Chapter 1
Introduction

1.1 Crops and Their Distribution

Crucifers occupy prominent place in world's agrarian economy as vegetables, oilseeds, feed, and fodder, green manure, and condiment. Oilseed *Brassica*, also known by their trade name of rapeseed-mustard, include *Brassica napus*, *B. juncea*, *B. carinata*, and three ecotypes of *B. rapa* (*B. rapa* var. *brown sarson*, *B. rapa* var. *yellow sarson*, *B. rapa* var. *toria*). Major crucifers susceptible to downy mildew disease grown all over the world are given in Table 1.1 with their botanical name, common name, and usages. Global production of oilseed *Brassica* crops exceeded 63.76 mt, making them the second most valuable source of vegetable oil in the world. The leading oilseed *Brassica* producers in the world are the European Union, China, Canada, and India (USDA 2015). Different forms of oilseed *Brassica* are cultivated throughout the world. Winter-type *B. napus* predominates in Europe, parts of China, and Eastern USA, while spring-type *B. napus* is cultivated in Canada, Australia, and China. Spring forms of *B. rapa* are now mainly grown in the Indian subcontinent. Winter-type *B. rapa* has largely been replaced by higher-yielding winter-type *B. napus* and spring crops in its traditional production zones. Only winter type of *B. juncea* is cultivated in the Indian subcontinent and has now been actively considered as an option in drier areas of Canada, Australia, and even Northern USA. In India, *B. juncea* predominates and is grown on an over 80% of the area under rapeseed-mustard crops. The goal of developing canola forms has been accomplished for *B. rapa*, *B. napus*, and *B. juncea* but remains as an important objective in *B. carinata*. Almost all rapeseed produced in Australia, Canada, and Europe, and to a very large extent in China, is now canola. The cultivation of canola rapeseed-mustard has just begun in India (Chauhan et al. 2010). Crucifer vegetable forms an important group of vegetable crops of the world. These include a wide array of crops that span numerous genera and species in the family Brassicaceae. However, cole crops belonging to *B. oleracea*, viz. cauliflower, cabbage, broccoli,

G. S. Saharan et al., *Downy Mildew Disease of Crucifers: Biology, Ecology and Disease Management*, https://doi.org/10.1007/978-981-10-7500-1_1

Table 1.1 Commonly cultivated crucifers and *Brassica* species susceptible to downy mildew disease

Botanical name	Common name	Usages
B. nigra	Black mustard	Condiment (seed), vegetable fodder (leaves)
B. oleracea		
B. oleracea var. *acephala*	Kale	Vegetable (head)
B. oleracea var. *capitata*	Cabbage	Vegetable (head)
B. oleracea var. *sabauda*	Savoy cabbage	Vegetable (head)
B. oleracea var. *gemmifera*	Brussels sprouts	Vegetable, fodder (stem)
B. oleracea var. *botrytis*	Cauliflower	Vegetable (inflorescence)
B. oleracea var. *gongylodes*	Kohlrabi	Vegetable, fodder (stem)
B. oleracea var. *italic*	Broccoli	Vegetable (inflorescence)
B. oleracea var. *fruticosa*	Branching bush kale	Fodder (leaves)
B. oleracea var. *alboglabra*	Chinese kale	Vegetable (stem, leaves)
B. rapa		
B. rapa subsp. *oleifera*	Turnip rape	Oilseed
B. rapa var. *brown sarson*	Brown sarson	Oilseed
B. rapa var. *yellow sarson*	Yellow sarson	Oilseed
B. rapa var. *toria*	Toria	Oilseed
B. rapa subsp. *rapifera*	Turnip	Fodder, vegetable (root)
B. rapa subsp. *chinensis*	Bok choy	Vegetable (leaves)
B. rapa subsp. *pekinensis*	Chinese cabbage	Vegetable, fodder (head)
B. rapa subsp. *nipposinica*	–	Vegetable (leaves)
B. rapa subsp. *parachinensis*	–	Vegetable (leaves)
B. carinata	Ethiopian mustard	Vegetable, oilseed
B. juncea	Mustard	Oilseed, vegetable
B. napus		
B. napus subsp. *oleifera*	Rapeseed	Oilseed
B. napus subsp. *rapifera*	Rutabaga, swede	Fodder
Eruca sativa	Rocket, taramira	Oilseed, fodder(leaves)
Raphanus sativus	Radish	Vegetable, fodder
Raphanus raphanistrum	Wild radish	Fodder

Brussels sprouts, kohlrabi, and kale, are most susceptible to downy mildew disease.

As these crops are grown in a wide array of climate and cropping systems, these require general or specific adaptation to specific situations. Varieties with varying maturity duration are required to escape frost (Canada) or late-season drought (Southern Australia) or to fit in multiple cropping sequences (India, China). Breeding programmes are also concerned with the cultivar suitability for existing or emerging management practices, e.g. herbicide resistance or mechanical harvesting, resistance to pod shattering, etc. (Kumar et al. 2015). *Brassica* species relationship has been given in Fig. 1.1. Crucifers are confronted with several biotic and

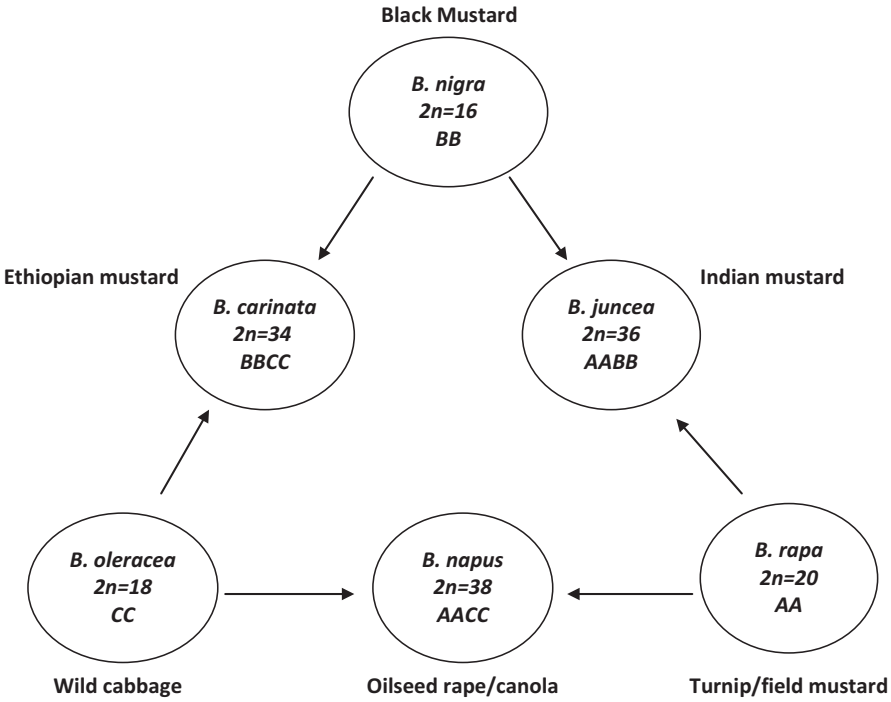

Fig. 1.1 U's triangle showing species relationship among different *Brassica* species (UN 1935)

abiotic stresses (Table 1.2). Among biotic stresses (diseases), downy mildew ranks the third most widespread devastating disease all over the world (Kolte 1985; Saharan et al. 1997, 2005).

1.2 The Disease and Pathogen

The term 'mildew' was first used in the USA to denote a wide group of parasitic fungi with little in common except their appearance as a white or lightly coloured delicate outgrowths caused by the proliferation, and fructification of mycelium on the surface of green, and necrotic plant tissues. Downy mildew quickly adapted to European conditions when vine mildew was introduced from North America. Downy mildew or members of the family Peronosporaceae are a distinctive group of obligate plant pathogens classified within the Mastigomycotina in the Oomycete order Peronosporales. In the family Cruciferae, about 50 genera and more than 100 different species are susceptible to infection by downy mildew pathogen.

Originally Gaumann (1918), on the basis of conidial measurements and cross-inoculation tests, recognized 52 species of *Peronospora* on crucifer hosts. Later studies by Yerkes and Shaw (1959) concluded that there are no reliable morphological

Table 1.2 Biotic and abiotic stresses of crucifers (Saharan 1984, 1992; Kolte 1985; Williams 1985)

Sr. No.	Common name	Symbol[a]	Pathogen or cause
1.	*Alternaria* disease, black spot, leaf stem or pod spots, and blight	Ab	*Alternaria* spp. (*A. brassicae* (Berk.) Sacc.; *A. brassicicola* (Schw.) Wiltsh.; *A. raphani* Groves & Skolko; *A. alternata*
2.	Anthracnose	Ch	*Colletotrichum higginsianum* Sacc.
3.	Aster yellows	AY	*Mycoplasma*-like organism or phytoplasma organism (MLO)
4.	Autogenic necrosis	AN(G)	Genetic disorder
5.	Bacterial leaf spot	Psm	*Pseudomonas syringae* pv. *maculicola* (McCulloch) Young et al.
6.	Bacterial soft rot, *Erwinia* stalk rot	Ec	*Erwinia carotovora* pv. *carotovora* (Jones) Bergey et al.
7.	Bacterial soft rot, *Pseudomonas* rot	Pmm	*Pseudomonas marginalis* pv. *marginalia* (Brown) Stevens
8.	Black leg and phoma root rot	Lm	*Leptosphaeria maculans* (Desm.) Ces. & de not. (anamorph: *Phoma lingam* (Tode: Fr.) Desm.
9.	Black mould rot	Rst	*Rhizopus stolonifer* (Ehr.: Fr.) Vuill.
10.	Black root	Ar	*Aphanomyces raphani* Kendrick
11.	Black rot	Xcc	*Xanthomonas campestris* pv. *campestris* (Pammel) Dowson
12.	Black speck	BS	Physiological disorder
13.	Bottom rot, damping off, head rot, seedling root rot, wire stem, basal stem rot	Rs	*Rhizoctonia solani* Kuhn (teleomorph: *Thanatephorus cucumeris* (Frank) Donk.)
14.	Broomrape	Oa	*Orobanche aegyptiaca* Pers.
15.	*Cercospora* leaf spot	Cb	*Cercospora brassicicola* Henn.
16.	Club root	Pb	*Plasmodiophora brassicae* Wor.
17.	Crown gall	At	*Agrobacterium tumefaciens* (Smith & Townsend) Conn.
18.	Damping off, *Fusarium*	F	*Fusarium* spp.
19.	Damping off, *Pythium*	PT	*Pythium* spp.
20.	Downy mildew, stag head	Hp	*Hyaloperonosporaparasitica* (Gaum.) Gokar
21.	Grey mould	Bc	*Botrytis cinerea* Pers.: Fr. (teleomorph: *Botryotinia fuckeliana* (de Bary) Whetz.
22.	Gummosis	G	Physiological disorder
23.	Light leaf spot	Pyb	*Pyrenopeziza brassicae* Sut. & Rawl

(continued)

Table 1.2 (continued)

Sr. No.	Common name	Symbol[a]	Pathogen or cause
24.	Nematode		
	(a) Awl	D(N)	*Dolichodorus* spp.
	(b) Cyst	Hs(N)	*Heterodera schachtii* Schmidt
			Heterodera cruciferae Franklin
	(c) Pin	PA(N)	*Paratylenchus* spp.
	(d) Root knot	M(N)	*Meloidogyne* spp.
	(e) Root lesion	P(N)	*Paratylenchus pratensis* (de Man) Filipjev
			Paratylenchus spp.
	(f) Sting	B(N)	*Belonolaimus* spp.
25.	Phyllody	PLO	*Phytoplasma*
26.	Pod malformation	GP	Genetical and physiological disorder
27.	Powdery mildew	Ec	*Erysiphe cruciferarum* Opiz. ex. Junell
28.	Ring spot	Mb	*Mycosphaerella brassicicola* (Duby) Lindau
29.	Root rot *Phymatotrichum*	Po	*Phymatotrichum omnivorum* (Shear) Dug.
30.	Root rot *Phytophthora*	Pm	*Phytophthora megasperma* Drechs.
31.	Scab	Sts	*Streptomyces scabies* (Thaxter) Waksman & Henrici
32.	*Sclerotinia* stem rot Watery soft rot	Ss	*Sclerotinia sclerotiorum* (Lib.) de Bary
33.	Southern blight, root rot	Sr	*Sclerotinia rolfsii* Sacc. (teleomorph: *Athelia rolfsii* (Curzi) Tu & Kimbrough)
34.	Stem blight	No	*Nigrospora oryzae* (Berk. & Brown) Peteb
35.	Tip burn	TB(P)	Calcium deficiency
36.	*Verticillium* wilt	Vd,Va	*Verticillium dahliae* Kleb. *V. albo-atrum* Reinke & Berth.
37.	Virus diseases		
	(a) Cauliflower mosaic	CAM (V)	Cauliflower mosaic virus
	(b) Mustard mosaic	MM	Mustard mosaic virus
	(c) Radish mosaic	RM(V)	Radish mosaic virus
	(d) Turnip mosaic	TUM(V)	Turnip mosaic virus
	(e) Yellows	BMY(V)	Beet western yellows virus
38.	White rust, stag head	Ac	*Albugo candida* (Pers. Ex. Lev.) Kuntze (*Hyaloperonospora* sp. commonly present in stag head phase)
39.	White leaf spot, grey stem	Pc	*Pseudocercosporella capsellae* (Ell. & Ev.) Deighton
40.	*Xanthomonas* leaf spot	Xca	*Xanthomonas campestris* pv. *armoraciae* (Mc Culloch) Dye
41.	Yellows	Fo	*Fusarium oxysporum* Schlecht. spp.
42.	Chlorosis	LC	Loss of chlorophyll; S, Mg, N, P, K deficiency

[a]Pathogen symbol: *G* genetic disorder, *P* physiological disorder, *N* nematode, *V* virus

criteria for distinguishing *Peronospora* isolates from different host species, and all collections of downy mildew from the Cruciferae are currently grouped in the single aggregate species *P. parasitica* (Pers. ex Fr.) Fr. Constantinescu (1989) later proposed a new genus, *Paraperonospora*, to accommodate several species of *Peronospora* pathogenic on hosts in the family Compositae. Constantinescu and Fatehi (2002) splitted *Peronospora* into three separate genera, *Peronospora* s. str., *Hyaloperonospora*, and *Perofascia*, on the basis of morphology, ITS 1, ITS 2, and 5.8 S rDNA sequence analysis. The latest revisions are based on molecular phylogenetic methods. Interestingly, the latter two genera were found to be almost entirely restricted to a single host family, Brassicaceae. Within *Hyaloperonospora*, only six species were accepted because of the differences in morphology of conidia and conidiophores. This concept is similar to that of Yerkes and Shaw (1959) and de Bary (1863). There are two different patterns of host colonization: systemic and localized. Systemic infection is characterized by colonization of leaves, stems, and sometimes roots, mostly through the infection of the seedlings by primary inoculum. The symptoms vary from chlorotic discolouration to stunting and distortion of the whole plant. Localized infections are characterized by the occurrence of lesions on leaves, surrounded by a conspicuous characteristic white 'down' on the abaxial surface (Lucas and Sherriff 1988).

1.3 The Downy Mildew of Crucifers

The term mildew was first used in the USA to denote fungal growth on the surface of green and necrotic plant tissues. The word downy mildew came from the vine mildew introduced from North America which was quickly adapted by researchers of different countries. Persoon (1796) was the first person who described downy mildew of crucifers on *Capsella bursa-pastoris*. All isolates obtained from cruciferous hosts were ascribed to *Peronospora parasitica* (Pers. ex. Fr.) Fr. However, Gaumann (1918) named isolates of *Peronospora* affecting plants of *Brassica* species as *P. brassicae* Gaum. After several arguments and controversies among mycologists and taxonomists and following an extensive biometric study, over 80 species names were reduced to one synonym, and a single species, i.e. *P. parasitica*, was recognized on cruciferous hosts (Dickinson and Greenhalgh 1977; Hiura and Kanegae 1934; Waterhouse 1973; Yerkes and Shaw 1959). Based on molecular and morphological features, the large genus *Peronospora* was segregated by Constantinescu and Fatehi (2002) into two genera, i.e. *Hyaloperonospora* and *Perofascia*. Based on molecular, morphological, ITS1, ITS2, and 5.8 S rDNA sequence analysis and phylogenetic investigations, *Hyaloperonospora* was demonstrated to be the pathogen on cruciferous hosts with specificity of *H. parasitica* to *Capsella bursa-pastoris*, *H. brassicae* to Brassicaceae, and *H. arabidopsidis* to *Arabidopsis thaliana* (Choi et al. 2003; Goker et al. 2003, 2004, 2007, 2009a, Voglmayr 2003). Since, the first report of downy mildew disease on crucifers in 1796, periodic milestones set, and significant historical development in the downy mildew research have been outlined in Table 1.3. It is quite clear from the

Table 1.3 Significant historical developments in the downy mildew research of crucifers

Period	Milestones in discovery/first report of downy mildew
1796–1918	Downy mildew as a disease of crucifers was reported for the first time on *Capsella bursa-pastoris* (Persoon, 1796; Corda, 1837; Gaumann, 1918)
1918	On the basis of conidial measurements and cross-inoculation tests, 52 species of *Peronospora* on crucifers were recognized (Gaumann, 1918). Pathogen was identified as *P. parasitica* (Pers. Ex. Fr.) Fr. Sum. Veg. Scand. 193, 1849
1959	All collections of downy mildew from the crucifers were grouped in the single aggregate species *P. parasitica* (Pers. Ex. Fr.) Fr. (Yorkes and Shaw, 1959)
1918–2005	Yield losses ranging from 50 to 60 percent were estimated due to downy mildew infections in *Brassica* oilseeds and vegetables (Butler, 1918; Vasileva, 1976; Bains and Jhooty, 1979; Kolte, 1985; Achar, 1992; Saharan, 1984, 1992; Koike, 1998; Davis et al., 1997; Paul et al., 1998; Mahajan and Gill, 1993; Singh and Singh, 2005)
1918–2006	Symptoms of downy mildew on crucifers crops were described in details (Butler, 1918; Vasudeva, 1958; Kolte, 1985; Saharan et al., 1997; Sherf and Macnab, 1986; Ramsay and Smith, 1961; Natti et al., 1956; Gram and Weber, 1952; Jafar, 1963; Chou, 1970; Slusarenko and Schlaich, 2003; Larren et al., 2006)
1967–2012	Different disease assessment scales to assess leaf and stag head infection were suggested (Natti et al., 1967; Sadowaski, 1987; Ebrahini et al., 1976; Dickinson and Greenhalgh, 1977; Knight and Furber, 1980; Nashaat and Rawlinson, 1994; Saharan, 1992; Williams, 1985; Kruger, 1991; Brophy and Laing, 1992; Jensen et al., 1999; Monterio et al., 2005; Coelho et al., 2012)
1918–1990	Reproduction and reproductive structures of *H. peronospora* were described in details (Butler, 1918; Fraymouth, 1956; Holliday, 1980; Ohguchi and Asada, 1990; Chu, 1935; Walker, 1946, Channon, 1981; Preece et al., 1967; Wager, 1900)
1956–2003	Host penetration, haustorium development, host-pathogen interface, conidia, and conidiophore development, cytology, and genetics were studied through light and electron microscopy to reveal fine structures (Fraymouth, 1956; Chou, 1970; Davison, 1968; Shiraishu, 1975; Sansome and Sansome, 1974; Ehrlich and Ehrlich, 1966; Soylu and Soylu, 2003)
1920–2012	Pathogenic variability and specificity in the downy mildew pathogen on crucifers were determined (Gardener, 1920; Kabel, 1921; Gaumann, 1926; Thung, 1926; Lucas et al., 1988, 1994; Uknes et al., 1992; Nashaat et al., 1995; Hiura and Kanegae, 1934; Wang, 1944; Felton and walker, 1946; Natti, 1958; Knox-Davis, 1980; Dzhanuzakov, 1963; Chang et al., 1964; Semb, 1969; Dickinson and Greenhalgh, 1977; Bains and Jhooty, 1983; Mehta and Saharan, 1994; Masheva et al., 1996; Moss et al., 1991, 1994; Silue et al., 1996; Nashaat and Rawlinson, 1994; Sherriff and Lucas, 1990, 1994; Hill et al., 1988; Sequeira and Monteiro, 1996; Yashida and Ohguchi, 1998; Silve et al., 1996; Lackie et al., 1996; Rehmany et al., 2000; Vishunavat et al., 1998; Coelho et al., 2012)
1937–1996	Heterothallism and homothallism both were observed for sexual reproduction in the pathogen (De Bruyn, 1937; Mc Meekin, 1960; Kluczewski and Lucas, 1983; Sherriff and Lucas, 1989; Sequeira and Monteiro, 1996)

(continued)

Table 1.3 (continued)

Period	Milestones in discovery/first report of downy mildew
1926–1998	Survival and perpetuation through mycelium, conidia, and oospores; germination and dispersal of conidia and oospores were observed (Gaumann, 1926; Chang et al., 1963; Jang and Safeeulla, 1990; Krober, 1970, 1981; Le beau, 1945; McMeekin, 1960; Jang and Safeeulla, 1990; Vishunavat and Kolte, 1993; Ohguchi and Asada, 1989; Lin and Liang, 1974; Shao et al., 1990; Pinckard, 1942; Lin, 1981; Hartman et al., 1983; Achar, 1995, 1998; Badul and Achar, 1998; Karuna and Kolte, 1993; Paul et al., 1998; Guo and Ohguchi, 1996)
1918–2003	The process of infection and pathogenesis of downy mildew pathogen in different crucifers was described (Butler, 1918; Preece et al., 1967; Chou, 1970; Shiraishi et al., 1975; Kroher, 1969; Achar, 1992; Chu, 1935; Felton and walker, 1946; Jonsson, 1966; Le Beau, 1945; Chang et al., 1963; Jang and Safeeulla, 1990; Kluczewski and Lucas, 1982; Singh et al., 1980; Koch and Slusarenko, 1990; Soylu and Soylu, 2003)
1945–2005	Disease cycle of downy mildew on cruciferous hosts was explained (Le Beau, 1945; Chang et al., 1963; Shiraishi et al., 1975; Lucas et al., 1995; Saharan et al., 2005; Holub et al., 1994)
1935–2010	Epidemiology of downy mildew was initiated with the influence of temperature, humidity, rainfall, and leaf wetness on infection and disease development (Chu, 1935; Eddins, 1943; Walker,1946; Nashaat, 1997; Chou, 1970; Nakov, 1972; Williams and Leung, 1981; Jonssen 1966; D'Ercole, 1975; Bains and Jhooty, 1979; Mehta et al., 1995; Kolte et al., 1986; Vladimirskaya et al., 1975; Achar, 1998; Alaonso and Alonso, 1995; Sangeetha and Siddaramaiah, 2007; Banerjee et al., 2010)
1928–2015	The development of disease in relation to planting time and host nutrition was recorded (Saharan, 1984; Kolte et al., 1986; Mehta, 1993; Quanjer, 1928; Townsend, 1935; Butler and Jones, 1949; Falton and walker, 1946; Hammarlund, 1931; Petraitiene and Brazauskiene, 2005; Sochting and Verret, 2004; Jiang and Caldwell, 2015)
1986–1998	Downy mildew prediction models were developed (Kolte et al., 1986; Mehta and Saharan, 1998)
1918–2014	The association and mixed infection of downy mildew with white rust especially at leaf and stag head stage was recorded (Butler, 1918; Wiese, 1927; Boning, 1936;Bains and Jhooty, 1978, 1985; Chaurasia et al., 1982; Saharan and Verma, 1992; Choudhary and Verma, 1987; Mehta et al., 1995; Singh et al., 2002; Saharan et al., 2014)
1974–2003	Biochemistry of host pathogen interaction was studied in relation to metabolic changes and role of natural biochemical compounds in host resistance (Thorntan and Cooke, 1974; Kluezewski and Lucas, 1982; Singh et al., 1980; Davis and Ausubel, 1989; Delaney et al., 1994; Mansfield, 2000; Scheideler et al., 2002; Slusarenko and Schlaich, 2003)
1949–1997	Mechanism of host resistance against downy mildew was studied at various levels, viz. pre-penetration and postinfection (Wang, 1949; Kluezewski and Lucas, 1992; Ohguchi and Asada, 1991; Saharan et al., 1997)
1993–1996	Host-pathogen recognition system was studied using *Arabidopsis-Hyaloperonospora* model system (Davis and Hammerschmidt, 1993; Lebeda and Schwinn, 1994; Holub et al., 1994; Reignault et al., 1996; Joos et al., 1996)

1992–1998	Systemic acquired resistance was demonstrated in *Arabidopsis* plants treated with chemical inducers (Uknes et al., 1992; Lawton et al., 1996; Zimmerli et al., 2000; Bowling et al., 1997; Clarke et al., 1992; Cao Hui et al., 1998; Mauch–Mani and Slusarenko, 1996)
1967–2012	Growth of host-pathogen relationship studies indicated that host resistance was governed by dominant genes (Natti et al., 1967; Hoser-Krause et al., 1991, 1995; Niu et al., 1983; Bennet and Blancard, 1987; Lucas et al., 1988; Nashaat et al., 1995; 1996,1997, 2004; Jensen et al., 1999; Reignoult et al., 1996; Joos et al., 1996; Dickson and Petzoldt, 1996; Carvalho and Montario, 1996; Vicente et al., 2012)
1983–1999	Quantitative or partial resistance to downy mildew was identified (Leung and Williams, 1983; Dickson and Petzoldt, 1996; Leung and Williams, 1983; Jensen et al., 1999)
1976–1999	Biochemical basis of resistance was demonstrated (Greenhalgh and Mitchell, 1976; Rawlinson et al., 1989; Nashaat and Rawlinson, 1994; Daughty et al., 1995; Menard et al., 1999; Glazebrook et al., 1997)
1972–1994	Role of lignin formation in the host cell walls was demonstrated to provide resistance (Asada and Matsumoto, 1969, 1972; Ohguchi et al., 1974; Ohguchi and Asada, 1975; Matsumoto et al., 1978; Asada et al., 1975; Matsumoto and Asada, 1984; Matsumoto, 1994)
1966–1997	Sources of resistance to downy mildew were identified in cruciferous crops which had been used and are being used for breeding downy mildew resistance cvs. Through conventional and biotechnological techniques (Jonsson, 1966; Bonnet and Blancard, 1987; Lucas et al., 1988; Nashaat and Rawlinson, 1994; Nashaat and Awasthi, 1995; Nashaat et al., 1997; Silue et al., 1996; Saharan et al., 1997; Ebrahimi et al., 1976; Greenhalgh and Mitcheli, 1976; Greenhalgh and Dickinson, 1975)
1918–2005	To manage downy mildew of crucifers, several strategies (cultural, chemical, biological resistance) including integrated disease management were suggested (Butler, 1918; Vasudeva, 1958; Conroy, 1960; Schmidt, 1960; Sherf and Macnab,1986;Downy and Bolton, 1996; Kolte, 1985; Saharan, 1984,1992; Saharan et al., 1997, 2005; Kupryanova, 1957; Pauls and Nelson, 1977; Crute, 1984; White et al., 1984; Mehta et al., 1996; Ryan, 1977; Chiu, 1959; Davies and Wafford, 1987; Chann, 1981; Channon et al., 1970; Whitewell and Griffin, 1967; Ryan et al., 1984; Brophy and Laing, 1992; McKay et al., 1992; Yang et al., 1983; Sharma and Sohi, 1982; Mehta, 1993; Ark and Thompson, 1959; Nicolas and Aggery, 1940; Crute et al., 1985; Silue et al., 1996; Shao et al., 1991)
2003–2008	Phylogenetic investigations and molecular data were used to circumscribe species concept of downy mildew pathogen (Choi et al., 2003, 2005, 2006, 2007; Voglonayr, 2003; Goker et al., 2004, 2007; Scott et al., 2004; Cunnington, 2006; Spring et al., 2006; Voglmayr et al., 2006; Landa et al., 2007; Garcia-Blazguez et al., 2008)
2002–2007	On cruciferous hosts, the downy mildew pathogen species identified and established were *Hyaloperonospora parasitica* on *Capsella bursa-pastoris*, *H. brassicae* on Brassicaceae, and *H. arabidopsidis* on *Arabidopsis thaliana* (Constantinescu and Fatehi, 2002; Choi et al., 2003; Goker et al., 2003, 2004, 2007; Voglmaur, 2003)

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Table 1.3 (continued)

Period	Milestones in discovery/first report of downy mildew
1990 to date	Era of genomics and molecular genetics/molecular plant pathology. Natural variation of DM resistance in *Arabidopsis thaliana* (At) was described as a model for molecular genetic investigation (Koch and Slusarenko, 1990). The DM isolate Emoy2 was borne from an oospore in a seedling of *Arabidopsis thaliana* 'Columbia'. This isolate would be used to establish genetics in the organism and provide the first reference genome of downy mildew parasites (Holub, 2006). Mutation of NDR1 demonstrates that H.H. Flor's notion of disease resistance being conferred by single R genes can actually be a multigenic process and involve common links in the signalling of defence against diverse pathogens. This established a precedent for using oomycete and bacterial pathogens of *Arabidopsis thaliana* in comparative laboratory experiments (Century et al., 1995, 1997). Systemic acquired resistance to bacterial disease and DM in *Arabidopsis thaliana* was found to require salicylic acid (Lawton et al., 1995). Mutation of EDS1 demonstrates that species level barriers (non-host resistance) to biotrophic parasites can be amenable to mutation and genetic analysis. This gene and PAD4, which is also typically required for DM resistance in *Arabidopsis thaliana*, were found to encode lipase-like proteins (Parker et al., 1996; Glazebrook et al., 1997; Falk et al.,1999; Jirage et al., 1999; Holub and Cooper, 2004). The first DM resistance gene RPP5 was cloned and found to encode a member of the previously described TIR-NBS-LRR class of cytoplasmic receptor-like proteins (Parker et al., 1997). Major R-gene clusters were revealed on four chromosomes of *Arabidopsis thaliana* using a powerful combination of recombinant inbred *Arabidopsis thaliana* populations and DM isolates as physiological probes to map *RPP* loci (Holub and Beynon, 1997). R-like homologues provide a powerful class of molecular markers for map-based new oomycete resistance genes in *Arabidopsis thaliana* and in crops such as lettuce and potato (Aarts et al., 1998a, b; Botella et al., 1997; Speulman et al., 1998). The multicopy locus *RPP1* contains several DM resistance genes (TIR-NBS-LRR subclass) that differ in specificity (Botella et al., 1998). DM resistance genes vary in how they confer defence via different regulatory proteins (Aarts et al., 1998a, b; Eulgem et al., 2004). A single DM resistance gene (*RPP7*) can confer accumulative (salicylic acid dependent and independent) defence responses (McDowell et al., 2000; Tor et al., 2002; Eulgem et al., 2007). DM isolates collected from *Arabidopsis thaliana* appear to be phylogenetically distinct from *Brassica* isolates: referred to hence as subsp. *Arabidopsidis* (*HpA*) or *Brassica* (*HpB*) (Rehmany et al., 2000). *RPP13* was cloned, encoding a protein homologous to *RPP8* and providing the most extreme benchmark for allelic diversification of a receptor-like gene in *Arabidopsis thaliana* (Bittner-Eddy et al., 2000; Rose et al., 2004). 'Gene-for-gene' paradigm was established in the *At-HpA* pathosystem. An outcross of *HpA* enables genetic evidence for five independent At-recognizable effectors (*ATR1*, *ATR4*, *ATR5*, *ATR8*, and *ATR13*) that correspond with different cloned DM resistance genes (Gunn et al., 2002). The DM parasite of crucifer species (previously *Peronospora parasitica*) was renamed as *Hyaloperonospora parasitica* (Constantinescu and Fatehi, 2002). *SGT1b* and *RAR1*/*PBS2* provide evidence for highly conserved regulators (also found in monocots) and the likely involvement of proteolysis in defence signalling (Austin et al., 2002; Muskett et al., 2002; Tor et al., 2002; Tornero et al., 2002; Warren et al., 1999). *A. thaliana*-oomycete molecular ecology was launched (Damgaard and Jensen, 2002). The first *Arabidopsis thaliana* recognized effector (*ATR13*) was cloned from *HpA* isolate Maks9 and found to encode a small secreted protein that exhibits a high degree of sequence variation among UK isolates of *HpA* (Allen et al., 2004). Enhanced downy mildew resistance (DMR) mutants were described and launched the genetic analyses of induced accessibility for oomycete parasites in *Arabidopsis thaliana* (Van Damme et al., 2005; Holub, 2006). Differential expression of R genes has been identified (Xiao et al., 2016). 129 TDFs have been identified of which 121 TDFs are upregulated and 8 are downregulated (Xiao et al., 2016)

The significant historical developments in the downy mildew research of crucifers have been arranged in chronological order after analysing the published literature so far. Reference details are given in respective chapters

milestones set by Gaumann (1918) on various aspects/areas of research which were further pursued and strengthened to comprehend the downy mildews on cruciferous crops all over the world by various researchers. The validation of Gaumann's research by others on crucifer downy mildew warrants that Gaumann may be considered as father of crucifers downy mildew disease.

The third largest group of downy mildews, which is mostly restricted to one plant family, is the brassicolous downy mildews (BDM), even though a few species of this group are parasitic to other plant families, such as Capparaceae, Resedaceae, Limnanthaceae, Cistaceae, and Zygophyllaceae (Constantinescu and Fatehi 2002; Goker et al. 2009b). The BDM contain about 100 known species (Constantinescu 1991), but there are several species level clades which have not yet been formally described (Goker et al. 2009b). Importantly, it also includes an economically relevant but yet undescribed species occurring on arugula (or rocket; *Eruca sativa*). Without quarantine regulations in effect, it is fast-spreading throughout the world (Goker et al. 2009b; Koike 1998). The BDM contain two genera, *Hyaloperonospora* and *Perofascia*, of which the monotypic genus *Perofascia* is characterized by sporangiophores that often intertwine, thick-walled oospores, and hyphal haustoria. The genus seems to be restricted to the Brassicaceae tribe Lepidieae (Constantinescu and Fatehi 2002). On plants belonging to the same tribe, but also on plants of about 20 other tribes of Brassicaceae, the genus *Hyaloperonospora* can be found.

Hyaloperonospora is characterized by treelike sporangiophores, comparatively thin-walled oospores, and globose to lobate haustoria. Like *Perofascia*, this genus was segregated from *Peronospora* only after molecular phylogenetic analyses were able to prove distinctiveness from *Peronospora* (Constantinescu and Fatehi 2002; Riethmuller et al. 2002; Voglmayr 2003), rendering them the first two downy mildew genera described with the aid of molecular phylogenies. The genus *Hyaloperonospora* is the third largest genus of downy mildews, containing more than 100 species, which can infect economically important Brassicaceae crops, e.g. horseradish (*Armoracia rusticana*), mustard greens (*Brassica juncea*), rapeseed (*B. napus*), cabbage (*B. oleracea*), Chinese cabbage (*B. rapa*), arugula (*Eruca sativa*), wasabi (*Eutrema japonicum*), watercress (*Nasturtium officinale*), and radish (*Raphanus sativus*). The downy mildew of *Arabidopsis thaliana* and *Hyaloperonospora arabidopsidis* (Goker et al. 2009b) has become a model organism to dissect plant pathogen interactions (Baxter et al. 2010; Coates and Beynon 2010). The BDM have been monographed by Gaumann (1918), and this almost 100-year-old work is still the most important source of reference for this group, together with the list of downy mildew names at that time placed in *Peronospora* by Constantinescu (1991). Since this last monograph, only few new species of this genus have been published (Voglmayr et al. 2014a), despite the fact that numerous new species-level clades have been found in phylogenetic analyses (Goker et al. 2009b; Thines and Choi 2016).

The downy mildew of crucifers is a very widely destructive disease all over the world wherever cruciferous plant species, cultivated or wild, are available (Table 1.1). The disease is very devastating causing yield losses in oil-yielding *Brassica* crops and cruciferous vegetable crops ranging from 50 to 100% depending

upon amount of pathogen inoculum present in the soil or near vicinity of host, favourable environmental conditions present for infection, and development, cultural practices adopted, and disease management practices followed. The information generated by the dedicated research workers so far on downy mildew of crucifers have been arranged in the present book in 16 chapters with appropriate headings and subheadings in numerical order. The present manuscript on downy mildew of crucifers deals with the various aspects, viz. the disease and its synonymous; geographical distributions and symptomatology on oil-yielding *Brassica* crops, cruciferous vegetables, and other economically important host plants; host ranges; yield losses and disease assessment scales/procedures; the pathogen, its taxonomy, and classification, phylogeny, and pathogenic variability; pathogen morphology, sporulation, perpetuation, and germination; host-parasite interaction; seed infection, process of infection, and pathogenesis; disease cycle; epidemiology and forecasting; fine structures observed though light and electron microscopy; biochemical changes during host-parasite interaction; host resistance, its sources, and genetical, biochemical, morphological, and histological mechanism of inheritance; molecular aspects dealing with cloning, mapping, and identification of resistance genes; disease management practices like cultural, chemical, biological, and integrated control; and standardized techniques on all aspects have been included. Chapter 15 deals with the future priorities of research on downy mildew of cruciferous crops for resolving unanswered questions by the researchers in the time to come. In the last Chapter, 16, subject index has been given to facilitate the readers to search desired information included in different chapters of the book.

1.4 The Pathogen/Causal Organism of Downy Mildew of Crucifers

In the literature published up to 2002, the pathogen/causal organism of downy mildew of cruciferous crops has been referred as *Peronospora parasitica* (Pers. Ex. Fr.) Fr under a very large genus *Peronospora* infecting large number of diverse kinds of plant families. In the present book, to keep the coherence of text, the pathogen has been referred as *Hyaloperonospora parasitica* (Gaum.) Goker. Based on molecular, morphological, and phylogenetical studies, taxonomists and mycologists have established three different species of downy mildew pathogen on crucifers, viz. *Hyaloperonospora parasitica* (Gaum.) Goker on *Capsella bursa-pastoris*, *H. brassicae* (Gaum.) Goker on Brassicaceae, and *H. arabidopsidis* (Gaum.) Goker on *Arabidopsis thaliana*.

1.5 Taxonomy and Classification of Downy Mildew Pathogen

The downy mildews are very large group of obligate parasite infecting the hosts of very diverse families of plants. The downy mildews (Peronosporaceae) in the traditional sense are morphologically diverse group which is mainly united by obligate parasitism in combination with more or less complex conidio- or sporangiophores with determinate growth. Even after recent molecular and phylogenetic analysis, it is still uncertain whether downy mildews are monophyletic or stem from different groups. The comparison of downy mildews order, family, and generic level by renowned taxonomists/mycologists is given in Table 1.4 including downy mildew

Table 1.4 Comparison of some ordinal, family, and generic classifications of downy mildews, white blister/rusts, and relatives

Waterhouse (1973)	Kirk et al. (2001)	Riethmuller et al. (2002)	Goker et al. (2007), Thines and Spring (2005)
Peronosporales	Peronosporales	(no order name)	Peronosporales
Peronosporaceae	Peronosporaceae	Peronosporaceae	Peronosporaceae
Basidiophora	*Basidiophora*	*Basidiophora*	*Basidiophora*
Bremia	*Benua*	(*Benua*)	*Benua*
Bremiella	*Bremia*	*Bremia*	*Bremia*
Peronospora	*Bremiella*	*Paraperonospora*	*Graminivora*
Plasmopara	*Paraperonospora*	*Peronophythora*	*Hyaloperonospora*
Pseudoperonospora	*Peronospora*	(*Peronosclerospora*)	*Paraperonospora*
Sclerospora	*Plasmopara*	*Peronospora*	*Perofascia*
Albuginaceae	*Pseudoperonospora*	*Phytophthora*	*Peronosclerospora*
Albugo	Albuginaceae	*Peronospora*	*Peronospora*
Pythiaceae	*Albugo*	*Plasmopara*	*Peronospora*
Phytophthora	Pythiales	*Pseudoperonospora*	*Plasmopara*
Pythiogeton	Pythiaceae	*Sclerospora*	*Plasmoverna*
Pythium	*Halophytophthora*	Albuginaceae	Protobremia
Sclerophthora	*Peronophythora*	*Albugo*	*Pseudoperonospora*
Trachysphaera	*Phytophthora*	Pythiaceae	Sclerospora
	Pythium	*Lagenidium*	Viennotia
	Trachysphaera	*Pythium*	(family not formally classified)
	Pythiogetonaceae	(*Pythiogeton*)	*Phytophthora*
	Pythiogeton	(*Trachysphaera*)	Albuginales
	Sclerosporales	(*Sclerophthora*)	Albuginaceae
	Sclerosporaceae		*Albugo*
	Peronosclerospora		*Pustula*
	Sclerospora		*Wilsoniana*
	Verrucalvaceae		
	Sclerophthora		

of crucifers. The taxonomic classification of *Hyaloperonospora parasitica* causing downy mildew of crucifers is as follows:

Kingdom	Mycota
Subkingdom	Eumycota
Division	Mastigomycotina
Class	Oomycete
Order	Peronosporales
Family	Peronosporaceae
Genus	*Hyaloperonospora parasitica* (Gaum.) Goker (*Peronospora parasitica*) *H. brassicae* (Gaum.) Goker on Brassicaceae (*Peronospora brassicae*) *H. arabidopsidis* (Gaum.) Goker on *Arabidopsis thaliana* *H. arabidopsidis* (Gaum.) Goker, Rieth:, Voglmayr, Weiss and Oberw [as *Arabidopsis*]. Mycol. Prog. 3(2): 89 (2004). Synonymy *Peronospora arabidopsidis* Gaum. (1918)

1.6 Current Generic Status of Downy Mildew of Crucifers

Generic concepts in downy mildews were (and still are) mainly based on conidio−/sporangiophore morphology in combination with conidial/sporangial morphology. Dichotomous versus monopodial branching of conidio−/sporangiophore, shape of the terminal branches, and presence of conidia or sporangia were the primary features used for genus classification. However, interpretation of these morphological features was not always unequivocal and dependent on the observer's vision, which sometimes resulted in conflicting generic concepts and delimitation.

With the availability of molecular phylogenies, it soon became apparent that current generic classification and circumscription contained numerous problems and had to be adapted if standards of phylogenetic classification were applied. Based on molecular and morphological features, the genera *Hyaloperonospora* and *Perofascia* were segregated from the large genus *Peronospora* (Constantinescu and Faheti 2002).

1.7 Species Concepts in Crucifer's Downy Mildew

The species concept is probably the most controversial issue in downy mildew systematics, partly due to experimental difficulties to test it and partly due to its profound implications for researchers outside the systematic research community. In

downy mildews, several species concepts were applied, which resulted in highly different numbers of accepted species depending on the criteria used. The main problem in species delimitation in downy mildews is that there are numerous indications that, due to their obligate parasitism, they often have narrow host ranges and, therefore, represent genetically distinct species. On the other hand, host specificity is not always paralleled by morphological distinctness. Therefore, if morphology is used as a primary criterion for species definition, only a few species can be defined and accepted in many lineages, resulting in genetically heterogeneous species. Historically, two approaches were commonly followed, which were both mainly based on host ranges: the splitting approach of Gaumann (1918, 1923) versus the lumping approach of Yerkes and Shaw (1959). Gaumann (1918, 1923) advocated a narrow species concept in *Peronospora*, based on his results of cross-inoculation studies and minute morphological differences. Each species was usually assumed to be confined to one host genus or even a few host species (one host-one species concept). Conversely, Yerkes and Shaw (1959) argued that host specificity is neither sufficient nor suitable for the recognition of a species without clear-cut morphological differences. As a consequence, the numerous *Peronospora* species were recognized on Brassicaceae and Chenopodiaceae each into a single species (*Peronospora parasitica* and *P. farinosa*, respectively), resulting in a wide one host family-one species concept.

Both the splitting and the lumping approach have sincere shortcomings. Using the narrow species concept, identification of morphologically similar species is often difficult or impossible without correct identification of the host. In addition, high host specificity has rarely been conclusively demonstrated, weakening the primary underlying assumption of the narrow species concept. In a wide species concept, there is the problem that genetically distinct or even unrelated entities may be classified in the same species, raising incorrect assumptions on biology and host ranges. This is especially problematic if host jumps are common and parasitism on the same host family has evolved multiple times, resulting in polyphyletic species. However, due to its easier applicability, the approach to classify all accessions of a given host family within a single species was widely followed by phytopathologists and molecular biologists.

1.8 Broad and Narrow Species Concepts

With respect to species concepts in downy mildews and in particular in the genus *Peronospora,* there have long been two conflicting views – the narrow species concept, advocated by Gaumann (1918, 1923) and Gustavsson (1959), and the broad species concept advocated by de Bary (1863) and Yerkes and Shaw (1959). While the narrow species concept was followed by most taxonomists, the broad species concept, which ascribed the host specificity of some downy mildew pathogens to specialized forms (formae speciales) of the same species, was mostly followed by applied plant pathologists. This schism made the sequencing of the *Arabidopsis*

downy mildew seems to be of direct importance to plant pathologists working on economically important Brassicaceae crops. However, due to the fact that the downy mildew species are only distantly related, even though the genome of *Hyaloperonospora arabidopsidis* has provided important insights into downy mildew evolution (Baxter et al. 2010), the findings are difficult to translate into applications in *Brassica* crops. In general, molecular phylogenetic analyses have provided solid evidence for a high degree of specialization for most downy mildew species, also including the genera *Hyaloperonospora* (Goker et al. 2009b; Voglmayr et al. 2014a) and *Peronospora* (Belbahri et al. 2005; Choi et al. 2007b, 2008, 2009, 2010, 2015; Thines et al. 2009; Voglmayr et al. 2014b). There are only rare exceptions – a few downy mildew species seem to have broad host ranges (Kenneth 1981; Runge et al. 2011). However, in line with molecular phylogenetics, infection trials have shown that downy mildews are generally highly host specific (Byford 1967; Gaumann 1918, 1923; Lebeda and Syrovatko 1988; Sherriff and Lucas 1990). Thus, it seems reasonable to treat emerging downy mildew diseases on new hosts as separate species – especially with respect to quarantine – until infection trials and phylogenetic investigations have revealed whether or not they are highly host specific. Had this been done in the past, it might have been possible to restrict pandemic downy mildew agents, such as *Peronospora belbahrii* from sweet basil, *Peronospora somniferi* from opium poppy, *Peronospora salvia officinalis* from sages, and *Plasmopara obducens* s.l. from cultivated *Impatiens* species, preventing high yield losses around the world (Thines and Choi 2016).

1.9 Use of Molecular Data for Downy Mildew Species Concept

Recently, molecular phylogenetic investigations have enabled the evaluation of the species problem using new perspectives and have led to the shift from a morphological to a phylogenetic species concept. A biological species concept directly addressing mating barriers has never been applied to downy mildews due to sincere methodological difficulties, and it is unlikely that these can be overcome. Therefore, reproductive isolation can only be indirectly assessed, e.g. by genetic distance of sequence data. The impact of molecular data is manifold: (1) numerous additional characters are available for recognition and distinction; (2) presence and amount of reproductive isolation can be assessed; (3) presence and amount of genetic distances provide indirect but strong evidence for host specificity and host ranges; (4) molecular data are less variable and prone to subjective interpretation than morphological data; (5) molecular data provide a sound basis for species identification even if morphological data are missing or incomplete; and (6) pathotypes or races, the basic entities for experiments in applied sciences, can be properly attributed to a species, and their phylogenetic relationships can be assessed. Therefore, in the absence of sound morphological characters, the species concept is increasingly based on

molecular evidence of reproductive isolation, which is a general tendency within mycology. Consequently, morphologically similar cryptic species are often recognized as distinct species if reproductive isolation and genetic distinctness can be demonstrated. However, evaluation of species boundaries by molecular data requires thorough sampling throughout the distribution area to assess genetic variability as well as reproductive isolation, and at best several molecular markers should be used for corroboration of species boundaries. Due to easy amplification and variability, the ITS rDNA region has been used in most investigations addressing the species concept in downy mildews and white blister rusts (Choi et al. 2003, 2005, 2006, 2007a, b, c, d; Voglmayr 2003; Goker et al. 2004; Scott et al. 2004; Cunnington 2006; Spring et al. 2006; Voglmayr et al. 2006; Landa et al. 2007; Garcia Blazquez et al. 2008). However, the mitochondrial COX2 region may also be a promising candidate to resolve species boundaries and for species identification (Choi et al. 2006, 2007d). Interestingly, the current evidence from molecular phylogenetic investigations often supports a narrow species concept as advocated by Gaumann (1918, 1923), although there are sometimes marked differences in detail.

1.10 *Hyaloperonospora* Species on Crucifers

According to Constantinescu and Faheti (2002), about 140 species names were published attributable to this genus. In their separation of *Hyaloperonospora* from *Peronospora*, Constantinescu and Faheti (2002) recognized only six morphologically distinct species. The accessions from most hosts of Brassicaceae were placed in *Hyaloperonospora parasitica*. However, subsequent molecular phylogenetic investigations demonstrated that the latter was a paraphyletic assemblage with respect to the other five *Hyaloperonospora* species and that many more species should be accepted based on the high genetic distances observed (Choi et al. 2003; Goker et al. 2003, 2004; Voglmayr 2003). Usually, these genetically distinct entities deserving species rank have a narrow host range and are confined to host genera or even species; however, in some cases, accessions from the same host do not form a monophylum (*Armoracia rusticana*; Goker et al. 2004). Therefore, it is problematic when species are determined solely on host association, as this is often but not always conclusive. The case study of *Hyaloperonospora* is also relevant for investigations at the molecular level of plant-pathogen interactions, as numerous studies are performed with the plant model organism *Arabidopsis thaliana* and its *Hyaloperonospora* parasite. The parasite is usually named *H. parasitica*, but it is genetically quite distinct from *H. parasitica* sensu stricto which is confined to *Capsella bursa-pastoris* (Goker et al. 2004); therefore, the name *H. arabidopsidis* should be used for the *Arabidopsis* parasite.

1.11 Strategies to Breed Downy Mildew Resistance Cultivars of Crucifers

The incompatible interaction between host and pathogen results into agriculturally important resistant phenotypes. Thus, any strategies that can contribute to the incompatible interaction are potentially useful in plant disease resistance breeding. On the one hand, incompatible interaction (R) can be converted into compatible interaction (S) in the case that host loses the related genes in immunity or pathogen evolves new virulence effectors (genes), especially in the gene-for-gene interaction of race-specific resistance in crucifer host-patho (*H. parasitica*) system. On the other hand, compatible interaction (S) can be converted into incompatible interaction (R) when the host or the pathogen loses the function of certain genes that are essential for pathogenesis or plant/host gains novel resistance genes. Incompatible interaction due to the loss of function mutation in a certain host gene is often of high value for disease resistance breeding programmes because the resistance is usually durable and non-race specific. Based on current information generated and understanding on the molecular mechanisms of crucifers, *H. parasitica* interactions following strategies for developing downy mildew resistance cvs. of crucifers may be adopted.

1.11.1 Identification and Utilization of Receptor-Like Kinases Involved in Plant Immunity

The first layer of immunity, termed pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI), is initiated upon the recognition of PAMP's by plant pattern recognition receptors (PRR) at the cell surface (Dodds and Rathjen 2010); PTI is often phenotypically reflected by basal resistance that is able to prevent infection by diverse potentially pathogenic pathogen (Catanzarite et al. 2010). The second layer of immunity is triggered upon the recognition of specific effector proteins from invading pathogens by host immune receptor proteins traditionally called resistance (R) proteins. Because a R protein specifically recognizes one or a few pathogen effectors (and the recognized effectors are termed avirulence factors or Avr), effector-triggered immunity (ETI) typically endows hosts with race-specific resistance, also known as R gene-mediated resistance against well-adopted pathogen carrying the recognized Avr genes. PTI is highly conserved at or above the species level. ETI is often polymorphic within a particular plant species with some cvs. Being resistant and others being susceptible. Due to its importance and genetic amenability, ETI has been extensively studied, and R genes have been widely exploited in crop production. However, large-scale deployment of elite cultivars carrying an R gene (i.e. monoculture) imposes higher selection pressure on the pathogen carrying the cognate Avr gene to survive, resulting in the modification or depletion of the recognized Avr gene or generation of novel effector gene that can escape the

recognition of the old R gene. This R-Avr interactive co-evolution explains why many R cvs. in field lose their resistance in a relatively short period of time. In this regard, exploiting new knowledge on the molecular mechanisms of PTI and durable and broad-spectrum resistance has become important in disease resistance breeding programme of crucifers host – downy mildew pathosystem (Jones and Dangl 2006; Li et al. 2013). Thus, screening and employment of receptor-like proteins involved in plant immunity appear to be very promising strategies for creating crop cultivars or germplasm with broad-spectrum and durable resistance in cruciferous crops.

1.11.2 Identification and Utilization of R Genes Involved in ETI

Most of the characterized R proteins involved in ETI belong to NB-LRR family and are extensively exploited in crop breeding and production. Some R genes contribute to broad-spectrum resistance because they confer resistance against a large part of strains of a pathogen. Identification of novel broad-spectrum resistance R genes involved in ETI and using molecular markers for such genes can highly improve selection efficiency in breeding programme for disease resistance. Whole-genome sequencing of plant pathogens in generating an increasing list of effector proteins that can facilitate the identification of new R genes in crop plants or in their wild relatives is essential. A strategy of 'pyramiding' R genes can be exploited in which several R genes each of which recognizes a specific range of strains of a pathogen are introduced into a single plant via marker-assisted selection (Xiao et al. 2008; Dangl and Jones 2001).

1.11.3 The Utilization of Quantitative Trait Loci (QTLs)

Resistance-associated QTLs provide abundant resources for disease resistance breeding because they generally render non-race-specific and durable resistance. Some resistance-related QTL genes can be directly used in breeding and production of cruciferous crops.

1.11.4 Screening and Utilization of Recessive Gene-Mediated Broad-Spectrum Resistance

In host-pathogen interaction, there are some host genes whose functions are required for the pathogenesis of certain pathogens. When mutation occurs in such a gene, host containing the mutant gene usually confers broad-spectrum or non-race-specific resistance to that pathogen. It is possible to screen for resistance mutants

from mutagenized susceptible plant populations under conditions favourable for pathogenesis of certain diseases (Li et al. 2013). The mutated genes can be used in the investigation of the underlying mechanisms and breeding programme. Identification of such genes can be a novel approach in crucifiers.

1.11.5 Engineering Broad-Spectrum Resistance Through Biotechnology

The mechanism of host-pathogen interaction has been more thoroughly investigated in model host plant *Arabidopsis thaliana* and then in other crucifers. NPR1 is a key regulator of the expression of pathogenesis-related (PR) genes; PR1, PR2, and PR5 are of disease resistance response termed as systemic acquired resistance (SAR) in *Arabidopsis*. Ectopic expression of NPR1 leads to broad-spectrum resistance or enhanced resistance against pathogens. The resistance obtained due to NPR1 – or NPR1 homologous – overexpression is usually associated with faster and greater expression of the PR genes (Xiao 2012; Lacombe et al. 2010; Cao et al. 1997, 1998; Mukhtar et al. 2009). Another potential resource of broad-spectrum resistance is RPW 8.2 that confers salicylic acid-dependent resistance to a wide range of pathogens at the host pathogen interfacial membrane in *Arabidopsis*. RPW 8.2 enhances the callose deposition and induces the H_2O_2 accumulation in the invaded cell to limit the invasion of the pathogen (Wang et al. 2007, 2010; Collier et al. 2011). RNA interference (RNAi)-based host-induced gene silence (HIGS) is another promising biotechnology to create resistance in cvs. by knockdown of either the host or pathogen-originated virulence-related regulators. The principle of this strategy is to express a small RNA *in planta* that can target genes of pathogen to suppress the virulence (Li et al. 2013). Mathematical modelling that combines ecological parameters (to explain spatial and temporal changes in population) with evolutionary genetics (natural selection acting on multiple loci on interacting species) represents an enormous challenge for breeding downy mildew resistance cvs. of crucifers.

1.11.6 Designation and Nomenclature of Downy Mildew Resistance Genes (R Genes) and Isolates (Races/ Pathotypes)

Breeders have interogressed disease resistance (R) genes from both cultivated and wild cruciferous plants in their efforts to produce more resistant varieties. Even so, new races of downy mildew pathogen regularly evolve through sexual reproduction of the pathogen that can overcome individual R genes. As per the term ‘gene-for-gene’ hypothesis, a plant to exhibit resistance (incompatibility) to a pathogen, a R gene must be present in the plant, and a corresponding avirulence (AVR) gene must

be present in the pathogen. An absence of either leads to disease (compatibility). This led to the hypothesis (elicitor/receptor model) that R genes encode receptors that enable plant to detect the ingress of pathogens whose avirulence genes cause them to produce the corresponding legends. Thus, R-gene products might be expected to have two functions: first, molecular recognition, and second, activation of plant defence upon recognition. Historically, and as per convention, R genes have been designated in different host-pathogen interactions on the basis of name of a disease and/or a pathogen/host. To narrate some of the R genes, viz. Sr for stem rust, Lr for leaf rust, Yr for yellow rust, Pm for powdery mildew resistance of wheat, and WRR for white rust resistance of crucifers, are based on the name of the diseases caused in respective hosts. Resistant genes (R-genes) Hm1 confers resistance to maize leaf blight (*Helminthosporium maydis*, *Cochliobolus carbonum*), Xa 21 confers resistance to *Xanthomonas* of rice, Cf9, Cf2 confers resistance to *Colletotrichum fulvum* of tomato, RPP1, RPP5 confers resistance to *Peronospora parasitica* (*H. parasitica*) of crucifers are based on the names of the pathogens. R genes ATR1 and ATR13 conferring resistance to downy mildew pathogen (*H. arabidopsidis*) of *Arabidopsis thaliana* are based on the name of hosts generic and specific names (AT). In the past specificity loci (R genes) of *A. thaliana* were named as RPP loci (abbreviation of recognition of *P. parasitica* or else recognized by *P. parasitica* (AVR)) and were numbered consecutively (i.e. RPP1, RPP2, etc.). Specificity loci (AVR genes) of *P. parasitica* had been named ATR loci (abbreviation of *A. thaliana* recognized or else *A. thaliana* recognition) and were numbered the same as the corresponding RPP locus (R genes). This nomenclature is descriptive of an interaction regardless of which partner is actively recognizing the other. Ideally new loci (R gene) should be named strictly on the basis of genetic recombination. Unfortunate consequence of change of pathogen name from *P. parasitica* to *H. parasitica* to *H. arabidopsidis* is no longer intuitively connected with the downy mildew pathogen from its current name to recognize *P. parasitica* (RPP) gene designation. Such changes in the names of host (*Sisymbrium thalianum* (L.) Grey to *Arabidopsis thaliana* (L.) Heynh) and pathogen (*P. arabidopsidis* Gaum. to *P. parasitica* (Pers. ex. Fr.) Fr. to *H. parasitica* to *H. arabidopsidis* (Gaum.) Goker) may be confusing and irritating for students and researchers, but it is inevitable in this modern era of molecular genetics and phylogenetic analysis of living beings. However, there is a need to develop and adopt a standardized system and procedure for the designation of R genes. On the basis of host and pathogen (both) recognition which can reflect both in their interaction phenotype, i.e. ATHA1 and ATHA2 for recognition of R genes by *H. arabidopsidis* from *A. thaliana*; ATAC 1, ATAC 2, etc. for recognition of R genes by *A. candida* from *A. thaliana*; and BJHP 1 and BJHP 2 for recognition of R genes by *H. parasitica* from *B. juncea*. The downy mildew resistance genes (R genes) recognized by *H. parasitica* isolates (pathotypes) from *A. thaliana* accessions are given in Table 1.5. Similarly, for other crucifers, a uniform system may be adopted, viz. BNHP for *B. napus-H. parasitica*, BOHP for *B. oleracea-H. parasitica*, and BRHP for *B. rapa-H. parasitica* interaction phenotypes (R genes).

Designation and nomenclature of pathogenic isolates (races/pathotypes) have gone through evolutionary process and methods. (1) Initially, physiologic races were

Table 1.5 Resistance genes (R genes) identified in crucifers (*A. thaliana*) against downy mildew (*H. arabidopsidis*) isolates (pathotypes)

Arabidopsis accessions	R genes	Downy mildew isolates (pathotypes)
Col-0 (Columbia)	RPP-4	EMOY 2, EMWA 1
Col-0	RPP 2	CALA-2
WS-0 (Wassilewskija)	RPP 1A,B	EMOY 2
Ler-0 (Landsberg erecta)	RPP 5, RPP 8	EMOY 2, NOCO 2, EMWA 1
Ws-0	RPP 1A	CALA 2
Ws-0	RPP 1A, B, C	NOCO 2
Nd-1 (Niederzenz)	RPP 13 (ATR 13Nd)	MAKS 9, ASWA, EDCO, EMCO, GOCO
Ws-0	RPP 1 (ATR 1 Ws B)	MAKS 9
Rld 2 (Reschew)	RPP 11	WELA 1
Col-0	RPP 6	WELA
Col-0	RPP 7	HIKS
Nd-0	RPP 25	AHCO
Ler-0	RPP 27	HIKS
Ws-0	RPP 10	NOCO, EMOY, MAKS, COLA
Ws-0	RPP 14	NOCO, EMOY, MAKS
Nd-1	RPP 26	WACO
Nd-1	RPP 16	ASWA
Nd-1	RPP 17	EMCO
Ws-0	RPP 12	WELA
Col-0	RPP 18	HIND
Ler-0	RPP 23	GOWA
Ler-0	RPP 21	MADI, MAKS
Ler-0	RPP 22	ASWA
Ler-0	RPP 24	EDCO
Oy-0 (Oystese)	RPP 3	CALA
Wei-0 (Weiningen)	RPP 9	HIKS
Cola-0	RPP 19	HIND 4
Cola-0	RPP 20	WAND
Cola-0	RPP 28	HIND 2
	Tightly linked genes	
	RPP 1- Ws A	CALA, EMOY, HIKS, MAKS, NOCO
	RPP 1- Ws B	
	RPP 1-Ws C	

generally designated as numbers or letters in an arbitrary manner in order of their discovery, i.e. *Puccinia* spp., *Melampsora lini*, *Albugo candida*, *Peronospora parasitica*, etc. (2) An improvement over the use of arbitrary numbers or letters was Black's nomenclature in which the races were designated on the basis of their virulence on particular genes for resistance, i.e. an isolate of *Phytophthora infestans* attacking a potato cv. carrying the R genes, R1, was designated as race 1, the one attacking R4 as race 4, and an isolate attacking both R1 and R4 as race 1 and 4. The race which was avirulent on all the genes for resistance was designated as race 0. (3) Virulence formulae were proposed to designate races of stem, and leaf rust of wheat

virulent or avirulent on particular genes for resistance, e.g. the formula 6,7,10/5,8,9, 9a, 11 for a race of *P. graminis tritici*, indicates that the race is virulent on Sr6, Sr7, and Sr10 but avirulent on Sr5, Sr8, Sr9 a, and Sr11. (4) A very complicated method was proposed by Habgood using binary and decanary values. (5) A virulence analysis method was suggested using mobile nurseries in case of powdery mildew of barley.

Like with any other host-pathosystem, the designation and nomenclature of downy mildew of crucifers pathogenic isolates/races/pathotypes have not been standardized at International level. No standard method and procedure has been adopted. Each researcher has used his own vision and system to name the pathogenic isolates collected from different locations/countries from cruciferous host species/varieties/accessions (Table 5.12). However, a naming system for the isolates of *H. arabidopsidis* from *A. thaliana* was introduced by Dangl et al. (1992), Holub et al. (1994), and Slusarenko and Schlaich (2003) on the basis of geographical location and ecotypes infected. As, for example, an isolate collected from suburb of Zurich called *We*iningen and virulent on (among others) the ecotype *La*ndsberg erecta was named WELA using the first two letters of the location where the isolate was found (WE), combined with the first two letters of susceptible ecotype (LA). Thus, NOCO was found in Norwich and is virulent on Columbia, EMWA at East Malling, and is virulent on Wassilewskija. The isolate EM (East Malling, UK), CA (Canterbury, UK); WE (Weiningen), CH, and NO (Norwich, UK) and the susceptible host line used for the isolates, third and fourth letters OY=OY-0, LA=LA-*er*, ND=*Nd*-0, CO=*Col*-0, etc have been used. New isolates collected from the same location and maintained on the same host genotypes were distinguished by a number (e.g. EMOY1 and EMOY 2). However, the system and procedure of naming of an isolate should reflect both host-pathogen interactions to recognize avirulence gene (AVR gene) of the pathogen along with R gene of the host, i.e. HPBJ 1, HPBJ 2, etc., indicating *H. parasitica* isolate/pathotype recognized R genes 1 and 2 from *B. juncea* after interaction of isolate (pathotype) HPBJ1 and HPBJ2. Like international code of botanical nomenclature for naming an organism, a pattern or system of designation and nomenclature of R genes and AVR genes should be developed at International level with code of conduct so that researchers can compare/confirm and validate each other's results. It will avoid unnecessary repetition and confusion among *Brassica* scientists.

1.12 Importance of *Hyaloperonospora arabidopsidis* in Molecular Plant Pathology

Hyaloperonospora arabidopsidis is a prominent pathogen in natural populations of *Arabidopsis thaliana* (Coates and Beynon 2010; Holub 2008). As such, it was adopted in the 1980s as one of the two pathogens of *Arabidopsis*, together with the bacterium *Pseudomonas syringae* (Koch and Slusarenko 1990). The Top 10 ranking of *H. arabidopsidis* reflects the subsequent success of the *Arabidopsis-H. arabidopsidis* pathosystem. *H. arabidopsidis* was initially utilized as a 'physiological probe' of the *Arabidopsis* immune system (Holub et al. 1994). This research led to the cloning of the first plant disease *R* genes against an oomycete, better understanding

of the evolutionary dynamics of *R* genes, the definition of broadly important immune system regulators, the identification of downy mildew-resistant mutants, and genetic definition of the complexity of the plant immune signalling network (Coates and Beynon 2010; Lapin and Van den Ackerveken 2013; Slusarenko and Schlaich 2003). On the pathogen side, research is hampered by the lack of protocols for culture and genetic transformation, established techniques with other oomycetes such as *P. infestans*. However, work in the early 2000s led to the development of genetic maps and DNA libraries that enabled the discovery of the first avirulence effector (Allen et al. 2004) and later to the R X LR effector family (Rehmany et al. 2005).

Genome sequencing of *H. arabidopsidis* isolate Emoy2, completed in 2010, unveiled 134 predicted RXLR effectors and other components of the *H. arabidopsidis* secretome (Baxter et al. 2010). Notably, this report also revealed important genomic signatures of obligate biotrophic that have evolved convergently in other obligate oomycete and fungal lineages (McDowell 2011). Protein interaction assays have shown that *H. arabidopsidis* effectors target highly interconnected host machinery, helping to define a representative plant-pathogen interaction network (Mukhtar et al. 2011). In addition, several high-throughput functional studies have investigated effector subcellular localizations, suppression of immune responses, molecular targets, and cognate immune receptors (Cabral et al. 2011, 2012; Caillaud et al. 2011; Fabro et al. 2011).

Future studies with the *H. arabidopsidis* experimental system will include (i) direct or *Agrobacterium*-mediated transformation for genetic manipulation required for the molecular analysis of downy mildew pathogenicity; (ii) the establishment of the temporal hierarchy of effectors during penetration, colonization, and sporulation, which may serve as a blueprint for a better understanding of the molecular basis of biotrophy; (iii) the role of genetic recombination and epigenetic on the emergence of new effectors; (iv) the development of tools to understand how plant-originated molecules regulate pathogen response; and (v) the relevance of interspecies transfer of small RNAs. These investigations on *H. arabidopsidis* will continue to provide new insights into the molecular mechanisms of downy mildew pathogenicity and contribute to comparative and functional analysis of (obligate) biotrophic oomycete and fungal pathogens (Kamoun et al. 2015).

1.13 Impact of Climate Change on the Diseases of Crucifers

Climate change has become a household topic of discussion with more scientists getting involved in scientific research on the aspect, while politicians are trying to derive mileage from the paradigm. The last decade of the twentieth century and the beginning of the twenty-first century have been the warmest period in the entire global instrumental temperature record. Climate change is defined as any long-term significant change in the ‘average weather’ that a given region experiences, or in other words, it is the shift in the average statistics of weather for long term at a specific time for a specific region. Average weather may include temperature,

precipitation, and wind patterns. It involves changes in the variability or average state of the atmosphere over durations ranging from decades to millions of years. These changes can be caused by dynamic process on earth, external forces including variations in sunlight intensity, and human activities. Climate change in the usage of the Intergovernmental Panel on Climate Change (IPCC) refers to a change in the state of the climate that can be identified (e.g. using statistical tests) by changes in the mean and/or the variability of its properties that persists for an extended period, typically for decades or longer. It refers to any change in climate over time, whether due to natural variability or as a result of human activity (IPCC 2007).

Increased emission of carbon dioxide (CO_2) and other greenhouse gases, predominantly methane (CH_4) and nitrous oxide (N_2O), has been ascribed as the main agents causing increase in global temperature. The second assessment report (AR2) of IPCC indicated that the increase of greenhouse gas concentrations leads to an additional warming of the atmosphere and the earth's surface. Concentration of CO_2 has increased from about 280 to almost 360 ppmv since preindustrial time, CH_4 from 700 to 1720 ppmv, and N_2O from about 275 to about 310 ppmv. This development is ascribed to the magnitude of human intervention mostly in terms of fossil-fuel use, change in land-use pattern, and agriculture. Global mean surface temperature has increased by 0.3–0.6 °C since the late nineteenth century, a change that is unlikely to be entirely natural in origin. The temperature increase is widespread over the globe and is greater at higher northern latitudes (http://www.ipcc.ch). According to IPCC, cold days and cold nights have become less frequent and hot days, hot nights, and heat waves more common. Rising temperature also affect the pattern of precipitation. Changes in rainfall pattern have already been noticed. The IPCC reports that the frequency of heavy precipitation has increased over most land areas, which is consistent with warming and increase of atmospheric water vapour. Based on the trends since 1900, precipitation significantly increased in eastern parts of North and South America, northern Europe, and northern and central Asia whereas declined in the Sahel, the Mediterranean, Southern Africa, and parts of southern Asia. Globally, the area affected by drought has increased since the 1970s. Effect of climate change on agriculture or more precisely on insect pests and diseases of agricultural crops is multidimensional. Magnitude of this impact could vary with the type of species and their growth patterns. With the change in the temperature and rainfall pattern, the natural vegetation over a region is facing a new phase of competition for survival. The fittest species are more likely to dominate in the changing pattern of climate. It may be assumed that the vegetation tolerating high temperature and salinity and having high CO_2-use efficiency could fair better than other species. Any change in the managed vegetation system, i.e. agriculture and forestry, will directly affect the socio-economic implications of the regions involved. IPCC in its report of 1995 predicted that a double increase in the CO_2 level will increase yield by 30% in several crops. The elevated production could be off-set partly or entirely by the insect pest, pathogens, or weeds. It is, therefore, important to consider all the biotic components under the changing pattern of climate. There is also thought about shorter winters, which may affect the oil yields of the rapeseed-mustard crops.

World over research on effect of climate change on pests and diseases of crops is inadequate (Huda et al. 2005). In India, there is limited effort in this area for any insect pest or disease of any crop (Subba Rao et al. 2007; Chattopadhyay and Huda 2009). However, at the genomic level, advances in technologies for the high-throughput analysis of gene expression have made it possible to begin discriminating responses to different biotic and abiotic stresses and potential trade-offs in responses. At the scale of the individual plant, enough experiments have been performed to begin synthesizing the effects of climate variables on infection rates, though pathosystem-specific characteristics make such synthesis challenging. At the population level, the adaptive potential of plant and pathogen populations may prove to be one of the most important predictors of magnitude of effects of climate change. Ecologists are now addressing the role of plant disease in ecosystem processes and the challenge of scaling up from individual infection probabilities to epidemics and broader impacts (Garrett et al. 2006). Swaminathan (1986) indicated that the number of diseases on the same crops was much higher in tropics than under temperate conditions to indicate how rising temperatures could impact occurrence of plant diseases on agricultural crops. Presently, most of the work related to climate change vis-à-vis plant diseases is going on in rice (blast, bacterial leaf blight), wheat (*Puccinia*, *Septoria*), and horticultural (*Meloidogyne*) crops. The trend indicates that severity of majority of diseases is found to be higher with elevated CO_2 levels (Chakraborty et al. 2008), an off-shoot of climate change. It is also being opined that climate change could lead to a changed profile (variants) of pathogen, insect pest ('climate change can activate "sleeper" pathogens, while others may cease to be of economic importance' – Bergot et al. 2004). The facultative pathogens with broad host range may survive better. There is also possibility of broadening of host range of the facultative pathogens. The need for further work in this area has been highlighted in adaptation experiments using twice-ambient CO_2, which increased the aggressiveness (Chakraborty and Datta 2003) and fecundity (Chakraborty et al. 2000) of *Colletotrichum gloeosporioides*, which causes anthracnose of tropical legumes. Elevated CO_2 may modify pathogen aggressiveness and/or host susceptibility and affect the initial establishment of the pathogen, especially fungi, on the host (Coakley et al. 1999; Plessl et al. 2005; Matros et al. 2006). In most examples, host resistance has increased, possibly due to changes in host morphology, physiology, and composition. Increased fecundity and growth of some fungal pathogens under elevated CO_2 have also been reported (Hibberd et al. 1996; Coakley et al. 1999; Chakraborty et al. 2000). However, it has been reported that greater plant canopy size, especially in combination with humidity, and increased host abundance can increase pathogen load (Manning and Tiedemann 1995; Chakraborty and Datta 2003; Mitchell et al. 2003; Pangga et al. 2004). Sporulation by the pathogenic fungi could be 15–20-folds higher, leading to massive increase in the pathogen (Mitchell et al. 2003). New strains may develop, with adaptation occurring faster, and their evolution may get accelerated (Coakley et al. 1999). Among the 27 diseases examined under elevated CO_2 levels, 13 caused higher crop losses than expected. Ten of the diseases had a reduced impact, and four had the same effect as they do now (NSW DPI 2007). Low solar radiation and short-day periodicity could

result in higher infections by *Fusarium*, *Sclerotinia*, and *Verticillium* (Nagarajan and Muralidharan 1995). Root rot is an emerging threat for rapeseed-mustard production system, recently reported from the farmers' field in some pockets of the country (Meena et al. 2010), which was initially identified as stand-alone and along with bacterial or fungal incidence or in combinations (*Erwinia carotovora* pv. *carotovora*, *Fusarium*, *Rhizoctonia solani*, and *Sclerotium rolfsii*). Keeping in view the fact that some isolates of *Alternaria brassicae* sporulated at 35 °C and several isolates had increased fecundity under higher RH, it seems that as per recent changes towards warmer and humid winters, being in line with current projections for future climate change (Waugh et al. 2003), existence of such isolates could pose more danger to the oilseed *Brassica* due to *Alternaria* blight in times to come. The immense variation available among 20 representative isolates of *A. brassicae* also indicates their ability to adapt to varied climatic situations (Meena et al. 2012). Powdery mildew (*Erysiphe cruciferarum*) disease in oilseed *Brassica* was mostly occurring in Gujarat state barring stray incidences elsewhere, and the appearance of the disease used to occur from late January onwards in other parts of the country. However, in recent times, the disease has been found to be occurring in other oilseed *Brassica* growing states, *viz.* Haryana, Central UP, MP, parts of Rajasthan, Jharkhand, and Bihar, with the disease making its appearance even in December possibly due to shortening of cold spell during the crop period. Bihar hairy caterpillar (*Spilarctia obliqua*) surprisingly on mustard has been noted to be on the rise. Oilseeds *Brassica* have been affected a lot by the painted bug (*Bagrada cruciferarum*) in the Western and by saw fly (*Athalia proxima*) in the Eastern India. Presently, the Indian Meteorological Department (IMD, GOI) and the National Centre for Medium Range Weather Forecasting (NCMRWF) in coordination with scientists from other agencies as ICAR, etc. are regularly issuing location-specific weather forecast and agrometeorological advisory as per different climatic conditions and cropping systems. The Indian Council of Agricultural Research (ICAR) recently launched National Initiative on Climate Resilient Agriculture (NICRA) in February 2011 to boost research on the impact of climate change and its mitigation at national level. The project aims to enhance resilience of Indian agriculture to climate change, climate vulnerability through strategic research, and technology demonstration. Research on adaptation and mitigation covers crops, livestock, fisheries, and natural resource management. It also demonstrates site-specific technology packages on farmers' fields for adapting to current climate risks. This will certainly enhance the capacity of scientists and other stakeholders in climate-resilient agricultural research and its application (http://www.icar.org.in). The mitigation of the adverse effect of climate is challenging. Acquaintances between pragmatic and modelling studies could prop up swift advancement in perception and prediction of climate change effects (Chattopadhyay et al. 2011).

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