls. In culture, sclerotia are most common. The fungus is facultative necrotrophs. Mycelium is superficial or immersed, hyaline to brown, branched, septate, often tree like in form. Pycnidia are separate, globose, dark brown, immersed within one cavity, thick walled, erumpent, 100–200 µm in diameter. Conidiophores are absent, conidiogenous cells, enteroblastic, phialidic, determinate, cylindrical, multicellular, 5–13 x 4–6 µm. Conidia are hyaline, aseptate, straight cylindrical to fusiform, thin walled, smooth of 14–30 x 5–10 µm. Sclerotia are brown to dark brown, smooth hard, formed of brown thick walled cells. The basic characters of the genus are formation of sclerotia. Sclerotia are dark brown to black, smooth, hard, and are formed of thick walled cells of irregular size but of uniform texture. The cells of the hyphae are barrel shaped, anatomizing frequently, branching more or less at right angles and pale brown to brown colour. Perfect stages are known as *Thanatephorus* or *Ceratobasidium* and belong to basidiomycotina. *R. bataticola* also produces imperfect state (pycnidial state) i.e., *Macrophomina phaseolina* belonging to Sphaeropsidales. In case of perfect state of *Rhizoctonia solani (Thanatephorus cucumaris)* pycnidia are formed which are separate, globose, dark brown, immersed within one cavity, thick walled, erumpent, 100 – 200 µm in diameter.

### Host range and Distribution

The fungi generally grouped as *Rhizoctonia* types occur in all parts of the world and are probably indigenous to uncultivated areas and multiply and spread through agricultural operations. The host plants infected by *Thanatephorus cucumeris* reported in Japan alone belong to 35 orders, 52 families, 125 genera and comprise more than 142 species from the Cycadopsida to the Monocotyledons. .

*R. solani* and binucleate *Rhizoctonia* spp. anastomosis groups (AGs) have been identified in relation to many diseases. Isolates of certain AGs were found to infect many host plants, while those of other AGs have a rather limited host ranges. AG 1 and its subgroups infect many different crops. Anastomosis groups of *R. solani,* binucleate *Rhizoctonia* spp. or *Waitea* species have been reported in 47 countries or districts; but more likely at least some AGs are present in every country. Distribution of anastomosis groups depends for pathogenic groups on the host plant distribution and on the climate and vegetation types for saprophytic groups. However, they are basically cosmopolitans, since they have a relatively wide host range and a flexible capability for adaptation.

*Rhizoctonia* spp. has been extensively studied, due to the introduction of new techniques, research and new ideas, the following aspects have received special attention and continued to do basic research. As opposed to other groups, isolates of AGs 5, 6, 7, 9, 10 and BI of *R. solani* have been reported to be weakly pathogenic or not pathogenic. Isolates of AG 7 have been reported as a severe pathogen on carnation (Lo *et al.,* 1990) in Taiwan and on soybean in Indonesia (Naito *et al.,* 1991). Therefore, the possibility that isolates of AGs 6, 9, 10 and BI are severe pathogens on some plants under certain environmental conditions should not be overruled.

Genetic studies regarding the pathogenicity of *Rhizoctonia* species and host-parasite relationships were relatively scarce compared with other pathogens. *R. solani* is basically multinucleate, and its hyphae are primarily heterokaryotic. Multinucleate heterokaryotic cells are less suitable for genetic studies. Therefore,

the use of uninucleate basidiospores as inoculum and consecutive infection on host plants, have enabled researchers to obtain more useful information of the genetic basis of pathogenicity, similar to genetic studies performed with airborne pathogens. Transformations of isolated genes from one isolate to the other can be expected to contribute to the understanding of the genetics of pathogenicity and physiology of *Rhizoctonia* spp.

It would be interesting to find out the destiny of fused protoplasts obtained from isolates of different AGs or different subgroups. Protoplasts obtained from isolates of different *R. solani* AGs are capable of fusing with each other. Can these fused protoplasts survive and establish new strains? What characteristics are displayed by isolates originating from these fused protoplasts? Such investigations today have elucidated characteristics and relationships of AGs and subgroups. In Australia, AG 8 has been divided into 5 groups according to the zymogram patterns of the isolates (Mac Nish *et al*., I993*).* It is quite possible that additional groups will be found in AG 8 in future. Cultural appearances are diverse among isolates from USA, Australia, and Scotland and may deserve special attention in this respect. AG 8 is known as a bridging group. Isolates of this group were found to be capable to fuse with isolates of AGs 2-1, 2-2 and BI (Rovira *et al.,* 1986). The bridging behavior has not been sufficiently studied. In South Africa, the "Crater rot" disease of wheat has been known for a long time (Stott *et al.,* 1979). It should be determined to which AG the isolates causing this disease, belong. Researchers in South Africa claim that this pathogen is *R. solani,* but the isolates do not fuse with any representative tester isolates of the known AGs (Lubbe and Meyer, 1995).

International distribution of *Rhizoctonia* isolates is basically not acceptable for quarantine reasons. Over the years, Ogashi (1996) sent isolates to 34 countries, 77 locations and 85 researchers. In a sense this is dangerous with regard to dissemination and infestation of the pathogens. It is suggested to establish a system, where at least one *Rhizoctonia* researcher in every country, district or continent will assume the responsibility to maintain and distribute *Rhizoctonia* spp. isolates. The key researchers should keep isolates of *R. solani,* binucleate *Rhizoctonia* spp. and *Waitea* AGs from their own countries or districts and determine their AGs with the known authentic isolates carefully. The local isolates could then be made available to researchers in the country, district or continent as tester strains in the country, district or continent.

The wide host range of *Rhizoctonia* spp., which includes roots of volunteer plants, grassy weeds and rotation crops, allows this pathogen to survive in the soil from season to season (Smiley *et al.,* 1992). *Rhizoctonia solani* AG2-1 is the principal pathogen causing damping-off and seedling and mature plant root rot (brown girdling root rot) in oilseed rape and canola (*Brassica napus* and *B. rapa*) in western Canada and the United States (Verma, 1996). Several brassicaceous weeds, including bail mustard [Nesliapaniculatad.) Desv.], stinkweed (*Thlaspi arvense* L) and shepherd's purse [*Capsella bursa-pastoris* (L.) Medic], also are affected by the brown root rot caused by *R. solani*. Besides root and collar rot, *R. solani* also causes a severe leaf blight of oilseed rape and mustard in India (Chauhan and Saksena, 1974). In Finland, damping-off due to *R. solani* occurred in 90% of spring rape (*B. napus* and *B. rapa*) fields surveyed during 1981-82 (Tahvonen *et al.,* 1984).

## Economic importance

The predominant population of *R. solani* causing severe seedling disease associated with establishment losses of up to 80–100% and final yield loss of up to 30% in oilseed rape (OSR, *Brassica napus*). The pathogen belongs to AG2-1 throughout world (Tahvonen *et al.,* 1984; Kataria and Verma, 1992; Khangura *et al.,* 1999).

## Epidemiology

Incidence and severity of pre- and post-emergence damping-off and root rot in rapeseed fields is influenced by weather and soil conditions and by inoculum density of the anastomosis groups of *R. solani* in the soil. Teo *et al.* (1988) showed that early planting of rapeseed, before the soil had warmed to 15°C, was more favourable to damping-off by AG 2-1 than AG 4, while damping-off due to AG 4 increased as the soil warmed to 20°C. Temperatures in the range of 18-30°C support luxuriant growth of both AGs, and AG 4 isolates have a higher growth rate than AG 2-1 isolates at these temperatures (Verma ,1996).

## Pathogenic variability

From the foregoing sampling of the literature on variation, it is clear that *R. solani* is a highly variable fungus and that any satisfactory species concept must accommodate this variability. It is also clear that our present state of knowledge does not permit the formulation of precise species concept, since we yet do not know the entire range of variability in the species.

No single feature, excepting the perfect state serves to distinguish *R. solani* from other similar fungi. Recognition of the species depends rather on the presence of a combination of several features. Mycelia possessing the usual rapid growth, branching habit, colour, septation, nuclear condition, monilioid cells, sclerotia and pathogenic habit of *R. solani* can be assigned to that species with confidence. Mycelia

in which one or more of these features are wanting or vary from the usual are more difficult to place. Available evidence indicates that the mycelium of *R. solani* invariably possesses multinucleate cells, branching near the distal septum of a cell, constriction of the branch arid formation of a septum near the point of branch origin, and a prominent septal pore apparatus in the septum. Mycelia lacking any of these features cannot be assigned to *R. solani.* Monilioid cells, sclerotia, rapid growth, virulence, or similar variable features may be lacking in some isolates of *R. solani* and cannot be considered essential to the placement of a mycelium in R. *solani.* These features are usually present in field isolates, however, and care should be exercised in including in *R. solani* fungi in which one or more of these features are absent. As has been suggested (Parmeter, 1970), it is difficult if not impossible to describe a mycelium with absolute assurance that another worker can identify that mycelium from the written description. It is possible only to provide a description with sufficient detail as to exclude most other mycelia. The following summary of characteristics for *R. solani* serves to delineate the species with as much accuracy as present information permits.

Characteristics consistently present; multinucleate cells in young vegetative hyphae; prominent septal pore apparatus; branching near the distal septum of cells in young vegetative hyphae; Constriction of the branch and formation of a septum in the branch near the point of origin; Some shade of brown. Characteristics usually present, but one or more of which may occasionally be lacking in individual isolates (usually single-spore isolates); Monilioid cells; Sclerotia (without differentiated rind and medulla); Hyphae greater than 5 µ in diameter; Rapid growth rate; and Pathogenicity. The characteristics never possessed; Clamp connections; Conidia; Sclerotia differentiated into a rind and medulla; Rhizomorphs; Red, green, blue, bright yellow, orange, or other pigments except brown; any perfect state other than *T. cucumeris.* One feature, habitat, was not included in the above list despite the fact that most workers associate *R. solani* with soil. Although many strains are soil inhabiting, we know very little about the behavior of aerial strains. Some of these may not be associated with soil. Furthermore, fungi frequently are obtained from soil as casual contaminants. Thus, the origin of an isolate in or outside of the soil is not a definitive taxonomic feature.

## Disease management

The lack of adequate genetic resistance to *R. solani* in currently grown rapeseed and canola cultivars, and the absence of cultural methods for suppressing *R. solani* populations in the field, caused research to be directed towards investigating chemical control. Carboxin (Vitavax RS) and iprodione (Rovral ST) in mixtures with insecticide gamma-HCH (Lindane) are although recommended as seed treatments to control damping-off and seedling root rot, Howevr, they do not provide control against the mature plant root rot.

## 4. *Verticillium longisporum* Stark comb. nov. Karapapa (Verticillium stem striping)

*Verticillium longisporum* (ex. *V. dahliae* var. *longisporum* Stark; comb. nov. Karapapa) is a soil borne fungal pathogen causing vascular diseases of cruciferous plants (Karapapa *et al.,* 1997; Zeise and von Tiedemann, 2001, 2002a, b). Verticillium wilt is a novel disease on oilseed rape (*Brassica napus* L. sp. *oleifera*), threatening its production particularly in the northern European countries (Dunker *et al.,* 2008). Oilseed rape crop, colonized of pathogen *V. longisporum* does not develop wilting symptoms, and therefore the common name of Verticillium wilt is unsuitable for this crop. Therefore, (Depotter *et al.,* 2016) proposed 'Verticillium stem striping' as the common name for *Verticillium* infections of oilseed rape.

*Verticillium longisporum* is a member of the Flavnonexudans lineage and thus lacks the ability to produce yellow hyphal pigmentation. The pathogen produces durable microsclerotia which accumulate in the soil and from which plant roots are attacked (Schnathorst, 1981). On germination of resting structures, which is triggered by root exudates, fungal hyphae grow toward the root surface and penetrate the root epidermal cells near the root tips (Eynck *et al.,* 2007; Zhou *et al.,* 2006). Before entering into the xylem, the fungus traverses the root cortex inter- as well as intracellularly. During most of its life cycle, *V. longisporum* is confined to the vascular system, a nutrient-poor environment to which the fungus is well adapted (Pegg, 1985; Van Alfen, 1989). The pathogen spreads with growing hyphae and/or conidiospores conveyed with the transpiration stream into upper parts of the plant vascular system. As the host tissue turns to senescence, the pathogen enters a final saprophytic growth stage in which microsclerotia are abundantly formed in the dying stem parenchyma.

**Economic importance:** Although, the impact of *V. longisporum* infection on oilseed yield remains unclear because Verticillium stem striping on oilseed rape appears towards the end of the cropping season. Significant yield reductions upon *V. longisporum* infection only occurred in a single field trial on all tested oilseed rape cultivars. These preliminary data suggest that Verticillium stem striping does not consistently impact oilseed rape yield, despite the occurrence of abundant disease symptoms (Depotter *et al.,* 2019). Yield losses by Verticillium stem striping have been anticipated to range from 10 to 50%, yet experimental

verifications of such estimations are lacking (Dunker *et al.,* 2008). In contrast, Verticillium stem striping did not impact yield significantly in field studies despite the presence of stem striping symptoms (Dunker *et al.,* 2008).

**Disease management:** The pathogen responsible for disease in oilseed rape cannot be controlled efficiently with fungicides on account of their ability to survive as microsclerotia in the soil (Heale and Karapapa, 1999) restricting the control measures of the disease can be done by either cultural practices such as wider crop rotations or the use of resistant cultivars. The breeding for resistance has been hampered by the lack of sufficient resistance in commercially available breeding material as well as in the different varieties. However, a promising result on account of level of resistance to *V. longisporum* has been identified in cabbage (*B. oleracea)* crop (Dixelius *et al.,* 2005; Happstadius *et al.,* 2003; Rygulla *et al.,* 2007a). The significant improvement of resistance in *B. napus* against *V. longisporum* has been achieved by hybridization of resistant *B. oleracea* with *B. rapa* (Rygulla *et al.,* 2007b). Resistance to wilt pathogens such as *Verticillium* is supposed to be depending predominantly on the physical restriction and chemical inhibition of the pathogen during the systemic phase of colonization (Nicholson and Hammerschmidt, 1992). Unlike many biotrophic and some necrotrophic interactions, complete resistance to vascular infection has been considered to be unlikely (Beckman *et al.,* 1987). Thus, host plants may lack severe symptoms although being systemically colonized which would normally be denoted "tolerant" instead of "resistant" (Bishop *et al.,* 1984; Hammond-Kosack and Jones, 1996; Pegg and Brady, 2002). Resistance to vascular pathogens is thus expressed "internally,"representing an exceptional phenomenon in the realm of plant–pathogen interactions. Usually, this type of resistance is based on the rapid build-up of mechanical barriers, vascular gels or tyloses delaying or even preventing the spread of the pathogen in the vascular system (Sinha and Wood, 1968). Due to lacking of resistant sources as well as effective chemical control of this disease, biochemical basis of disease is being under taken. The first histological and biochemical characterization of quantitative resistance in a rapeseed genotype against *V. longisporum* was documented by Eynck *et al.* (2009). Phenolic compounds delivered along the phenylpropanoid pathway play an important role in defense to pathogen infection either as preformed or post-infectional defense factors. They have been assigned to various important biological functions in defense such as cell wall reinforcement and antimicrobial activity (Tuncel and Nergiz, 1993), as modulators of plant hormones in defense signaling or as scavengers of reactive oxygen species (Dixon and Paiva, 1995). Particularly the phenolic polymer lignin is an important principal structural component of secondary vascular tissue and fibers in higher plants (Humphreys and Chapple, 2002; Whetten and Sederoff, 1995). It is known to play a fundamental role in mechanical support, solute conductance and disease resistance (Harakava, 2005). Deposition of lignin, lignin-like polymers, and other wall-bound phenolic materials is reported to be a response to mechanical damage, wounding, or microbial infection (Boudet *et al.,* 1995). In addition to cell wall strengthening and increased cell wall rigidity (Wardrop, 1971), lignin deposition is supposed to decrease the diffusion of enzymes and toxins released from pathogenic fungal hyphae to the host, and of water and nutrients from the host to the fungus, thus essentially starving the intruder (Vance *et al.,* 1980). The data on histochemical and physiological changes generated during the elucidation of mechanisms involved in the genotype-specific internal resistance of a *B. napus* genotype to *V. longisporum* showed that resistance is related to changes in the phenolic composition of root and stem tissues, to alterations in the vascular cell walls and to vessel occlusions occurring in the vascular tissue during particular stages of infection and systemic colonization. Pathogen spread in a susceptible and a resistant *B. napus* cultivar was followed by quantitative polymerase chain reaction (qPCR).

## 5. *Fusarium* species (Fusarium wilt)

*Fusarium oxysporum* f. sp. *conglutinans* is causing wilt in many plant species worldwide. *Fusarium* species including *Fusarium oxysporum*, *Fusarium equiseti* (Corda) Sacc. have been considered to be weak pathogen reported to cause disease on Brassicaceae family. Based on disease ratings in pathogenicity tests, six isolates of *F. avenaceum* showed high aggressiveness on canola in Canada (Chen *et al.,* 2014).

This pathogen specifically attacks *Brassica* species and is morphologically indistinguishable from other *F. oxysporum* strains. Sporodochia and macroconidia are abundant on CLA (Carnation leaf agar) medium. Macroconidia are short to medium in length and usually 3-septate. Microconidia usually 0-septate, oval or reniform and are formed in false heads. Chlamydospores are formed abundantly on agar medium. *Fusarium oxysporum* usually winters as soil borne chlamydospores. Two other spore types (macro- and microconidia) are also formed. Spores germinate, and penetrate roots directly or through cracks formed by emerging lateral roots. The expanding mycelium then infects the vascular tissue, which becomes discoloured and blocked. This blockage of the vascular tissue prevents water upwards movement in the stem, resulting in turn in wilting of the plant. Spores are also

formed on aerial plant parts, which fall to the ground and re-infest the plant. Occasionally, seed is infected via the vascular tissue, but usually the seed aborts before this can occur.

*Fusarium equiseti* is a soil inhabitant and can transmit a disease to seeds, roots, tubers, and fruit of numerous crop plants. This new species is unique in having colonies on PDA medium at 25±2$^{O}$C, fast growing, rosy pink to vanacous, reverse pell leuteus to light ochraceous. A dense white mycelium developed, that turned first beige, finally buff brown in colour. Micromorphology showed that the hyphae were hyaline, smooth, pigmented and 2.75-4.77µm wide. Single-spore pure culture produced chlamydospores were intercalary, fusoid, hyaline and smooth walled. While, macroconidia were abundantly produced, fusoid, smooth walled, hyaline, foot cell present and sickle shaped, 2-septate with 14.59-45.48 x 2.88-4.10µm in size. Though, microconidia were fusoid, 1-septate and 2.5-2.67 x 2.67- 13.7 µm in size (Meena *et al.,* 2019).

## Distribution

The pathogen causing Fusarium wilt, *Fusarium oxysporum* f. sp. *conglutinans* race 1, is known to widely occur in many countries. The pathogen has been reported to cause Fusarium wilt of oilseed mustard (*B. juncea*) in India, rapeseed (*B. napus*) in Russia and Canada (Klassen *et al.,* 2007). The formae speciales that can attack canola is *conglutinans*, which includes all crucifer infecting isolates. Fusarium wilt is a serious biosecurity threat to Australia's canola industry. This pathogen was also reported from Queensland, Australia in 1961 causing 'Yellows disease' on cabbages.

*F. equiseti* generally occurs in tropical and subtropical regions (Booth, 1978; Bosch and Mirocha, 1992), but it has also been recovered from cereals in temperate areas, including the Soviet Union, Europe, and North America (Kosiak *et al.,* 2003; Stack *et al.,* 1997; Tekauz *et al.,* 2005; Wing *et al.,* 1993; Xue *et al.,* 2006).

## Host range

The pathogen causing canola wilt mainly affects canola and mustard. It is also known to cause "*Fusarium Yellows'* on a range of other crucifers in particular vegetable Brassicas. *Fusarium equiseti* has been formerly concerned as a causal agent of disease on different plant species, such as canola (*B. napus*) (Goswami *et al.,* 2008) and Indian mustard (*B. juncea*) in India (Meena *et al.,* 2019).

## Economic importance

Fusarium wilt can reduce yield by up to 30%, however, in worst cases complete yield loss can occur (Lange *et al.,* 2007). The symptoms of the disease include poor seed set, premature senescence and premature shattering of siliquas. Sometimes, symptoms may be present on one side of the stem, or affecting only one or a few branches, while other plant parts appear normal. Vascular discoloration of the main stem is the key diagnostic symptom. *Fusarium avenaceum* is less well documented as a *Brassica* pathogen. *F. roseum* has been implicated several times in Canada and France as a seedling blight and root rot pathogen of *B. napus* and *B. rapa*. *Fusarium roseum* is an obsolete epithet that has since been divided into several species, including *F. avenaceum*. *Fusarium avenaceum* probably over winters as mycelium in plant diseased debris. Host range studies indicate that isolates that caused Fusarium wilt of canola can infect cereals, and may cause disease in those crops. *F. avenaceum* has long been known to cause root rots and seedling blights on canola. The pathogen also known to causes wilt-like symptoms in canola, although whether it infects the xylem vessels is still unknown.

## Disease management

Diagnosis is based upon fungal morphological characters on Special Minimal Medium (SNA). Since the pathogen is soil borne in nature, it is quite difficult to manage, however, deep summer ploughing, seed treatment with fungicides, alteration/ modification in cultural practices are some of the ways to manage this disease. Crop rotation and good weed control is vital to minimize the inoculum levels. Control options are limited and use of resistant varieties is the best option.

## 6. *Pseudocercosporella capsellae* (*Syn. Neopseudocercosporella capsellae* (Ellis & Everhart) Videira & Crous (White leaf spot)

White leaf spot disease caused by *Pseudocercosporella* (*Syn. Neopseudocercosporella capsellae* (Ellis & Everhart) Videira & Crous is an important disease on many Brassicaceae including oilseed, wild cruciferous crops, vegetable, condiment, and fodder *Brassica* species (Petrie and Vanterpool, 1978; Sumner *et al.,* 1978; Barbetti and Sivasithamparam, 1981; Cerkauskas *et al.,* 1998; Ocamb 2014; Gunasinghe *et al.,* 2020).

*Pseudocercosporella capsellae* has both sexual and asexual life cycles. When present, the teleomorph stage (*Mycosphaerella capsellae*), produced at the end of the crop season is primarily responsible for survival of the pathogen over the period between crops and for production of primary inoculum for the next season (Inman *et al.*, 1991).

On artificial media, the pathogen's growth rate is noticeably slow, taking 3 weeks for a colony to reach

1–2 cm in diameter (Crossan ,1954). Slow growth is a likely reason for under-reporting of the pathogen as the disease causal agent. On a variety of culture media, it produces dark to olivaceous-gray stromatic colonies with dentate margins (Inman, 1992). Optimum growth from hyphae occurs between 20–24$^{o}$C and at pH 5.5–7.0 on potato dextrose agar (PDA) (Okullo'kwany, 1987). Young colonies produce thin and hyaline hyphae becoming thick-walled, septate, brown hyphae with stroma-like or sclerotia-like structures, which give rise to conidia (Crossan, 1954). However, *N. capsellae* does not sporulate on commonly used artificial media including PDA (Miller and McWhorter, 1948; Crossan, 1954), but produces conidia when the growing pathogen on V8 or distilled water agar are exposed to near-UV light around 365–370 nm (Inman *et al.,* 1991). Petrie and Vanterpool (1978) observed and extracted a red/purple/pink pigment produced by *N. capsellae* mycelial mats. Now this pigment has been confirmed as the mycotoxin cercosporin (Gunasinghe *et al.,* 2016a).

## Distribution

*Neopseudocercosporella capsellae* has been recorded from all subcontinents except Antarctica, over a wide range of climatic conditions from temperate to tropical, including the principal oilseed rape or mustard producing countries of the European Union, China, India, Canada, Australia, and Japan (CMI, 1986). However, the disease severity and incidence differ geographically due to variation in pathogen populations, host species, cultivars grown, different agricultural practices adopted, and prevailing local climatic conditions (West *et al.* 2001), in particular, temperature, humidity, and rainfall (Siebold and Von Tiedemann, 2012).

There has been a worldwide increase in pathogen activity and the disease is now identified as a re-emerging disease on oilseed rape and on oriental *Brassica* vegetables, particularly in the UK (Inman, 1992; Anonymous, 2016), the USA (Ocamb 2014, 2016) and in Australia (Van de Wouw *et al.,* 2016; Murtza *et al.,* 2019). While *N. capsellae* is commonly a leaf pathogen, it also produces pod and stem lesions resulting in "*gray stem*" disease (Petrie and Vanterpool, 1978; Inman *et al.,* 1999). The disease occur across the oilseed *Brassica* growing countries including France (Penaud, 1987; Perron and Souliac, 1990), the UK (Inman *et al.,* 1992), Canada (Petrie and Vanterpool, 1978), Germany (Amelung and Daebeler, 1988; Sochting and Verreet, 2004) and Australia (Barbetti and Sivasithamparam, 1981; Howlett *et al.,* 1999; Eshraghi *et al.,* 2005).

## Host range

*Pseudocercosporella capsellae* has a wide host range (Boerema and Verhoeven 1980; Gudelj *et al.,* 2004), infecting diverse wild and cultivated crucifers, including oilseed, forage (Sumner *et al.,* 1978) and vegetable *Brassicas* (Deighton, 1973; Cerkauskas *et al.,* 1998). In Western Australia, *P. capsellae* was first recorded in 1942 on *Brassica napus* (Shivas, 1989), subsequently on turnip rapeseed (*B. rapa*), Chinese cabbage (*B. rapa* var. *chinensis*), broccoli (*B. oleracea* var. *italica*), swede (*B. napus* var. *napobrassica*) (Shivas, 1989), and more recently on *B. juncea* (Eshraghi *et al.,* 2005). Some species of the genus *Cercospora* are known as producers of cercosporin, visible as a purple-pink pigment adjacent to the margins of colonies growing on artificial media (Petrie and Vanterpool, 1978; Daub and Ehrenshaft, 2000).

The pathogen *N. capsellae* has been isolated from leaf lesions on "wild" or "weedy" crucifers such as wild radish (*Raphanus raphanistrum*) and wild turnip (*Brassica rapa* ssp *sylvestris*) (Marchionatto, 1947; Deighton, 1973; Morris and Crous, 1994; Francis and Warwick, 2003; Maxwell and Scott, 2008). It has also been recorded causing disease on false flax (*Camelina sativa*), a wild oilseed crop in Europe (Foller and Paul, 2002), chinese cabbage, mustard type *Brassica* vegetables, and cauliflower (Lancaster, 2006). Moreover, *N. capsellae* can produce leaf spots interspersed with symptoms caused by other *Brassica* pathogens.

## Economic importance

Severe losses due to white leaf spot disease can occur at the seedling stage (Ocamb, 2014) to older plants when susceptible varieties are grown under environmental conditions favorable for disease development (Reyes, 1979; Penaud, 1987; Barbetti and Khangura, 2000). In both these situations, and particularly when the environment is conducive, white leaf spot causes significant yield losses of at least 30% in oilseed Brassicas predominantly through defoliation and the development of pod lesions (Penaud, 1987; Barbetti and Khangura, 2000). Pod infections by *N. capsellae* can cause 15% yield losses in France (Penaud, 1987).

Variation of pathogen populations can be related to differences in pathogenicity and to virulence of isolates of the same pathogen as well (McDonald and Linde, 2002), and such differences can be attributed to differences in genetic makeup of the fungal isolates (Kuninaga *et al.,* 1997). The variation within populations of agronomically significant pathogens is an essential criterion towards developing and applying management strategies, and in understanding the pathogen's ability to respond to implemented management practices.

## Epidemiology

Under favorable environmental conditions, *N.*

*capsellae* conidia or ascospores produce lesions on the leaves, while only conidia are responsible for stem and pod lesions that appear later in the crop season ((Petrie and Vanterpool, 1978; Penaud, 1987; Barbetti and Khangura, 2000; Inman *et al.,* 1991, 1999). Optimal conidial germination occurs at 20–24$^{O}$C and is inhibited below 8$^{O}$C or above 28$^{O}$C (Crossan, 1954). Germination is usually from apical cells, or less frequently from basal cells (Morris and Crous, 1994) are occur. The conidia can produce multiple germ tubes from each conidial cell (Petrie and Vanterpool, 1978; Gunasinghe *et al.,* 2016b) and cleavage at a septum of a conidium can produce multiple conidia, each producing a germ tube to infect the host (Gunasinghe *et al.,* 2016b). Hyphae of germinating spores invade the host tissue through natural openings such as stomata (Crossan, 1954; Gunasinghe *et al.,* 2016b). Temperatures of 18–19$^{O}$C and high humidity (100%) with at least 8 h of continuous leaf wetness are ideal for white leaf spot infections (Inman, 1992).

### *Nigrospora oryzae* (Stem blight)

The pathogen *Nigrospora oryzae* causing stem blight revealed dark brown, septate, branched mycelium and flexuous, branched conidiophores ranging from 3.75 to 4.30 µm in diameter and 14.80 to 26.25 µm in length. Conidia are smooth, globose to sub spherical in shape, dark brown to black in colour; the mean size of 50 conidia is 13.25 µm (11.2– 15.3) x 12.60 µm (10.6–14.6) (Sharma *et al*., 2013). Based on morphological and cultural characteristics (Ellis, 1971), the fungus was identified as *Nigrospora oryzae* (Berk. & Br.) Petch (teleomorph, *Khuskia oryzae*). Identification was confirmed by the National Fungal Culture Collection of India (NFCCI), Agharkar Research Institute, Pune, India, and the culture was deposited (accession number NFCCI 2306).

The first time occurrence of a stem blight disease caused by *Nigrospora oryzae* (Berk. & Broome) Petch was observed on the lower portions of *Brassica juncea* stems in India (Sharma *et al.,* 2013). In advanced stages, the lesions are up to 120 cm in length on the stems and also spread to petioles and midribs of leaves. The disease spreads in the last week of December, and high disease severity (70%) was recorded in several *B. juncea* cv. Rohini fields. Symptoms of the disease included the formation of small (2–7 mm), circular to irregular, dark grey to black lesions with a slight bluish cast. The discoloration was observed as numerous separate, irregular blotches. In the advanced stages of disease, lesions reached a length of 120 cm on stems and spread onto petioles and midribs (Sharma *et al.* 2013). The pathogen survives on diseased plant debris in soil (Plate 1).

### Host range

*Nigrospora oryzae* was reported earlier on stems of Polish rapeseed (*Brassica campestris* L.) in Alberta, Canada (Vaartnou *et al.,* 1974), on rice in India (Prasad *et al.,* 1960), Pakistan (Hajano *et al.,* 2011), on Kentucky bluegrass (*Poa pratensis*) in Ontario, Canada (Zheng *et al.,* 2012), on Giant Parramatta grass (*Sporobolus fertilis*) (Officer, 2012), on wheat, sorghum and barley (Fakhrunnisa *et al.,* 2006) and has been associated with post-harvest diseases of citrus (Jagdish *et al.,* 1985).

### 8. *Sclerotium rolfsii* Sacc. (Root rot)

Root rot caused by *Sclerotium rolfsii* Sacc. have been reported on Indian mustard. Normally the pathogen causing severe disease on leguminous crops but has been reported in Brassicas also. The disease caused upto 80% yield losses at farmer's field in Rajasthan, Madhya Pradesh, Uttar Pradesh and Haryana states of India in favorable environmental conditions (Meena *et al.,* 2013). The disease appears at budding stage as topple down of neck of plants, inward leaves, and as

**Plate 1. Symptoms of stem blight of rapeseed- mustard incited by *Nigrospora oryzae***

**Plate 2. Symptoms caused *by Sclerotinia rolfsii* on *Brassica juncea* plant and mycelial growth at collar region of plant**

whitish growth of the fungus at ground level on stem (Plate 2). Death of infected plants due to increased rotting of roots in not uncommon.

The pathogen produces survival structures, called sclerotia which are typically brown to tan in colour and measure 0.5 to 2.0 mm in diameter. Germinating sclerotia initiate new infection under suitable conditions for fungal growth.

The disease development is favoured by wet (>90% RH), 25-29$^{O}$C maximum and 8-13$^{O}$C minimum temperature. The disease can be managed efficiently by seed treatment with carbendizim @ 2g/kg seed and spray of this chemical @ 0.1% at initial stage of the disease.

### 9. *Xanthomonas campestris* pv. *campestris* (Pam.) Dowson (Black rot)

Black rot of crucifers incited by *Xanthomonas campestris* pv. *campestris* (Pam.) Dowson (Xcc) is one of the most widespread and devastating worldwide. Black rot is predominantly on vegetable crops cauliflower, cabbage, knol kohl etc. Black rot caused by the vascular, gram-negative bacterium, *X. campestris* pv. *campestris*, is one of the most devastating seed borne epidemic diseases worldwide (Vicente *et al.,* 2001; Taylor *et al.,* 2002; Tonguc and Griffiths, 2004). The disease can also be spread through infected seedlings, infested soil, crop debris and other host weeds species (Schaad and Dianese, 1981; Vicente and Holub, 2013). Bacterium is a small, rod shaped, aerobic, gram negative, non-spore forming. The bacterium has a single polar flagellum and it is catalase positive, hydrogen sulphide positive, oxidase negative and does not produce nitrate or indole. It produces a yellowish extracellular polysaccharide (EPS) called xanthan on media containing glucose (Gupta *et al.,* 2013). *Xanthomonas campestris* is seed borne and overwinters in infected residue in the soil. Bacteria are usually splashed by water and enter plants through stomates. Little is known of the life cycle of X. *campestris* as it relates to disease development on rapeseed and mustard.

The bacteria caused systemic infection in the susceptible host plants and bacterium penetrate the host plants species via natural openings (hydathodes on the leaf margins) or wounds and replicates in the vascular tissues (Cook *et al.,* 1952a; Ignatov *et al.,* 1999). Once the bacteria invades the plant through hydathodes, *Xcc* multiply and colonize the vascular system, and extracellular polysaccharide (EPS) called xanthan are produced, which can obstruct the xylem vessels, lead to necrosis of tissue and severe leaf wilting (Williams, 1980). The point of the V-shaped lesions is directed toward a vein. Eventually, the pathogen affected regions on the plants become necrotic and the veins turn black or brown. Plant metabolic activities are severely affected with the advancement of the disease which results in stunted growth.

The pathogen moves systemically through the vascular bundles turning them black and produces main symptoms on leaves as described by various workers. The pathogen produces two types of symptoms. Initially, the marginal chlorotic spots appear on the leaves followed by darkening or blackening of mid rib and veins (Cook *et al*., 1952a). However, Sutton and Williams (1970) reported that vein blackening was the first visible symptom which was due to the accumulation of melanin among xylem cells. The pathogen colonizes the vascular system after its entry into the plant and produces plentiful extracellular polysaccharide called xanthan (Jeanes 1973) which along with the bacterial cells plug the xylem vessels, restricting the water flow and resulting in the characteristic V-shaped chlorotic lesions originating from the margins of the leaves. Typical disease symptoms are 'V' shaped necrotic lesions appearing from the leaf margins with blackened veins has also been reported. The disease has a wide

geographical distribution and is destructive to crucifer vegetables causing yield (50–65%) and quality loss due to cultivar susceptibility (Williams 1980; Dhar and Singh 2014).

## Distribution

Black rot disease was recorded initially on cabbage in USA by Garman (1894). In 1898, the disease was reported on cabbage from Wisconsin by Russel. Since then, there has been a growing recognition of seriousness of black rot on cruciferous crops throughout the world. In India, the disease was recorded for the first time on cauliflower by Patel *et al.* (1949).

This disease is reported to cause huge loss from different regions of world such as Brazil, Ethiopia, South Africa, Belgium, Germany, Sweden, Hungry, France, Netherlands, Italy, the USA, the UK, Nepal, China, Taiwan, and Canada along with Australia and India (Saharan, 1993; Mulema *et al.,* 2012; Singh *et al.,* 2016; Akhtar *et al.,* 2017).

## Host range

*Xanthomonas campestris* pv. *campestris* (Xcc) belongs to the genus that causes disease on at least 124 monocotyledonous and 268 dicotyledonous plant species including all major crop plants (Lcyns *et al.,* 1984). Several other plant species in the Brassicaceae family including radish, turnip, rutabaga, vegetable mustard, black mustard, wild mustard, ornamentals and cruciferous weeds also serve as a host of this pathogen. The bacterium is found to attack all members of Cruciferae family including rutabaga, cabbage, savoy cabbage, broccoli, turnip, radish, kohlrabi, *Lapidium sativum*, *Raphanus sativus* var. *caudatus*, *Brassica juncea*, fodder cabbage, *Capsella bursa-pastoris* and *Cantella asiatica* (Mahiar and Khalif, 1999).



## 10. *Erwinia carotovora* pv. *carotovora* (Jones) Bergy (Stalk rot)

Stalk rot on rapeseed-mustard incited by *Erwinia carotovora* pv. *carotovora* (Jones) Bergy, or *Erwinia carotovora* pv. *atroseptica* (van Hall) Dye (syn. *E. atroseptica* (van Hall) Jennison).was reported from various parts of India viz., Rajasthan, Madhya Pradesh, Uttar Pradesh and Haryana (Satyavir *et al.,* 1973; Bhowmik and Trivedi (1980). According to them, the disease appeared in epiphytotic proportion on the commonly cultivated *B. juncea* variety Varuna in 1979 in the Pali district of Rajasthan state, India.

## Economic importance

In India, the yield loss due to *E. carotovora* in partially and completely diseased *B. juncea* was 92.29 and 96.06% and in *B. rapa,* it was 86.6 and 89.04%, respectively (Sobti, 1983). The disease has recently become a threat to successful cultivation in some parts of Haryana and Rajasthan states of the country. The bacterium was reported with the incidence of 60 to 80 per cent in Indian mustard at farmer's field in Mahua Simpani village of Bharatpur district of Rajasthan, India (Meena *et al.,* 2010).

## Symptoms

Symptoms of the disease were characterized by the appearance of water-soaked lesions at the collar region of plants, which was usually accompanied by a white frothing. Leaf tissue becomes chlorotic with veins in the chlorotic area dark in color.The tender branches were also affected as the lesions advance further to cover larger areas. The leaves showed signs of water stress and withering. The affected stem and branches, particularly the pith tissues, became soft, pulpy and produced dirty white ooze with a foul smell (Plate 3). The central part of the stem may be partially or completely rotted at flowering or before, causing an infected plant to wilt. Wilting is associated with a mottled leaf appearance. The rot starts where larvae of *Hylemyia* sp. damages the stem at soil level or where branching occurs. The infected collar region became sunken and turned buff-white to pale-brown in colour (Meena *et al.,* 2010, 2013). The pathogen survives on diseased plant debris in soil. The disease usually appears after first irrigation in mustard. Warm and humid weather favours disease development.

## Disease management

Crop rotation with non-host crops, burning of diseased debris, deep ploughing in summer months

**Plate 3. Symptoms of stalk rot incited by *Erwinia carotovora* pv. *carotovora* on rapeseed mustard**

**Plate 4. Symptoms on (A) *Brassica juncea* and (B) *B. rapa* ssp. Yellow Sarson**



helps in minimizing inoculum build up. Timely sown crop (10-25th October) is likely to have less disease incidence. Removal of weeds, rogouing and destruction of affected plants and avoidance of over irrigation has been found to reduce the incidence of disease.

## 11. *Turnip mosaic virus* (TuMV) (Turnip mosaic)

*Turnip mosaic virus* (TuMV) is a member of the genus Potyvirus of family Potyviridae. TuMV is a positive strand RNA virus with a genome of 9830–9833 nucleotides (Ohshima *et al.,* 1996) and well characterized molecular biology (Walsh and Jenner 2002). The earliest report of occurrence of mustard mosaic was made by Bennett in 1944. This virus was obtained from *Brassica adpressa* (Moench.) Bioss near Riverside, California and is related to mild mosaic virus of annual stock mentioned by Tompkins (1939).

### Symptoms

Symptoms of the mosaic disease on rapeseed and mustard are similar to Turnip mosaic symptoms as described by Tompkins (1938). According to Ling and Yang (1940), the infection manifests itself in the beginning as a systemic infection. On rape and *B. juncea*, conspicuous clearing of veins, usually commencing at or near the base of the leaf and gradually spreading over the entire leaf. Generally, this stage may last for 2 to 3 weeks. The affected plants do not produce flowers or very few flowers are produced on such infected plants. Azad and Sehgal (1959) observed that when siliquae are formed, they remain poorly filled and show shriveling. Ling and Yang (1940) further observed that in certain cases particularly under high temperatures vein-clearing is soon replaced by vein-banding. During the later stages of infection, numerous raised or non-raised, dark-green islands of irregular outline appear in the chlorotic area between the veins, giving rise to a mottled appearance, often curvature of the midrib and distortion of the leaf blade. Low temperatures favour the expression of symptoms and temperatures higher than $20^0$C usually induce their masking. Plants infected early are usually severely stunted and often killed but those infected late in their development are stunted only or not at all. More or less similar symptoms have been described (Plate 4) (Dale 1948; Rao *et al*., 1977; Takahashi 1949).

In addition to rape and mustard, Chinese radish (*Raphanus sativus* var. *longipinnatus*), turnip (*Brassica rapa*) and Chinese cabbage (*Brassica chinensis)* have also been reported to be found infected with identical symptoms. Initial symptoms develop on seedlings as chlorotic spots on leaves, mottling followed by systemic vein clearing, necrosis, leaf distortion and often stunting.

### Host range

TuMV possesses an exceptionally broad host range; it infects 156 plant genera representing many arable, vegetable, and ornamental crops including oilseed and vegetable brassicas, peas, lettuce, rhubarb, and stocks (Edwardson and Christie 1991a). Natural infection by TuMV was common in Brassicaceae weeds. Infected crops included mustard (*Brassica juncea*), field pea (*Pisum sativum*), chickpea (*Cicer arietinum*), and coriander (*Coriandrum sativum*). Forage turnip, (*B. rapa*) was also infected (Schwinghamer *et al.,* 2014). It infects mainly Brassicaceae hosts, including *Brassicas*

and non-Brassicas such as radish (*Raphanus sativa* L.). Canola is the Brassicaceae crop of most concern to grain growers, but TuMV also infects at least 39 other dicotyledonous plant families and three mono-cotyledonous families either naturally or by controlled inoculation (Edwardson and Christie, 1991b; Walsh and Jenner, 2002; Tomimura *et al.,* 2004).

The virus is also common on certain other species of mustard in southern California. Two more common mustard mosaic diseases have been described as Turnip mosaic poty virus (TuMV), Beet western yellows luteo virus (BWYV) and Cauliflower mosaic caulimovirus (CaMV) by Chen *et al.* (2000). Among the three viruses, TuMV is the most common. Often mixed infection of TuMV and BWYV have been observed on *Brassica juncea* and *Brassica napus*.

A mosaic disease on rape, *B. napus* L. var. *biennis* (Schubl. and Mart.) Reichb, Winter rape var. Dwarf Essex, *B. campestris* L. var. *napobrassica* (L) D.C. and Rutabaga var. American Purple Top is wide spread in different districts of China (Ling and Yang, 1940), U.S.A. (Sylvester, 1953), United Kingdom (Hunter *et al*., 2002; Hardwick *et al*., 1994; Hauser *et al*., 2000b; Schubert *et al*., 1998) and Canada (Rao *et al*., 1977). Beet western yellows luteo virus in the United Kingdom on oilseed rape with higher incidence than Cauliflower mosaic (CaMV) has also been reported. The average plant infection due to BWYV has been reported to be in the range of 49-73 per cent in the United Kingdom (Hardwick *et al*., 1994). Alternative names, Turnip yellows virus (TuYV), *Brassica* yellow virus (BrYV) and *Brassica* yellowing virus (BrYV) have recently been proposed for BWYV (Hauser *et al.,* 2000a). The BrYV does not infect beet but infects a large number of plants belonging to the genus *Brassica* and the virus has been identified as one of three distinct polerovirus species infecting oilseed rape (Hauser *et al*., 2000a).

Although, TuMV is naturalized in all states of Australia (McLean and Price 1984), it is reported to cause significant disease only in vegetable crops and one ornamental species (stock: Brassicaceae: *Mattiola incana* L.). Affected hosts from the eastern states of NSW, Tas, Queensland (Qld), Victoria (Vic), and South Australia (SA) include a range of *Brassica* vegetables, rocket (Brassicaceae: *Eruca vesicaria* L. subsp. *sativa*), lettuce, and rhubarb (Polygonaceae: *Rheum rhabarbarum* L.), with NSW represented among voucher specimens for all of these hosts (Gibbs *et al.,* 2008). In NSW, TuMV was first recorded causing serious disease in cabbage, broccoli, and cauliflower (*Brassica oleracea* L. varieties) in the Sydney metropolitan area in 1957 and subsequently in irrigation areas in other parts of the state (Letham *et al.,* 1975; Conroy, 1959).

**Economic importance:** The mosaic diseases of rapeseed and mustard cause considerable losses of yield, especially the Turnip yellows virus (Beet Western Yellows Virus) in European Winter oilseed rape (Dreyer *et al*., 2001). A 90 per cent loss in yield due to the disease in eastern China was reported by Wei *et al.* (1960). Considerable losses in yield from different parts of the world have been reported but true picture has not been mentioned. Since, BWYV is prevalent on oilseed rape in the U.K, the continued infection foci appear to have great implications for the sugar beet crop. TuMV caused a loss of 30% in *B. napus* production in Canada (Shattuck and Stobbs, 1987), as well as seed yield losses of up to 70% in *B. napus* in the UK (Hardwick *et al.,* 1994) and 50% reductions in *B. oleracea* var. *capitata* (cabbage) head production in Kenya (Spence *et al.,* 2007).

## Distribution

TuMV is one of the most prevalent viruses, which was first discovered in the United States, and is threatening *Brassica* vegetables around the world, especially in Europe, Asia and North America (Tomlinson, 1987; Walkey and Pink, 1988; Walsh and Jenner, 2002). TuMV disease was first described in *B. rapa* in the USA (Gardner and Kendrick, 1921; Schultz, 1921), while it was later found in *B. oleracea* in the UK (Smith, 1935), and in *B. napus* in China (Ling and Yang, 1940). Now, TuMV is one of the most prevalent viruses and is threatening *Brassica* vegetables around the world, especially in Europe, Asia and North America (Tomlinson, 1987; Walkey and Pink, 1988; Walsh and Jenner, 2002).

## TuMV diversity

Turnip mosaic virus is a member of the genus Potyvirus (type species Potato virus Y) in the family Potyviridae. TuMV is the only potyvirus known to infect Brassicas (Walsh and Tomlinson, 1985). The TuMV genome consists of a positive-sense single-strand RNA molecule of about 9830 nucleotides in length. The gene contains a single open reading frame (ORF) flanked by two untranslated regions (Basso *et al.,* 1994). The ORF is translated into a single polyprotein, which is co-and post-translationally processed by three virus-encoded proteases (Walsh and Jenner, 2002). The proteins released comprise P1, helper component protease (HCPro), P3, 6K1, cytoplasmic/cylindrical inclusion (CI), 6K2, virus-encoded genome linked (VPg) protein, nuclear inclusion protein a (NIa), nuclear inclusion protein b (NIb) and the coat protein (CP) (Walsh and Jenner 2002). Turnip mosaic virus probably originated from a virus of wild orchids in Germany and, while adapting to wild and domestic brassicas, spread to Asia Minor via southern Europe no more than 700 years ago (Yasaka *et al.,* 2017). Various strains and pathotypes of

TuMV have been defined based on their ability to infect certain plant species or cultivars (Shattuck, 1992). An early scheme used symptom type on cabbages and *Nicotiana glutinosa* to distinguish TuMV strains (Yoshii *et al.,* 1998). Liu *et al.* (1996) also used symptom types and disease severity indices on a variety of *Brassica* species to define seven strains (Tu1–7). Other schemes were based on differential lines of a single species, either *B. rapa* (strains C1–C4; Provvidenti 1980; strains C1–C5; Green and Deng, 1985) or *B. napus* (pathotypes 1–12) (Walsh, 1989; Jenner and Walsh, 1996).

## Disease management

It is difficult to control TuMV because it is mainly transmitted in a non-persistent mode by at least 89 aphid species. Specifically, TuMV is introduced into plant cells via the stylet of aphids in a typical non-persistent transmission mode during aphid probing or feeding. The wide host range, high genetic variability and transmission mode of TuMV make the virus difficult to control through traditional methods such as chemicals. Chemical insecticides may control one or multiple aphid species but, as there are more than 89 aphid species capable of transmission, these aphids are soon replaced by other species leading to a continuation of host infection. The cultivation of resistant varieties, which can provides a more effective and environmental friendly approach is recommended. In particular, virus-resistant cultivars can be important in controlling TuMV epidemics, although resistance-breaking TuMV pathotypes that overcome single gene resistance may arise (Jenner and Walsh, 1996; Hughes *et al.,* 2002; Walsh *et al.,* 2002; Gładysz and Hanus-Fajerska, 2009). It is necessary to combine control measures (including identification of symptoms and virus by molecular, serological and electron microscopically methods) with host resistance to ensure effective TuMV-induced disease management (Jones, 2004, 2006). In disease control studies against mosaic when seedlings of *Brassica juncea var. crispifolia* and *Brassica juncea* var. *foliosa* are covered with a polyethylene insect- proof net provides 51.2-73.3 per cent control. Application of 10 per cent imidacloprid at 1:1500 is effective for control of aphids the vector of the virus (Chen *et al.*, 2000).

## 12. Phyllody (Phytoplasma Disease)

Phyllody disease in oilseed *Brassica* is mainly caused by *Phytoplasma*, which is devastating worldwide. The disease is prevalent in Toria (*Brassica rapa* L. subsp. *dichotoma* (Roxb.) since 1958 (Bhowmik, 2003; Bindra and Bakhetia, 1967) and Yellow Sarson growing regions of the country. It may cause yield losses of 70-90 per cent in susceptible toria (*Brassica rapa* var. Toria) cultivars, if infection occurs at early stage of plant growth (Azadvar and Baranwal, 2010).

**Host range and distribution:** The occurrence of 16SrVI-A phytoplasma has been reported to infecting spring oilseed mustard (*B. juncea*) in Inner Mongolia, China (Zhang *et al.,* 2020). Azadvar *et al.* (2011) confirmed the symptoms of phytoplasma infection were induced only in toria, yellow sarson [*Brassica rapa* L. subsp. *trilocularis* (Roxb.)], brown sarson [*Brassica rapa* L. subsp. *sarson* (Prain)], rapeseed (*B. napus* subsp. *oleifera*), and rocket or taramira (*Eruca sativa*). Phytoplasma pathogens that infect rapeseed/canola in Canada, Czech, Italy and Greece (Bertaccini *et al.,* 1998; Maliogka *et al.,* 2009; Olivier *et al.,* 2006; Wang and Hiruki 2001), identified as the 16SrI aster yellows group, cause stunting, leaf yellowing or purpling. The association of aster yellows phytoplasma with canola and its transmission to the progeny plants has been reported (Olivier *et al.,* 2008). Mycoplasma-like organisms (MLOs) were found in field-grown pak-choi cabbage (*Brassica chinensis* L.) in Alberta, Canada, in September 1993 (Chang *et al.,* 1995). In Poland, a phytoplasma belonging to the subgroup 16SrI-B of '*Ca*. Phytoplasma asteris' species causes disease in *Brassica napus* (Zwolinska *et al.,* 2019).

## Symptoms

The diseased plants can be seen only at flowering stage (Plate 5). Symptoms generally include phyllody (development of floral parts into leafy structures), virescence (development of green flowers and the loss of normal pigments), witches'-broom, extensive malformation of floral part, formation of bladder-like siliquae and flower sterility (Azadvar *et al.,* 2009; Bhowmik, 2003). The corolla becomes green and saploid, while stamens are green and become indehiscent. The affected parts of the raceme do not form silquae (Plate 5). If the infection occurs at early stage, infected plants remain stunted and produce more branches, giving bushy appearance (Meena *et al.,* 2013).

## The pathogen

The pathogen survives on alternate host like sesame and several other plants which serves as primary source of infection. The disease is transmitted through leafhopper (*Orosius albicinctus =Deltocephalus* sp.). The disease spreads by repeated cycles of secondary infection through the process of transmission. The insect vector is an important factor that can influence the host range of phytoplasmas in nature (Kakizawa *et al.,* 2010), so identification of the vector is important. *Laodelphax striatellus* was the major plant hopper in the toria field, and its detection with TP phytoplasmas in the present is the first record of it as a potential vector of 16SrIX phytoplasma.

**Plate 5. Symptoms of phyllody of rapeseed incited by phytoplasmas**



## Disease management

Prolong dry and warm weather is favourable for phyllody disease development through insect vector. Toria sown around mid of September is likely to escape infection. Rouging and destruction of phyllody affected plants helps to reduce further spread of disease. Spray twice with Rogor or Metasystox @ 0.1% at an interval of 15 days starting from the initiation of symptoms to control the insect vector.

## 13. Aster Yellows (Phytoplasma Disease)

Aster yellow is a virus-like disease caused by a phytoplasma, an organism similar to a bacterium. *Candidatus* phytoplasma asteris" and related strains (i.e., aster yellows group 16SrI) have been associated with diseases of numerous plant species worldwide. This is another phytoplasma disease having similar characteristics of phytoplasmas which is causing phyllody in Brassicas. The aster yellows phytoplasma is vectored by the aster leafhopper, an olive-green or straw-colored leafhopper with six dark spots on its forehead. The aster leafhopper will not fly at temperatures below 60°F. Aster leafhopper feeding itself is not economically damaging, but the aster yellows phytoplasma is damaging. Aster yellows affects over 300 crops and weeds, including carrot, celery, cucurbits, potato, sage, tomato, quack grass, plantain, chickory, knotweed, sowthistle, ragweed, Kentucky bluegrass and wild carrot. The pathogen survives between susceptible crops in alternate hosts or in its vector.

Typical aster yellows symptoms are phyllody, the transformation of floral parts into leaf like tissues. As in case of phyllody, symptoms generally include stunting, virescence, leaf yellowing or purpling, phyllody, and formation of bladder-like siliques. Symptoms are generally appearing on scattered plants mid to late in the season. Pods of infected plants are hollow and no seeds are produced.

## Disease management

Plant resistant varieties, if available are one of the best and effective methods of disease management. Control weeds in and around fields that serve as alternate hosts for aster yellows. Increasing planting population may reduce the incidence of aster yellows. Insecticides applied when aster leafhoppers populations are at their peak can reduce the incidence of aster yellows.

## Conclusion

Diseases caused by various pathogens either major or minor remain impediment to attainment of superior productivity of oilseed *Brassica* crops. The oilseed *Brassica* crops are faced by several fungal, bacterial and viral pathogens. Among them, major fungal pathogens are *Albugo, Alternaria, Hyaloperonospora, Erysiphe, Leptosphaeria, Plasmodiophora,* and *Sclerotinia,* while several other minor pathogens including *Colletotrichum higginsianum*. *C. capsici*, *Pyrenopeziza brassicae*, *Rhizoctonia solani*, *Verticillium longisporum*, *Fusarium oxysporum* f. sp. *conglutinans*; *F. equisiti*, *Xanthomonas campestris* pv. *campestris*, and mosaic (*Turnip mosaic virus*: TuMV infecting Brassicas. A proper prioritization of plant diseases in relation to the magnitude of the economic losses is utmost necessary to make economically effective and feasible mitigation over large areas in rapeseed-mustard. There is need for proper vigilant surveillance on occurrence of newer pathogens and severity of existing pathogens during crop season to protect the crop from huge losses. Occurrence of any biotic stress depends on existing favorable micro-

climatic conditions during crop season. There may be chances of occurrence of newer pathogens introduced either by seed or any means of transportation. Recent changing climatic situations responsible for shifting of the disease scenario and the minor pathogens are becoming major pathogen causing considerable yield losses. Therefore, the knowledge of minor pathogens may be helpful for oilseed *Brassica* growers.

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