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Historical perspectives of white rust caused by *Albugo candida* in Oilseed Brassica

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Abstract

Albugo candida (Pers. Ex. Lev.) Kuntze is a wide spread pathogen of cruciferous crops causing heavy yield losses all over the world. Molecular and phylogenetic studies of the family Albuginaceae revealed four distinct lineages: *Albugo* s.str., *Albugo* s.l., *Pustula* s.l. and *Wilsoniana* s.l. It's host range is more than 300 hosts. The host specificity of A. candida has been recorded from more than eight countries of the world. Studies on host-pathogen interaction, fine structures of hyphae, mycelium, haustoria, sporangia, zoospores and oospores have been conducted through histopathology, electron microscopy, scanning electron microscopy and transmission electron microcopy. The pathogen survives through mycelium, sporangia and oospores. Germination of sporangia and oospores has been determined. Biochemical host-pathogen interaction studies have been conducted. Studies on identification and cloning of plant defense resistance genes are in progress. Genome sequencing of *A. candida* and *A. laibachii* have been made. Very useful and reproducible techniques have been developed on the aspects viz., growth chamber inoculation, oospore germination, induction of stag-heads, detached leaf culture, *In vitro* callus culture, temperature effects on disease development and oospore formation, process of infection, association of white rust and downy mildew, pathogenic variability, virulence spectrum, host resistance, genetic of host-parasite interaction, slow white rusting and chemical control. Future research areas have been suggested.

Keywords : White Rust, *Albugo* candida, Oil Seed *Brassica*

1. Introduction

Albugo candida (Pers. Ex. Lev.) Kuntze. (*A. cruciferarum S.F. Gray*), a member of the family Albuginaceae in the order Albugonales of class Peronosporomycetes is an obligate parasite responsible for causing white rust (WR) disease of many cruciferous crops (Saharan and Verma, 1992). Local infection produces white to cream coloured pustules on leaves, stems and pods, while general or flower bud infection (Verma and Petrie, 1980) causes extensive distortion, hypertrophy, hyperplasia and sterility of inflorescences generally called "stagheads". The staghead phase (SP) accounts for most of the yield loss attributed to this disease.

Depending on the severity of both foliar and SP of the disease, the percent yield losses ranging from 1-60 % in Polish or Turnip rape (*Brassica rapa* L.) in Canada (Berkenkamp, 1972; Petrie and Venterpool, 1974; Harper and Pittman, 1974; Petrie, 1973), from 23-89.8 % in Indian mustard [*B. juncea* (L.) Czern and Coss] in India (Bains and Jhooty, 1979; Lakra and Saharan, 1989a), and from 5-10 % in Australia (Barbetti, 1981; Barbetti and Carter,

1986) have been reported; substantial yield losses in radish (*Raphanus sativus* L.) have also been reported (Kadow and Anderson, 1940; Williams and Pound, 1963).

Although Canadian and European *B. napus* cultivars are not attacked in some countries, many cultivars of this species grown in China are susceptible (Fan *et. al.*, 1983). The wide range of yield losses caused by this disease in many host species needs assessment of genotypes under suitable environmental conditions (Saharan, 2010). In the present manuscript the progress made in white rust research on biology, ecology, epidemiology and management of *A. candida* on oilseed Brassica and future priority research areas have been discussed.

2. Taxonomy and nomenclature

The first species of *Albugo* described by Gmelin in 1792 as *Aecidium candidum* was later placed in genus *Uredo*, subgenus *Albugo* by Persoon in 1801. Based on differences in symptom development, Persoon (1801) described two different species of white blister rust, with *Uredo candida*, and subdivided into three varieties, parasitic to Brassicaceae and Asteraceae. After a few years, *Albugo* was established as an independent genus by de Roussel (1806), although Gray (1821) is often still given as the author for this genus. De Candolle (1806) added the species *Uredo portulaceae* (now *Wilsoniana portulacae*), and *Uredo candida* beta *tragopogi* to species rank (*Uredo tragopogi*, now *Pustula tragopogonis*) and renamed *Uredo candida cruciferarum.* Leveille (1847) described the genus *Cystopus*, and later de Bary (1863) described the sexual state of *Albugo*, adopting the generic name *Cystopus. Albugo* has been typified by Kuntze (1891), who gave *Uredo candida* (Pers) Pers. as the type species.

Before Biga (1955) pointed out that names of sexual form have no antecedence over anamorphs in the class Oomycetes, many researchers considered white blister rusts to be members of the superfluous genus *Cystopus* (Wakefield, 1927), although the older genus name, '*Albugo'* also persisted. Subsequently, in the early 20th century, numerous other species of genus *Albugo* were described. Wilson (1907) and Biga (1955), respectively recorded 13 and 30 species of this genus about 50 years later (Mukerji, 1975). The recent key to the genus *Albugo* published by Choi and Priest in 1995 recognised only 10 species of genus *Albugo*.

Until molecular phylogenetic studies of the Albuginaceae became possible, *Albugo* was generally treated as a member of the Peronosporales (Dick, 2001), in which it was placed along with the second group of obligate plant parasites, the downy mildews (DM). The Albuginaceae family contains four distinct lineages: *Albugo* s.str., parasitic to Brassicales; *Albugo* s.l., parasitic to Convolvulaceae; *Pustula* s.l., parasitic to Asterales, and *Wilsoniana* s.l., parasitic to Caryophyllales. *Albugo cruciferarum* is regarded as a synonym of *A. candida* (Choi *et al*., 2007). Till now, the white blister pathogen on oilseed rape has been considered *A. candida* (Farr and Rossman, 2010). The high degree of genetic diversity exhibited within *A. candida* complex warrants their division into several distinct species (Choi *et al*., 2006).

3. Host range

The first record of *A. candida* on Brassicaceae seems to be by Colmeiro (1867). *Albugo candida* has been reported in Brassicaceous hosts over widely different geographical areas of the world with host range of some 63 genera and 241 species (Biga, 1955; Saharan and Verma, 1992; Choi *et al*., 2007; Farr *et al*., 1989). According to the USDA-ARS Systemic Botany and Mycology Laboratory, *A. candida* was recorded on more than 300 hosts (Farr *et al*., 2004). Based on recent molecular phylogenetic investigations, *A. candida* has an extraordinarily broad host range, extending from numerous genera of the families Brassicaceae to Cleomaceae, and Fabales to Capparoceae (Choi *et al*., 2006, 2007, 2008, 2009, 2011a). *A. candida* and *A. tragopogonis* each may consist of several distinct lineages (Voglmayr and Riethmuller, 2006).

The host specificity of *A. candida* has been recorded from Australia (Kaur *et al*., 2008), Britain (Happer, 1933), Canada (Verma *et al*., 1975), Germany

(Eberhardt, 1904), India (Saharan, 2010), Japan (Hiura, 1930), Romania (Savulescu and Rayes, 1930), and U.S.A. (Pound and Williams, 1963). *Albugo candida* isolates from *Brassica* can infect *Amaranthus viridis* (Amaranthaceae), *Cleomev viscosa* (Capparaceae, now included in Brassicaceae; APG, 2003), as well as *B. rapa* var. Rapa (Khunti *et al.*, 2000). Recently, Saharan (2010) has listed all pathotypes reported globally (Table 1).

4. Geographical distribution

White rust on cultivated oilseed Brassicas and other hosts have been reported worldwide. Countries where the disease occurs include the U.K. (Berkeley, 1848), U.S.A. (Walker, 1957), Brazil (Viegas and Teixeira, 1943), Canada (Greelman, 1963; Petrie, 1973), Germany (Klemm, 1938); India (Chowdhary, 1944), Japan (Hirata, 1954), Pakistan (Perwaiz *et al*., 1969), Palestine (Rayss, 1938), Romania (Savulescu, 1946), Turkey (Bremer *et al*., 1947), Fiji (Parham, 1942), New Zealand (Hammett, 1969), China (Zhang *et al*., 1984) and Korea (Choi *et al*., 2011a). White rust on sunflower occurs in Russia (Novotel'Nova, 1962), Uruguay (Sackston, 1957), Argentina (Sarasola, 1942), Australia (Middleton, 1971; Stovold, and Moore, 1972), and in many other countries (Kajomchaiyakul and Brown, 1976). White rust of salsify occurs in Australia, Canada, U.S.A., S. America, Europe, Asia and Africa (Wilson, 1907), and on water spinach occurs in India, Hong Kong (Ho and Edie. 1969; Safeefulla, and Thirumalachar, 1953), and also in Texas (Wiant, 1937; Williams and Pound, 1963).

5. Structures and reproduction

Studies on host-pathogen-interaction, fine structures of hyphae, mycelium, sporangia, zoospores and oospores have been conducted through histopathology using electron microscopy, scanning electron microscopy and transmission electron microscopy (Berlin and Bown, 1964; Davison, 1968; Coffey, 1975; Hughes, 1971; Khan, 1976, 1977; Tewari *et al*., 1980; Kaur *et al*., 1984; Baka, 2008). The members of the Albuginaceae are distinguished from those of related families by the formation of the asexual sporangia in basipetal chains.

5.1 Mycelium

The non-septate and intercellular mycelium of *Albugo* species feeds by means of globose or knob-shaped intracellular haustoria, one to several in each host cells (Verma *et al*., 1975). The detail of haustorial formation and development has been given by Berlin and Bowen (1964); Coffey (1975); Davison (1968); Fraymouth (1956), and Wager (1896).

5.2 Asexual organs

5.2.1 Sporangiophore

The sporangiophores are short, hyaline, clavate, thick-walled, especially towards the base, 30-45 x 15-18 µm diameter, basally branched, club-shaped and give rise to simple chains of sporangia. The number of sporangia produced is indefinite. They are formed in basipetal succession; that is, the sporangiophore forms a cross-wall or septum, cutting off that portion which is to become a sporangium. The sporangiophore increases in length, a second sporangium is cut off, and the process continues, resulting in the simple chains of multinucleate sporangia.

5.2.2 Sporangia

The number of sporangia produced is indefinite in basipetal succession; that is, the sporangiophore forms a cross-wall or septum, cutting off that portion which is to become a sporangium, which is globose to oval, hyaline with uniform thin wall, and 12-18 µm diameter. As sporangial production continues, the older, terminal portions of the chain breaks, releasing the individual sporangia. The sporangia germinate by the formation of zoospores and, on rare occasions, by means of a germ tube (Heald, 1926; Wager, 1896; Walker, 1957; Zalewski, 1883).

5.2.3 Zoospores

Sporangia absorb water and swell, develop vacuoles in the granular protoplasm, and finally 4-12 uninuleate polyhedral portions of the protoplasm are delineated by fine lines. In the mean time, an obtuse papilla is formed at one side of the sporangium, which produces zoospores. The zoospores, still immobile, emerge usually one by one, with final cleavage following complete emergence

of the sporangium's contents. The flagella soon become apparent by an oscillatory motion of the entire zoospore mass. These single-nucleated spores formed in sporangia are released only in aqueous environment. The slightly concave-convex zoospore contains a disc-like vacuole on one side, near which are attached two flagella, one short and one long, by which the zoospore soon detaches itself from the mass and swims away if liquid is present. They have one tinsel flagellum, and one whiplash flagellum. Only the tinsel flagellum has distinctive flagellar hairs. Zoospore formation occurs within minutes and is considered one of the fastest developmental processes in any biological system. Once released from the sporangium, zoospores exhibit chemotactic, electrotaxis, and autotaxis or auto-aggregation responses to target new hosts for infection (Walker and West, 2007). Zoospores soon come to rest, retract their flagella, encyst and germinate by the formation of a germ tube. If germination occurs on a susceptible host, the germ tube penetrates through stomata to form an intercellular mycelium (Heald, 1926; Wager, 1896; Walker, 1957).

5.3 Sexual organs

The oogonia and antheridia are formed from the mycelium in the intercellular spaces of the host, particularly in a systemically invaded tissue (Wager, 1896). Oogonia are globose, terminal or intercalary, each contains upto 100 nuclei and its contents clearly defined into a peripheral zone of periplasm and a single central oosphere. Antheridia are clavate, each contains 6 to 12 nuclei, and are applied to the sides of an oogonium (Heald, 1926, Heim, 1959, Walker, 1957).

5.3.1 Gametogenesis, fertilization, and oospore formation

One or more antheridia come to occupy a position close to an oogonium. There are two types of egg organization within an oogonium. In *A. candida*, the protoplast becomes differentiated into a peripheral or extemal zone, the periplasm, which contains many nuclei, and a central mass, the egg cell or ooplasm, which contains a single nucleus. The antheridium, which is a multinucleate cell, produces a short, tube-like outgrowth, the fertilization tube, which penetrates the periplasm and comes in contact with the egg cell or ooplasm. The antheridial or male nuclei are discharged through this tube into the egg cell. In the uninucleate egg, the female nucleus fuses with a single male nucleus, where as in the multinucleate egg, female and male nuclei fuse in pairs. This nuclear union constitutes the process of fertilization (Heald, 1926; Walker, 1957). Following fertilization, the egg is gradually transformed into a thick-walled oospore. The periplasm is absorbed, the oospore wall darkens and thickens, and develops a characteristic external ridges, reticulations or knobs, while the interior of the oospore becomes filled with an abundance of reserve food in the form of oily or fatty globules. The fully developed oospore lies within the old empty oogonial cell. The oospores are released only by weathering and decay of the host tissues (Heald, 1926). The characteristics of oospores are useful criteria for distinguishing species of *Albugo*, in which the epispore is tuberculate or ridged, and is a more specialized group, where there is complete development of the epispore with cytological phenomena (Zalewski 1883; Stevens, 1901).

6. Survival

6.1 Mycelium

It is believed that in perennial hosts such as horse-radish, the mycelium is capable of overwintering in the infected crowns and lateral roots (Endo and Linn, 1960; Kadow and Anderson, 1940; Walker, 1957). Remaining dormant during the winter, the mycelium resumes its activity and grows into the new shoots the host produces in the spring.

6.2 Sporangia

At 30°C temperature, viability of sporangia is lost after 4 h when attached, and after 2 h when detached from host tissues (Lakra *et al*., 1989). They observed that sporangia of *A. candida* can survive for 4.5 days at 15°C on detached-infected *B. juncea* leaves, but loses their viability after 18 h if separated and incubated without host tissues. However, sporangia can be stored for 105 days at -40°C as a dry powdered mass.

6.3 Oospores

Oospores are formed in the hypertrophied tissues

(leaves, stems, inflorescences, pods, roots) of infected host plants. Overwintered oospores in infected plant debris in soil function as the source of primary inoculum of the pathogen (Butler, 1918; Butler and Jones, 1961; Chupp, 1925; Kadow and Anderson, 1940; Verma *et al*., 1975; Walker, 1957). Oospores have also been observed in naturally infected senesced leaves of *B. juncea* and *B. rapa var.* Toria. Lakra and Saharan (1989b) estimated 8.75 x 10^5 oospores in one gram of hypertrophied cup-shaped leaves, and 21.85 x 10^5 in one gram of hypertrophied staghead portions. Verma and Petrie (1975) found that oospores can remain viable for over 20 years under dry storage conditions. Petrie (1975) reported 1500 oospores per gram seed of rapeseed and reported the possibility of survival and spread of the pathogen by means of oospores carried extrnally on seeds. According to Tewari and Skoropad (1977), oospores have a highly differentiated, 5- layered cell wall and that their greater longevity is probably due to the heavily fortified cell wall.

7. Spore germination

7.1 Oospores

de Bary (1866) first observed germination of *Albugo* oospores via asessile vesicle. Vanterpool (1959) confirmed this and described a second mode of germination by means of a terminal vesicle; however, maximum germination was only 4% and its occurrence was unpredictable. Petrie and Verma (1974) and Verma and Petrie (1975) described a very reliable and reproducible technique for germination of *A. candida* oospores. Oospores germinated by the production of one or two simple or branched germ tubes, by the release of zoospores from vesicles formed at the ends of germ tubes (terminal vesicles), and by the release of zoospores from sessile vesicles. Germination by sessile vesicles was the most common. Verma and Bhowmik (1988) observed that the treatment of oospores with 200 ppm KMn04 for 10 minutes induced increased germination. Oospores do not appear to require any dormancy period. Recently gut enzymes (1% b-glucuronidase and arylsulfatase, Sigma make) were used in studies for germination of the oospores from hypertrophied plant tissue (Meena and Sharma, 2012).

7.2 Sporangia

Sporangial germination in *A. candida* was studied by several researchers. In 1911, Melhus reviewed the earlier work on sporangial germination. Prevost (1807), and De Bary (1860) found that sporangial germination occurs via the production of zoospores. Harter and Weimer (1929) stated that sporangia may germinate by the direct production of germ tubes, but germination via zoospores was more frequent. Eberhardt (1904), Melhus (1911), and Napper (1933) found that sporangia of *A. candida* germinate invariably by the production of zoospores; which was confirmed by Lakra *et al*. (1989). De Bary (1860), and Melhus (1911) reported that sporangia did not germinate above 25°C or below 0°C; the best germination was at lower temperatures. Napper (1933) did not observe sporangial germination above 20°C. Melhus (1911) suggested 10°C as the optimum temperature for sporangial germination, but Napper (1933) found that germination takes place as readily at 1-18°C. Endo and Linn (1960) reported the overall optimum temperature range for sporangial germination to be 15-20°C, with maximum germination occurring between 0 and 28°C. However, Lakra and Saharan (1988b), and Lakra *et al*. (1989) observed >75% sporangial germination in *A. candida* at 12-14°C after 8 h incubation. Sporangia ceased to produce zoospores below 6°C and above 22°C. Sporangial germination started after 4 h and reached their maximum 8 h after incubation. A quadratic equation, Y -103.16 + 26.99 x -1.01 x2, where Y = % sporangial germination and x = temperature in °C was proposed to estimate the frequency of sporangial germination of *A. candida* from *B. juncea* at any known temperature. The variation in the cardinal temperatures for sporangial germination among different studies is probably due to the involvement of different host specific biological races of *A. candida*. Germination of *A. candida* sporangia from naturally-infected *B. juncea* and *B. rapa* var. Toria leaves occurred within one hour at 13°C.

Although Melhus (1911), and Holliday (1980) reported that sporangial germination is not affected by light or darkness, Lakra *et al*. (1989) demonstrated that exposure to light of 150 $\mu EM^{-2}s^{-1}$ slightly

delays sporangial germination in *A. candida* infecting *B. juncea*. Melhus (1911) found that sporangia germinated readily in both saturated and non-saturated atmosphere, while Lakra and Saharan (1988b), and Lakra *et al.* (1989) found that a film of free water is essential for germination of sporangia. Melhus (1911), and Napper (1933) found that chilling and a reduction of 30% water content in sporangia were essential for germination. Lakra *et al*. (1989), however, states that it is not a prerequisite, since up to 75% of sporangia germinated without chilling or dehydration. According to Uppal (1926), sporangia of *A. candida* require oxygen for germination. Takeshita (1954) reported that sporangia of *A. candida* from horseradish germinated best at pH 4.5-7.5 at 10-20°C. Light did not affect germination. However, Endo and Linn (1960) found that sporangia of *A. candida* from horseradish require pH of 3.5-9.5 with an optimum of about 6.5; optimum temperature range was 15-20°C. Only a few studies have been carried out on sporangial germination of species other than *A. candida*. Edie and Ho (1970) demonstrated that although the sporangial germination in *A. ipomoeae-aquaticae* is nearly identical with that of other *Albugo* species with regard to the method of sporangial germination and host penetration, it requires a slightly higher germination temperature in the range of 12-30°C with an optimum of about 25°C. However, Saffeefulla and Thirumalachar (1953) mentioned that sporangia germinated at 15°C, but not at 24°C. Sporangia of *A. ipomoeae-panduratae* germinate at 8-25°C (Harter, and Weimer, 1929) and optimum of 12-14°C. Sporangia of *A. tragopogonis* germinate at 4-35°C with an optimum range of 4-15°C. Encysted zoospores germinate best at 10°C (Kajomchaiyakul and Brown, 1976). Sporangial germination of *A. tragopogonis* from *Senecio squandus* occurs at 5-15°C, with an optimum of 10-15°C and very little germination occurs at 20°C (Whipps, and Cooke, 1978a, 1978b). Sporangia of *A. bliti* germinate at a temperature range of 2-25°C, but optimum at 18°C (Mishra and Chona, 1963). Chilling of sporangia, increases germination but mature sporangia from just-opened pustules, or those naturally-detached, germinated best. Sporangia of *A. occidentalis* germinate at 2-25°C with an optimum near 12°C (Raabe and Pound, 1952). Light, water content of sporangia, and pH also have little effect on sporangial germination.

8. Fine structures

Electron microscopy, particularly when used in association with physiological, biochemical and genetic studies, provides valuable information on the complex relationships which exist between host and pathogen. The fine structures of *A. candida* were studied by Berlin and Bowen (1964a, b), Davison (1968) and Coffey (1975).

8.1 Haustoria :

The small stalked capitate haustoria of *Albugo* are connected to the much larger haustorial mother cell by a slender cylindrical neck. Haustoria contain mitochondria with tubular cristae, ribosomes and occasional cisternae of rough endoplasmic reticulum. Nuclei and perinuclear dictyosomes, although present in the mother cells, are absent in the haustoria. The fungal plasma membrane and cell wall are continuous from an intercellular hypha to the haustorium except that there is no evidence of a fungal cell wall around a portion of the haustorial stalk proximal to the haustorial head (Saharan and Verma, 1992). In the host mesophyll cell, the haustorium is invariably surrounded by host plasma membrane and/or a thin layer of host cytoplasm. The host cell wall invaginates at the point of haustorial penetration to form a short sheath around the penetration site, but the host cell wall is absent from rest of the haustorium. A collar consisting of fibrillar material is commonly found around the proximal portion of the neck. An electron-opaque capsulation lies between the haustorium and the host plasma membrane, and extends into the penetration region between the sheath and the fungal cell wall. An electron-opaque sheath surrounds the thin wall of the haustorial body, but is absent from the neck region. A series of tubules is continuous with the invaginated host plasma membrane which surrounds the haustorial body. These tubules contain an electron-dense core similar in appearance to, and continuous with, the sheath matrix. Host dictyosomes and their secretory vesicles are not involved in formation of the haustorial sheath

(Saharan and Verma, 1992). A constant feature of the haustorial apparatus is the association of flattened cistenae of host endoplasmic reticulum with the distal portion of the haustorial neck. Woods and Gay (1983) provide evidence for a neckband delimiting structural and physiological regions of the host plasma membrane associated with haustoria of *A. candida*. Coffey (1983) demonstrated cytochemical specialization at the haustorial interface of *A. candida*. Soylu (2004) observed that ultrastructural nature of the haustorium produced in *Arabidopsis* clearly differ from the DM, rust or the powdery mildew (PM) fungi.

8.2 Sporangia :

In sporangia, the paramural bodies are formed by elaborations of the plasma membrane and break away from the plasma membrane and undergo autodigestion. In vegetative hyphae, the tubules and lamellae of paramural bodies break up into vesicles and are finally sequestered into the cell wall (Khan, 1976, 1977). The surface layer of the cell wall of the sporangia and sporangiophores of *A. candida* is composed of a series of lamellae. Evidence from freeze-fracture, freeze-etch, and single-stage replicas demonstrated that the lamellae are bilayered, an organization associated with the presence of lipids. This multilamellate layer on the surface of the cell wall facilitates air dispersal and protects the sporangia from desiccation (Tewari *et al*., 1980). In *Albugo* sporangia are produced in basipetal chains at the apices of sporangiophores and are released by the dissolution of the septa that delimit them. Hughes (1971) suggested that sporangiophores of *Albugo* produce sporangial chains by percurrent proliferation, and they are "apparently the morphological equivalents of annellophores (annellides)" (Hughes, 1971). A sporangial initial buds out from a fixed locus at the tip of the sporangiophore. After reaching a certain size, it is delimited by a basal septum and converted into a sporangium. A new initial grows out from the sporogenous locus, pushing the newly formed sporangium upward. By repetition of this process, a basipital chain of sporangia is formed. Both layers of the sporangiophore wall grow out and take part in forming the sporangial wall. In conidium ontogeny this mode of development is called holoblastic. During sporangial formation in *A. candida* the sporangiophores do not increase in length; however, abnormally long sporangiophores are sometimes seen among the smaller, regular ones. There are no annellations on the sporangiophore surface and no increase in the thickness of the sporangiophore wall at its apex. Thus, none of the characteristics that have been shown to be associated with percurrent proliferation are present during the development of sporangia in *Albugo* (Khan, 1977). In maturing sporangia a burst of activity was observed by Khan (1976). Even after formation of sporangia, the numbers of mitochondria and the amounts of endoplasmic reticulum increase. Perinuclear vesicles and smooth surface cistenae differentiate into well developed Golgi apparatuses, which remain secretory until complete maturation of sporangia. Maturing sporangia have autophagic vacuoles containing various cell organelles. Nuclear degeneration and mitosis proceed simultaneously. All activities decline towards the end of sporangial maturation.

8.3 Oospores

The structure and development of oospores of *A. candida* in the stagheads on rapeseed (*B. rapa*) were investigated by light microscopy, transmission electron microscopy of ultrathin sections and scanning electron microscopy (Tewari and Skoropad, 1977). A reaction zone forms on the oogonial wall at the point of contact by the fertilization tube of the antheridium. The oospore has a highly differentiated, five-layered cell wall. The periplasm appears to play an active role in the deposition of the oospore cell wall. The contents of the periplasm do not disappear after maturation of the oospore; instead, they form a persistent material between it and the oogonial wall. Hence, functionally, the oospore wall complex has two additional layers which may contribute to the longevity of the oospore. In a histochemical study of cytoplasmic changes during wall layer formation on the oospore of *A. candida*, Kaur *et al*. (1984) reported that the young multinucleate oogonium is double-walled. The oospore nuclei are large and prominent, and have an outer shell or sheath of proteinaceous material surrounding a central core of nucleoplasm. The first wall of the fertilized oospore is laid at the interphase

of the periplasm and the ooplasm. Subsequent wall layers are formed both on the inner and outer side of the first oospore wall. The second oospore wall is formed just internal to the first one. The third wall of the oospore is formed external to the first one and appears ridged. The last wall to be formed is the Innermost one which completely surrounds the central ooplasm. This wall layer is callosic in nature.Oospore morphology is basically reticulate.

9. Biochemistry of host pathogen interaction

Biochemical studies of the growth and survival of a pathogen, and of the changes it induces in its host can ultimately lead to a better understanding of epidemiology, disease development and control. With a few exceptions, such studies on WR lag far behind those for diseases caused by other major groups of biotrophs. Ideal prerequisites for meaningful studies of the biochemistry of host-parasite interaction are a) a clear understanding of the genetic control of virulence and avirulence in the parasite and of susceptibility and resistance in the host, b) precise histological and cytological descriptions of spore germination, infection and the establishment and development of infection, and c) the availability of methods for growing the parasite alone and in combination with its host under controlled conditions. Unfortunately, these criteria have not been fully satisfied for any WR disease. Reduction in sugar content was proportionate to the disease severity, and maximum reduction was observed in the infected leaves. Total free amino acids increased after infection in all the infected plant parts, and this increase was proportionate to the disease severity (Singh, 2005).

9.1 Carbohydrate metabolism and respiration

A number of reports indicate that the respiration rates of tissues infected by members of the Albuginaceae also rise dramatically (Black *et al*., 1968, Williams and Pound, 1964). Long and Cooke (1974) suggested that host-fungus movement of carbohydrates in *Albugo-Senecio squalidus* system is maintained by hydrolysis of host sucrose and uptake of hexoses, followed by accumulation of trehalose within the mycelium and spores. Trehalose was synthesized within pustules by the fungus but no acyclic polyols were found. Accumulation of hexoses around pustules together with increased hydrolysis of exogenous sucrose by pustular material indicated increased invertase activity within infected tissues. Accumulation of dark-fixed carbon compounds in WR pustules of *Senecio squalidus* infected with *A. tragopogonis* has been reported (Thomton and Cooke. 1970).

Quantitative imaging of chlorophyll fluorescence revealed that the rate of photosynthesis declined progressively in the invaded regions of the leaf. Images of nonphotochemical fluorescence quenching (NPQ) suggested that the capacity of the Calvin cycle had been reduced in infected regions, and that there was a complex metabolic heterogeneity within the infected leaf. *Albugo candida* also caused localized changes in the carbohydrate metabolism of the leaf; soluble carbohydrates accumulated in the infected region whereas the amount of starch declined. There was an increase in the activity of invertases which was confined to regions of the leaf invaded by the fungal mycelium. The increase in apoplastic invertase activity was of host origin, as mRNA levels of the ATb FRUCT1 gene (measured by semiquantitative RT-PCR) increased 40-fold in the infected region. The increase in soluble invertase activity resulted from the appearance of a new isoform in the invaded region of the leaf. The resistant and moderately resistant cultivars contained higher amounts of chlorophyll, sugars and total phenols than the susceptible cultivar at all growth stages. However, total proteins and free amino acids were higher in the susceptible cultivar at all growth stages (Singh, 2000). Information on chromosome number and meiotic chromosome configuration is tabulated for 3 *B. juncea* lines developed at the Agriculture Canada Research Centre in Saskatoon: TO97-3360 (BC4F4) with high oleic acid content (68.6%), TO97-3400 (BC3F4) which is resistant, and TO97-3414 (BC3F4) has a low alkenyl glucosinolate content (28 micro moles/g defatted meal) (Cheng *et al*., 1999). Higher starch contents were found in noninfected tissues, and it is suggested that this could be due to the higher alpha amylase activity in diseased tissues (Debnath *et al*., 1998). Chlorophyll has a positive role in *A. candida* resistance in Indian mustard (Gupta *et al*., 1997). Thaumatin-like

protein (PR-5), associated with the resistance of *B. juncea* towards *A. candida* which is not found previously. One protein, peptidyl-prolyl cis/trans isomerase (PPIase) isoform CYP20-3, was only detected in the susceptible variety and increased in abundance in response to the pathogen. PPIases have recently been discovered to play an important role in pathogenesis by suppressing the host cell's immune response (Kaur *et al*., 2011a).

9.2 RNA content

In *lpomoea* WR there was greater reduction in the RNA content of infected tissues than in the healthy, adjacent tissues (Misra and Padhi, 1981).

9.3 Photosynthesis

Black *et al*. (1968) used infrared CO2 analysis to demonstrate that a decline in the photosynthetic rate of cotyledons of radish infected with *A. candida* preceded the rise in respiration rate reported by Williams and Pound (1964). In another study, Harding *et al*. (1968) examined the pattem of pigment retention during green island development following infection of *B. juncea* cotyledons with *A. candida*. They found that labelled glycine 2-14C was incorporated into chlorophyll a and b in both infected and non-infected tissue. Both tissue fixed $^{14}CO_2$ in the light, but 4 days after infection green islands fixed five times more $^{14}CO_2$ in the light than did noninfected tissue. The maintenance of chlorophyll and continued photosynthetic activity in green island tissue was parallaled by delayed breakdown of chloroplasts. Extensive research has indicated that the overall activity of photosynthetic pathways declines in leaves infected by rusts and PM, and is accompanied by a decrease in chlorophyll content of the tissue (Cooke, 1977; Daly, 1976).

9.4 Accumulation of metabolites

Long *et al*. (1975) suggested that invertase may play a key role in the provision of substrate for the accumulation of starch at infection sites: where there is a surplus of soluble carbohydrate, particularly sucrose, hydrolysis by invertase might provide hexose for starch synthesis within chloroplasts. Invertase may thus mediate a system by which the excess soluble carbohydrate at infection sites is converted to osmotically inactive polysaccharides. Dhingra *et al*. (1982) found decreased amounts of free protein, total protein and total phenolic compounds in floral parts and floral axes of *B. rapa* infected with WR.

Dhawan *et al* (1981) correlated resistance of *B. juncea* cv. RC-781 with higher concentrations of phenols when compared with the susceptible cvs. Prakash and RH-30, where greater amount of sugar was present. Singh *et al*. (1980) demonstrated that cellulase, endo-PMG and endo-PG were produced in *B. juncea* leaves infected with *A. candida*. Maheshwari and Chaturvedi (1983) found that the swelling and disruption of subcellular particles rich in lysomal acid hydrolases was produced by acid phosphatase activity centered primarily in the infected tissues of *B. juncea*. Acid phosphatase activity in antheridia, oogonia and oospores of *A. candida* indicates that this enzyme plays a role in the synthesis of fungal organs.

9.5 Growth substances:

Infection of host plants with *Albugo* causes hyperplasia and hypertrophy of leaf, stem and floral parts. Kiermayer (1958) found that these symptoms are produced in plants infected with *A. candida* due to the production of indolacetic acid (IAA). Hirata (1954, 1956) found that infection with *A. candida* causes an initial increase in diffusible auxin in diseased stems and leaf sections, followed by a decrease before maximum development of the galls. The auxins in healthy and *Abugo*-infected inflorescences of *B. napus* have now been identified and estimated quantitatively by Srivastava *et al*. (1962). Malformed *B. napus* inflorescences produce IAA, IAN, accelerator L, and an ether-insoluble growth substance designated as A.

Kumari *et al*. (1970), and Lal *et al*. (1980) studied the quantitative and qualitative changes in the amino acid contents of diseased (hypertrophied) and healthy tissues of mustard and radish. The infection causes the breakdown of plant proteins, releasing small quantities of tryptophan, which reacts with endogenic phenolic acid to produce IAA which is responsible for hypertrophied growth. It was possible to recover waxy or medium waxy *B. juncea* types with WR

resistance, though in low frequencies (Subudhi and Raut, 1994). More research is needed to gather basic information concerning the effects of WR on respiration, photosynthesis, accumulation and transfer of carbohydrates, production of growth regulators, and the role of phenolics and other growth substances in infected host tissues.

9.6 Plant defense resistance genes

Plant defenses against colonization by a pathogen thought to be triggered by either direct, or indirect interaction between proteins encoded by the pathogen avirulence (Avr) gene and a corresponding plant resistance gene.

From previous molecular genetic analyses of downy mildew resistance, there are numerous examples of receptor-like genes in *A. thaliana* that vary in different modes of defense regulation (Eulgem *et al*., 2004; Holub, 2001; McDowell *et al.,* 2000; Tör *et al.,* 2002). The majority of plant *R* genes encode nucleotide-binding site leucine-rich repeat (NB-LRR)-type proteins which can be further grouped into two subclasses based on their N-terminal sequence: those containing a coiled-coil (CC) domain (CC-NB-LRR), or those containing a domain with similarity to Drosophila toll and mammalian interleukin-1 receptor (TIR) (TIR-NB-LRR) (Hammond-Kosack and Jones, 1997; Jones and Jones, 1997; Young, 2000). LRR is involved in protein–protein interactions and occur in a number of proteins with different function (Kobe and Deisenhofer, 1994, 1995). Domain exchange between LRR of closely related *R* genes supports their role in pathogen recognition (Ellis *et al.,* 1999; Wulff *et al.*, 2001). Variation among *R*-genes occurs mainly in their LRR domain, typically in the solvent exposed â-strand/â-turn structure within the LRR domain.

Based on their similarity with some of the animal proteins involved in apoptosis and innate immunity, The N-terminal domain of plant R-proteins are thought to have to function as a signaling domain (Rairdan and Moffet, 2007). However, several recent reports indicate that the N-terminal domains of NB-LRR proteins may be involved in recognition specificity (Moffett, 2009). The high variability of LRR domains and their role in protein-protein intreraction led to the idea that R-proteins interact directly with their congnate Avr proteins. However there is a limited evidence for such a direct intreation which led to the development of Guard and Decoy hypotheses which propose that R-proteins detect interaction of Avrs with host proteins (Dangl and Jones, 2001; van der Hoorn and Kamoun, 2008).

Recognition of a pathogen by a plant initiates a rapid response localized to the infection site and manifested by changes in ion flux and production of reactive oxygen species that lead to induction of downstream signals and defense genes (Kombrink and Schmelzer, 2001; Morel and Dangl, 1997). Initiation of local defense also results in signals that induce systemic acquired resistance (SAR) in non-infected distal parts of the plant, resulting in broad-spectrum resistance (Dong, 2001; Shah and Klessig, 1999). The role of salicylic acid (SA) in plant defense and induction of SAR has been shown by treatment of plants with SA or its synthetic analogs such as 2, 6-dichloroisonicotinic acid (INA) and benzothiadiazole (Klessig *et al*., 1994). Furthermore, transgenic plants expressing the bacterial SA-degrading enzyme, NahG, are unable to induce SAR (Delaney *et al*., 1995).

Several mutants in *A. thaliana* have been identified that affect disease resistance responses associated with defense regulatory genes such as: *AtSGT1b* (homolog of the yeast gene SGT1) (Austin *et al*., 2002; Tör *et al*., 2002), *EDS1* (enhanced disease susceptibility) (Parker *et al.* 1996), *NDR1* (non-race-specific disease resistance) (Century *et al*., 1997), *PAD4* (phytoalexin deficient) (Glazebrook *et al*., 1997), and *RAR1* (homolog of a barley gene required for Mla powdery mildew resistance) (Muskett *et al.*, 2002; Tornero *et al.*, 2002). Resistance specified by the *RPS4* gene to the bacterial pathogen *Pseudomonas syringae* expressing avr*Rps4* (Gassmann *et al*., 1999), and the oomycete *Peronospora parasitica* specified by *RPP1*, *RPP2*, *RPP4*, and *RPP5*, which all encode TIR-NB-LRR proteins, is abolished by *eds1* (Aarts *et al*., 1998; Parker *et al*., 1996; Rusterucci *et al*., 2001).

White rust in natural populations of *A. thaliana* has

been attributed to distinct Albugo species, *A. candida* and A. *laibachii* (Thines *et al*. 2009). Three resistance genes to *A.candida* (recently renamed as *A. laibachii*; Kemen *et al*., 2011) *(RAC*) isolate *Acem1* were identified (Borhan *et al*., 2001). Cloning was reported of the first WR resistance gene to isolate *Acem1* of *A. candida* (*RAC1*) from *Ksk-1* accession of *A. thaliana*. They also describe the effect on *RAC*-mediated resistance of standard mutations that previously were used to characterize defense signaling in DM resistance.

RAC1 is a member of the Drosophila toll and mammalian interleukin-1 receptor (TIR) nucleotide-binding site leucine-richrepeat (NB-LRR) class of plant resistance genes. Strong identity of the TIR and NB domains was observed between the predicted proteins encoded by the Ksk-1 allele and the allele from an Acem1-susceptible accession Columbia (Col) (99 and 98 %, respectively). However, major differences between the two predicted proteins occur within the LRR domain and mainly are confined to the â-strand/â-turn structure of the LRR. Both proteins contain 14 imperfect repeats. *RAC1*-mediated resistance was analyzed further using mutations in defense regulation, including: *pad4-1*, *eds1-1,* and *NahG,* in the presence of the *RAC1* allele from Ksk-1. White rust resistance was completely abolished by *eds1-1,* but was not affected by either *pad4-1* or *NahG* (Borhan, 2004).

A second white rust resistance gene (WRR) named WRR4 was also cloned form *A. thaliana* accession Col (Borhan *et al*., 2008). WRR4 encodes for a TIR-NB-LRR protein and is resistance to *A. candida* races from Brassica (races 2, 7 and 9 from *B. juncea, B. rapa* and *B. oleracea* respectively) as well as race 4 from *Capsella bursa-pastoris*. WRR4 resistance is dependent on the functional expression of eds1. Expression of the *A. thaliana* WRR4 in *B. juncea* (susceptible to race 2) and a *B. napus* lines susceptible to race 7, provided full immunity (Borhan *et al*., 2010).

A single gene (*Acr*) responsible for conferring resistance to *A. candida* was mapped on a densely populated *B. juncea* RFLP map. Two closely linked RFLP markers identified (X42 and X83) were 2.3 and 4 cM from the *Acr* locus, respectively (Cheung *et al*., 1998).

9.7 Albugo Genome

Genetic and genomics research on *Albugo* was hampeered by the fact that it is an obligate biotroph pathogen and could not be cultured axenically. However recent technological advances in genome sequencing and advent of next generation sequencing technology has made it possible to sequence the genome of two *Albugo* species, *A. candida* (Links *et al.,* 2011), and *A. laibachii* (Kemen *et al*., 2011). *Albugo candida* has a compact geneome (approximately 45 Mb) and it is almost half of the the oomycete *Hyaloperonospora arabidopsidis* genome (99Mb). Another feature of *A. candida* geneome is reduction in the number of pathogenicity factors and reduced retention of certain bosynthetic pathways, fatures that are for *A. laibachii* and are signature of biotrophy.

10. Techniques

10.1 Growth chamber inoculation technique

In a most widely used growth chamber inoculation technique Verma *et al.* (1975) and Verma and Petrie (1979) described that seeds of the susceptible *Brassica* cultivar are planted 2 cm deep in a soil-free growth medium (Stringham, 1971) in 10 cm square plastic pots. Seedlings are thinned to ten plants per pot. Plants are grown in a growth chamber with an 18-h photoperiod ($312\mu EM^{-2}S^{-1}$) and at day-night temperatures of 21°C and 16°C, respectively. Pots are placed in metal trays and watered by flooding the trays.

Inoculum was prepared by dispersing zoosporangia from pustules from-infected fresh or frozen leaves in deionized distilled water, filtered through cheese cloth, germinated for 2-3h at 5°C, and adjusted to 75000-100,000 zoopores per ml. The inoculum was sprayed on to plants with an atomizer until leaf run-off. Control plants were sprayed with distilled water. The plants were placed in a mist chamber (100 % relative humidity) in the growth chamber for 72 h at 16°C to promote infection, and disease incidence and severity recorded 10-days after inoculation. Several greenhouse and growth

chamber inoculation techniques with similar parameters have also been reported (Goyal *et al*., 1996 b; Singh *et al*., 1999; Bansal *et al*., 2005).

Rimmer *et al.* (2000), and Li *et al.* (2007) used WR pustules collected 10 days prior to inoculation (dpi) and stored at -80°C. For use as an inoculum, zoosporangia were dispersed from infected cotyledons into deionized water and filtered through cheesecloth to remove plant debris. The concentration of the zoosporangia was determined using a haemocytometer and adjusted to 10^5 zoosporangia ml^{-1}. Fully expanded cotyledons from seedlings, 10 days after sowing, were inoculated by spotting 10µl of the zoosporangial suspension onto the adaxial surface of each of the two lobes of each cotyledon. Plants were then subjected to 4 days of enhanced humidity (=95 % RH). At 4–5 leaf stage (4 weeks after sowing when the 5th leaf was emerging), plants were inoculated by spraying a suspension of 10^5zoosporangia/ml until run-off. Plants were subjected to 4 days of enhanced humidity by placing each pot into a sealed plastic bag that had been pre-moistened with DI water. The propagators were placed in an air-flow-bench under spore free conditions in the glasshouse at 18°C ± 2°C (Jenkyn *et al*, 1973; Nashaat & Rawlinson, 1994) with supplementary light to maintain a 16 h light/ 8h dark; day/ night cycle. The seedlings were sprayed with sterilised distilled water (SDW) to clean the surface of cotyledons 24 h prior to inoculation.

Meena (2007) prepared sporangial suspension by adding 1 to 2 ml SDW to glass vial containing excised frozen or freshly sporulating cotyledons. The vial was shaken vigorously on a vortex shaker to facilitate the release of sporangia from the sporangiophores. The concentration of the sporangia was determined using haemocytometer and adjusted to 2.5 x 10^4 sporangia/ml. Each cotyledon was inoculated with two 5ml droplets of sporangial suspension using a micropipette. Alternatively, the plants were sprayed to run off with the spore suspension using an atomiser. After inoculation, the propagators were covered with clear plastic lids and sealed with insulation tape to maintain approximately 100% RH. The plants were then placed in a growth chamber for 12 days at 16°C with 8 hours darkness initially, followed by 16 hours photoperiod with 70-120µmol/m^2/s irradiance.

10.2 Oospore germination

The most conspicuous symptoms of WR and probably the major cause of yield loss are distortion and hypertrophy of infected inflorescence called "staghead". When ripe, stagheads are almost entirely composed of numerous brown, thick-walled oospores, the form in which the pathogen survives during the off-season, and also the source of primary infection. Despite their importance in the epidemiology, conditions under which the oospores germinate have largely been a mystery until the reports of Petrie and Verma (1974) and Verma and Petrie (1975). Prior to this report, only De Bary (1866) and Vanterpool (1959) have described oospore germination in *A. candida*. Vanterpool (1959) reported germination as "always irregular and uncertain", never exceeding 4 % of the spores. Verma and Bhowmik (1988) observed that treatment of oospore with 200ppm $KMNO_4$ for 10 minutes induces increased germination. Petrie and Verma (1974); Verma and Petrie (1975), however devised three reproducible techniques which all gave very high percentage of germination.

In the first method, a small amount of finely ground staghead powder consisting largely of oospores was scattered over moist filter paper placed on wet cotton in a petri dish; the lid of the dish was also lined with moist cotton. The plates were incubated at 10-15°C for the period of up to 3 weeks. In the second method, sterile deionized water or strile or non-strile tap water was allowed to drip slowly onto sintered glass filters of ultrafine porosity where small amounts of oospore powder were scattered. This was done in an attempt to mimic the leaching action that might occur during spring from melting snow or rain. Most of these experiments were run at 10-15°C. In the third method, which the authors most routinely used, a small amount of oospore powder was placed in 50 ml sterile water in a 125 ml flask and incubated at 200 rpm on a rotary shaker at 18-20°C for a period of 3-4 days. The spore suspension was then poured into a petri dish and kept stationary at 13°C for 24h or more. Counts of germinated oospore were made on materials mounted in lactophenol-aniline blue.

All three techniques induced germination of oospores in large numbers. Washing of oospores on a rotary shaker for 3-4 days followed by a day in still culture was the most rapid method and gave the highest percentage germination. Oospores required 2 weeks of washing on a sintered glass filter before maximum germination was obtained. On moist filter paper, maximum germination occurred after an incubation period of 21 days.

Three distinct types of germination were observed. In the most common type, the oospore content was divided into numerous zoospores which were then extruded into a globular, thin-walled sessile vesicle. Zoospores subsequently escaped from the vesicle. Initiation of a vesicle to zoospore escape was completed in 3.0-5.2 minutes with an average elapsed time of 4.1 minutes. Between 40 and 60 zoopsores were formed per vesicle (Verma and Petrie, 1975).

In the second germination type, observed only infrequently, a germ tube was produced from the germinating oospore and zoospores which were differentiated in the oospore were discharged through the tube into a so-called "terminal vesicle" formed at the end of the tube. Zoospores subsequently escaped from the vesicle (Verma and Petrie, 1975). A less commonly observed mode of germination was by a simple or branched germ tubes. Occasionally up to three branches were observed on a germ tube (Verma and Petrie, 1975).

We still do not know how long oospore can remain viable in soil or plant debris. In their extensive studies on viability of oospores, Verma and Petrie (1975) however, reported that more than 50% of the oospores germinated in all staghead samples with the exception of 1953, 1956 and 1959. Germination of 43 % of oospores from staghead material kept in storage for 20 years (1953 material) does indicated their potential longevity. Since the authors recorded the highest percentage of germination (70 %) in 1973 samples which had been collected only 2 weeks prior to the test, their results suggest that oospores do not appear to require any dormancy period.

10.3 Oospores as primary source of inoculum

Oospores are formed in the *A. candida*-infected hypertrophied tissues of inflorescence, stem, pod, roots (Lakra and Saharan, 1989b; Goyal *et al*., 1996b) and senesced leaves (Verma and Petrie, 1978). The oospores are important both for initation of the disease (Butler, 1918; Butler and Jones, 1961; Chupp, 1925; Kadow and Anderson, 1940; Walker, 1957), as well as for the survival of the pathogen in the absence of the host (Verma and Petrie, 1975).

However, in the absence of a reliable method of germination, the role of oospores both as over-wintering agent, as well as incitant of primary infection have largely been speculated. Even after the germination of the oospores, information is still locking whether the zoospores from germinating oospores are capable of infecting rapeseed host plants. Since the emerging cotyledons are the most likely infection sites in the field, Verma *et al*. (1975) grew plants of susceptible *B. rapa* cv. Torch in the growth chamber (under conditions described earlier) kept them at cotyledon stage by removing the growing points. Cotyledons of 10-day-old plants were drop-inoculated with zoospore suspension derived from germinating oospores. Plants were kept under a mist for 3 days. Ten days after inoculation nearly every inoculated plant showed heavy infection in the form of white pustules on the underside of cotyledons. These infection studies suggest that zoospores from germinating oospore are the main infecting units for initiation of primary infection.

Verma and Petrie (1980) also investigated the importance of oospore as a source of primary inoculum in a field experiment conducted under irrigated and dry land conditions. The treated plots were seeded with seeds of susceptible *B. rapa* cv. Torch mixed with an equal weight of oospore powder. The control plots received no oospore powder. Both number of pustules per infected leaf, and the percentage of plants with stagheads were significantly higher in oospore-infested than those in the non-infested plots. These results convincingly suggest that oospores over-wintered in soil, or carried on the seed as contaminant, are most likely the primary source of infection.

Recently, Meena and Sharma (2012) used a mixture of 1 % b-glucuronidase arylsulfatase (available from Sigma) for germination of oospores from hypertrophied plant tissues in 1:9 ratio in SDW which was then stored in 10ml vials in the refrigerator. 100 mg of staghead powder was suspended in 10 ml enzyme dilution and incubated on a rotary shaker (200 rpm) at room temperature for 24 h. On the second day the suspension was centrifuged to pellet the spores and washed three times with 20 ml SDW (mixing them by centrifuging after each wash). The oospore suspension was returned to the rotary shaker for 48-72 h, centrifuged, resuspended in fresh water daily till the sixth day, suspension transferred to empty flask, and chilled at 10ºC for 24 h. The suspension was removed from refrigerator and brought to room temperature before inoculation.

10.4 Induction of staghead in flower-bud inoculated plants

In the past, it was a common blief that the hypertrophies or stagheads are produced as a result of early infection of young seedlings and systemic development of the fungus in the plant. However, this theory was rejected when Verma and Petrie (1979) and Goyal *et al*. (1996b) routinely obtained stagheads by artificially inoculating flower buds of plants grown under growth chamber and greenhouse conditions. These results of growth chamber and of several field experiments (Verma and Petrie, 1979, 1980) conclusively proved that a large percentages of stagheads in the field are produced as a result of secondary infection of flower buds rather than a systemic development of the fungus in the plant. This flower bud inoculation technique at growth stage 3.1 (Goyal *et al*., 1996b) is now routinely being used for screening advanced breeding lines at the Agriculture Canada Research Station, Saskatoon, Canada. Results of these studies are also useful in determining actual time of application of both protectant and systemic fungicides to control WR.

10.5 Detached-leaf culture technique

In order to make more economic use of growth chamber space for screening germplasm for resistance, and to determine effects of abiotic factors on temporal development of *A. candida* infection and oospores development, Verma and Petrie (1978) investigated use of detached-leaf-culture-technique. Healthy leaves from the rosette of 12-14-day-old *B. rapa* seedlings are detached and transferred to petri dishes containing 20-25 ml of autoclaved medium consisting of 0.5 ppm benzyl adenine and 0.8% agar. Leaves are placed in the dishes with their lower surface on the medium usually within 15 minutes of detachment. Four leaves are placed in a plate and at least 20 leaves are used per treatment. Leaves are drop-inoculated with a zoospore suspension (75,000-100,000 zoospore/ml) derived from zoosporangia of *A. candida* race-7. Control leaves are treated with distilled water. A clean but generally non-sterile technique is used and no attempt is made to manipulate leaves aseptically or to sterilize the inoculum. Leaves are kept under 100 % relative humidity for 72-h with day-night temperatures of 21 and 16ºC, respectively. Following an initial 24-h dark period, an 18-h day ($312\mu EM^{-2}S^{-1}$) is maintained for the duration of the experiment. Observations are recorded 14 days after inoculation.

Plant susceptibility ratings of various *Brassica* species and breeding lines on the inoculated detached leaves are essentially the same as when intact plants are used as the host. In addition, the detached-leaf-culture-technique has several advantages to the researchers. The method facilitates the establishment and maintenance of single zoospore cultures and should enable almost complete isolation from extraneous inoculum, including races of *A. candida.* Detached-leaf culture also results in greater uniformity of experimental units, more economic use of growth and mist chamber space, and allows greater use of environmental control. From the plant breeder's point of view, the program efficiency is increased, since the breeder can select resistant material for inter-crossing from among a vigorous growing plant population rather than a weak group of resistant plants that have survived the unfavourable environment necessary to obtain differential infection on potted plants.

10.6 *In vitro* callus cultures of *A. candida*

Preliminary dual *in-vitro*-culture of *A. ipomoeae-*

panduraneae and species of *Ipomoea* (Singh, 1966), *A. candida* race 2 and *B. juncea* (Lahiri and Bhowmik, 1993), and unidentified race of *A. candida* and *B. juncea* (Goyal *et al*., 1995) have been established. Although, investigators report the presence of zoosporangia and oospores in callus tissues derived from hypertrophied stems (Singh, 1966, Goyal *et al*., 1995), or hypertrophied peduncles or thickened terminal leaves (Lahiri and Bhowmik, 1993), but the origin of both sexual and asexual spores is questionable, because the hypertrophied tissues used as explants in their studies are known to almost entirely composed of thick-walled oospores (Verma and Petrie, 1975; 1979; Saharan and Verma, 1992; Verma and Bhowmik, 1988).

Using explants from freshly-inoculated leaves, Goyal *et al.,* (1996c) very successfully established dual-*in vitro* callus cultures of *A. candida* race 7V and *B. rapa* cv. Torch on MS medium (Murshige and Skoog, 1962) supplemented with 1.0mgL^{-1} ´-naphthalene acetic acid and 1.0 mgL^{-1} benxylamino-purine. These authors have provided evidence for: a) production of zoosporangia, oospores, and parthenogenetic-like oospores; b) establishment of haustorial-connections with host cells; c) origin and development of both antheridia and oogonia; and d) the pathogenicity of the zoospores from *in-vitro*-produced zoosporangia and oospores.

Goyal *et al.* (1996c) reported that : a) callogenesis was observed within 7-8 days of incubation; b) proportion of callused explants was significantly affected by the type and concentration of growth regulators; c) under both light and dark conditions, the length of incubation period significantly affected the presence and development of haustoria, zoosporangia, oogonia, antheridia and oospores; and d) the callus tissues incubated in the light were hard, nodular, and green, compared-to soft, watery, and become yellow in the dark.

Zoosporangia were observed in the longest numbers of calli at 8 days incubation, and after this their numbers declined consistently; zoosporangiophores without zoosporangia grew out of the callus cells after 18 days of incubation. In callus cells, the zoosporangiophores were long, knotted, branched, and indeterminate, compared to the short, club-shaped, unbranched, and determinate in infected leaves. By subculturing the calli every two weeks, for 18 weeks, the *A. candida- B. rapa* dual cultures were maintained. After 18 days of incubation and until the end of the observation period, haustoria similar to those reported in infected leaf tissues (Verma *et al.,* 1975) were observed in the cytoplasm of callus cells, or between the cell wall and the cell membrane.

The development of antheridia and oogonia among the callus cells were observed after 13-days of incubation and until the end of the observation period. Two types of oospores, mature oospores with characteristic features including wall layers and a coenocentrum, or two coenocentra, and parthenogenetic-like oospores were observed after 18-days of incubation. The parthenogenetic-like oospores were oval, devoid of warty layers like typical mature oospores, often germinated by a germ tube, and were associated with haustoria inside the callus cells. Pathogenecity test on seedlings of *B. rapa* cv. Torch using zoospores derived from *in-vitro*-produced zoosporangia and germinating oospores confirmed the viability and the virulence of *A. candida* in dual callus cultures (Goyal *et al.,* 1996c).

The *A. candida –B. rapa* dual culture system reported by these authors has potential for sexual studies of the fungus. Because it was possible to trace the development of antheridia and oogonia from the mycelium, which support the view that isolates of *A. candida* race 7V are homothallic. This dual culture system can also be useful *in vitro* selection studies for recovering resistant cells.

Debnath *et al*., (2001) reported that the host callus and the pathogen establishes a complete balance in culture, and the morphology of the mycelium, haustoria, zoosporangia, antheridia, oogonia and oospores in dual culture is identical to that of infected intact plant. Oospore formation is favoured over that of sporangia, and oospore germination by germ-tube is evident. Growth of dual culture is influenced by light quality, temperature, vitamins, carbohydrates and amino acids in the medium. These

differential responses can be used for future studies on host pathogen interactions and for breeding of disease resistant plants. Lahiri and Bhowmik (1993) maintained *A. candida* in infected callus tissue for prolonged periods by periodic subculturing and it kept pace with the growth of the callus tissue.

11. Epidemiology

Temperature gradient plates (Smith and Reiter, 1974) and detached-leaf-culture technique (Verma and Petrie, 1978) were used to determine effect of temperature on temporal progression of white rust on a) leaves of different ages, b) leaves detached at the end of light and dark periods, c) type and number of zoosporangial pustules on abaxial and adaxial leaf surfaces, and development of oospores (Verma *et el.,* 1983; Goyal *et al.*, 1996a; Bartaria and Verma, 2001). It is essential to determine exact parameters for disease development before detached-leaf-culture technique can be used to screen rapeseed-mustard cultivars for resistance against *A. candida.*

11.1 Temperature effects on disease development:

Temperature, leaf age, time of leaf detachment, and the interaction of these factors had a significant effect on the temporal development of *A. candida* race 7 on detached leaves (Verma *et al.,* 1983). Of the temperatures tested (3-32^{o}C), 21^{o}C gave the best disease development, with 18.5^{o}C being the calculated optimum. The disease did not develop at 3^{o}, 29^{o}, and 32^{o} C, and was slow to develop at 9^{o}, 12^{o}, and 27^{o}C. There was a highly significant ($p<0.01$) interaction between length of incubation period and temperature. Unlike intact plants, detached leaves developed pustules on both surfaces. Infection occurred on leaves of all ages, but medium-aged leaves supported the maximum number of pustules, followed by the younger leaves. Leaves detached at the end of a dark period developed more pustules than those detached at the end of light period. While using detached leaf culture technique for screening germplasm for resistance to white rust, Verma *et al.* (1983) advised inoculation of adaxial surface of cotyledons of medium aged leaves, with an incubation temperature of 18-22^{o}C. Sullivan *et al.* (2002) observed that only 3 h of leaf wetness is required for disease development at optimum temperature range of 12 to 22°C.

The nonavailability of forecast system for major diseases of oilseed Brassicas in India does not allow farmers to make timely and effective fungicidal sprays. In one of the multilocation study conducted for 8 years, Chattopadhyay *et al.* (2011) observed the initiation of WR disease on leaves of mustard during 29-131 days after sowing (DAS), highest being at 54 DAS. Severity of WR disease is favoured by >40 % minimum afternoon and >97 % maximum morning relative humidity (RH) and 16-24^{o}C maximum temperature. Staghead formation is significantly and positively influenced by 20-30^{o}C maximum (>12^{o}C minimum) temperature and >97 % maximum morning RH.

11.2 Temperature effects on oospore development

Epidemiological studies on *A. candida* have focused on the production, viability and germination of zoosporangia (Melhus, 1911; Endo and Linn, 1960; Lakra *et. al.,* 1989), and the influence of host age and time of leaf detachment on development of the disease (Verma *et. al.,* 1983).

Little is known about the sexual reproduction and genetics of the fungus due to the difficulty in determining the factors responsible for induction of the sexual reproductive phase. The effect of temperature on *in vitro* germination of oospore has been reported (Verma and Petrie, 1975), however, information on the optimum temperature and the time required for production of oogonia, antheridia and mature oospore in leaf tissue would assist in designing experiments for the study of oogenesis, fertilization and karyogamy. Using temperature gradient plate (Smith and Reiter, 1974) and detached leaf culture technique (Goyal *et al.*, 1996a) established effect of temperature and incubation period on progressive development of oospores of *A. candida* race 2V in *B. juncea* leaves.

The progressive development of *A. candia* oospores in detached leaves of *B. juncea* is largely

dependent on incubation temperature. Oogonia and oospore production occurred over the entire range of incubation temperatures of 10-27°C. The earliest development of oogonia is observed at 25°C, 7days after inoculation and incubation. The largest number of oogonia at the 21°, 23°, 24° and 25°C treatments is observed 12 days post inoculation and numbers decreased thereafter; at lower and higher temperatures; development of oogonia occurred later. Maximum numbers of oogonia are recorded after 17 days at 15°C treatment at the end of the experiments. Mature oospores are observed 12 days after incubation at 23° and 24°C. The number of mature oospores was still increasing at 17 days post-inoculation in all treatments. Mature oospores developed later and more slowly at lower and higher incubation temperatures.

The production of *A. candida* oospores in leaf tissues can be important in disease perpetuation. Hypertrophied tissues (staghead) are quite resistant to decomposition and the release of oospores can take 3-4 years. Leaf tissues are quick to decompose, and thus oospore release from such material could be expected the following year. In naturally-infected leaves, oospores are produced in the later part of the season when temperatures are warm (Verma, 1989). Warm temperatures hasten leaf senescence, which in turn enhances tissue decomposition and early release of oospores.

The knowledge of an optimum temperature and time for the development of oospores in detached leaves in their study made it possible to compare the sequential events of oogenesis, fertilization and karyogamy in various *Albugo* species at the earliest stages of their development. These comparative investigations in *Albugo* species can also be useful in fungal taxonomy. The detached leaf culture technique for oospore development can also be used to determine the heterothallic nature of *A. candida*.

11.3 Temporal development of *A. candida* infection in cotyledons:

Verma *et al.,* (1975) determinied temporal progression of WR infection in cotyledons of susceptible (*B. rapa, B. juncea*), moderately resistant (*B. hirta*), and immune (*B. napus*) cultivars. Cotyledons of all four *Brassica* species were inoculated with zoospores of *A. candida* produced from germinating oospores or zoosporangia. At different times after inoculation, whole cotyledons were fixed in 95 % ethanol-acetic acid (v/v) solution, cleaned in 70 % lactic acid at 40°C for 3-4-days, and stained with cotton blue in lactophenol. The preparation was examined under the compound microscope. Generally, the sequence of events from zoospore encystment to formation of the first haustorium was the same in all hosts, although under field conditions, *Brassica hirta* is moderately resistant and *B. napus* is essentially "immune". In *B. juncea* the first haustorium was observed 16-18 h after inoculation, while in *B. rapa, B. hirta* and *B. napus* the first haustorium was observed about 48 h after inoculation. In the susceptible hosts, after the formation of the first haustorium, the hyphae grew rapidly and produce variable number of haustoria in each cell. The profusely branched, nonseptate mycelium appeared to fill all available intercellular spaces, and in five to six days after inoculation, the club-shaped zoosporangia develop from a dense layer of mycelium.

In the immune host, usually only one haustorium was formed, after which the hyphae ceased to elongate. At about 72 h after inoculation, a fairly thick, densely stained encapsulation was usually detected around each haustorium, and later only "ghost" outlines of hyphae and haustoria were observed. Encapsulations were not observed around haustoria of susceptible hosts.

From these observations (Verma *et. al.,* 1975) it seems probable that zoospores derived from germinating oospores constitute the primary inoculum for infection of cotyledons of susceptible *Brassica* species. No evidence of direct infection by the germ tubes was seen (Verma and Petrie, 1975).

The establishment and maintenance of compatible relationship between *A. candida* and its hosts hinges on the successful formation of the first haustorium. A similar sequence of events in both susceptible and immune hosts upto this point suggests that there

appears to be no morphological barrier to zoospore encystment, germination and subsequent penetration through stomata. In the incompatible combination it is not clear whether the parasite fails to produce a functional haustorium, or whether a viable haustorium is formed within the host cell and is subsequently killed by the host's defence mechanism. The fairly dense, thick encapsulation observed around haustorium of immune host tissue suggests that the later may be the case. In any event it does seem that the decision between compatibility and incompatibility is made within 48 h after inoculation.

Studies using whole mounts (Verma *et.al.,* 1975) can provide a rapid and useful quantitative means of measuring fungal development and can be useful in screening for disease resistance or testing the effects of environmental changes or fungicide treatments. Whole mounts may also provide a useful perspective for ultrastructural studies where the total amount of fungal thallus present in a susceptible host is not always appreciated. Certainly, the massive amount of intercellular mycelium, particularly the much-branched sporangiophore "base", which the host is capable of supporting while still actively photosynthesizing, emphasizes the highly integrated and delicate control occurring in the type of parasitism that has evolved in *A. candia.*

12. Association of *Albugo* and *Hyaloperonospora*

The association or mixed infection, or simultaneous occurance of *A. candida* and *Hyaloperonospora brassicae* pathogens on leaves, inflorescence and silique of oilseed *Brassica* in nature is very common (Saharan and Verma, 1992). The intensity of mixed infections varies from 0.5 to 35.0 per cent. It is reported that *A. candida* predisposes the host tissues to infection by *H. brassicae* (Bains and Jhooty, 1985; Saharan and Verma, 1992; Saharan and Mehta, 2002). However, Soylu *et al.* (2003) reported that the *H. parasitica* infections are first apparent to the naked eye as a carpet or ''down'' of conidiophores covering the upper and lower surfaces of leaves and petioles, a symptom characteristic of DM diseases. The zoosporangia of *H. parasitica* emerge in profusion from stomata without forcible damage of host tissue (Borhan *et al*., 2001). While both these pathogens usually exist as specialized pathotypes on different cruciferous species, and even on different cultivars within a species, asexual reproduction, in general, is most prolific on the particular host of origin (Mathur *et al*., 1995a; Nashaat and Awasthi, 1995; Petrie, 1988; Pidskalny and Rimmer, 1985; Saharan and Verma, 1992; Silue *et al*., 1996). Normally *A. candida* occurs in intimate association with *H. parasitica* (Holub *et al*., 1991) including on stagheads (Awasthi *et al*., 1997) in crucifers. Bains and Jhooty (1985) found that *H. parasitica* colonies commonly occurring among those of *A. candida* on plant tissues. They studied the association of *H. parasitica* with *A. candida* on *B. juncea* leaves and proposed that *A. candida* biochemically pre-disposes the host plants to *H. parasitica*. However, because the incubation period of *H. parasitica* is shorter than that of *A. candida*, they found that *H. parasitica* colonies tend to develop first, followed by *A. candida* under glasshouse conditions. In contrast, they found that the situation could be the reverse under natural conditions (Bains and Jhooty, 1985). Under field conditions, *A. candida* possesses the capacity to elevate the incidence and severity of infection by *H. parasitica* in crucifers (Constantinescu and Fatehi, 2002), and similar situations have been described for *H. arabidopsis* in *Arabidopsis thaliana* (Holub *et al.,* 1991) and in *B. juncea* (Cooper *et al*., 2008) after pre-inoculation with *A. candida.* Singh *et al*. (2002a) studied that the infection of *B. juncea* with a virulent isolate of *H. parasitica* inhibited or adversely affected the development of a virulent isolate of *A. candida* after simultaneous coinoculation of *B. juncea*, while an avirulent isolate of *A. candida* induced host resistance toward *H. parasitica*. Previous findings suggest that the inoculation order of the two pathogens may be a critical factor in determining the outcome of the interaction of two pathogens. Kaur *et al*. (2011b) observed that the inoculation of *B. juncea* with an asymptomatic isolate of *H. parasitica* and subsequently with a virulent isolate of *A. candida*, not only reduced the incubation period but also increased the severity of disease caused by the WR pathogen. They also

determined that although *H. parasitica* was asymptomatic in the host, it systemically colonized host tissues away from the site of inoculations.

13. Pathogenic variability in *A. candida*

Physiological specialization has long been known in *A. candida*. Eberhardt (1904) recognized two specialized groupings of *Albugo* one attacking *Capsella, Lepidium* and *Arabis*, and other attacking *Brassica, Sinapis* and *Diplotaxis;* he was however, hesitant to use the phrase biological forms. Melhus (1911) also suggested the existence of specialization in *A. candida*. Pape and Rabbas (1920) demonstrated that the fungus on *Capsella bursa-pastoris* should be considered a distinct form. Savulescu and Rayss (1930) distinguished eight morphological forms within *A. candida,* and in 1946, Savulescu estabilished 10 varieties of *A. candida* based on host specialization and morphology. Hiura (1930) distinguished three biologic forms of *A. candida* on *Raphanus sativus, B. juncea* and *B. rapa sp. chinensis.* Napper (1933) described 20 races of *A. candida* in Britain. Togashi and Shibasaki (1934) found that sporangia of *Albugo* from *Brassica* and *Raphanus* were 20 x 18 µm in size, while those from *Cardamine, Capsella, Draba* and *Arabis* measured 15.5 x 14.5 µm, and classified these as *macrospora* and *microspora,* respectively. Results of these two Japanese studies (Hiura, 1930; Togashi and Shibaskaki, 1934) suggested that five distinct biological forms of *Albugo* were present.

Subsequently, Ito and Tokunaga (1935) elevated the forms with larger spores to the rank of the species *A. macrospora* (Togashu) Ito. Biga (1955) recognized two morphological texa: *A. candida macrospora* and *A. candida microspora,* as proposed by Togashi and Shibaskaki (1934), but renamed them *A. candida microspora* and *A. candida candida,* respectively. On the basis of conidial measurements from 63 host species, Biga (1955) reported that *A. candida* microspora (15-17.5 µm diam.) was restricted to *Armoracia, Brassica, Erucastrum, Raphanus* and *Rapistrum,* whereas *A. candida candida* (12.5-15 µm diam.) had a wide range of cruciferous hosts. Endo and Linn (1960) reported a race of *Albugo* on *Armoracia rusticana.*

It is clear that each of the above authors were hesitant in describing specialized races of *A. candida.* Pound and Williams (1963) identified six races of *A. candida*: race I from *Raphanus sativus* var. Early Scarlet Globe; race 2 from *B. juncea* var Southern Giant Curled; race 3 from *Armoracia rusticana* var Common; race 4 from *Capsella bursa-pastoris;* race 5 from *Sisymbrium officinale,* and race 6 from *Rorippa islandica*. Verma et al (1975) and Delwiche and Williams (1977) added race 7 from *B. rapa* Turnip or Polish rapeseed and race 8 from *B. nigra,* respectively. Novotel'nova (1968) from USSR while analyzing intra-specific texa, reported that *A. candida* species consisted of separate morphological specialized forms confined to a particular range of host plants. Within the morphological forms, races can be differentiated, while within heterogeneous populations, both races and forms can be differentiated. It was considered that geographic and climatic conditions leave their distinguishing mark on the processes of form and populations of the fungus encountered by investigators from different countries. Novotel'nova and Minasyan (1970) and Burdyukova (1980) studied the biology of *A. candida* and *A. tragopogonis* in former USSR and conducted an in-depth study on the extent of specialization of *A. candida.*

In India, Singh and Bhardwaj (1984) tested 12 *Brassica* species and identified 9 races from four hosts, viz, *B. juncea, B. rapa* var. Toria, *B. campestris* var Bbrown Sarson and *B. rapa* var. Pekinensis. Lakra and Saharan (1988c) identified five races of *A. candida* on the basis of its reaction on a set of 16 host differentials. They identified two distinct races from *B. juncea* which were different from the previous records. One (race 2), attacked *B. nigra, B. juncea* and *B.*

rapa var. Brown Sarson, and the other (race 3) infected only *B. juncea* and *B. rapa* var Toria. Bhardwaj and Sud (1988) tested 26 cultivated and wild cruciferous hosts and identified nine new biological races from nine hosts, viz, *B. rapa* var. Brown Sarson cv. BSH 1, *B. rapa* var. Toria cv. OK-I, *B. juncea* cv. Varuna, *B. chinensis. B. rapa* var. Pekinensis cv. Local, *B. rapa* cv. PTWG,

Raphanus sativus cv. Chineses Pink, *Raphanus raphanistrum* wild radish and *Lepidium virginicum* wild. They reported that reaction of nine isolates of *A. candida* differed from each other on 26 differential hosts revealing thereby, that the monotypic pathogen *A. candida* on crucifers existed in the form of different biological races designated as new biological races or forms 1 to 9.

The concept of races in *A. candida.* as proposed by Pound and Williams (1963) was based on species relationships. Studies have, however, clearly demonstrated that cultivars of *Brassica* crops must be included in a set of host differentials to distinguish isolates of the pathogen within a present accepted race (Burdyukova, 1980; Pidskalny and Rimmer, 1985). There is an urgent need to standardize host differentials keeping in mind the homogenity and purity of species and varieties. Petrie (1988) using North American race 2 and 7 from *B. juncea* and *B. rapa,* respectively, have screened accessions of several *Brassica* species including *B. rapa* var. Yellow Sarson, *B. rapa* var. Brown Sarson, *B. rapa* var. Toria and *B. juncea* from India, both yellow and brown sarson, were equally highly susceptible to both races, toria only to race 7, and *B. juncea* only to race 2.

A detailed study is needed to determine whether the races of *A. candida* attacking *B. juncca* and several *B. rapa* crop in India are similar to race 2 and 7 from Canada and the USA. Kolte *et al*. (1991) reported that the WR isolate obtained from *B. rapa* appeared to be distinct in pathogenicity from the one obtained from *B. juncea* under Indian conditions. Petrie (1994) in Saskatchewan and Alberta, Canada discovered new races 7v in 1988 and race 2v in 1989. Verma et al (1999) reported two new races of *A. candida* in India viz., race 12 from *B. juncea* and race 13 from *B. rapa* var. Toria using 14 (including 6 standard) crucifer host differentials.

Mathur *et al.* (1995b) and Rimmer *et al*. (2000) collected isolates of *A. candida* from different geographic locations in Western Canada and tested virulence on a number of cultivars and accessions of *Brassica* species. Most isolates were identified as race 7, which could be subdivided into 7a and 7v on the basis of their virulence on *B. rapa* cv. Reward. Isolates 28-7 and 29-1 were avirulent to all the differentials except the rapid cycling *B. rapa* CrGCI-I8. Tower isolates, 11-6 and 41-4, which could infect cultivars of both *B. rapa* and *B. juncea,* appeared to be hybrids between race 2 and race 7. Wu *et al.* (1995) studied genetic variation among isolates of *A. candida* using randomly amplified polymorphic DNA (RAPD) with five selected random primers fingerprint patters generated for each isolates. Most polymorphism was found between different races than among isolates within a single race. Most Canadian field isolates were grouped as race 7 and could be further subdivided into two groups (7a and 7v). Classification of *A. candida* isolates based on the results from the RAPD analysis was identical to the virulence classification on 10 *Brassica* differentials.

Four distinct and new pathotypes of *A. candida* viz, ACI4 from RL 1359, AC 15 and AC 16 from Kranti, and AC 17 from RH 30 cultivars of *B. juncea* have been identified on the basis of their differential interactions on 11 host differentials by Gupta and Saharan (2002). Jat (1999) identified 20 distinct pathotypes of *A. candida,* 17 from *B. juncea* (AC 18 to AC 34), 2 from *B. rapa* var. Brown Sarson (AC 35 to AC 36), and one from *B. nigra* (AC 37). From Western Australia, Kaur *et al*. (2008) identified pathotype AC 2A from *B. juncea* and pathotype AC 2v from' *Raphanus raphanistrum.*

The pathogenic variability recorded in *A. candida* in the form of races are: 2 from Australia, 20 from Britain, 4 from Canada, 2 from Germany, 49 from India, 8 from Japan, 18 from Rumania and 7 from USA. However, nomenclature of *A. candida* races came into practice after the use of host differentials to distinguish races by Pound and Williams (1963). Global virulence of *A. candida* based on primary host is documented in Table 2. In *A. candida,* the sexual reproduction in the form of oospores is very common especially on *B. juncea*. Therefore, numerous races are expected to exist. In addition to this, other mechanism of variability including recombination, mutation and heterokaryosis are also in operation in the nature. To get the true picture of *A. candida* races and virulence spectrum, there is

Table 1: Global virulence of *A. candida* pathotypes (Saharan, 2010)

Pathotype designate	**Country**	**International primary host**	**Reference**
AC1	North America	*Raphanus sativus*	Pound and Williams, 1963
AC2	North America	*Brassica juncea*	Pound and Williams, 1963
AC2V	North America	*B. napus*	Petrie, 1994
AC3	North America	*Armoracia rusticana*	Pound and Williams, 1963
AC4	North America	*Capsella bursa-pastoris*	Pound and Williams, 1963
AC5	North America	*Sisymbrium officinale*	Pound and Williams, 1963
AC6	North America	*Rorippa islandica*	Pound and Williams, 1963
AC7	North America	*B. rapa*	Verma *et al*., 1975
AC7V	North America	*B. rapa* cv. Reward	Petrie, 1994
AC8	North America	*B. nigra*	Delwiche and Williams, 1977
AC9	North America	*B. oleracea*	Williams, 1985
AC10	North America	*Sinapis alba*	Williams, 1985
AC11	North America	*B. carinata*	Williams, 1985
AC12	India	*B. juncea*	Verma *et al*., 1999
AC13	India	*B. rapa* var. Toria	Verma *et al*., 1999
AC1 to 9	India	*Brassica* species	Singh and Bhardwaj, 1984
AC1to 5	India	*Brassica* species	Lakra and Saharan, 1988c
AC14	India	*B. juncea* cv RL 1359	Gupta and Saharan, 2002
AC15	India	*B. juncea* cv Kranti	Gupta and Saharan, 2002
AC16	India	*B. juncea* cv Kranti	Gupta and Saharan, 2002
AC17	India	*B. juncea* cv RH 30	Gupta and Saharan, 2002
AC18 to 34	India	*B. juncea* cv RH 30; EC 182925; DVS 7-3-1	Jat, 1999
AC35 and AC36	India	*B. rapa* var. Brown Sarson	Jat, 1999
AC37	India	*B. nigra*	Jat, 1999
AC2A	Western Australia	*B. juncea* cv Vulcan; Commercial Brown	Kaur *et al*., 2008
AC2V	Western Australia	*Raphanus raphanistrum*	Kaur *et al*., 2008

an urgent need to standardize host differentials for each crucifer species in the form of isogenic lines at international level. Standard nomenclature of the races viz, Acjun 1, 2,- for *B. juncea* isolates, AC rap 1, 2- for *B. rapa* isolates, AC nig 1,2 - for *B. nigra* isolates and ACol 1,2, - for *B. oleracea* isolates, and so on, appears to be a very useful beginning.

13.1 Virulence spectrum of *Albugo candida*

As per the gene-for-gene hypothesis, interaction of *Albugo*-crucifers for compatibility and incomtability phenotype determines number of virulence genes in the pathotype and resistance genes in the host genotype. It has been observed that pathotypes of *A. candida* from *B. juncea* have wide range of virulence genes. Pathotypes like AC 23, AC 24, AC 17 infects only one, two and three differential hosts respectively, indicating a limited virulence potential. However, pathotypes of wider virulence viz., AC 29, AC 27, AC 30, AC 18 and AC 21 infected 21, 18, 16, 12 and 10 host differentials, respectively (Jat, 1999; Gupta and Saharan, 2002).

Availability of virulence variability in pathotypes from *B. juncea* has suggested the possibility of identification of more number of resistant genes in the genotypes including identification of loci and alleles. In the absence of isogenic lines, it is not clear weather the races with wider virulence attack the same genes in the differentials, or genes for susceptibility are different or situated on different loci or tightly linked.

14. Host resistance

The transfer of resistance from different sources in *Brassica* crops is possible and is being done through conventional as well as modern technologies all over the world (Saharan *et al*., 2005).

15. Genetics of host-parasite interactions

Studies on the genetics of host-parasite interactions in WR disease have been concentrated largely on the level of host genotypes without considering pathogens' races. Although, All India Co-ordinated Research Project on Rapeseed-Mustard (AICRP-RM) have identified several genotypes with stable resistance, but very few have been utilised for developing WR resistant cultivar (Table 2). Thus, understanding of *Brassica* genotypes by *A. candida* interactions is of vital importance in identifing resistant genotypes for specific adaptability. GSL-1, EC 414299 and EC 399299 showed additive gene for horizontal resistance to WR which can prove good donors in further genetic improvement programmes. Varuna, JMM 07-2, JMM 027-1 and JYM 10 had non-additive gene action for pathogenicity to WR. PBC 9221, GSL 1, EC 414299 and EC 399299 were very similar in genetic make-up for disease resistance while Varuna showed maximum divergence in genetic constitution from these strains (AICRPRM, 2009).

Even within the confines of race cultivar specificity, the studies have been one-sided in that no genetic information has been generated on *Albugo*, the causal organism. Interest in such studies was stimulated by Hougas *et al*. (1952), who investigated on the genetic control of resistance in WR of horse radish. The exhaustive work of Pound and Williams (1963) clearly demonstrated that resistance to white rust was controlled by a single dominant gene in radish cv. China Rose Winter (CRW) and Round Black Spanish (RES). Histological studies revealed that resistance in CRW was manifested as a hypersensitive reaction, which might be modified to a sporulating tolerant reaction by environmentally-controlled minor genes.

Humaydan and Williams (1976) while studying the inheritance of resistance in radish to *A. candida* race 1, changed the gene designation R into the more descriptive symbol AC-l derived from the initials and race number of *A. candida*. The resistance to *A. candida* race 1 in *Raphanus sativus* cv. Caudatus was controlled by a single dominant gene, *A C-l.* The resistance gene *AC-I* and the gene *Pi*, controlling pink pigmentation was found to be linked with a recombination value of 3.20 per cent. Bonnet (1981) found that WR resistance in radish variety Rubiso-2 was also controlled by one dominant gene. Among *Brassica* species monogenic dominant resistance to *A candida* race 2 has been found in *B. nigra*. *B. rapa, B. carinata* and *B. juncea* (Delwiche and Williams, 1974; Ebrahimi *et al.*, 1976; Thukral and Singh, 1986; Tiwari *et al*.,

1988). A single dominant gene, *AC-2*, controlling resistance to *A. candida* race 2 in *B. nigra* was identified by Delwiche and Williams (1981). In a study to select quantitatively inherited resistance to *A. candida* race 2 in *B. rapa*, CGS-l, Edwards and Williams (1982) found that variability in reaction to *A. candida* race 2 among susceptible *B. rapa* strain PHW-Aaa-l was due to quantitative genetic regulation and suggested that rapid progress in resistance breeding could be made via mass selection when starting with a susceptible base population.

Canadian cultivars of *B. napus* were resistant to WR, but many cultivars of this species grown in China were susceptible (Fan *et al*., 1983). The inheritance of WR resistance in *B. napus* cv. Regent was conditioned by independent dominant genes at three loci, designated as *AC-7-1, AC-7-2* and *AC-7-3*. Resistance was conferred by dominance at anyone of these loci, while plants with recessive alleles at all loci were susceptible (Fan *et al*., 1983). Verma and Bhowmik (1989) were in part agreement with those of Fan *et al*. (1983) who suggested that resistance of BN-Sel *(B. napus)* to the *B. juncea* pathotype of *A. candida* found in India was conditioned by dominant duplicate genes.

The host-pathogen-interaction-genetics studies indicates, that resistance in host is governed by one, two or more than two dominant genes (*AC*-7-1, *AC*-7-2, *AC*-7-3), additive genes with epistatic effects, and single recessive gene (*WPr*) alongwith a single gene (*WRR4*) confirring broad spectrum resistance to races, *AC*-2, 4, 7 and 9 (Pound and Williams, 1963; Fan *et al.*, 1983; Liu *et al*., 1996; Saharan and Krishnia, 2001; Bansal *et al*., 2005; Borhan *et al*., 2008). The inheritance of virulence in *Albugo- Brassica* system suggested that a single dominant gene controls avirulence in race *AC*-2 to *B. rapa cv*. Torch (Adhikari *et al*., 2003). Systemic resistance in *B. juncea* to *A. candida* can be induced by pre-or co-inoculation with an incompatible isolates of *A. candida* (Singh *et al*., 1999). Resistant genes have been mapped and identified on the chromosomes of *B. juncea* viz., *ACr* (Cheung *et al.*, 1998), *AC*-2_1 (Prabhu *et al*., 1998), *AC*-2 (Varshney *et al*., 2004), *ACB*1-*A*4.1, *ACB*1-*a*5.1 (Massand *et al.*, 2010), *B. rapa* viz., *ACA*1 (Kole *et al*., 1996), *B. napus* viz., *ACA*1 (Ferreira *et al*., 1994), *AC* 2*V1* (Somers *et al.*, 2002) and *A. thaliana viz. RAC*-1, *RAC*-2, *RAC*-3 and *RAC*-4 (Borhan *et al*., 2001; 2008) effective against one or more than one race of *A. candida*.

In a study of inheritance of resistance to *A. candida* race 2 in mustard, Tiwari *et al.* (1988) found that resistance was dominant, monogenic, controlled by nuclear genes, and was easily transferred to adapted susceptible genotypes via back crossing. In a study evaluating performance of 15 advanced generation (F_6) progenies of two interspecific crosses of *B. juncea* and *B. carinata* against *A. candida*, Singh *et al*. (1988) showed significant differences among the hybrid progenies which all gave a resistant reaction. A later study on five interspecific crosses between *B. juncea* and *B. carinata* revealed that the dominant gene which conferred resistance to WR was located in C genome of *B. oleracea* a progenitor of *B. carinata* (Singh and Singh, 1988). Williams and Hill (1986), and Edwards and Williams (1987) have opened unusual potential for resolving many problems relating to host-parasite interactions and breeding for disease resistance through development of rapid cycling *Brassica* populations. Their preliminary studies demonstrated considerable isozyme variations among individuals in a population which when inoculated with several pathogens, showed a wide range of plant to plant variation in the levels of resistance and susceptibility. This will assist plant breeders in developing cultivars with genetic resistance to plant diseases. Gene pools of both major and minor genes for resistance to various crucifer pathogens have been constructed (Edwards and Williams 1987; Hill *et al*., 1988; Williams and Hill 1986) which will be of immense value to plant breeders seeking sources of resistance.

Thukral and Singh (1986) studied the inheritance of WR resistance in two crosses involving resistant (R) and susceptible (S) types of *B. juncea* namely EC 12749 x Prakash and EC 12749 x Varuna under normal and late-sown cohditions and found that analysis of six generations revealed the importance of additive, domi-

nant and epistatic effects. Reciprocal recurrent selection was also advocated for exploiting the additive and non-additive gene effects for resistance to WR. Singh and Singh (1987) reported that when *A. candida* resistant Ethiopian mustard *(B. carinata)* was crossed with *B. juncea*, the interspecific hybrids showed tolerance to *A. candida*. In the study on the inheritance to *A. candida* race 7 in *B. napus*, Liu *et al*. (1987) found that a digenic model with dominant resistance is confirred by R_J and *R2* gene. Presence of a dominant allele at either of the two loci will confer resistance to a plant, whereas homozygous recessive at both loci will result in a susceptible phenotype expression. Liu and Rimmer (1992) studied the inheritance of resistance to an Ethiopian isolate of *A. candida* collected from *B. carinata* using two *B. napus* lines and suggested that resistance to the *B. carinata* isolate was conditioned by a single dominant resistant gene.

Pal *et al*. (1991) evaluated the genetic component of variation for WR resistance through a 12 x 12 diallel crosses involving resistant and susceptible parents of Indian and exotic origin mustard under four sets of environmental conditions *viz*, normal-sown in natural conditions, normal-sown in artificially-created-epiphytotic conditions, late-sown in natural conditions and late-sown in artificially-epiphytotic conditions. Based on these results, they suggested that in all four sets of environments, both additive and non-additive components of variation were significant but an over dominance under the late-sown environment. Gadewadikar *et al* (1993) in their study suggested that resistance to *A. candida* was governed by a single dominant nuclear gene pair which could easily be transferred via back crossing. Paladhi *et al*. (1993) also concluded that the resistance to *A. candida* in an Indian mustard genotype PI-15 was controlled by a single dominant gene.

Bains (1993) reported that resistance in the leaves differed from that of resistance in the young flowers; in the leaves it was due to the CC genome transferred from Indian mustard. Rao and Raut (1994) observed that the susceptibility of *B. juncea* cv. Varuna to the local Delhi pathotype of *A. candida* was conditioned by two genes, with dominant and recessive gene interaction. Interspecific crosses between *B. juncea* and *B. napus* suggested that resistance in WW 1507 and ISN 114 to *A. candida* was controlled by a single dominant gene (Jat, 1999). In their study of three interspecific crosses between *B. juncea* and *B. napus*, Subudhi and Raut (1994) revealed digenic control with epistatic interaction for WR resistance trait and a close association of parental species and different grades of leaf waxiness. Sachan *et al*. (1995) in their study using diallel fashion crosses between two WR resistant Canadian *B. juncea* cvs. Domo and Cutlass, and two susceptible *B. juncea* Indian cvs. Kranti and Varuna, reported that F1 hybrids, except susceptible x susceptible, were resistant; segregation pattern for resistance in F2 and test crosses was under the control of a single dominant gene in Domo and Cutlass, and that a recessive gene for susceptibility was present in Kranti and Varuna. Liu *et al*. (1996) in Canada developed monogenic lines for resistance to *A. candida* from a Canadian *B. napus* cultivar, and suggested that these monogenic lines could be used to study the mechanism of resistance response conditioned by the individual genes. These lines also facilitate molecular mapping of the loci in *B. napus* for resistance to *A. candida* race 7.

In an inter-varietal cross between a susceptible Indian cv. Pusa Bold and a resistant genotype DIRA 313. Mani *et al*. (1996) showed a significant additive x additive interaction for the a) final intensity of WR on plant (FIP), b) final intensity of WR on leaf (FIL), and c) area under disease progress curve (AUDPC) along with the association of complimentary epistatic interactions indicating close association between the nature of inheritance for AUDPC on one-hand, and FIP and FIL on the other. This was also substantiated by a significant correlation between FIP and FIL, and AUDPC suggesting ease in selection for lower AUDPC (slow rusting) through FIP or FIL. Sridhar and Raut (1998) reported a monogenic inheritance showing complete dominance in four crosses and lack of dominance in seven crosses attempted between *B. juncea* and resistance sources derived

from different species. According to Jat (1999), the resistance was dominant in all the crosses except susceptible x susceptible where it was recessive. Under controlled conditions, inoculation with three different races of *A. candida* on F_2 population of crosses from R x R revealed that the resistant genes may be located on the same locus or on different loci. In both intra-specific and interspecific crosses between *B. juncea* X *B. carinata,* Saharan and Krishnia (2001) showed that resistance was dominant in all the crosses. They confirmed that resistance to *A. candida* was governed by one dominant gene or two genes with either as dominant, recessive or epistatic interaction or complete dominance at both gene pairs. Partial resistance in *B. napus* to *A. candida* was controlled by a single recessive gene designated as *wpr* with a variable expression (Bansal *et al*., 2005). Dominant alleles at three unlinked loci (ACh AC7z, and $AC7_3$) conferred resistance in *B. napus* cv. Regent to race AC 7 of *A. candida* (Fan *et al.*, 1983; Liu *et al*., 1996). Two loci also controlled resistance in *B. napus* to *A. candida* race AC2 collected from *B. juncea* (Verma and Bhowmik, 1989). The Chinese *B. napus* accession 2282-9, susceptible to AC7 has one locus controlling resistance to an isolate of *A. candida* collected from *B. carinata* (Liu and Rimmer, 1992). These studies Indicated that only one allele for resistance was sufficient to condition an incompatible reaction in this pathosystem (Ferreira *et al*., 1995). In addition, a single locus controlling resistance to AC2 in *B. napus* and *B. rapa* was mapped using restriction fragment length polymorphism (RFLP) marker (Ferreira *et al*., 1995). A dominant allele at a single locus or two tightly linked loci were reported to confer resistance to both races AC 2 and AC 7 of *A. candida* (Kole *et al.*, 2002). According to Borhan *et al*. (2008), a dominant WR resistant gene, *WRR* 4, encodes a TIR-NB-LRR protein that confers broad-spectrum resistance in *A. thaliana* to four races (AC2, AC4, AC7 and AC9) of *A. candida.*

15.1 Slow white rusting in crucifers

Rate of infection or disease spread is influenced by incubation and latent periods of *A. candida* in its compatible host. In WR, the sporangia become visible after the host epidermis is ruptured as a white powdery mass which can readily be dispersed by wind or rain drops to cause secondary infection. In rapeseed, WR pustules become visible in 5-6 days after inoculation (Liu *et al.*, 1989), while in cabbage symptoms appear in 8 days after inoculation (Coffey, 1975). Slow-rusting requires longer incubation and latent periods. In *B. juncea* cvs. Rajat and RC 781, incubation and latent periods of 11/14, and 11/15 have been observed; similarly, in *B. rapa* cvs. Candle, Tobin and Span, longer incubation and latent periods of 11/115, 15/1l8, and 1l/ 18 days, respectively have been reported (Lakra and Saharan 1988d; Jat, 1999; Gupta and Saharan, 2002). There is a need to identify genotypes with slow-rusting attributes to curb the epidemic development of WR in the field. Partial resistance to *A. candida* in crucifer genotypes can be identified through lower infection frequency, lower spore production, and longer incubation and latent periods.

16. Chemical control

16.1 Efficacy of fungicides on germination of *A. candida* oospores *in vitro*

Albugo candida oospores occur as common contaminant in *Brassica* seed samples (Petrie, 1975). Inoculum levels on seeds may be considerably higher than actually required for initiation of infection considering that on germination a single oospore releases 40-60 zoospores (Verma and Petrie, 1975). Germination of oospores following a period of washing in water, infection of *Brassica* cotyledons by zoospores from germinating oospores, and field experiments showing more foliar and staghead infection in oospore-treated plots than in the controls, support the view that oospores contaminated seeds constitute a primary inoculum for infection of *Brassica* species (Verma *et al.*,1975). Thus treatment even by a protectant fungicide can be important in controlling WR infections either by inhibiting oospore germination or by killing the zoospores on emergence.

An oospore germination technique was used to study the effectiveness of 27 protectant fungicides in inhibiting oospore germination at various stages (Verma and Petrie, 1979). Among the chemicals tested, the three mercurial fungicides, mersil, PMA-

10 and panogen, were the best inhibitors of oospore germination. The total inhibition with any of these fungicides at a concentration of 500 ppm active ingredient (a.i.) was about 75 %. Among the non-mercurial compounds, mancozeb and ethazol were the most effective giving total inhibition of about 60%. The inhibition provided by bromosan and pyroxychlor was about 50 %. Since none of the fungicides tested in this study was 100 % effective, the search for a completely effective, preferably systemic, fungicide needs to be continued.

16.2 Efficacy of protectant fungicides in controlling both the foliar and staghead phase of WR disease

Using protectant fungicides, several researchers around the globe have reported varied degree of control of *A. candida*-induced foliar infections in various cruciferous hosts (Verma and Petrie, 1979; Sharma and Sohi, 1982; Sharma and Kolte, 1985; Sharma, 1983; Singh *et al*., 2002; Dainello *et al*., 1986; 1990; Chambers *et al.*, 1974, Singh and Singh, 1990; Meena and Jain, 2002; Pandya *et al*., 2000; Saharan *et al*., 1990). In a detailed growth chamber study, Verma and Petrie (1979) reported that of the nine protectant fungicides tested, application of either chlorothalonil or mancozeb, at 250 or 500 ppm, respectively, 6 h before inoculation and then a week later, controlled the disease effectively. In view of their mainly protectant action, failure to control WR by either fungicide applied 24 h and 7 days after inoculation was not surprising, as establishment of *A. candida* infection on rapeseed cotyledons, and perhaps leaves, would normally be completed within 24 h of inoculation (Verma *et. al.*, 1975).

Two foliar spraying of chlorothalonil (Bravo) in June under Canadian conditions when the plants were 3-4 weeks old significantly reduced both foliar and staghead infections in the field (Verma and Petrie, 1979). However, in view of the growth room studies on successful initiation of stagheads (Verma and Petrie, 1980), a third application at the time of flowering is also advised. Multiple applications, however, may not be economically feasible under commercial rapeseed production.

16.3 Efficacy of metalaxyl in controlling both the foliar and staghead phase of WR disease

Among the systemic chemicals, metalaxyl is probably the best fungicide currently available for WR control. Metalaxyl was active against *A. candida* race 7 in *B. rapa* cv. Torch (Stone *et. al.*, 1987a, b). Treating the seed with metalaxyl at 5.0 g a.i. /kg controlled foliar infection in the growth chamber up to the sixth leaf stage, 22 days after planting. When sprayed on the plants up to 4 days after inoculation, metalaxyl reduced foliar infection by 95 %. Foliar infection was also controlled when applied as a soil drench, but phyto-toxicity was evident. Foliar spray application at 2.0 kg a.i. /ha or higher reduced foliar infections in three years of field studies. Foliar applications also reduced staghead infections when applied at growth stages 3.2 or 4.1.

Growth chamber and field studies (Stone *et. al.*, 1987a) showed that metalaxyl possesses both protective and eradicative activity against *A. candida.* Control of disease in tissues remote from the site of application indicated that the fungicide moves systemically in rape plants. Disease control was obtained on the foliage, either by seed treatment or soil drenching, and disease eradication was successful when the fungicide was sprayed within 4days of inoculation, a further evidence of systemicity (Stone *et. al.*, 1987b). The best cost benefit ratio was obtained by Mehta *et al.*, (1996) when seed treatment with Apron SD-35 (2 g a.i. / kg) was followed by three sprays of mancozeb (0.2 %) at 40, 60 and 80 days after seeding. However, best disease control was obtained when three sprays of Ridomil MZ-72 (0.25 %) were given at 40, 60 and 80-days after seeding.

Seed treatment results were promising but in field situation it provided adequate protection only in the early stages of plant growth. The decline in the activity of metalaxyl with increasing age of plants in seed treatment experiments may have been the result of fungicide dilution as the volume of plant tissue increased. Accordingly, infection of flower buds by wind-borne zoosporangia was not controlled by seed dressing.

In the growth chamber, metalaxyl was active as a foliar eradicant for up to 4 days, but when applied 5

or 6 days after inoculation, the fungicide did not prevent sporulation (Stone *et. al.*, 1987a). It would appear, therefore, that after 4 days the fungus had reached a stage of development when fungicide treatment could not completely arrest growth, although pustule size and development were restricted with these late applications.

Results of studies by Verma and Petrie (1979, 1980) and Stone *et al.* (1987a) suggest that *A. candida* does not require early infections to develop systemically but can produce stagheads from infections of young flower buds by zoospores arising from wind-borne zoosporangia after plant growth stage 2.6. Successful disease control with metalaxyl, therefore, requires that a sufficient quantity of the fungicide be available well into the growing season. Seed dressings only provide protection for a limited period of time, and if conditions favour disease development throughout the season, staghead development will not be controlled. By providing early disease control, however, seed treatment could reduce the secondary inoculum potential in the crop, and thereby limit initiation of stagheads from newly infected flower buds.

Bioassay and gas chromatographic analyses of plant tissue extract confirmed the presence of metalaxyl in tissue remote from the site of the treatment (Stone *et. al.*, 1987b). Both bioassay and chemical analyses of plants grown in metalaxyl-drenched soil showed that the fungicide was readily taken up by plants from the soil solution, that the greatest accumulation was in the lower leaves, and that metalaxyl was found in decreasing amount in leaves furthrest from the roots and in only small concentrations in the stem and inflorescence. These results indicate that root absorption is an efficient means of metalaxyl uptake because when applied to a single leaf it was not detected in the leaves below or above the treated leaf; thus, it is concluded that negligible symplastic translocation occurs. Different levels of control of WR using metalaxyl treated seeds (Verma and Petire, 1979; Rod, 1985; Sokhi *et al.*, 1997; Pathak and Godika, 2005), soil drenching (Stone *et al.*, 1987a; Dainello *et al.*, 1990) and foliar application (Verma and Petire, 1979; Sharma and Sohi, 1982; Sharma and Kolte, 1985; Sharma, 1983; Srivastava and Verma, 1989; Singh *et al*., 2002; Dainello *et al.*, 1986; Singh and Singh, 1990; Meena and Jain, 2002, Mehta *et al*., 1996) have also been reported globally.

17. Suggestions for future research

i. Information regarding production of oospores inside the seeds, and their possible importance both in the survival and initiation of primary infection are lacking.

ii. Role of simple or branched germ tubes from germinating oospores need to be studied.

iii. Single zoospore cultures from germinating sporangia and oospores must be prepared and their pathogenicity compared.

iv. After screening lines for resistance against foliar infections, some select advanced lines must also be screened for production of stagheads using flower-bud inoculation technique.

v. Based on host specificity, mycologists may consider classifying *A. candida* complex into different species.

vi. Host differentials in each crucifer species in the form of isogenic lines must be standardized internationally.

vii. Nomenclature of the *A. candida* races should be standardized internationally viz. AC jun I, 2- for *B. juncea*, AC rap 1, 2, - for *B. rapa*, AC nig I, 2, - for *B. nigra*, A C ol 1,2, etc., for *B. oleracea*.

viii. Identification of sources of resistance should be based on broad spectrum effectiveness of a genotype against specific races, and inheritance of resistance should be studied alongwith the virulence spectrum of *A. candida* isolates.

ix. Efforts should be made to identify resistant loci in the genotypes along with alleles for resistance in each locus.

x. Genotypes exhibiting attributes of slow white-rusting, disease tolerance, and partial resistance may be categorized.

xi. Mapping, cloning, characterization and identification of genes for resistance and virulence with markers at molecular level may be strengthened.

xii. Genetics of *Albugo-Hyaloperonospora* association may be determined both at phoenotypic and genotypic levels.

xiii. Strong and weak genes for resistance in the host and their suitable combinations for durable resistance should be studied.

xiv. Sources of multiple disease resistance should be explored.

References

Aarts, MGM, Hekkert, BL, Holub, EB, Beynon, JL, Stiekema, WJ and Pereira, A. 1998. Identification of R- gene homologous DNA fragments genetically linked to disease resistance loci in *Arabidopsis thaliana*. *MPMI* **11**: 251–258.

Adhikari, TB, Liu, JQ, Mathur, S, Wu, CX and Rimmer, SR. 2003. Genetic and molecular analyses in crosses of race 2 and race 7 of *A. candida*. *Phytopathol* **93**: 959-965.

APG (Angiosperm Phylogeny Group). 2003. An update of the Angiosperm Phylogeny Group classification for the orders and families of flowering plants: APG II. *Botl. J. Linnean Soc* **141**: 399–436.

Austin, MJ, Muskett, P, Kahn, K, Feys, BL, Jones, JD and Parker, JE. 2002. Regulatory role of SGT1 in early *R* gene-mediated plant defenses. *Science* **295**:2077-2080.

Awasthi, RP, Nashaat, NI, Heran, A, Kolte, SJ and Singh, US. 1997. The effect of *Albugo candida* on the resistance to *Peronospora parasitica* and *vice versa* in rapeseed-mustard. *In*: Abstr. ISHS Symp. Brassicas: 10th Crucifer Genet. Workshop. ENSARINRA, Rennes, France: p 49.

Bains, SS. 1993. Differential reaction of leaves and young flowers of different cruciferous crops to *A. candida*. *Plant Dis Res* **8**: 70-72.

Bains, SS and Jhooty, JS. 1979. Mixed infections by *Albugo candida* and *Peronospora parasitica* on *Brassica juncea* inflorescence and their control. *Indian Phytopathol.* **32**: 268-271.

Bains, SS and Jhooty, JS. 1985. Association of *Peronospora parasitica* with *Albugo candida* on *B. juncea* leaves. *Phytopathol Z* **112**: 28-31.

Baka, ZAM. 2008. Occurrence and ultrastructure of *Albugo candida* on a new host, *Arabis alpina* in Saudi Arabia. *Micron* **39**: 1138–1144.

Bansal, VK, Tewari, JP, Stringam, GR and Thiagarajah, MR. 2005. Histological and inheritance studies of partial resistance in the *Brassica napus–Albugo candida* host–pathogen interaction. *Plant Breed* **124**: 27-32.

Barbetti, MJ. 1981. Effects of sowing date and oospore seed contamination upon subsequent crop incidence of white rust (*Albugo candida*) in rapeseed. *Aus. J. Plant Pathol* **10**: 44-46.

Barbetti, MJ and Carter, EC. 1986. Diseases of rapeseed. Rapeseed in Western Australia. Western Australian Department of Agriculture, Bulletin No. 4105. (Ed JA Lawson) : pp 14-19.

Bartaria, AM and Verma, PR. 2001. Effect of temperature on viability of zoosporangia of *Albugo candida*. *J. Mycol. Plant Pathol* **31**: 236-237.

Berkeley. 1848. On the white rust of cabbages. *J. Hort. Soc. London* **3**: 265-271.

Berkenkamp, B. 1972. Diseases of rapeseed in central and northern Alberta in 1971. *Can. Plant Dis. Surv* **52**: 62-63.

Berlin, JD and Bowen, CC. 1964a. Centrioles in fungus *Albugo candida*. *Amer. J. Bot* **51**: 650-652.

Berlin, JD and Bowen, CC. 1964b. The host-parasite interface of *Albugo candida* on *Raphanus sativus*. *Amer. J. Bot* **51**: 445-452.

Bhardwaj, CL and *Sud*, AK. *1988.* A study on the variability of *Albugo candida* from Himachal Pradesh. *J. Mycol. Plant Pathol* **18**: 287-291.

Bhardwaj, CL and Sud, AK. 1993. Reaction of Brassica cultivars against *Albugo candida* isolates from Kangra valley. *Indian Phytopathol* **46**: 3: 258-260.

Biga, MLB. 1955. Review of the species of the genus *Albugo* based on the morphology of the conidia. *Sydowia* **9**: 339-358.

Black, LL, Williams, PH and Pound, GS. 1968.

Anaerobic metabolism of *A. candida* - infected radish cotyledons. *Phytopathol* **58**: 672-675.

Bonnet, A. 1981. Resistance to white rust in radish (*Raphanus sativus* L.). *Cruciferae News Lett* **6**: 60.

Borhan, MH, Brose, E, Beynon, JL and Holub, EB. 2001. White rust (*Albugo candida*) resistance loci on three Arabidopsis chromosomes are closely linked to downy mildew (*Peronospora parasitica*) resistance loci. *Mol. Plant Pathol* **2**: 87–95.

Borhan, MH, Gunn, N, Cooper, A, Gulden, S, Tor, M, Rimmer, SR and Holub, EB. 2008. WRR4 encodes a TIR-NB-LRR protein that confers broad-spectrum white rust resistance in *Arabidopsis thaliana* to four physiological races of *Albugo candida*. *MPMI* **21**: 757–768.

Borhan, MH, Holub, EB, Beynon, JL, Rozwadowski, K and Rimmer, SR. 2004. The *Arabidopsis* TIR-NB-LRR Gene *RAC1* Confers Resistance to *Albugo candida* (White Rust) and Is Dependent on *EDS1* but not *PAD4*. *MPMI* **17**: 711–719.

Borhan, MH, Holub, EB, Kindrachuk, C, Omidi, M, Bozorgmanesh-Frad, G and Rimmer, SR. 2010. *WRR4*, a broad-spectrum *TIR-NB-LRR* gene from *Arabidopsis thaliana* that confers white rust resistance in transgenic oilseed *Brassica* crops. *Mol. Plant Pathol* **11**: 283-291.

Bremer, H, lsmen, H, Karel, G, Ozkan, H and Ozkan, M. 1947. Contribution to knowledge of the parasitic fungi of Turkey. *Rev. Faculty Sci., Univ Istanbul, Series-B* **13**: 122-172.

Burdyukova, LI. 1980. Albuginacea fungi. Taxonomy, morphology, biology, and specialization. *Ukrainskyi Botanichnyi Zhumal* **37**: 65-74 (Russian).

Butler, EJ. 1918. White rust [*C. candidus* (Pers. Lev.)]. IN: Fungi and Disease in Plants. Thacker, Spink & Co., Calcutta, India: 291-297.

Butler, EJ and Jones, SG. 1961. *Plant Pathology*. McMillan, London.

Century, KS, Shapiro, AD, Repetti, PP, Dahlbeck, D, Holub, EB and Staskawicz, BJ. 1997. *NDR1*, a pathogen-induced component required for *Arabidopsis* disease resistance. *Science* **278**: 1963-1965.

Chambers, AY, Hadden, CH and Merrill, S. 1974. Control of white rust of spinach with fungicides. *Tennessee Farm and Home Sci. Progress Report* **90**: 30-31.

Chattopadhyay, C, Agrawal, R, Kumar, A, Meena, RL, Faujdar, K, Chakravarthy, NVK, Kumar, A, Goyal, P, Meena, PD and Shekhar, C. 2011. Epidemiology and development of forecasting models for White rust of *Brassica juncea* in India. *Archives Phytopathol. Plant Protect* **44**: 751-763.

Cheng, BF, Seguin-Swartz, G, Somers, DJ and Rakow, GFW. 1999. Meiotic studies on Indian mustard (*B.juncea*) germplasm possessing the fatty acid composition and white rust resistance of *B. napus*. *Cruciferae Newslett* **21**: 45-46.

Cheung, WY, Gugel, RK and Landry, BS. 1998. Identification of RFLP markers linked to the white rust resistance gene (*Acr*) in mustard (*Brassica juncea* (L.) Czern. and Coss.). *Genome* **41**: 626-628.

Choi, D and Priest, MJ. 1995. A key to the genus *Albugo*. *Mycotaxon* **53**: 261–272.

Choi, YJ, Hong, SB and Shin, HD. 2006. Genetic diversity within the *Albugo candida* complex (Peronosporales, Oomycota) inferred from phylogenetic analysis of ITS rDNA and COX2 mtDNA sequences. *Mol. Phylogenetics Evol* **40**: 400-409.

Choi, YJ, Park, MJ, Park, JH and Shin, HD. 2011. White blister rust caused by *Albugo candida* on Oilseed rape in Korea. *Plant Pathol J* **27**: 192.

Choi, YJ, Shin, HD, Hong, SB and Thines, M. 2007. Morphological and molecular discrimination among *Albugo candida* materials infecting *Capsella bursa-pastoris* world-wide. *Fungal Diversity* **27**: 11–34.

Choi, YJ, Shin, HD, Hong, SB and Thines, M. 2009. The host range of *Albugo candida* extends from Brassicaceae through Cleomaceae to Capparaceae. *Mycol. Progress* **8**: 329–335.

Choi, YJ, Shin, HD, Ploch, S and Thines, M. 2008. Evidence for uncharted biodiversity in the *Albugo candida* complex, with the description of a new species. *Mycol. Res* **112**: 1327–1334.

Chowdhary, S. 1944. Some fungi from Assam. *Indian J. Agri. Sci* **14**: 230-233.

Chupp, C. 1925. *Manual of Vegetable Garden Diseases*. MacMillan, New York.

Coffey, MD. 1975. Ultrastructural features of the haustorial apparatus of the white blister fungus *Albugo candida*. *Can. J. Bot* **53**: 1285-1299.

Coffey, MD. 1983. Cytochemical specialization at the haustorial interface of a biotrophic fungal parasite *Albugo candida*. *Can. J. Bot* **61**: 2004-2014.

Colmeiro, M. 1867. Enumeración de las criptógamas de España y Portugal, II. *Rev. Progr. Ci. Exact. Fis. Nat* **17–18**: 63–164.

Constantinescu, O and Fatehi, J. 2002. Peronospora-like fungi (Chromista, Peronosporales) parasitic on Brassicaceae and related hosts. *Nova Hedwigia* **74**: 291–338.

Cooke, R. 1977. *The biology of symbiotic fungi*, John Wiley and Sons, London.

Cooper, AJ, Latunde-Dada, AO, Woods-To¨r, A, Lynn, J, Lucas, JA, Crute, IR and Holub, EB. 2008. Basic compatibility of *Albugo candida* in *Arabidopsis thaliana* and *Brassica juncea* causes broad-spectrum suppression of innate immunity. *MPMI* **21**: 745–756.

Dainello, FJ and Jones, RK. 1986. Evaluation of use-pattern alternatives with metalaxyl to control foliar diseases of spinach. *Plant Dis* **70**: 240-242.

Dainello, FJ, Black, MC and Kunkel, TE. 1990. Control of white rust of Spinach with partial resistance and multiple soil applications of metalaxyl granules. *Plant Dis* **74**: 913-916.

Daly, JM. 1976. *Physiological Plant Pathology*. Ed. R. Heitefuss and P.H. Williams. Springer-Verlag, Berlin: pp 27-50 and 450-479.

Dangl, JL and Jones, JDG. 2001. Plant pathogens and integrated defence responses to infection. *Nature* **411**: 826-833.

Davison, EM. 1968. Cytochemistry and ultrastructure of hyphae and haustoria of *P. parasitica* (Pers. ex Fr.) Fr. *Ann. Bot* (Lond.) **32**: 613-621.

De Bary, A. 1860. Sur la formation de zoospores chez quelques champignons, premier memoire. Ann. des Sci. Nat. Bot. Tome 13, 4th series. 236-254.

De Bary, A. 1863. Recherches sur le developpement quelques champignons parasites. *Ann. des Sci. Nat. Bot. Tome 20, 4th series.* 5-148.

De Bary, A. 1866. Morphologie und physiologic der Pilze, Flechten und Myxomyceten. Welhelm Engelmann, Leipzig: pp 426-439.

De Candolle, AP. 1806. Flore française, Lyon.

de Roussel, HFA. 1806. Flore du Calvados et des terreins adjacens. IIe Edition, Caen.

Debnath, M, Sharma, SL and Kant, U. 1998. Changes in carbohydrate contents and hydrolysing enzymes in white rust of *B. juncea* (L.) Czern. and Coss. caused by *A. candida in vivo* and *in vitro*. *J. Phytol. Res* **11**: 81-82.

Debnath, M, Sharma, SL and Kant, U. 2001. Growth of *Albugo candida* infected mustard callus in culture. *Mycopathologia* **152**:147-53.

Delaney, TP, Friedrich, L and Ryals, JA. 1995. *Arabidopsis* signal transduction mutant defective in chemically and biologically induced disease resistance. *Proc. Natl. Acad. Sci. USA* **92**: 6602-6606.

Delwiche, PA and Williams, PH. 1974. Resistance to *Albugo candida* race 2 in *Brassica* sp. *Proc. Amer Phytopathol Soc* **1**: 66 (Abstr).

Delwiche, PA and Williams, PH. 1977. Genetic studies in *Brassica nigra* (L.) Koch. *Cruciferae NewsLett* **2**: 39.

Delwiche, PA and Williams, PH. 1981. Thirteen marker genes in *Brassica nigra*. *J. Hered* **72**: 289-290.

Dhawan, K, Yadava, TP, Kaushik, CD and Thakral, SK. 1981. Changes in phenolic compounds and sugars in relation to white rust of Indian mustard. *Crop Improv* **8**: 142-144.

Dhingra, RK, Chauhan, N and Chauhan, SVS. 1982. Biochemical changes in the floral parts of *Brassica campestris* infected with *Albugo candida*. *Indian Phytopathol* **35**:177-179.

Dick, MW. 2001. Straminipilous Fungi: Systematics of the Peronosporomycetes Including Accounts of the Marine Straminipilous Protists, the Plasmodiophorids and Similar Organisms. Kluwer Academic Publishers, Dordrecht/ Boston/ London.

Dong, X. 2001. Genetic dissection of systemic acquired resistance. *Curr. Opin. Plant Biol* **4**: 309-314.

Eberhardt, A. 1904. Contribution attitude de *Cystopus candidus* Lev. *Zentr. Bakteriol. Parasitenk* **12**: 235-249.

Ebrahimi, AG, Delwiche, PA and Williams, PH. 1976. Resistance in *Brassica juncea* to *Pemnospora parasítica* and *Albugo candida* Race 2. *Proc. Amer. Phytopathol. Soc* **3**: 273.

Edie, HH and Ho, BWC. 1970 Factors affecting sporangial germination in *A. ipomoea eaquaticae. Trans. Br. Mycol. Soc* **55**: 205-216.

Edwards, M and Williams, PH. 1982. Selection for quantitatively inherited resistance to *Albugo candida* race 2 in *B. canpestris*, CGS-1. *Cruciferae NewsLett* **7**: 66-67.

Edwards, MD and Williams, PH. 1987. Selection of minor gene resistance to *A. candida* in rapid-cycling population of *Brassica campestris*. *Phytopathol* **77**: 527-532.

Ellis, JG, Lawrence, GJ, Luck, JE and Dodds, PN. 1999. Identification of regions in alleles of the flax rust resistance gene *L* that determines differences in gene-for-gene specificity. *Plant Cell* **11**: 495-506.

Endo, RM and Linn, MB. 1960. The white rust disease of horseradish. *Illinois Agri. Exp. Station Bull* **655**: 56.

Eulgem, T, Weigman, VJ, Chang, HS, McDowell, JM, Holub, EB, Glazebrook, J, Zhu, T, and Dangl, JL. 2004. Three genetically separable *R* gene-signaling pathways converge to regulate a largely overlapping transcriptome. *Plant Physiol* **135**: 1129-1144.

Fan, Z, Rimmer, SR and Stefansson, BR. 1983. Inheritance of resistance to *Albugo candida* in rape (*Brassica napus* L.). *Can. J. Genet. Cytol* **25**: 420-424.

Farr, DF and Rossman, AY. 2010. Fungal Databases, Systematic Mycology and Microbiology Laboratory, ARS, USDA. Retrieved May 18, 2010, from http://nt.arsgrin.gov/fungaldatabases.

Farr, DF, Bills, GF, Chamuris GP and Rossman AY. 1989. *Ipomoea*. In: Farr DF, Billis FG, Chamuris GP, Rossman AY (eds) *Fungi on plants and plant products in the United States*. APS Press, St Paul: 142–143.

Farr, DF, Rossman, AY, Palm, ME and McCray, EB. 2004. Online Fungal Databases, Systematic Botany & Mycology Laboratory, ARS, USDA. Available at http://nt.ars-grin.gov/fungaldatabases.

Ferreira, ME, Williams, PH, and Osborn, TC. 1994. RFLP mapping of *Brassica napus* using doubled haploid lines. *Theor. Appl. Genet* **89**: 615–621.

Ferreira, ME, Williams, PH, and Osborn, TC. 1995. Mapping of locus controlling resistance to *Albugo candida* in *Brassica napus* using molecular markers. *Phytopathol* **85**: 218-220.

Fraymouth, J. 1956. Haustoria of the Peronosoporales. *Trans. Br. Mycol. Soc* **39**: 79-107.

Gadewadikar, PN, Bhadouria, SS and Bartaria, AM. 1993. Inheritance of resistance to white rust (*Albugo candida)* disease in Indian mustard (*Brassica juncea*). IN: *Natl. Sem., Oilseeds Research and Development in India: Status and Strategies*. Aug 2-4, 1993.

Gassmann, W, Hinsch, ME, and Staskawicz, BJ. 1999. The *Arabidopsis RPS4* bacterial-resistance gene is a member of the TIR-NBS-LRR family of disease-resistance genes. *Plant J* **20**: 265-277.

Glazebrook, J, Zook, M, Mert, F, Kagan, I, Rogers, EE, Crute, IR, Holub, EB, Hammerschmidt, R, and Ausubel, FM. 1997. Phytoalexin-deficient mutants of *Arabidopsis* reveal that *PAD4* encodes a regulatory factor and that four *PAD* genes contribute to downy mildew resistance. *Genetics* **146**: 381-392.

Gmelin, JF. 1792. Systema Naturae. **2** (2). GE, Beer, Leipzig.

Goyal, BK, Kant, U and Verma, PR. 1995. Growth of *Albugo candida* (race unidentified) on *Brassica juncea* callus cultures. *Plant and Soil* **172**: 331-337.

Goyal, BK, Verma, PR and Spurr, DT. 1996. Temperature effects on oospore development of *Albugo candida* race 2V in detached *Brassica juncea* leaves. *Indian J. Mycol. Plant Pathol* **26**: 224-228.

Goyal, BK, Verma, PR, Spurr, DT and Reddy, MS. 1996. *Albugo candida* staghead formation in *Brassica juncea* in relation to plant age,

inoculation sites, and incubation conditions. *Plant Pathol* **45**: 787-794.

Goyal, BK, Verma, PR, Swartz, G, Seguin and Spurr, DT. 1996. Growth of *Albugo candida* in leaf callus cultures of *Brassica rapa. Can. J. Plant Pathol* **18**: 225-232.

Gray, SF. 1821. A natural arrangement of British plants: according to their relations to each other as pointed out by Jussieu, De CandoIle, Brown: with an introduction to botany. Baldwin, Cradock and Joy, London. 72.

Greelman, DW. 1963. New and noteworthy diseases. *Can. Plant Dis. Surv* **43**: 61-63.

Gupta, K and Saharan, GS. 2002. Identification of pathotypes of *Albugo candida* with stable characteristic symptoms on Indian mustard. *J. Mycol. Plant Pathol* **32**: 46-51.

Gupta, ML, Singh, G, Raheja, RK, Ahuja, KL and Banga, SK. 1997. Chlorophyll content in relation to white rust (*A. candida*) resistance in Indian mustard. *Cruciferae Newslett* **19**: 105-106.

Gupta, RBL and Singh, M. 1994. Source of resistance to white rust and powdery mildew of mustard. *Int. J. Trop. Plant Dis* **12**: 225-227.

Gupta, S, Sharma, TR and Chib, HS. 1995. Evaluation of wild allies of *Brassica* under natural conditions. *Cruc Newslett* **17**: 10–11.

Hammett, KPW. 1969. White rust diseases. *New Zealand Gardner* **26**: 43.

Hammond-Kosack, KE and Jones, JDG. 1997. Plant disease resistance genes. *Ann. Rev. Plant Physiol. Plant Mol. Biol* **48**: 575-607.

Harding, H, Williams, PH and McNabola, SS. 1968. Chlorophyll changes, photosynthesis, and uttrastructure of chloroplasts in *Albugo candida* induced "green islands" on detached *Brassica juncea* cotyledons. *Can. J. Bot* **46**:1229-1234.

Harper, FR and Pittman, UJ. 1974. Yield loss by *Brassica campestris* and *Brassica napus* from systemic stem infection by *Albugo curciferarum*. *Phytopathol* **64**: 408-410.

Harter, LL and Weimer, JL. 1929. White rust. IN. A Monographic study of sweet-potato diseases and their control. *Tech. Bull. U.S. Dept. Agric. No* **99**: 53-56.

Heald, FD. 1926. Diseases due to downy mildew and allies. IN: Manual of Plant Diseases. McGraw Hill Book Company, Inc. New York. Ch **16**: 390-426.

Heim, P. 1959. On the sexual reproduction of *Cystopus portulacae* D.C. C.R. *Acad. Sci. Paris* **248**: 1012-1014.

Hill, C, Crute, I, Sherriff, C and Williams, PH. 1988. Specificity of *Albugo candida* and *Peronospora parasitica* pathotypes toward rapid-cycling crucifers. *Cruciferae Newslett* **13:** 112-113.

Hirata, S. 1954. Studies on the phytohormone in the malformed portion of the diseased plants. I. The relation between the growth rate and the amount of free auxin in the fungous galls and virus-infected plants. *Ann. Phytopathol. Soc. Jpn* **19**: 33-38.

Hirata, S. 1956. Studies on the phytohormone in the malformed portion of the diseased plants. II. On the reformation and the situation of free-auxin in the tissues of fungous galls. *Ann. Phytopathol. Soc. Jpn* **19**: 185-190.

Hiura, M. 1930. Biologic forms of *Albugo candida* (Pers.) Kuntze on some cruciferous plants. *Japan J. Bot* **5**: 1-20.

Ho, BWC and Edie, HH. 1969. White rust (*Albugo ipomoeae-aquaticae*) of *Ipomoea aquatic* in Hong Kong. *Plant Dis. Reporter* **53**: 959-962.

Holliday, P. 1980. Fungus diseases of tropical crops. Cambridge Univ. Press 2-5 p.

Holub, EB. 2001. The arms race is ancient history in *Arabidopsis*, the wild flower. *Nat. Rev. Genet* **2**: 516-527.

Holub, EB, Williams, PH and Crute, IR. 1991. Natural infection of *A. thaliana* by *A. candida* and *Peronospora parasitica*. *Phytopathol* **81**:1226.

Hougas, RW, Rieman, GH and Stokes, GW. 1952. Resistance to white rust in horseradish seedlings. *Phytopathol* **42**: 109-110.

Hughes, SJ. 1971. Annel'whores. In Taxonomy of fungi Imperfecti. Univ. Toronto Press. 132-139.

Humaydan, HS and Williams, PH. 1976. Inheritance of seven characters in *Raphanus sativus*. *Hort. Sci* **11**: 146-147.

Ito, S and Tokunaga, Y. 1935. Notae mycologicae Asiae orientalis. *I. Trans. Sapporo. Nat. Hist. Soc* **14**: 11-33.

Jain, JP. 1995. Management of white rust and alternaria blight diseases of mustard. Proc. Global Conference on Advances in Research on Plant Diseases and their management. Rajasthan College of Agriculture, Udaipur, Rajasthan, India: p 80 (Abstr).

Jain, KL, Gupta, AK and Trivedi, A. 1998. Reaction of rapeseed-mustard lines against white rust pathogen *Albugo candida*. *J. Mycol. Plant Pathol* **28**: 72-73.

Jat, RR. 1999. Pathogenic variability and inheritance of resistance to *Albugo candida* in oilseed *Brassica*. PhD Thesis, CCSHAU, Hisar: pp 129.

Jenkyn, JF, Hirst, JM and King, G. 1973. An apparatus for the isolated propagation of foliar pathogens and their hosts. *Ann. Appl. Biol* **73**: 9-13.

Jones, DA and Jones, JDG. 1997. The role of leucine-rich repeat proteins in plant defenses. *Adv. Bot. Res* **24**: 89-167.

Kadow, KJ and Anderson, HW. 1940. A study of horseradish diseases and their control. *Univ. Illinois Agri. Exptl. Station Bull* **469**: 531-543.

Kajomchaiyakul, P and Brown, JF. 1976. The infection process and factors affecting infection of sunflower by *Albugo tragopogi*. *Trans. Br. Mycol. Soc* **66**: 91-95.

Kaur, D, Bhandari, NN and Mukerji, KG. 1984. A histochemical study of cytoplasmic changes during wall layer formation in the oospore of *A. candida*. *Phytopathol. Z* **109**: 117-130.

Kaur, P, Jost, R, Sivasithamparam, K and Barbetti, MJ. 2011. Proteome analysis of the *Albugo candida–Brassica juncea* pathosystem reveals that the timing of the expression of defence-related genes is a crucial determinant of pathogenesis. *J. Exptl. Bot* **62**: 1285–1298.

Kaur, P, Sivasithamparam, K and Barbetti, MJ. 2008. Pathogenic behaviour of strains of *Albugo candida* from *Brassica juncea* (Indian mustard) and *Raphanus raphanistrum* (wild radish) in Western Australia. *Aus. J. Plant Pathol* **37**: 353-356.

Kaur, P, Sivasithamparam, K, Li, H and Barbetti, MJ. 2011. Pre-inoculation with *Hyaloperonospora parasitica* reduces incubation period and increases severity of disease causedby *A. candida* in a *B. juncea* variety resistant to downy mildew. *J. Gen. Plant Pathol* **77**: 101–106.

Kemen, E, Gardiner, A, Schultz-Larsen, T, Kemen, AC, Balmuth, AL, Alexandre, Robert-Seilaniantz, Kate Bailey, Holub, E, Studholme, DJ, MacLean, D, Jonathan, Jones, DG. 2011. Gene Gain and Loss during Evolution of Obligate Parasitism in the White Rust Pathogen of *Arabidopsis thaliana*. *PLoS Biol* 9: e1001094. doi:10.1371/journal.pbio.1001094.

Khan, SR. 1976. Ultrastructural changes in maturing sporangia of *Albugo candida*. *Ann. Bot* **40**: 1285-1292.

Khan, SR. 1977 Light and electron microscopic observations of sporangium formation in *Albugo candida. Can. J. Bot* **55**: 730-739.

Khunti, JP, Khandar, RR and Bhoraniya, MF. 2000. Studies on host range of *Albugo cuciferarum* the incitant of white rust of mustard. *Agri. Sci. Digest* **20**: 219–221.

Kiermayer, 0. 1958. Paper chromatographic studies of the growth substances of Capsella bursa pastoris after infection by *Albugo candida* and *Peronospora parasitica*. *Osterr. Bot. Z* **105**: 515-528.

Klemm, M. 1938. The most important diseases and pests of Colza and Rape. *Dtsch. Landw. Pr.* IXV **19**: 239; **20**: 251-252.

Klessig, DF, Malamy, J, Hennig, J, Sanchez-Casas, P, Indulski, J, Grynkiewicz, G, and Chen, Z. 1994. Induction, modification, and perception of the salicylic acid signal in plant defense. *Biochem. Soc. Symp* **60**: 219-229.

Kobe, B, and Deisenhofer, J. 1994. The leucine-rich repeat: a versatile binding motif. *Trends Biochem. Sci* **19**: 415-421.

Kobe, B, and Deisenhofer, J. 1995. Proteins with leucine-rich repeats. *Curr. Opin. Struct. Biol* **5**: 409-416.

Kole, C, Teutonico, R, Mengistu, A, Williams, PH, and Osborn, TC. 1996. Molecular mapping of a locus controlling resistance to *Albugo candida* in *Brassica rapa*. *Phytopathol* **86**: 367–369.

Kole, C, Williams, PH, Rimmer, SR and Osborn, TC. 2002. Linkage mapping of genes controlling resistance to white rust (*Albugo candida*) in *Brassica rapa* (syn. *campestris*) and comparative mapping to *Brassica napus* and *Arabidopsis thaliana. Genome* **45**: 22-27.

Kolte, SJ. 1985. White rust: IN: *Diseases of Annual edible oilseed crops*, Vol. II. *Rapeseed-mustard and sesame diseases.* CRC Press, Boca Raton, Florida, USA. Ch **2**: 27-35.

Kolte, SJ and Tewari, AN. 1980. Note on the susceptibility of certain *oleiferous Brassicae* to downy mildew and white blister diseases. *Indian J. Mycol. Plant Pathol* **10**: 191-192.

Kolte, SJ, Bordoloi, DK, Awasthi, RP. 1991. The search for resistance to major diseases of rapeseed and mustard in India. *GCIRC 8th International Rapeseed Congress,* Saskatchewan, Canada 219–225.

Kombrink, E, and Schmelzer, E. 2001. The hypersensitive response and its role in local and systemic disease resistance. *Eur. J. Plant Pathol* **107**: 69-78.

Kumari, K, Varghese, TM and Suryanarayana, D. 1970. Qualitative changes in the amino-acid contents of hypertrophied organs in mustard due to *Albugo candida*. *Curr. Sci* **39**: 240-241.

Kuntze, O. 1891. *Revisio generum plantarum,* Pars I & II. Arthur felix, Leipzig, Germany, 2 pp. i-cccx.

Lahiri, I and Bhowmik, TP. 1993. Growth of the white rust fungus *Albugo candida* in callus tissue of *Brassica juncea. J. Gen. Microbiol* **139**: 2875-2878.

Lakra, BS and Saharan, GS. 1988. Efficacy of fungicides in controlling white rust of mustard through foliar sprays. *Indian J. Mycol. Plant Pathol* **18**: 157-163.

Lakra, BS and Saharan, GS. 1988. Influence of host resistance on colonization and incubation period of *Albugo candida* in mustard. *Cruciferae NewsLett* **13**: 108-109.

Lakra, BS and Saharan, GS. 1988. Morphological and pathological variations in *Albugo candida* associated with *Brassica* species. *Indian J. Mycol. Plant Pathol* **18**:149-156.

Lakra, BS and Saharan, GS. 1988. Progression and management of white rust of mustard in relation to planting time, host resistance and fungicidal spray. *Indian J. Mycol. Plant Pathol* **18**: 112 (Abstr).

Lakra, BS and Saharan, GS. 1989. Sources of resistance and effective screening techniques in *Brassica-Albugo* system. *Indian Phytopathol* **42**: 293 (Abstr).

Lakra, BS and Saharan, GS. 1989. Correlation of leaf and staghead infection intensities of white rust with yield and yield components of mustard. *Indian J. Mycol. Plant Pathol* **19**: 279-281.

Lakra, BS and Saharan, GS. 1989. Location and estimation of oospores of *Albugo candida* in infected plant parts of mustard. *Indian Phytolpath* **42**: 467.

Lakra, BS, Saharan, GS and Verma, PR. 1989. Effect of temperature, relative humidity and light on germination of *Albugo candida* sporangia from mustard. *Indian J. Mycol. Plant Pathol* **19**: 264-267.

Lal, BB, Prasad, M and Ram, RP. 1980. Amino acid constituents of inflorescence tissue of crucifers in health and disease, due to *A. candida* (Pers.) Kuntze. *Zbl. Bakt. II. Abt.* **135**: 240-245.

Leveilla, JH. 1847. On the methodical arrangement of the Uredineae. *Ann. Sci. Nat. Ser.* 3 **8**: 371.

Li, CX, Sivasithamparam, K, Walton, G, Fels, P, Barbetti, MJ. 2008. Both incidence and severity of white rust disease reflect host resistance in *Brassica juncea* germplasm from Australia, China and India. *Field Crops Res* **106**: 1-8.

Li, CX, Sivasithamparam, K, Walton, G, Salisbury, P, Burton, W, Banga, SS, Banga, Shashi, Chattopadhyay, C, Kumar, A, Singh, R, Singh, D, Agnohotri, A, Liu, SY, Li, YC, Fu, TD, Wang, YF and Barbetti, MJ. 2007. Expression and relationships of resistance to white rust (*Albugo candida*) at cotyledonary, seedling, and flowering stages in *Brassica juncea* germplasm from Australia, China, and India. *Aus. J. Agri. Res* **58**: 259-264.

Links, MG, Holub, E, Jiang, RHY, Sharpe, AG, Hegedus, D, Beynon, E, Sillito, D, Clarke, WE, Uzuhashi, S and Borhan, MH. 2011. *De novo* sequence assembly of *Albugo candida* reveals a small genome relative to other biotrophic oomycetes. *BMC Genomics* **12**: 503. doi: 10.1186/1471-2164-12-503.

Liu, JQ, Parks, P and Rimmer, SR. 1996. Development of monogenic lines for resistance to *Albugo candida* from a Canadian *Brassica napus* cultivar. *Phytopathol* **86**: 1000–1004.

Liu, O, Rimmer, SR and Scarth, R. 1989. Histopathology of compatibility and incompatibility between oilseed rape and *Albugo candida*. *Plant Pathol* **38**: 176-182.

Liu, O, Rimmer, SR, Scarth, R and McVetty, PBE. 1987. Confirmation of a digenic model of inheritance of resistance to *Albugo candida* race 7 in *Brassica napus*. *Proc. 7th Intern. Rapeseed Congress*, Poznam, Polland: 1204-1209.

Liu, Q and Rimmer, S.R. 1992. Inheritance of resistance in *Brassica napus* to an Ethiopian isolate of *Albugo candida* from *Brassica carinata. Can. J. Plant Pathol* **14**: 116-120.

Long, DE and Cooke, RC. 1974. Carbohydrate composition and metabolism of seneciosqualidus leaves infected with *A. tragopogonis* (Pers.) S.F. Gray. *New Phytol* **73**: 889-899.

Long, DE, Fung, AK, MacGee, EEM, Cooke, RC and Lewis, DH. 1975. The activity of invertase and its relevance to the accumulation of storage polysaccharides in leaves infected by biotrophic fungi. *New Phytol* **74**: 173-182.

Maheshwari, DK and Chaturvedi, SN. 1983. Histochemical localizaiton of acid phosphatase in two fungus galls. *Indian Phytopathol* **36**:167-170.

Mani, N, Gulati, SC, Raman, R and Raman, R. 1996. Breeding for genetic resistance to white rust in Indian mustard. *Crop Improv* **23**: 75-79.

Massand, PP, Yadava, SK, Sharma, P, Kaur, A, Kumar, A, Arumugam, N, Sodhi, YS, Mukhopadhyay, A, Gupta, V, Pradhan, AK and Pental, D. 2010. Molecular mapping reveals two independent loci conferring resistance to *Albugo candida* in the east European germplasm of oilseed mustard *Brassica juncea. Theor. Appl. Genet* **121**: 137–145.

Mathur, S, Wu, C and Rimmer, SR. 1995. Virulence of isolates of *Albugo candida* from western Canada to *Brassica* species. *Proc. 9th Intern. Rapeseed Congr*., Cambridge, UK **2**: 652-654.

Mathur, S, Wu, C, Liu, JQ and Rimmer, SR. 1995. Inheritance of virulence and fungicide resistance in *Albugo candida*. *Can. J. Plant Pathol* **17**: 360 (Abstr).

McDowell, JM, Cuzick, A, Can, C, Beynon, JL, Dangl, JD and Holub, EB. 2000. Downy mildew (*Peronospora parasitica*) resistance genes in *Arabidopsis* vary in functional requirements for *NDR1*, *EDS1*, *NPR1*, and salicylic acid accumulation. *Plant J* **22**: 523-529.

Meena, PD. 2007. Report on Research Attachment. DFID Indo-UK Collaboration on Oilseeds (Rapeseed-mustard) Phase II (1998-2007): pp 83.

Meena, PD and Sharma, P. 2012. Methodology for production and germination of oospores of *Albugo candida* infecting Oilseed *Brassica*. *Vegetos* **25**: 115-119

Meena, RL and Jain, KL. 2002. Fungicides and plant products in managing white rust of Indian mustard caused by *A. candida* (Pers. Ex. Lev.) Kuntze. *Indian J. Plant Protect* **30**: 210-212.

Mehta, N, Saharan, GS, Kaushik, CD and Mehta, N. 1996. Efficacy and economics of fungicidal management of white rust and downy mildew complex in mustard. *Indian J. Mycol. Plant Pathol* **26**: 243-247.

Melhus, IE. 1911. Experiments on spore germination and infection in certain species of Oomycetes. *Wisconsin Agri. Exptl. Stan. Res. Bull* **15**: 25-91.

Middleton, KJ. 1971. Sunflower diseases in South Queensland. *Queensland Agri. J* **97**: 597-600.

Mishra, MD and Chona, BL. 1963. Factors affecting sporangial germination and epidemiology of the white rust of Amaranths [*Albugo* bliti (Biv.) Bern. (Kuntze.]. *Indian Phytopathol* **16**: 333-343.

Misra, A and Padhi, B. 1981. Impact of brown rust and white rust on the RNA content of their host tissues. *Advancing Frontiers of Mycology*

and Plant Pathology. (Eds) KS, Bilgrami, RS, Misra, PC, Misra :pp 175-182.

Moffett, P. 2009. Mechanisms of recognition in dominant R gene mediated resistance. *In*: Advances in Virus Research **75**: 1-33 (Ed) Gad Loebenstein and John P. Carr, Elsevier Inc.

Morel, J and Dangl, JL. 1997. The hypersensitive response and the induction of cell death in plants. *Cell Death Differ* **4**: 671-683.

Mukerji, KG. 1975. *Albugo candida*. IMI Descriptions of Fungi and Bacteria **46**: 460.

Murashige, T and Skoog, F. 1962. A revised medium for rapid growth and bioassays with tobacco tissue cultures. *Physiol. Plant* **15**: 473-497.

Muskett, PR, Kahn, K, Austin, MJ, Moisan, LJ, Sadanandom, A, Shirasu, K, Jones, JDG, and Parker, JE. 2002. *Arabidopsis* RAR1 exerts rate-limiting control of R gene-mediated defenses against multiple pathogens. *Plant Cell* **14**: 979-992.

Napper, ME. 1933. Observations on spore germination and specialization of parasitism in *Cystopus candidus*. *J. Pomol. Hort. Sci* **11**: 81-100.

Nashaat, NI and Awasthi, RP. 1995. Evidence for differential resistance to *Peronospora parasitica* (downy mildew) in accessions of *Brassica juncea* (mustard) at the cotyledon stage. *J. Phytopathol* **143**: 157–159.

Nashaat, NI and Rawlinson, CJ. 1994. The response of oilseed rape (*Brassica napus* ssp. *oleifera*) accessions with different glucosinolate and erucic acid contents to four isolates of *Peronospora parasitica* (downy mildew) and the identificat ion of new sources of resistance. *Plant Pathol* **43**: 278-285.

Novotel'Nova, NS. 1962. White rust on sunflower. *Zashch. Rast. Moskva* **7**:57 (Russian).

Novotel'Nova, NS. 1968. Intraspecific taxa of *Cystopus candidus* (Pers.) Lev. *Novosti Sistematiki Nizsh. Rast*: 88-96 (Russian).

Novotel'Nova, NS and Minasyan, MA. 1970. Contribution to the biology of *Cystopus candidus* (Pers.) Lev. and *Cystopus tragopogonis* (Pers.) Schroet. *Trudy Vsesoyuznogo Nauchnoissledovaterskogo Instituta Zashchity rastenii* **29**: 121-128 (Russian).

Pal, Y, Singh, H and Singh, D. 1991. Genetic components of variation for white rust resistance in Indian x Exotic crosses of Indian mustard. *Crop Res* **4**: 280-283.

Paladhi, MM, Prasad, RC, Dass, B and Dass, B. 1993. Inheritance of field reaction to white rust in Indian mustard. *Indian J. Genet. Plant Breed* **53**: 327-328.

Pandya, RK, Tripathi, ML, Singh, R and Singh, R. 2000. Efficacy of fungicides in the management of white rust and Alternaria blight of mustard. *Crop Res. Hisar* **20**: 137-139.

Pape, H and Rabbas, P. 1920. Inoculation tests with *Cystopus candidus* Pers. *Mitt. Biol. R.-Anst. L. and U. Forstw. U* **18**: 58-59 (German).

Parham, BE. 1942. White rust of cruciferae (*Albugo candida*). *Agri. J. Dept. Agric. Fiji* **13**: 27-28.

Parker, JE, Holub, EB, Frost, LN, Falk, A, Gunn, ND and Daniels, MJ. 1996. Characterization of eds1, a mutation in Arabidopsis suppressing resistance to Peronospora parasitica specified by several different RPP genes. *Plant Cell* **8**: 2033–2046.

Pathak, AK and Godika, S. 2005. Efficacy of seed treatments for the control of Alternaria blight and white rust diseases of mustard. *Pestology* **29**: 19-20.

Persoon, CH. 1801. *Synopsis methodica fungorum.* Part I, II. Gottingen: 706 pp.

Perwaiz, MS, Moghal, SM and Kamal, M. 1969. Studies on the chemical control of white rust and downy mildew of rape (*Sarsoon*). *West Pak. J. Agri. Res* **7**: 71-75.

Petrie, GA. 1973. Diseases of *Brassica* species in Saskatchewan, 1970-72. I. Staghead and aster yellows. *Can. Plant Dis. Surv* **53**:19-25.

Petrie, GA. 1975. Prevalence of oospores of *Albugo cruciferarum* in *Brassica* seed samples from western Canada, 1967-73. *Can. Plant Dis. Surv* **55**: 19-24.

Petrie, GA. 1986. *Albugo candida* on *Raphanus sativus* in Saskatchewan. *Can. Plant Dis. Surv* **66**: 43-47.

Petrie, GA. 1988. Races of *Albugo candida* (white rust and staghead) on cultivated cruciferae in Saskatchewan. *Can. J. Plant Pathol* **10**: 142-150.

Petrie, GA. 1994. New races of *Albugo candida* (white rust) in Saskatchewan and Alberta. *Can. J. Plant Pathol* **16**: 251-252.

Petrie, GA and Vanterpool, TC. 1974. Fungi associated with hypertrophies caused by infection of Cruciferae by *Albugo cruciferatum*. *Can. Plant Dis. Surv* **54**: 37-42.

Petrie, GA and Verma, PR. 1974. A simple method for germinating oospores of *Albugo candida*. *Can. J. Plant Sci* **54**: 595-596.

Pidskalny, RS and Rimmer, SR. 1985. Virulence of *Albugo candida* from turnip rape (*Brassica campestris*) and mustard (*Brassica juncea)* on various crucifers. *Can. J. Plant Pathol* **7**: 283-286.

Pound, GS and Williams, PH. 1963. Biologal races of *Albugo candida*. *Phytopathol* **53**: 1146-1149.

Prabhu, KV, Somers, DJ, Rakow, G and Gugel, RK. 1998. Molecular markers linked to white rust resistance in mustard *Brassica juncea*. *Theor. Appl. Genet* **97**: 865-870.

Prevost, B. 1807. Memoire sur la causes immediate de la Carie ou charbon des bles. etc. Paris 33-35. (English translation by G.W. Keitt in Phytopath. *Classics No.* 6, **1939**: 48-55).

Raabe, RD and Pound, GS. 1952. Relations of certain environal factors to initiation and development of the white rust disease of spinach. *Phytopathol* **42**: 448-452.

Rairdan, G and Moffett, P. 2007. Brothers in arms? Common and contrasting themes in pathogen perception by plant NB-LRR and animal NACHT-LRR proteins. *Microbes Infect* **9**: 677–686.

Rao, MVB and Raut, RN. 1994. Inheritance of resistance to white rust (*Albugo candida*) in an interspecific cross between Indian mustard (*Brassica juncea*) and rapeseed (*Brassica napus*). *Indian J. Agril. Sci* **64**: 249-251.

Rayss, T. 1938. Nouvelle contribution altetude de la mycofbre de Palestine. *Palestian J. Bot* **1**: 143-160.

Rimmer, SR, Mathur, S and Wu, CR. 2000. Virulence of isolates of *Albugo candida* from western Canada to *Brassica* species. *Can. J. Plant Pathol* **22**: 229–235.

Rod, J. 1985. Effect of Zineb, metalaxyl and iprodion on the yield and germination characteristics of radish seeds. *Tests Agrochem. Cult* **6**: 64-65.

Rusterucci, C, Aviv, DH, Holt, BF, Dangl, JL, and Parker, JE. 2001. The disease resistance signaling components EDS1 and PAD4 are essential regulators of the cell death pathway controlled by LSD1 in Arabidopsis. *Plant Cell* **13**: 2211-2224.

Sachan, JN, Kolte, SJ and Singh, B. 1995. Genetics of resistance to white rust (*Albugo candida* race-2) in mustard (*Brassica juncea*). In: GCIRC *9th International Rapeseed Congress*, Cambridge, UK: pp 1295–1297.

Sackston, WE. 1957. Diseases of sunflowers in Uruguay. *Plant Dis Rep* **41**: 885-889.

Safeefulla, KM and Thirumalachar, MJ. 1953. Morphological and cytological studies in *Albugo species* on *Ipomoea aquatica* and *Merrimia emarginata*. *Celluler* **55**: 225-231.

Saharan, GS. 1996. Studies on physiologic specialization, host resistance and epidemiology of white rust and downy mildew disease complex in rapeseed-nustard. Final report of adhoc research project ICAR, Dept. of Plant Pathology, CCSHAU, Hisar, India: pp 1083.

Saharan, GS. 1997. Disease resistance. *In*: Recent Advances in Oilseed Brassicas. (Eds.) Kalia, HR and Gupta, SC, Kalyani Pub., Ludhiana Chap **12**: 233-259.

Saharan, GS. 2010. Analysis of Genetic Diversity in *Albugo*-Crucifer System. *Mycol. Plant Pathol* **40**: 1-13.

Saharan, GS and Krishnia, SK. 200l. Multiple disease resistance in rapeseed and mustard. In: *Role of resistance in intensive agriculture*. (S. Nagarajan and D.P. Singh, Eds.), Kalyani Pub., New Delhi: 98-108.

Saharan, GS and Mehta, N. 2002. Fungal diseases of rapeseed-mustard. *IN: Diseases of Field Crops*, Gupta VK and Paul, YS (Eds.): Indus Pub Co. New Delhi: 193-228.

Saharan, GS and Verma, PR. 1992. *White rust. A review of economically important species*. International Development Research Centre, Ottawa, Ontario, Canada, IDRC-MR315e, IV+65p.

Saharan, GS, Kaushik, CD and Gupta, PP. 1990. Optimum fungicidal spray schedule for the control of white rust of mustard. In: Plant Path. Res. Problem and Progress (M.P. Srivastava and G.S. Saharan, editors). Haryana Agric. Univ., Hisar, India: pp 25-29.

Saharan, GS, Kaushik, CD and Kaushik, JC. 1988. Sources of resistance and epidemiology of white rust of mustard. *Indian Phytopathol* **41**: 96-99.

Saharan, GS, Mehta, N and Saharan, MS. 2005. Development of disease resistance in rapeseed-mustard. In: *Diseases of oilseed crops* (Eds) GS, Saharan, N, Mehta and MS, Saharan). Indus Pub. Co., N. Delhi: pp 561-617.

Sarasola, AA. 1942. Sunflower disease. Publication Directorate Agrio. *B. Aires:* 14 p.

Savulescu, 0. 1946. Study of the species of *Cystopus* (Pers.) Lev. Bucharest, 1946. *Anal. Acad. Rous. Mem. Sect. Stimtiface. Soc* **21**: 13.

Savulescu, T and Rayss, T. 1930. Contribution to the knowledge of the Peronsoporaceae of Romania. *Annals Mycol* **28**: 297-320.

Shah, J and Klessig, DF. 1999. Salicylic acid: signal perception and transduction. *In*: Biochemistry and Molecular Biology of Plant Hormones. MA, Hooykaas and KR, Libbenga, eds. Elsevier Science B.V., New York: 513-541.

Sharma, KD and Kolte, SJ. 1985. Metalaxyl in the control of downy mildew and white rust of rapeseed and mustard. *Pestol* **9**: 31-35.

Sharma, SR. 1983. Effect of fungicidal spray on white rust and downy mildew diseases and seed yield of radish. *Gertenbauwissenschaft* **47**: 108-112.

Sharma, SR and Sohi, HS. 1982. Effect of fungicides on the development of downy mildew and white rust of radish. *Indian J. Agri. Sci* **52**: 521-524.

Silue´ D, Nashaat NI, Tirilly Y. 1996. Differential response of *Brassica oleracea* and *B. rapa* accessions to seven isolates of *Peronospora parasitica* at the cotyledon stage. *Plant Dis* **80**: 142–144.

Sinah, PK and Mall, AK. 2007. Screening of rapeseed and mustard genotypes against white rust (*Albugo cruciferarum* S.F., Gray). *Vegetos* **20**: 59-62.

Singh, US, Nashaat, NI, Doughty, KJ and Awasthi, RP. 2002. Altered phenotypic response to *Peronospora parasitica* in *Brassica juncea* seedlings following prior inoculation with an avirulent or virulent isolate of *Albugo candida. Eur. J. Plant Pathol* **108**: 555–564.

Singh, B and Singh, RV. 1990. Economic fungicidal control of white rust of mustard. *Indian Phytopathol* **43**: 272.

Singh, BM and Bhardwaj, CL. 1984. Physiologic races of *Albugo candida* on crucifers in Himachal Pradesh. *Indian J. Mycol. Plant Pathol* **14**: 25 (Abstr).

Singh, D and Singh, H. 1987. Genetic analysis of resistance to white rust in Indian mustard. *Proc. 7th International rapeseed congress*, Poznan, Poland, 11-14 May, 1987. Poznan, Poland: 126.

Singh, D and Singh, H. 1988. Inheritance of white rust resistance in interspecific crosses of *Brassica juncea L.* x *Brassica carinata L. Crop Res* **1**: 189-193.

Singh, D, Singh, H and Yadava, TP. 1988. Performance of white rust *(A. candida)* resistance genotypes developed from interspecific crosses of *B. juncea* L. x B. c*arinata* L. *Cruciferae NewsLett* **13**: 110.

Singh, Dharam, Maheshwari, VK and Gupta, Anuja. 2002. Field evaluation of fungicides against white rust of *Brassica juncea*. *Agric. Sci. Digest* **22**: 267 – 269.

Singh, G, Gupta, ML, Ahuja, I and Raheja, RK. 1998. Biochemical traits in relation to white rust resistance in Indian mustard (*Brassica juncea* L.). *Crop Improv* **25**: 48-52.

Singh, H. 1966. On the variability of the callus of Ipomoea infected with *Albugo*, grown under in vitro conditions. *Phytomorphol* **16**: 189-192.

Singh, HV. 2000. Biochemical basis of resistance in *Brassica* species against downy mildew and white rust of mustard. *Plant Dis. Res* **15**: 75-77.

Singh, HV. 2005. Biochemical changes in *Brassica juncea* cv. Varuna due to *Albugo candida* infection. *Plant Dis. Res. Ludhiana* **20**: 167-168.

Singh, SB, Singh, DV and Bais, BS. 1980. *In vivo* cellulase and pectinase production by *A. candida* and *P. parasitica*. *Indian Phytopathol* **33**: 370-371.

Singh, US, Doughty, KJ, Nashaat, N.I., Bennett, RN, and Kolte, SJ. 1999. Induction of systemic resistance to *Albugo candida* in *Brassica juncea* by pre- or coinoculation with an incompatible isolate. *Phytopathol* **89**: 1226-1232.

Smith, JD and Reiter, WW. 1974. A general-purpose illuminated temperature gradient plate. *Can. J. Plant Sci* **54**: E59-E64.

Sokhi, SS, Grewal, RK and Munshi, GD. 1997. A laboratory technique of fungicide evaluation against *Albugo candida* causing white rust of *Brassica juncea. Indian Phytopathol* **50**: 585-586.

Somers, DJ, Rakow, G and Rimmer, SR. 2002. *Brassica napus* DNA markers linked to white rust resistance in *Brassica juncea*. *Theor. Appl. Genet* **104**: 1121–1124.

Soylu, S, Keshavarzi, M, Brown, I, Mansfield, JW. 2003. Ultrastructural characterisation of interactions between Arabidopsis thaliana and *Albugo candida*. *Physiol. Mol. Plant Pathol* **63**: 201–211.

Soylu, S. 2004. Ultrastructural characterisation of the host–pathogen interface in white blister-infected *Arabidopsis* leaves. *Mycopathol* **158**: 457–464.

Sridhar, K. and Raut, RN. 1998. Differential expression of white rust resistance in Indian mustard (*Brassica juncea*). *Indian J. Genet. Plant Breed* **58**: 319-322.

Srivastava, BIS, Shaw, M and Vanterpool, TC. 1962. Effect of *Albugo candida* (Pers. Ex Chev.) Kuntze. On growth substances in *Brassica napus* L. *Can. J. Bot* **40**: 53-59.

Srivastava, LS and Verma, RN. 1989. Fungicidal control of white rust of mustard in Sikkim. *Indian Phytopathol* **42**: 84-86.

Stevens, FL. 1901. Gametogenesis and fertilization *in Albugo*. Contribution from the Hull Botanical Laboratory. XXIX. *Bot. Gaz* **32**: 77-98.

Stone, JR, Verma, PR, Dueck, J and Westcott, ND. 1987. Bioactivity of the fungicide metalaxyl in rape plants after seed treatment and soil drench applications. *Can. J. Plant Pathol* **9**: 260-264.

Stone, JR, Verma, PR, Dueck, J and Spurr, DT. 1987. Control of *Albugo candida* race 7 in *B. campestris* cv. Torch by foliar, seed and soil applications of metalaxyl. *Can. J. Plant Pathol* **9**: 137-145.

Stovold, GE and Moore, KJ. 1972. Diseases. *Agrio. Gaz. N.S.W* **83**: 262-264.

Stringam, GR. *1971. Genetics of four hypocotyl mutants in Brassica campestris* L. *Hered* **62**: 248-250.

Subudhi, PK and Raut, RN. 1994. White rust resistance and its association with parental species type and leaf waxiness in *Brassica juncea* x *Brassica napus* crosses under the action of EDTA and gamma-ray. *Euphytica* **74**: 1-7.

Sullivan, MJ, Damicone, JP, and Payton, ME. 2002. The effects of temperature and wetness period on the development of spinach white rust. *Plant Dis* **86**: 753-758.

Takeshita, RM. 1954. Studies on the white rust disease of horseradish incited by *Albugo candida* (Pers.) Kuntze. *Diss. Abstr* **14**: 1493-1494.

Tewari, JP and Skoropad, WP. 1977. Ultrastructure of oospore development in *Albugo candida* on rapeseed. *Can. J. Bot* **55**: 2348-2357.

Tewari, JP, Skoropad, WP and Malhotra, SK. 1980. Multi-lamellar surface layer of the cell wall of *Albugo candida* and Phycomyces blakesleeanus. *J. Bacteriol* **142**: 689-693.

Thines, M, Choi, YJ, Kemen, E, Ploch, S, Holub, EB, Shin, HD, Jones, JD. 2009. A new species of Albugo parasitic to Arabidopsis thaliana reveals new evolutionary patterns in white blister rusts (Albuginaceae). *Persoonia* **22**: 123-128.

Thomton, JH and Cooke, RC. 1970. Accumulation of dark-fixed carbon compounds in pustules of *Albugo* tragopogonis. *Trans. Br. Mycol. Soc* **54**: 483-485.

Thukral, SK and Singh, H. 1986. Inheritance of white rust resistance in *Brassica juncea*. *Plant Breed* **97**: 75-77.

Tiwari, AS, Petrie, GA and Downey, RK. 1988. Inheritance of resistance to *Albugo candida* race 2 in mustard [*Brassica juncea* (L.) Czem.]. *Can. J. Plant Sci* **68**: 297-300.

Togashi, K and Shibasaki, Y. 1934. Biometrical and biological studies of *Albugo candida* (Pers.) O. Kuntze in connection with its specialization.

Bull. Impl. College Agri. Forestry (Morioka, Japan) **18**: 88.

Tor, M, Gordon, P, Cuzick, A, Eulgem, T, Sinapidou, E, Mert-Turk, F, Can, C, Dangl, JL, and Holub, EB. 2002. *Arabidopsis* SGT1b is required for defense signaling conferred by several downy mildew resistance genes. *Plant Cell* **14**: 993-1003.

Tornero P, Merit, P, Sadanandom, A, Shirasu, K, Innes, RW, and Dangl, JL. 2002. *RAR1* and *NDR1* contribute quantitatively to the function of *Arabidopsis* disease resistance genes in both simple and non-linear pathways. *Plant Cell* **14**: 1005-1015.

Uppal, BN. 1926. Relation of oxygen to spore germination in some species of the Peronsoporales. *Phytopathol* **16**: 285-292.

Van der Hoorn, RAL and Kamoun, S. 2008. From guard to decoy: A new model for perception of plant pathogen effectors. *Plant Cell* **20**: 2009–2017.

Vanterpool, TC. 1959. Oospore germination in *Albugo candida*. *Can. J. Bot* **37**: 169-172.

Varshney, A, Mohapatra, T and Sharma, RP. 2004. Development and validation of CAPS and AFLP markers for white rust resistance gene in *Brassica juncea*. *Theor. Appl. Genet* **109**: 153–159.

Verma, PR. 1989. Report for the post-doctoral transfer of work on white rust (*Albugo candida*) conducted during Nov 1989 - March 1990 at the Jawaharlal Nehru Krishi Vishwa Vidyalaya, Regional Agricultural Research Station, Morena, M.P., India. Unofficial Report: Project No. 88-1004, IDRC, Ottawa, Canada: 8 p.

Verma, PR and Petrie, GA. 1975. Germination of oospores of *Albugo candida*. *Can. J. Bot* **53**: 836-842.

Verma, PR and Petrie, GA. 1978. A detached-leaf culture technique for the study of white rust disease of *Brassica* species. *Can. J. Plant Sci* **58**: 69-73.

Verma, PR and Petrie, GA. 1979. Effect of fungicides on germination of *Albugo candida* oospores *in vitro* and on the foliar phase of the white rust disease. *Can. Plant Dis. Surv* **59**: 53-59.

Verma, PR and Petrie, GA. 1980. Effect of seed infestation and flower bud inoculation on systemic infection of turnip rape by *Albugo candida*. *Can. J. Plant Sci* **60**: 267-271.

Verma, PR, Harding, H, Petrie, GA and Williams, PH. 1975. Infection and temporal development of mycelium of *Albugo candida* in cotyledons of four *Brassica spp*. *Can. J. Bot* **53**: 1016-1020.

Verma, PR, Saharan, GS, Bartaria, AM and Shivpuri, A. 1999. Biological races of *Albugo candida* on *Brassica juncea* and *Brassica rapa* var. Toria in India. *J. Mycol. Plant Pathol* **29**: 75–82.

Verma, PR, Spurr, DT and Petrie, GA. 1983. Influence of age, and time of detachment on development of white rust in detached *Brassica campestris* leaves at different temperatures. *Can. J. Plant Pathol* **5**: 154-157.

Verma, U and Bhowmik, TP. 1988. Oospores of *A. candida* (Pers. ex. Lev.) Kuntze: its germination and role as the primary source of inoculum for the white rust disease of rapeseed and mustard. *Int. J. Trop. Plant Dis* **6**: 265-269.

Verma, U and Bhowmik, TP. 1989. Inheritance of resistance to a *Brassica juncea* pathotype of *Albugo candida* in *Brassica napus*. *Can. J. Plant Pathol* **11**: 443-444.

Verma, Uma and Bhomik, TP. 1986. A simple method of inoculating white rust on rapeseed-mustard. *Ind. J. Trop. Plant Dis* **4**: 41-43.

Viegas, AP and Teixeira, AR. 1943. Alguns fungos do Brasil. *Bragantia, Sao Paulo* **3**: 223-269.

Voglmayr, H and Riethmüller, A. 2006. Phylogenetic relationships of *Albugo* species (white blister rusts) based on LSU rDNA sequence and oospore data. *Mycol. Res* **110**: 75–85.

Wager, H. 1896. On the structure and reproduction of Cystopus candidus, Lev. *Ann. Bot* **10**: 295-342.

Wakefield, EM. 1927. The genus *Cystopus* in South Africa. *Trans. Br. Mycol. Soc* **2**: 242-246.

Walker, CA and West, PV. 2007. Zoospore development in the oomycetes. *Fungal Biol. Rev* **21**: 10–18.

Walker, JC. 1957. Plant Pathology. McGraw-Hill Book Co., Inc. New York: pp 214-219.

Whipps, JM and Cooke, RC. 1978. Behaviour of zoosporangia and zoospores of *Albugo tragopogonis* in relation to infection of *Senecio squalidus*. *Trans. Br. Mycol. Soc* **71**: 121-127.

Whipps, JM and Cooke, RC. 1978. Interactions of species of Compositae with *Albugo tragopogonis* from *Senecio squalidus*. *Trans. Br. Mycol. Soc* **70**: 389–392.

Wiant, JS. 1937. White rust on Texas spinach. *Plant Dis. Rep* **21**:114-115.

Williams, PH. 1985. White rust [*Albugo candida* (Pers. ex. Hook.) Kuntze.] IN: Crucifer Genetics Cooperative (CRGC) Resource Book. University Wisconsin, USA: 1-7.

Williams, PH and Hill, CB. 1986. Rapid cycling populations of *Brassica*. *Science* **232**: 1385-1389.

Williams, PH and Pound, GS. 1963. Nature and inheritance of resistance to *Albugo candida* in radish. *Phytopathol* **53**: 1150-1154.

Williams, PH and Pound, GS. 1964. Metabolic studies on the host-parasite complex of *A. candida* on radish. *Phytopathol* **54**: 446-451.

Wilson, GW. 1907. Studies in North American Peronasporales. I. The genus *Albugo*. *Bull. Torrey Botl. Club* **34**: 61-84.

Woods, AM and Gay, JL. 1983. Evidence for a neckband delimiting structural and physiological regions of the host-plasma membrane associated with haustoria of *Albugo candida. Physiol. Plant Pathol* **23**: 73-88.

Wu, CR, Mathur, S and Rimmer, SR. 1995. Differentiation of races and isolates of *Albugo candida* by random amplification of polymorphic DNA. *IN*: *Proc. 9th Int. Rapeseed Congress*, GCIRC, Cambridge, UK: pp 655-657.

Wulff, BB, Thomas, CM, Smoker, M, Grant, M, and Jones, JD. 2001. Domain swapping and gene shuffling identify sequences required for induction of an Avr-dependent hypersensitive response by the tomato Cf- 4 and Cf-9 proteins. *Plant Cell* **13**: 255-272.

Yadav, R and Sharma, P. 2004. Genetic diversity for white rust (*Albugo candida*) resistance in rapeseed-mustard. *Indian J. Agril. Sci* **74**: 281–283.

Yadav, YP and Singh, H. 1992. The potential exotic sources for white rust resistance in India mustard [*Brassica juncea* (L.) Czern. & Coss.]. IDRC, Ottawa, Ontario, Canada (Pub.), A. Omran (Ed.). *Oil Crops NewsLett* **9**: 18.

Young, ND. 2000. The genetic architecture of resistance. *Curr Opin Plant Biol* **3**: 285-290.

Zalewski, A. 1883. Zur Kenntniss der Gattung *Cystopus*. *Bot. Centbl. Bd* **15**: 215-224.

Zhang, ZY, Wang, YX and Liu, YL. 1984. Taxonomic studies of the family Albuginaceae of China. II. A new species of *Albugo* on Acanthaceae and known species of *Albugo* on cruciferae. *Acta* Mycologica *Sinica* **3**: 65-71.