CHAPTER 4. INTEGRATED SYSTEMS OF PROTECTION OF CRUCIFEROUS CROPS AGAINST MAJOR DISEASES (DOMESTIC AND INTERNATIONAL EXPERIENCE)

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4.1. Integrated systems and methods of Alternaria control in agrocenoses of cruciferous crops (generalised world experience)

Modern intensive technologies of oilseed rape cultivation require clear, well-thought-out and decisive measures in the field of plant protection against diseases. Given the increase in rapeseed cultivation, there is a need to develop new effective systems and means of crop protection [1].

Monitoring of diseases of winter and spring rape in Ukraine over the past 5 years has shown that the most common and damaging disease is Alternaria [2]. The ability of the pathogen to persist in crop residues, seeds, and develop on winter and spring rape and other crops leads to the risk of epiphytic development of Alternaria under favourable conditions every year. This indicates the possibility of significant yield and quality losses.

It is noted [3]. that among the factors affecting crop yields, fungal diseases prevail, in particular, Alternaria, the symptoms of which appear throughout the growing season as spots on leaves, stems, and pods. Seeds affected by Alternaria pathogens die in the soil. In the early stages of ontogeny, Alternaria appears on seedlings in the form of dark brown spots, leading to their death. On the leaves, the disease is marked by light brown to dark brown, almost black spots of various sizes. The nature of the spots depends on the type of pathogen and the organ affected. On the stems, the spots are dark, on the pods – dark spots

that turn into ulcers, stretch marks over time, the pods are deformed and develop small seeds or do not form. If the top of the pod is affected or along the seams of the valves, it cracks, causing early yield losses [4]. In the literature on phytopathology, the following species are now more commonly described as pathogens of Alternaria: *Alternaria brassicae* (Berk.) Sacc., *A. brassicicola* (Schwein) Wiltshire and *A. japonica* Yoshii (Figure 4.1). In addition, alternaria pathogens are facultative parasites with a wide range of adaptive responses. As a rule, they are characterised by a saprophytic lifestyle, but when plants are weakened, they can switch to parasitism. Variability of pathogenicity of fungi of the genus Alternaria is known. For example, according to O.L. Gasych, out of 3 isolates of fungi of the genus *A. brassicae*, 11 varieties of rape showed pathogenicity to varying degrees on the leaves. The culture liquid of A. brassicae fungus can be phytotoxic to rapeseed sprouts. According to the research of F.B. Hannibal, A. alternata and A. tenuissima species are more toxic to plants than A. infectoria. Studies1058 conducted on Sarepta mustard indicate that A. brassicicola is more toxic than A. brassicae.

In the works of scientists who conducted research in Iran, Israel, Peru, and China, the pathogenicity of A. alternata fungi was noted [5–8].

Weak and damaged plants are more susceptible to damage, so it is important to select resistant, high-performance varieties for production, apply effective protection systems using pesticides and growth regulators to prevent damage at the initial stages of organogenesis [9].

In long-term studies of the structure of pathogenic alternaria in Poland and Ukraine [10] it is noted that the development of Alternaria in crops of varieties and hybrids undergoing competitive variety testing ranged from 1,8 to 43,3%, This indicator in winter rape crops, depending on the variety, reached in 2014 21,1% on leaves and 16,6% on pods; in 2015 – 4.5–22.1% and 6.9–8.8%; in 2016 – 3.5–13.6% and 12.4–24.4%; in 2017 – 8.5–12.1% on leaves and 35.6–48.8% on pods.

The harmfulness of Alternaria in winter rape crops is to reduce the length of the pod by 16.2–30.5%, the number of seeds in the pod by 13.8–31.0%, the weight per pod by 17.6–35.3%, the weight per plant by 53.3–81.4%, and the weight of 1000 seeds by 9.1–37.9%. It was noted that 64.3% of the plants examined were affected by the disease of moderate and severe degree. The main indicator that is significantly affected by Alternaria spp,

fungi is the weight of seeds per plant (r = -0.711). As the intensity of the disease increases, seed quality decreases: dry matter content – from 95.9 to 80.6%, fat – from 45.7 to 42.1%, but glucosinolates content – from 18.7 to 32,9% and protein content – from 17.7 to 20.5%.

The threshold of harmfulness of Alternaria or the level of disease development, from which a significant decrease in the weight of 1000 seeds is observed, is 2.7–6.1%, the relative harmfulness coefficient is 0.34–0.63.

Figure 4.1 – Alternaria (pathogen: *Alternaria brassicae, Alternaria brassicicola, Alternaria raphani* [11]

To clarify the species composition of fungi of the genus Alternaria, which parasitise winter rape, the following were isolated and analysed [5] more than 500 isolates. As a result of determining the species composition of Alternaria spp. isolated from parts of winter rape – leaves, stems, pods, roots, seeds, the dominance of isolates of *A. tenuissima* with a frequency of 32.3%, *A. brassicicola* – 26.6 and 19.6% – A. alternata was established. In the population structure of Alternaria spp. 21.5% of isolates caused difficulties in their identification.

It was found that the frequency of isolation of A. alternata in the maximum amount (83.9%) was noted when isolated from seeds, while the fungus was not isolated from the affected tissues of stems and pods (Table 4.1).

Table 4.1

Dominance structure of Alternaria spp. fungi isolated from parts of winter rape plants (laboratory experiment) [4]

Type	Frequency of occurrence, %			
	seed	leaves	stems	pod
A. brassicicola	40.5	45.3	7.1	7.1
A. tenuissima	33.3	33.3	21.6	11.8
A. alternata	83.9	16.1	0.0	0.0
Alternaria spp.	23.5	58.8	5.9	11.8

Table 4.2

Effect of Alternaria spp. isolates on the growth of sprouts and roots of winter rape (average for 2014–2017) [12]

Type	Number of isolates	Affected plant organs, %			
		sprouts		spines	
		length reduction	stimulation growth	length reduction	stimulation growth
A. brassicicola	42	90.5	9.5	95.2	4.8
A. tenuissima	51	82.3	17.7	86.3	13.7
A. alternata	31	96.8	3.2	96.8	3.2
Alternaria spp.	34	88.2	11.8	91.2	8.8

The fungus *A. brassicicola* was isolated from seeds (40.5%) and leaves (45.3%) with relatively equal frequency, the same dependence is characteristic of the fungus *A. tenuissima*, the latter was also isolated more often than others from the affected stem tissues (21.6%).

The differentiation of Alternaria spp. species isolated from the affected parts of winter rape plants by the frequency of occurrence necessitated the need to conduct studies to determine their effect on sprouts in case of seed damage.

The analysis of the data obtained indicates a significant suppressive effect of Alternaria spp. isolates on the length of sprouts and roots. Isolates of *A. alternata* reduced the length of the studied parameters in 96.8% of seedlings, while, for example, *A. brassicicola* – in 90.5% of sprouts and 95.2% of roots.

Isolates of A. tenuissima caused a decrease in the length of the sprout (82,3%) and roots (86.3%), and some isolates had a stimulating effect on seedlings. A higher number of such isolates was characteristic of A. tenuissima – 17.7%, which stimulated sprout growth, and 13.7% – root growth, for *A. alternata* isolates – 3.2 and 3.2%, respectively.

Symptoms of sprout damage by pathogenic fungi were in the form of dots and necrosis from brown to dark brown and black, located singly or in groups. The defeat of 50% or more of the sprout surface caused its decay or death. Symptoms of winter rape sprout damage are shown in Figure 4.2.

The analysis of Alternaria spp. isolates by pathogenicity showed (Table 4.3) that the overall population of Alternaria spp. is highly pathogenic.

All (100%) isolates of *A. brassicicola* and *A. tenuissima* are highly pathogenic, among the isolates of A. alternata group 83,9% are highly pathogenic, 9,7% are pathogenic and 6,4% are moderately pathogenic.

Figure 4.2 – Signs of fungal infestation of winter oilseed rape sprouts *Alternaria* spp [8]

Under artificial infection of winter rape plants with *A. brassicicola, A. tenuissima* and *A. alternata*, at different stages of crop development, their pathogenicity was confirmed and it was determined that the highest level of disease development on leaves was observed at the stage of BBCH 50 (appearance of the primary peduncle) and BBCH 61 (about 10% of open flowers on the main shoot, elongated peduncle, tightly closed by the upper leaves) – 45.9 and 72.9%, on pods – at stages 61 and 69 (end of flowering) – 72.7 and 84.2%.

The study of the effect of temperature on the growth of Alternaria spp. fungi revealed that the optimal temperatures for the growth of *A. alternata, A. tenuissima* and *A. brassicicola* are in the range of 20.0–25.0 °C.

The study of relationships between fungal pathogens of Alternaria during co-cultivation showed that the relationship between isolates of *A. brassicicola* and *A. alternata*, *A. tenuissima* is based on the type of territorial antagonism, and the pathogen *A. brassicicola* was the antagonist.

Table 4.3

Differentiation of Alternaria spp. fungal isolates by pathogenicity (laboratory test, average for 2014–2017) [13]

Type	Number of isolates	Proportion of isolates causing lesions, %					
		highly pathogenic	pathogenic	moderately pathogenic	moderately pathogenic	slightly pathogenic	avirulent
A. brassicicola	42	100	0.0	0.0	0.0	0.0	0.0
A. tenuissima	51	100	0.0	0.0	0.0	0.0	0.0
A. alternata	31	83.9	9.7	0.0	6.4	0.0	0.0
Alternaria spp.	34	91.2	8.8	0.0	0.0	0.0	0.0

The results obtained in clarifying the species composition of rapeseed pathogens, studying their developmental features, pathogenic properties and their impact on the crop made it possible to substantiate plant protection methods.

Oilseed crops are constantly exposed to a number of pathogens and, as a result, they have developed complex defence mechanisms to recognise and protect against a wide range of these pathogens through structural

defences [8] and induction of a complex of defence reactions that can defeat pathogens [14].

Table 4.4

Features of winter rape sprout damage due to infection with Alternaria pathogens [13]

Characteristics of the seedling impression	Occurrence (%) of seedling lesions, fungi		
	A. alternata	*A. brassicicola*	*A. tenuissima*
Healthy	28.6	4.0	5.0
Point necrosis of tissues	26.2	21.2	22.9
Necrosis of about 50 % of the surface	19.5	23.0	21.9
Total destruction	25.7	51.8	50.2

Structural defence against Alternaria is associated with factors that prevent conidia from being retained on the host surface, such as high epicuticular wax deposits that form a physical barrier in the form of a hydrophobic coating, which reduces the deposition of aqueous inoculum, reduces the rate of conidial germination and germ tube formation [15–16].

Species *B. napus, B. carinata* and *B. alba* have relatively more epicuticular wax than *B. rapa* and *B. juncea* and are generally less susceptible to infection by Alternaria pathogens [5].

It was found that biochemical defence against Alternaria of mustard leaves was associated with leaf enzymes related to the phenolic pathway and higher sugar content in the leaves. The resistance of Camelina sativa to A. brassicae was associated with the presence of chemical compounds, camalexins, somewhat similar to the fungicide available on the market. The resistance of Camelina sativa to A. brassicae through the production of the phytoalexin camalexin was also reported by Jejelowo et al. [10] and Thomma (1999) [7].

Camalexin also has an indirect effect on Alternaria resistance, as it was found that camalexin inhibits the production of the toxin destructin B in *A. brassicae* [5].

The cultivation of resistant varieties is important in controlling this pathogen. It has been established that the resistance index of cruciferous varieties has a significantly different value in different genotypes. For example, resistance to Alternaria was studied97 on 14 spring oilseed

rape varieties of domestic and foreign breeding at the Agronomic Research Station of the National University of Life and Environmental Sciences of Ukraine (NULES of Ukraine) (Table 4.5).

At the same time, these studies have established [8], that during the growing season, there was an increase in the values of indicators of the spread and development of the disease. According to the results of the research, a significant difference in the resistance of the studied varieties was found. No immune varieties were found.

If we place the studied varieties in order from lower to higher levels of resistance to Alternaria, this chain will look like this: Belinda, Salsa KL, Delight, Obriy, Olga, Magnat, Khuzar, Siesta F1, Oksamit, Aira, La Rissa, Cliff, Hidalgo, Maria. Thus, Belinda had the highest degree of damage (96.5% spread, 10.7% development), and Maria was the most resistant (54.5% and 4.7%, respectively). It is worth noting that weather conditions restrained the spread and development of the infection on plants, so the damage caused by Alternaria did not affect the yield data, with varietal characteristics playing a decisive role.

In long-term foreign studies on the virulence of Alternaria pathogens and the search for resistance donors, taking into account the chemistry of host-pathogen interaction, a number of important conclusions and generalisations have been made.

Due to the high losses caused by Alternaria in oilseeds, the goal of oilseed breeders is to develop Alternaria-resistant lines. Several attempts have been made in the past to identify sources of resistance to Alternaria, but no resistant sources have been identified in any of the cultivated castor oilseed rape species, but a high degree of resistance to Alternaria has been found in *B. alba,· Eruca sativa* [18] and *Sinapis alba* [19–22]

The highest degree of resistance to *A. brassicae* was found in wild relatives of cabbage outside the Brassicaceae tribe. This is false flax (*Camelina sativa*), shepherd's bag (*Capsella bursa-pastoris*) and panicle weed.

In search of sources of resistance for transferring resistance genes, thirty-eight species belonging to nine genera, including cultivated and wild cruciferous species, were evaluated under epiphytic conditions for two years. Eight species, namely *B. desnottesii, C. sativa, Coincya pseuderucastrum, Diplotaxis berthautii, Diplotaxis catholica, Diplotaxis cretacea, Diplotaxis erucoides* та *Erucastrum gallicum* were found to

be fully resistant, while others were classified as moderately resistant, susceptible and highly susceptible.

Table 4.5

Distribution and development of Alternaria in spring oilseed rape varieties [9]

Variety	Disease prevalence, %			Disease progression, %			Yield, t/ha
	Growth phase	Flowering	Achievements	Growth phase	Flowering	Achievements	
Belinda	39.6	43.4	96.5	5.4	6.3	10.7	2.03
Delight	33.0	34.2	99.0	4.8	6.0	9.9	1.69
Horizon	27.8	32.5	98.3	4.1	5.3	9.8	1.71
Cliff	19.1	22.1	84.8	2.8	3.2	7.0	2.18
Larissa	19.9	23.0	77.3	3.5	4.3	7.9	1.94
Siesta FI	23.3	34.9	94.8	3.6	5.5	9.2	2.45
Maria	13.1	21.8	54.5	1.6	2.2	4.7	2.37
Huzar	20.1	22.6	82.0	4.0	5.3	9.7	2.74
Olga	22.6	30.8	88.0	4.5	6.1	9.8	2.08
Velvet	18.3	24.3	79.8	2.7	3.4	9.0	1.82
Magnate	15.0	18.5	64.2	2.9	3.9	9.8	1.86
Hidalgo	14.1	26.3	75.4	1.8	2.6	5.9	2.68
Salsa KL	20.0	22.9	84.5	4.2	5.3	10.0	2.13
Aira	21.6	29.4	91.8	3.4	4.5	8.7	2.59
SSD_{05}	1.7	1.2	1.4	0.1	0.3	0.2	0.18

A wide range of variation was also observed among species of the genus *Diplotaxis*, but the genus Diplotaxis proved to be more resistant than the genus *Brassicas*. Based on this, we concluded that the sources of resistance to *A. brassicae* present within the cenotic species *Brassicas* (tribes), from which resistance genes can be introduced into cultivated cruciferous species. Seven species, except for *C. sativa*, have been identified as fully resistant to Alternaria leaf spot and belong to the cenotic species of Brassica (Brassicaceae). As resistance is not present in cultivated species, these eight

resistant wild species can be used as donor parents for introgression of resistance to Indian mustard leaf spot. The absence of a resistance gene in crossbred Brassica germplasm necessitates the use of genetic engineering strategies to develop genetic resistance to this pathogen.

Mondal et al. (2007) [23] obtained the integration and expression of the class 1 major glucanase gene in transgenic mustard plants and observed that the transgenes delayed hyphal growth of *A. brassicae* by 15–54%. Transfer of alternaria resistance genes in Brassica sp. Tuan and Garg (2001) [24] carried out gene transformation in Brassica sp. using particle bombardment. Cotyledons and hypocotyls of different Brassica species were used as target explants. Transient expression of the uidA gene was obtained by constructing it with CaMV35S or Actin promoters. The uid4 gene encodes enzyme 1, beta-glucuronidase (GUS). It is a reporter gene system, especially useful in plant molecular biology and microbiology. The highest expression was recorded between 10 and 15 hours after the bombardment. The plasmids pBI121, pBI221 and pDM803 were used to transfer the uidA gene. To obtain the highest transformation efficiency, further transformation steps are required.

Biochemical defence is triggered under any plant stress condition and is the most important tool of the plant defence mechanism. The hypersensitive reaction is one of the most effective plant defence reactions against pathogens [25]. Mustard Alternaria resistance has been reported to be associated with the synthesis of phenolic pathway-associated leaf enzymes and higher leaf sugar content. It was reported that the concentration of phenolic compounds at all stages of plant growth was higher in resistant genotypes compared to susceptible genotypes. However, the content of soluble sugars, reducing sugars and soluble nitrogen in resistant genotypes was lower [26–27]. Another study reported that total phenol, total sugar, reducing sugar, o-dihydroxyphenol, chlorophyll and flavonol content were higher in resistant genotypes. Plants can respond to pathogens by activating several defence responses that prevent the infection process. These include the production of reactive oxygen species (ROS), the accumulation of pathogenesis-related proteins (PRPs) and phytoalexins, and the synthesis of compounds that strengthen the plant cell wall [28]. In addition, the content of ascorbic acid, total phenol, enzymatic activity of superoxide dismutase and peroxidase, as well as cell-protective enzymes such as phenylalanine

ammonia kinase and polyphenol oxidase increased in resistant mustard genotypes [29]. β-Aminobutyric acid (BABA), a non-protein amino acid, is known to stimulate resistance to various pathogens in a number of plant species [30–31]. Pretreatment of Brassica oilseed rape plants with BABA-mediated resistance to the necrotrophic pathogen *A. brassicae* through enhanced expression of protein genes related to pathogenesis [32]. Higher and earlier accumulation of H_2O_2 was observed in resistant *C. sativa* and *S. alba* compared to *B. juncea*. Catalase activity increased in both *C. sativa* and *S. alba*, but in the case of *B. juncea*, the opposite phenomenon was observed [33].

Ethiopian mustard (B. carinata) has the highest level of resistance to Alternaria among cabbage crops. Among the wild cruciferous plants close to the genus Brassica, the highest level of resistance to *A. brassicae* was confirmed for white mustard (*Sinapis alba*) [34-37] However, the highest overall levels of resistance to Alternaria spp. were found in cruciferous plants more distant from Brassica, such as redhead (*Camelina sativa*; false flax), shepherd's purse (*Capsella bursa-pastoris*), arugula *(Eruca sativa*) and panicle mustard (*Neslia paniculata*) [38-39].

Resistance to Alternaria (black spot) has also been reported among other wild representatives of the family *Brassicacae* [40]: *Alliaria petiolata; Barbarea vulgaris; Brassica elongate, B. desnottessi, B. fruticulosa, B. maurorum, B. nigra, B. souliei, B. spinescens; Camelina sativa; Capsella bursa-pastoris; Coincya spp. berthautii, D. creacea, D. erucoides, D. tenuifolia; Erucastrum gallicum; Eruca vesicaria subsp. sativa; Hemicrambe fruticulosa, H. matronalis; Neslia paniculata; Rhaphanus sativus; S. alba ma S. arvensis*. Fully immune plants remained symptom-free both under natural field infection and controlled artificial inoculation. In comparison, broccoli and cauliflower varieties showed only moderate resistance to Alternaria, while cabbage was susceptible.

Depending on the plant material studied, *A. brassica/A. brassicicola* resistance is controlled by one or more nuclear genes with partially dominant interaction [41] or due to additive inheritance [42].

At the biochemical level, resistance to Alternaria pathogens is associated with high activity of phenolases (polyphenol oxidase, peroxidase, catalase), high sugar content in the leaves [43] and a thick epicuticular wax layer, which forms a hydrophobic coating that reduces the adhesion of water seed

and also limits the rate of spore germination. The presence of intense wax deposition on the leaves seems to correlate with the resistance shown by other plants of the cruciferous family.

It has been established that wild cruciferous plants secrete phytoalexins during inoculation. Among the species resistant to Alternaria, redhead stands out for its immunity against *A. brassicicola* infection, which is based on the plant's ability to synthesise camalexin, a compound with antibiotic properties, and thus prevent the pathogen from developing. Indeed, a camalexin-deficient Arabidopsis mutant, pad-3, has been shown to be more susceptible to *A. brassicicola* than wild-type plants [44].

Further evidence that camalexin plays an important role in resistance is the observation that different Arabidopsis ecotypes with different camalexin contents show correlated differential resistance [15]

Finally, the esa1 mutation affects resistance to *A. brassicicola* through a strong reduction in both camalexin production and jasmonate dehydrogenase gene induction, although the Esa-1 gene has not yet been cloned [18].

A direct method for determining resistance to Alternaria is phytopathological testing: field, greenhouse or phytotron. Field observations can be carried out during natural infection with the pathogen, or after controlled artificial inoculation with a fungal suspension. The advantages of greenhouse or phytotron tests are speed, reproducibility, and the ability to control the conditions. Phytopathological tests require conidia of Alternaria spp. collected directly from infected plant tissue or grown on artificial media. On the widely used artificial PDA media, fungal growth and efficient spontaneous sporulation occurs at a temperature of 25 ± 2ºC, in the dark. There is a choice of other methods for the growth and maintenance of Alternaria spp. *A. brassicicola* hyphae have been successfully cultured on V8A artificial medium (V8 juice – agar) at 25 ºC, which led to spontaneous sporulation under a 12-hour photoperiod [20].

Phytotests under controlled conditions are carried out on whole plants (in vivo) or on isolated leaves (in vitro) [45]. Plants are usually tested at the 3–6-week seedling stage, but phytotesting results for cotyledon leaves have also been published. The detached leaf method is one of the most common ways to assess the level of resistance to Alternaria spp. demonstrated by the plants under controlled conditions. However, there are differences in the method of inoculation and the conditions of the assay.

As described in several studies, an inoculum of 5 × 104 spores × ml^{-1} was placed on the upper (adaxial) side of the leaf, while other authors describe spraying the lower (abaxial) side of the leaf with an inoculum of 3 × 105 spores × ml^{-1} [17].

Others inoculated only the 4th and 5th leaves (45 days old seedlings). Wet swabs were used to remove the wax layers from both nerve sides on the adaxial surface of the leaf, so that the aqueous spore suspension was evenly distributed on the leaf surface, without the need for agar or adjuvants. Better adhesion of the aqueous suspension of spores to the waxed surface of cabbage leaves is ensured by the addition of agar [46] or tween. Small superficial incisions were made with a fine needle and a drop of inoculum (4 × 103 spores × ml^{-1}) was placed on them. Disease symptoms were assessed in 24-h increments at 3-days intervals. The assessment of the resistance of individual plants included three parameters: percentile of infected leaf surface (0–60 points), lesion size (0–30 points) and incubation time (0-10 points). Plants with maximum susceptibility received 100 points. Individual plants were grouped into resistance classes according to the scores: 0 – completely resistant; 1–15 points – moderately resistant; 16–25 points – susceptible; over 25 points – highly susceptible. As noted above, the optimal conditions for phytotesting are a temperature of about 20 °C, relative humidity of at least 90% for 6 hours or more, and an inoculation load of 6×104 spores×ml^{-1}.

Since resistance to Alternaria black spot is generally regulated by polygenes, breeding for resistance may involve pyramiding minor genes to provide additive/polygenic resistance. Rapid advances in tissue culture techniques, protoplast fusion, embryo rescue and genetic engineering have made it possible to transfer disease resistance traits across impenetrable incompatibility barriers that would otherwise be impossible. Disease-resistant transgenic plants that overexpress various antifungal compounds, such as pathogenesis-related proteins (PRs) (chitinase, glucanase, osmotin, etc.) and ribosome inhibitory proteins (RIPs) such as thionines, defensins and phytoalexins, to inhibit pathogen growth seem to be less effective. Somatic hybrids between *C. sativa* and *B. carinata* have been developed to introduce resistance to *A. brassicicola* derived from ryegrass into commercial varieties, but researchers have not been able to propagate the hybrids [47].

A similar strategy of fusion of *C. sativa* and *B. oleracea* protoplasts with subsequent regeneration of hybrids also failed [48]. Several research groups have attempted, but without success, to introduce black spot resistance derived from *E. sativa* into different species of cultivated cruciferous vegetables [49–51]. The first somatic hybrids obtained as a result of protoplast fusion were hybrids of *B. napus* (rapeseed) and *S. alba* [52]. None of the hybrids obtained in this way showed resistance to *A. brassicae* comparable to that of *S. alba*. Chevre et al. (1991) [53] used these species for interspecific crosses through somatic hybridisation and bidirectional crosses. Using the embryo rescue technique, the researchers were able to regenerate *B. napus* plants that carried 38 chromosomes characteristic of this species and demonstrated resistance to *A. brassicae* at a level close to that of *S. alba*, *B. oleracea* var. *botrytis* or *B. carinata* [54].

Seeds of the intertribal somatic hybrids between *B. napus* and *C. sativa* (using protoplast electrofusion) had an intermediate phenotype compared to the parental species. They also showed higher levels of linolenic and eicosanoic acids, but the hybrid plants are awaiting determination of their resistance to Alternaria [55]. In general, it has been suggested that the introduction of alternaria resistance genes into commercial cruciferous varieties depends on the accumulation of horizontal resistance genes. Therefore, it is crucial to identify the different sources of horizontal resistance among cruciferous plants and subsequently combine them to increase reliable protection against Alternaria. The strong cross incompatibility, polygenic origin of resistance (additive and dominant gene interactions), and differences in ploidy (different chromosome numbers) between the respective cruciferous species make it difficult to transfer resistance to Alternaria from wild species to cultivars. In addition, this is often associated with the use of modern in vitro hybridisation techniques, including somatic hybridisation, embryo and ovary preservation or protoplast fusion.

This has indeed been demonstrated in Colletotrichum and Magnaporthe, for which transcription factors involved in the regulation of melanin synthesis during development have been identified [56].

To date, there is not a single 100% resistant variety to Alternaria in various cruciferous species. Therefore, the use of resistance from wild species can be an effective breeding tool. Plant pathogens manage to infect different species, but cannot overcome host resistance [57]. Examples

of some non-host plants of *A. brassicae* include chickpea, lentil, wheat, sugarcane, barley, tomato, and potato.

Pre-infestation protection can include structural features of plants such as a large number of trichomes and chemical compounds that inhibit spore germination [58–60]. Previous studies have shown that spore germination occurs at the same rate on both host and non-host plants. Despite spot germination, pathogens may not reach stomata. Stomata in non-host plants may be misrecognised by the pathogen because the surface topography may differ significantly from that of the host leaf.

Another structural feature that can impede the penetration of Alternaria is the epicuticular wax [61–62]. Plants that are more resistant to damage may have more epicuticular wax than susceptible plants [63]. In addition, it was found that the non-native plant is able to induce stomatal closure, preventing the penetration of pathogens and creating an inducible chemical barrier that inhibits hyphal production and differentiation through the rapid formation of phytoalexins, antimicrobial compounds [64–66].

In a plant resistant to Alternaria, nutritional deficiencies and the presence of antimicrobial compounds in the apoplast can also prevent hyphae production into the mycelium. The pathogen also generates nonspecific or general toxins that can damage plant cells, ultimately leading to necrosis [67]. To avoid this, the plant is more resistant to damage – it can recognise these toxins and use defence mechanisms to detoxify them. Pathogenetically related genes PR-1, PR-2, PR-3 were highly expressed in Arabidopsis and *S. alba* after infection with Alternaria [68–70]. In addition, these two species have demonstrated resistance to *A. brassicicola*. Chitinase enzymes, which hydrolyse the fungal cell wall and release chitin fragments, are actively secreted by these two species [71–72]. The action of NHR involves stimulation of the signal transduction cascade by the plant cell after pathogen detection, which triggers the activation of protein kinases and mitogen-activated protein kinase (MAPK) members and, as a result, leads to the activation of defence genes in plants. The expression of MAPK was higher in S. alba and lower in *B. juncea*, indicating its possible role in resistance to Alternaria.

It was previously noted that during the interaction between the host and pathogen (Alternaria – crucifers), a number of biochemical changes occur in both the host and the pathogen. These biochemical changes lead to

the formation of various types of primary and secondary metabolites that affect the host defence system and the virulence of the pathogen. *Alternaria brassicicola* produces compounds such as the antitumour depudecin, the antibiotic complex brassicolin and the phytotoxic brassicins. Production of glucosinolates and phytoalexins correlates with host resistance [73].

Host resistance in cruciferous plants to Alternaria species has different components and is multilayered. The inheritance of resistance in inter- and intraspecific crosses of *B. juncea* and *B. carinata* with *A. brassicae* is regulated by additive genes, dominant genes, epistatic genes of additive x additive, additive x dominant and non-allelic interaction genes of dominant x dominant type. Crossing between resistant plants contributes to an increase in the level of resistance to A. brassicae due to the pyramiding of resistant genes. A high level of horizontal resistance in oilseed genotypes was recorded [74].

Epicuticular wax and a small number and narrow stomatal opening provide resistance to Alternaria infection in cruciferous species. Concentration of phenolic compounds, activation of polyphenol oxidase and catalase is higher in the tolerant mustard genotype [75].

The isolation and accumulation of phytoalexins in cruciferous plants after alternaria infection and their role in disease resistance have been demonstrated. The ability of *A. brassicae* to sequester calcium can be used to increase resistance to this pathogen in canola by applying calcium compounds to the soil or foliar application. Numerous sources of resistance to Alternaria species have been identified in different cruciferous species and their close and distant relatives, but very few have been used to develop resistant varieties. Despite several bottlenecks in the development of resistant varieties, various methods and technologies, including traditional as well as biotechnological approaches, are being used to incorporate desirable traits into cruciferous crops against *Alternaria disease* [76].

Alternaria species that are pathogenic to cruciferous plants produce host-specific and host-neutral toxins that contribute to their pathogenic process to become a successful pathogen. Prior to colonisation, necrotrophs must kill host cells by producing both toxins and lytic enzymes, often by triggering genetically programmed apoplastic pathways or by directly damaging cells, leading to necrosis. *Alternaria brassicae* and *A. brassicicola*, pathogenic to cruciferous plants, produce a number of toxins and metabolites belonging

to chemical groups containing terpenoids, pyranones, steroids and nitrogen. The effect of toxins on plants at the physiological, biochemical and molecular levels has been studied. The role of toxins in the process of infection, their biosynthesis, mode of action, chemical structure, role in host defence and transformation into phytoalexins have been determined [77–83].

Thus, common cultural practices such as the use of healthy and treated seeds of recommended varieties, long rotation crop rotation (3–4 years), sanitation, weed control, shallow sowing (2 cm depth) with proper sowing dates, use of balanced mineral nutrition, proper plant density, proper field drainage, management of plant residues, and use of tolerant (resistant) varieties should be recommended; the use of chemicals (bioagents) at appropriate times with adequate leaf coverage should be recommended for the management of Alternaria diseases of cruciferous crops. That is, the most appropriate is the integration of all control strategies, namely: cultural, chemical, biological, nutrient, biotechnology and genetic engineering.

Several biochemical components were found to confer resistance to *A. brassicae* in rapeseed and mustard. Total sugars, reducing sugars, flavonol and chlorophyll were present in high amounts in healthy leaves, while total phenol, o-dihydroxyphenol, carotenoids and protein increased with increasing *A. brassicae* infection [84–86].

However, total sugar, total phenol and ortho-dihydroxyphenol were higher in chlorotic areas than in necrotic areas of infected leaves. The content of flavonol and chlorophyll was lower in different infected parts of the leaves than in healthy ones, and significantly lower in necrotic areas than in chlorotic areas. The activity of some oxidative enzymes, namely peroxidase (PO) and polyphenol oxidase, increased in B. juncea leaves after infection [87]. The total phenol content and specific activity of phenylalanine ammonia kinase (PAK) and tyrosine ammonia kinase (TAK) were higher in leaves and pod walls infected with Alternaria compared to healthy leaves, indicating their possible involvement in plant defence against the disease [88]. Transpiration in oilseed rape decreases after infection with Alternaria species [89].

Several sources of tolerance to Alternaria have been reported among different Brassica species, with *B. juncea* and *B. rapa* being more susceptible than *B. carinata* and *B. napus*. Disease-resistant lines in B. juncea include PAB-9511, PAB-9534, JMM-915, EC-399296, EC-399301, EC-399299, EC-399313, PHR-1, PHR-2, Divya, PR-8988, PR-9024 and RN-490;

in B. carinata – HC-1, PBC-9221 (Kiran), NRCDR-515 and DLSC-1; in B. napus – PBN-9501, PBN-9502, PBN-2001 and PBN-2002 [90–91]. Sources of resistance to *A. brassicae* were found in wild cabbage species, namely: *Brassica alba*, *Camelina sativa, Capsella bursa-pastoris, Eruca sativa, Neslia paniculata, Brassica desnottesii, Coincya pseuderucastrum, Diplotaxis berthautii, Diplotaxis catholica, Diplotaxis cretacea, Diplotaxis erucoides ma Erucastrum gallicum*.

To genetically increase the level of resistance, was proposed [92] breeding programme using the number and size of lesions to identify differential responses of different genotypes to *Alternaria blight*. Resistance to Alternaria blight of mustard oilseed rape is associated with factors such as phenolic compounds, namely polyphenol oxidase, PO, catalase in leaves, higher sugar and nitrogen content, lower in resistant species, or inhibiting conidial retention on the plant surface, e.g., high epicuticular wax deposits, which form a physical barrier in the form of a hydrophobic coating that reduces the deposition of aqueous seed and also reduces the rate of conidial germination and germ tube formation. *B. napus* (Tower, HNS-3), *B. carinata* (HC-2) and *B. alba* have more wax on the plant surface compared to *B. rapa* (BSH-1, YSPB-24) and *B. juncea* (RH-30). Two phytoalexins, namely camalexin and brassinin, and two isothiocyanates (ITC), namely allyl and benzyl ITC, were reported to have antifungal activity at different stages of development of Alternaria pathogens, namely A. brassicae and A. brassicicola of cruciferous plants [93].

It was found that wild cruciferous plants produce phytoalexins during inoculation. Activity of some compounds related to camalexin ($C_{11}H_8N_2S$) and 6-methoxycamalexin ($C_{12}H_{10}N_2SO$), turned out to be the most toxic for *A. brassicae*.

The phytotoxin destruxin B induces a phytoalexin response in *B. alba* [94]. reported that destruxin B is not a selective host toxin and does not induce the availability of host plants to *A. brassicae*. Resistance to A. brassicae is multilayered and multicomponent, with sensitivity to the host-specific toxin destructin B, quantitative and qualitative elimination of phytoalexins, hypersensitive response and Ca sequestration determining the fate of the host-pathogen interaction.

Resistant cruciferous cultivars also produce some metabolites, namely sesquiterpenes, deoxyuvedin B, albrasitriol, isoalbrasitriol and brassicadiol [95].

As resistance to Alternaria is regulated by additive or polygenes, breeding for resistance to these diseases may include pyramiding of minor genes, introgression of genes from material found to be resistant, reciprocal re-selection or diallel selective crossing, extensive hybridisation (*B. alba*), molecular selection (e.g. from *C. sativa* by somatic hybridisation; transgenic expressing the endochitinase gene of *Trichoderma harzianum*), pollen culture and destructin B sensitivity testing.

Although some studies on the mechanism of resistance to Alternaria have pointed to the influence of additive genes or polygenic or cluster genes, with resistance controlled by nuclear partial dominance genes, it has also been found that resistance components are highly correlated with each other in terms of slow disease development, and dominance plays a predominant role in the genetic control of the timing of resistance emergence. Additive × dominance prevails for other factors of disease progression, namely, the area under the disease progression curve [96].

Idiotypic (morphological) signs of resistance of cruciferous plants to Alternaria were also established. Thus, variations in the number and size of stomata in resistant and susceptible varieties were recorded – the number of stomata was maximum in the susceptible variety. Stomatal aperture was also narrower in resistant varieties compared to susceptible varieties (Table 4.6). Significantly lower number of stomata per unit area and smaller stomatal aperture are important morphological resistance factors in reducing infection of cruciferous genotypes *A. brassicae*.

The structure of wax in cruciferous plants and its role in pathogen resistance has been studied by a number of researchers [97–102]. In the Candle, Tobin and Altex rape varieties, the wax is organised into an amorphous layer overlying a crystalline layer. The crystalline layer consists of lamellar crystals and a layer of upright weeping and rod-shaped crystals (Figs. 4.3–4.5) [103].

Cruciferous wax is complex both structurally and chemically [105]. Cruciferous waxes have the same nine main components (alkanes, esters, ketones, aldehydes, secondary alcohols, ketones, primary alcohols, triterpenols and fatty acids), but in different proportions [106].

In *B. napus* ssp. *oleifera*, the main wax constituents are C 29 alkanes, C 29 ketone, C 29 secondary alcohol and C 40 -C 48 esters [107].

Table 4.6

Correlation coefficients (R) between different components of Alternaria resistance and yield of mustard (*B. juncea*) genotypes [104]

Disease components and yields	The size of the stain	Disease index	Leaf defoli-ation	Spore-bearing	AUDPC	Contami-nation	Productivity
Number of spots	0.883**	0.897**	0.812**	0.923**	0.956**	0.864**	–0.788*
Size of spots	–	0.985**	0.934**	0.982**	0.956**	0.893**	–0.668*
Disease index	–	–	0.905**	0.974**	0.949**	0.876**	–0.684*
Defoliation of leaves	–	–	–	0.952**	0.884**	0.743*	–0.625 NS
Sporulation	–	–	–	–	0.973**	0.851**	–0.679*
AUDPC	–	–	–	–	–	0.903**	–0.790*
Infection rate	–	–	–	–	–	–	–0.770*

* Significant at the 5% level; ** significant at the 1% level; ns – not significant.

In providing host resistance against Alternaria spp. the wax appears to be a physical barrier without any direct chemical effect.

The wax forms a hydrophobic coating and reduces the deposition of the aqueous seed. The wax also reduces the germination rate of the conidia and the number of germ tubes formed by each conidium. The crystalline wax layer is made fluffy by nested air pockets, which may be responsible for the above two effects by impeding the movement of plant exudates.

B. napus ssp. oleifera plants are very waxy compared to *B. rapa* ssp. *oleifera* plants, which are more susceptible to *A. brassicae*. Leaves of *B. napus* cultivars resistant to Alternaria have a significant amount of epicuticular wax [108]. According to [109–110], Alternaria-resistant genotypes have more wax on the leaves at different growth stages compared to susceptible genotypes. Gomez-Campo and Prakash (1999) [111] identified three different epicuticular wax columns in Brassica species with chromosome number (n) = 9: long columns, short columns and reticulate columns. *B. napus* and *B. carinata* inherited the reticulated wax type present in *B. oleracea*. It was observed that intraspecific crosses

between *B. napus* and *B. juncea* had a high content of epicuticular wax (Table 4.7, Figs. 4.4–4.5).

It is noted that mutation selection may be one of the possible methods of developing pathogen-resistant varieties in the absence of a useful donor of pathogen resistance in the existing germplasm of crops. Some *B. napus* plants resistant to *A. brassicicola* were regenerated from selected and unselected calli after mutation using gamma rays (physical) and ethyl methane sulfonate (chemical) mutagens [112].

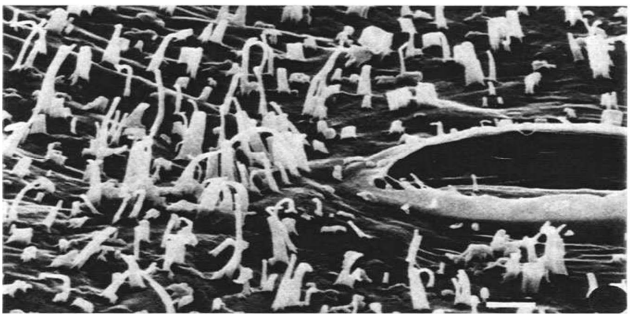

Figure 4.3 – Scanning electron micrograph of an air-dried, osmium vapour-fixed and gold-coated middle leaf of Brassica rapa, showing wax crystals (bar = 2 μm) [115]

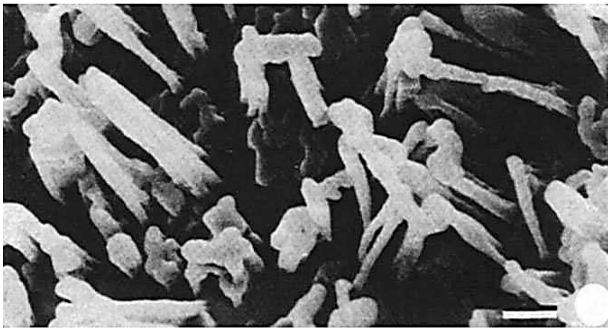

Figure 4.4 – Scanning electron micrograph of an air-dried, osmium vapour- and gold-coated Brassica rapa stem showing a gently sloping and vertical wax crystal (bar = 1 μm) [115]

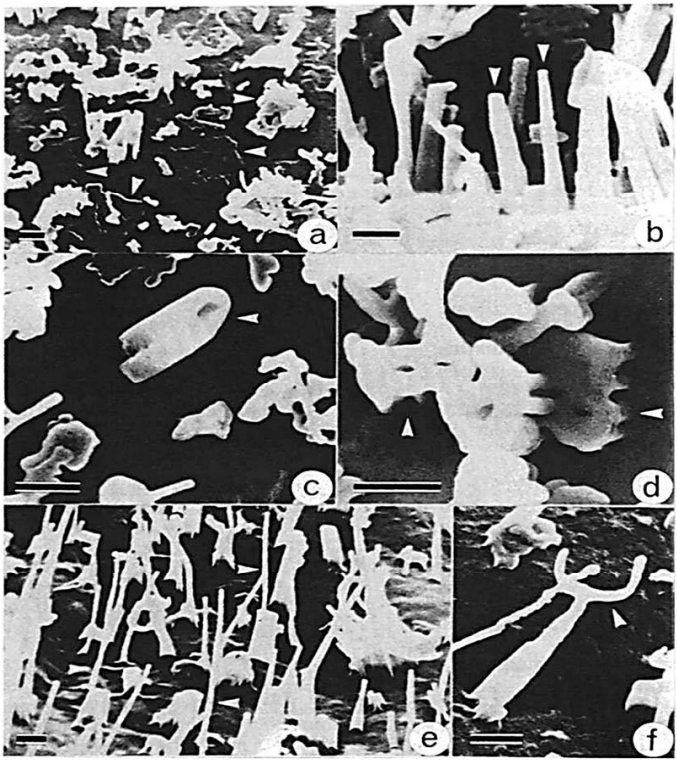

Figure 4.5 – (a) Adaxial surface of the upper leaf of Brassica napus cv. Altex, showing lamellar wax crystals (arrows); (b) stem surface of Altex from the middle of the plant, showing rods (arrows); (c) adaxial surface of the upper leaf of B. rapa cv. Tobin, showing fused rods (arrow); (d) adaxial surface of the upper leaf of *B. napus* cv. Westar, showing fused rods (arrows) and growth rings in wax crystals; (e) adaxial surface of the upper leaf showing small wax crystals (a); and (f) adaxial surface of the middle leaf showing a branched small wax crystal (arrow). The plant surfaces shown in Figures a-f were prepared for SEM by air drying (bar = 1 μm) [115]

Crop rotation is one of the easiest and most effective ways to control Alternaria in the field, as the main source of primary inoculum is infected plant residues in the soil. The minimum rotation duration is considered to be a three-year crop rotation with a non-cruciferous crop, which is necessary to avoid infection with a soil-borne pathogen [113–114].

For many producers, especially those with small acreages, it can be difficult to achieve a crop rotation in which host crops are adequately separated in both time and space due to the large number of host crops in the cruciferous family and their long overlapping growing seasons. In addition, studies of mortality of A. brassicicola and A. brassicae during overwintering have been conducted mainly in Europe, and it is possible that longer crop rotations may be required to break down lignin-rich stem tissue.

Cultural practices may also be important to minimise the secondary spread of the disease. Leaf and stem residues in the field containing infected plant parts can serve as a source of inoculum, as *A. brassicicola* and *A. brassicae* can continue to grow and reproduce as long as host tissue is present [116–117]. Therefore, it is important to manage crop residues in the piles, and cruciferous crops should be grown in fields that are as far away from these storage facilities as possible. Weed control is crucial in any system and can be an important component of an integrated approach to Alternaria management [118].

As mentioned earlier, fleas can transfer *A. brassicicola* spores from plant to plant within a field as well as from field to field, so controlling these insects can be an important part of an integrated disease management effort. Fleas are difficult to control and are a major limiting factor in organic production of vegetables and cabbage. They are controlled in these systems primarily by using floating covers to provide a physical barrier between the beetles and the plant leaves [119]. There are many insecticides available to conventional growers and those containing pyrethroids or carbamates that provide the best control, although fleas are still the main pest in cruciferous agrocenoses [120].

Biological control serves as the first line of defence against plant diseases in any production system, but in some cases it may be the only option available to reduce disease intensity in organic systems. Most biological control methods are aimed at reducing the amount of pathogen in the field, reducing plant-pathogen contact and creating an environment unfavourable

to disease development. Cultural practices that can be effective in reducing the damage caused by Alternaria leaf spot on cruciferous crops include the use of pathogen-free seeds, and maintaining crop rotations of at least three years, managing infected crop residues, reducing populations of quinoa weeds that can serve as alternative hosts, and controlling flea beetle (*Phyllotreta cruciferae*) populations as these insects can spread spores of the disease pathogen, *Alternaria brassicicola*.

Alternaria leaf blight spreads mainly through infected seeds and infected crop residues in the field. To prevent infection by soil-borne *A. brassicicola* spores, a three-year or longer crop rotation is recommended [121–122]. However, many farms, especially those with small areas, often find it difficult to maintain a crop rotation in which crops are adequately separated in time and space due to the large number of host crops of the Alternaria pathogen in the Brassicaceae family and their long growing season. Therefore, the use of mulch to create a physical barrier between the host plant tissue and overwintering *A. brassicicola* spores in the soil was investigated. Several mulch materials were tested to determine their effect on the growth of cruciferous crops from the perspective of alternaria infection. The management of Alternaria in organic systems depends on preventing the introduction of the pathogen into the field through infected seeds, as well as preventing infection of the crop by overwintering spores transported from the soil. Therefore, the hypothesis that mulch can reduce the incidence of Alternaria by creating a physical barrier between infected soil and susceptible plant tissues was tested. The effects of low-density film, Biotelo biodegradable plastic film and wheat straw on disease incidence and plant growth were measured and compared to the control without mulch [123].

All three mulches tested reduced the incidence of Alternaria, but only the straw mulch significantly reduced the incidence compared to the control without mulch. There may be several reasons for the differences in the effectiveness of the three mulch materials, but soil coverage at the base of the plants, leaf mulch breakdown during the growing season and surface moisture are likely to play an important role. In this regard, the physical barrier created by the straw mulch was probably much more complete than that of the plastic sheet mulch. In addition, sheet mulch can be torn or ripped by the user, animals or even the wind blowing over the field, creating more

exposed soil surface. This is especially true in the case of Biotelo mulch, which is quite fragile and was indeed torn during the mulch decomposition process and had many large tears by the end of the experiment. Incomplete soil coverage would likely have reduced the effectiveness of the mulch as a physical barrier to soil seed, and would have increased weed pressure, which would then have to be handled manually, which can be a time-consuming and therefore expensive task.

Another possible explanation for the difference in effectiveness between leaf and straw mulch is related to the moisture content of the soil surface. A. brassicicola spores can be spread across the field by wind, rain and rain splashes. Although conditions remained hot and dry for most of the growing season, temperatures dropped in September and October and rains became more frequent and heavy. During these wet periods, puddles of water could be seen on the surface of the LDPE mulch and Biotelo sheet mulch, while rainwater could seep through the straw mulch and no standing water accumulated on the soil surface. These puddles could have caused spores to be dispersed by spray or provided the free moisture and leaf moisture (sometimes low-hanging cabbage leaves were observed hanging in these puddles) needed for spores to germinate, cause infection and sporulation. It is possible that if a mechanical plastic layer had been used under the film to form raised beds, better soil-mulch contact would have been achieved (fewer air pockets that later became flooded areas) and water could have rolled off the film and been diverted away from the host crop. This was not done because the experimental plot was so small that randomisation of the mulch treatment would have been difficult to achieve with the tractor-mounted pile mulch spreading equipment.

Mulch can also serve as a soil barrier for the insect pest, the cruciferous flea beetle (*Phyllotreta cruciferae*). Adult flea beetles feed on the green tissue of host plants during the day and burrow under the soil at night [124]. Adult females lay their eggs in moist soil at the base of host plants, and when larvae emerge, they live in the soil where they feed on the roots of host plants until they pupate and become adults in about two to three weeks [125]. Although flea damage to plants was not measured in this study, plants in the control treatments had significantly more flea damage in the form of "through holes" than plants in the mulched treatments. Fleas may also be an important factor influencing the intensity of Alternaria outbreaks, as

these insects are able to physically carry A. brassicicola spores on their bodies and in their faeces, spreading the pathogen from plant to plant and field to field [126]. Thus, if mulch can reduce flea numbers in crops, it may also reduce the severity of Alternaria damage. There is also reason to believe that there may be differences in flea behaviour depending on the type of mulch, as it has been shown that *Phyllotreta pusilla* (western black flea) populations are affected by the colour and reflectivity of mulch [127]. Thus, this study demonstrated that mulch has the potential to reduce the incidence of Alternaria, possibly by reducing the initial infection of plants with overwintering *A. brassicicola* spores. Mulch also increased plant growth, and in a hot and dry year, straw mulch provided the greatest benefit to yield growth.

It is known that organic matter application causes changes in the density and composition of microbial communities, and thus can affect microbiologically mediated plant defence responses [128–131]. The biochar is the product of the thermal degradation of organic materials in the absence of air, a process known as pyrolysis, and is being studied extensively worldwide for its potential as an additive to soil organic matter to improve soil fertility, water and nutrient retention, and to sequester carbon and mitigate climate change. Although the biochar has been widely studied in agronomic settings, little is known about its impact on plant diseases. Several recent studies have reported that biosolids have been successfully used to reduce disease incidence in controlled environments and microplots, and while several mechanisms are likely responsible for the observed disease suppression, induced systemic resistance has been documented in several pathosystems [132–136]. Greenhouse study by Elad et al. in 2010 [137], showed a significant reduction in the intensity of the development of diseases caused by the pathogens Botrytis cinerea grey rot and powdery mildew Leveillula taurica on peppers and tomatoes when applying a biochar from citrus wood in concentrations of 3 and 5% to coconut fibre.

In a similar greenhouse study conducted by Harel et al. (2012) [138], A significant reduction in the severity of diseases caused by three strawberry leaf diseases (*Botrytis cinerea*, *Colletotrichum acutatum* and *Podosphaera apahanis*) was found when plants were grown in a medium with 3% (w/w) biochar. In addition, such "biochar", which is produced by burning plant or animal materials, is approved by the Organic Materials Review Institute and

is listed as a certified organic product for use as a soil fertiliser. Therefore, we investigated [139] The impact of biofuels on the severity of leaf spot by Alternaria. Biochar produced from lodgepole pine chips was used in greenhouse and field studies to investigate the sustainable management of Alternaria leaf spot in cruciferous vegetable crops. A detailed chemical analysis was carried out to determine the elemental composition and relevant physical and biochemical parameters of the biochar used in the greenhouse and field studies. The results showed a significant reduction in the prevalence of Alternaria and the degree of damage even under favourable conditions of pathogen development.

Treatment of seeds with hot water reduces the growth of Alternaria. However, the spores of these fungi can survive on leaf tissue for 8–12 weeks, and on stem tissue – up to 23 weeks. Therefore, in fields that are reseeded shortly after harvest, a large amount of seed is often available for rape-mustard disease, which is likely to affect the emergence of crop seedlings and early stages of growth. Thus, crop rotation with non-cruciferous crops and the eradication of cruciferous weeds can help control these pathogens.

Early sowing of well-preserved, clean, certified seeds after deep ploughing with 45 cm row spacing, deep tillage, timely weeding and maintaining an optimal plant population, avoiding irrigation during flowering and pod formation stages can help control the disease [140]. Sowing of seeds should not be carried out by the method of spreading, as this increases the intensity of the disease, which can be reduced on leaves and pods by applying 40 kg K/ha together with the recommended dose of nitrogen (N) or sulphur (40 kg/ha), or together with the recommended dose of NPK [141]. However, higher doses of N make crops more susceptible to disease. Soil application of K as the main fertiliser has been found to control mustard Alternaria disease. Sulphur, zinc and boron have been reported to reduce Alternaria development and increase mustard seed yields. The use of some trace elements, viz: B at a concentration of 1 g/l, Mo at a concentration of 1 g/l, S at a concentration of 2 g/l and Zn at a concentration of 2 g/l, in various combinations reduced Alternaria development and increased the yield of rapeseed and mustard.

Inorganic fertilisers, namely phosphorus (P) and potassium (K), also reduce the incidence, while higher rates of nitrogen increase it [142]. This is important at a time when growers are reporting an increase in the prevalence

of Alternaria and a decrease in control. The GR isolate of Trichoderma viride was on a par with manococcus in terms of efficacy when testing the intensity of damage to mustard leaves and pods. The conidial suspension of T. viride was more effective than the culture filtrate in reducing the intensity of the disease on leaves and pods [143].

The strain of *Bacillus subtilis* UK-9 isolated from reclaimed soil caused morphological changes in vegetative cells and spores by destroying and lysing the pathogen cell wall, which led to a decrease in the intensity of disease development and spore germination on leaves [144].

Treatment of seeds with bioagents led to an increase in the content of lipids (phospholipids, glycolipids and sterols) and proteins in the seeds of treated plants. However, seed treatment and foliar spraying of white mustard leaves with bioagents increased the content of dry matter, total phenol, orthohydroxyphenols, starch, total soluble sugars, reducing sugars total lipids and various membrane lipids in the leaves, but the total protein content decreased after treatment with biocontrol agents at 30 and 60 days after germination, which may be due to protective mechanisms and enhanced plant growth [145]. Extracts of several plants were evaluated against *A. brassicae* [146]. The level of effectiveness of *Azadirachta indica* extract increases with the number of sprayings [147]. Spraying of garlic bulbs and neem leaf extract at the flowering stage suppressed disease incidence and increased mustard yield [148]. Foliar spraying with *Calotropis procera* leaf extract, *A. indica* kernel and *A. sativum* bulbs can induce resistance against *A. brassicae* by increasing the content of soluble phenol, sugar and soluble proteins, namely polyphenol oxidase in mustard leaves [149]. Among the several essential oils evaluated, peppermint oil (*Mentha piperita*) provided complete inhibition of fungal growth at 2000 µg/ml, followed by cyperus oil (*Cyperus scariosus*).

Essential oil from radish roots (at a concentration of 1:2500) inhibits *A brassicae* [150]. Deproteinised leaf extracts *Acacia nilotica*, *Enicostema hyssopifolium*, *Mimosa hamata* and *Vitis vinifera* showed fungistatic activity against *A. brassicicola* [151]. Extracts obtained from the leaves *Lawsonia alba*, root of *Datura stramonium* and *Mentha piperita* inflorescences, have fungitoxic activity against *A brassicae* isolated from cabbage leaves. Extracts of the ferns *Adiantum caudatum*, *Diplazium esculentum* and *Pteris vittata* reduce the growth and germination of *A. brassicicola* [152–153].

The plant extracts of roots, leaves, stems, inflorescences and fruits of several species (Table 4.7) showed fungicidal activity in vitro and in vivo against the two main Alternaria species, namely *A. brassicae* and *A. brassicicola*, which cause Alternaria or black spot of cruciferous vegetables. In vitro, almost all plant extracts limit mycelial growth, sporulation and conidial germination. Some have shown efficacy in the field, limiting infection points, spot size and disease intensity.

Table 4.7

Plant extracts used against A lternaria spp. on cruciferous plants [154]

Plant species	Concentration (%)	Sources of the study
Acacia nilotica	–	[154]
Encostema hyssopifolium		
Mimosa hamata	–	
Vitis vinifera	–	
Datura stramonium	–	
Lawsonia alba	–	[155]
Mentha piperita	_	
Adiantum caudatum	–	[156]
Diplazium esculentum	–	
Pteris vittata	–	
Allium sativum	5	[154]
Azadirachta indica	10	[157]
Lawsonia inermis	5	[158]
Erythrina chiaposana	5	
Ricinus communis	5	
Zingiber offi cinale	5	
Euphobia pulchrima	5	[154]
Rumax dentatus	5	
Urtica dioica	5	
Eucalyptus globules	5	
Ocimum sanctum	5	[159]
Anagallis arvensis	5	
Solanum nigrum	5	
Rhus chinensis	–	[160]
Polygonum perfoliatum	1	
Agave americana	1	[161]
Solanum xanthocarpum	1	[162]
Lavandula pubescens, Calotropis procera	10	[163–164]

Eucalyptus spray gave significantly fewer spots per leaf (2.05), minimum spot size (1.28 mm), minimum spore intensity (1.22 x 105) and minimum disease index, followed by calotropis, ocimum and polyanthea extracts at 5% [165].

Mustard Alternaria can be controlled by spraying aqueous extracts of *Azadirachta indica*, *Allium sativum* and *Zingiber officinale* at concentrations of 5, 10, 15 and 20 %, which leads to higher yields. However, spraying with 15% *A. indica* extract gave the highest yield with the best cost-benefit ratio [166].

While, according to the [167–168], the use of *Allium sativum* onion extract in concentrations of 45 and 75 p.p. gave the highest yield of cruciferous seeds. *Allium sativum* extract is also the most effective in the fight against mustard fungi, which are transmitted with seeds [169].

Saprophytic phylloplanktonic fungi such as *Aureobasidium pullulans* and *Epicoccum nigru*m are pathogenic to *A. brassicicola*.

Verticillium state of Nectria inventa Pethybridge, a destructive myco-parasite, is one of the dominant phylloplane fungi of rapeseed seeds [170].

Among the leaf surface mycorrhizal fungi, the most antagonistic are *E. purpurascens*, *A. pullulans* and *Cladosporium cladosporioides* in the case of *A. brassicae*. The metabolites of Acremonium roseogriseum, *Aspergillus terreus* and *C. cladosporioides* inhibit *A. brassicae*. The most significant effects are observed when spraying with spores of surface fungi and their metabolites before inoculation of the pathogen on the leaves [171].

Pre-application of Streptomyces rochei spore suspension or its diffusate leads to a significant decrease in the intensity of *A. brassicae* and *A. brassicicola* leaf damage on *B. rapa* [172–173].

Jayant and Sinha (1981) reported that *S. hygroscopicus* is a strong antagonist of *A. brassicae* and *A. brassicicola*. When spraying the culture filtrate a week before or a week after spraying with a suspension of *A. brassicae* and *A. brassicicola* spores, germination and development of diseases are reduced.

The pigmented and xylose-utilising strain of *S. bobili* was found to be active against *A. brassicae*, *A. brassicicola* and *A. raphani* [174].

An isolate of Streptomyces spp. obtained from light *Finnish sphagnum* garden peat proved to be an effective biological agent for controlling plant pathogens [175].

Treatment of cauliflower seeds with isolates of *Trichoderma viride* and Streptomyces spp. inhibits or reduces desiccation caused by *A. brassicicola* [176–177].

Seed treatment with mycostop, a powdered preparation made from streptomycete spores and mycelium, was successful in controlling the drying out (80–90%) of seeds artificially infected with *A. brassicicola*. The seed treatment remains effective on seeds stored in dry conditions for 5-6 weeks, but its effectiveness slowly decreases thereafter. Streptomycetes treatment controls, as well as chemical thiram treatment, the prevention of desiccation caused by Alternaria fungi in seedlings grown from commercial seed lots of different origin [178].

It is reported [179], that Patostop, a biofungicide based on a selected isolate of S. griseoviridis from Finnish sphagnum peat, applied either by seed or soil treatment, controls *A. brassicae*.

Cruciferous seeds treated with Gliocladium virens-19, Trichoderma harzianum-22, T. harzianum-50, Penicillium corylophilum-36 and P oxalicum-76 gave a significantly higher yield of healthy seedlings. The hyphae of antagonist fungi were able to adhere to conidia or wrap around or penetrate germ tubes or hyphae.

It was found that *A. brassicicola* conidia shrivelled and plasmolysed in the presence of antagonistic fungi [180]. For the biological control of *A. raphani* and *A. brassicicola* of radish, which are transmitted by seeds, antagonists such as *Chaetomium globosum*, *T. harzianum*, *T. koningii* and Fusarium spp [181].

Wu i Lu (1984) Trichoderma, Gliocladium and Penicillium spp. were found to be parasitic on *A. brassicicola*. The use of *A. alternata* before inoculation with A. brassicae reduced the level of A. brassicae on rape by about 60 %, and after inoculation – by about 26% [182].

Spraying with *T. viride* conidial susceptibility inoculum reduces the damage to mustard by Alternaria on leaves (76%) and pods (68%), respectively. Bioagents survive on the leaves for up to 30 days at a relative humidity of 80–90% and a temperature of 20–35 °C [183]. У Польщі повідомлено про *Gonatobotrys simplex* як гіперпаразита *A. brassicae*.

Excellent control of *A. brassicae* in white cabbage and cauliflower seeds was obtained in Danish experiments by immersing them in water heated to 45 °C for 30 minutes, or for 20 minutes at 50 °C, or even 30 minutes at

40 °C. This treatment improves germination by up to 13% and also kills other fungi such as Penicillium and Mucor spp [184]. Treating seeds with hot water at 50 °C for 30 minutes controls cabbage diseases [185].

According to [186] treatment with hot water for 25 minutes at 50°C eliminates Alternaria infection from legume seeds. Treatment of seeds at 50 °C for 20 minutes is highly effective in controlling seed-borne fungi, including *A. brassicae* (B. juncea), without any significant effect on seed germination [187].

One of the most effective measures to control the disease caused by Alternaria is the effective use of fungicides.

Seed treatment is an effective measure in the control of Alternaria because it helps to reduce the number of primary inocula. Seed treatment with hot water at 50°C for 30 minutes to control Alternaria was recommended by Walker, while Ellis recommended the same temperature for 25 minutes to eliminate Alternaria infection from cabbage seeds. Seed treatment with thiram plus kaptan (1:1) 0,3% and four sprays with zineb (0,25%) proved to be quite effective in controlling the disease [188].

Chemical plant protection is known to be an integral part of integrated crop protection systems against pests. Winter rape seeds are a source of infection for many pathogens, including Alternaria and Fusarium, so seed treatment is an important element of preparing them for sowing. The treatment helps protect seeds and seedlings from damage in the early stages of plant ontogeny, which is the basis for healthy and friendly seedlings. The results of the phytosanitary examination of winter rape seeds indicate that the annual infection rate of fungi from the Alternaria genus ranges from 3 to 100% and Fusarium from 1.0 to 26.0%.

Infected seeds are the other main source of primary seed, and therefore the use of clean seeds and transplants is important to prevent the introduction of *A. brassicicola* and *A. brassicae* into clean fields. Treatment with hot water for 18 minutes at 50 °C resulted in a 98% reduction in disease incidence, but this method may be less effective in the presence of significant internal infection and seeds may lose viability. Hot water treatment is the only method currently available for seed disinfection in organic crop production systems.

In conventional systems, seeds can be treated with surface-active fungicides, but the best control is achieved with the locally applied fungicide

iprodione, which can also be absorbed by seed tissue to levels sufficient to kill established cotyledon infections, although further movement or toxicity is limited. Iprodione has been used for seed treatment since at least the 1970s and continues to be the main tool for controlling Alternaria worldwide [189]. Some biological control methods are known, especially in the form of seed inoculation, as discussed earlier. The effectiveness of several microbes isolated from broccoli seeds or from the rhizosphere of broccoli seedlings in reducing the incidence of seed blight has been investigated. The authors found that *Gliocladium roseum* and *Trichoderma harzianum* were effective in reducing seed infection, increasing germination and reducing leaf infection by *A. brassicicola*. An early study on the treatment of cabbage seeds infected with *A. brassicicola* with seed-borne antagonists showed that Periconia sp., Pencillium sp. and *Chaetomium globosum* strain Kunze increased the emergence of healthy seedlings equivalent to iprodione [190].

Although there is a great deal of interest in biocontrol during the seed stage of A. brassicicola and A. brassicae, very little is known about the effect of biocontrol organisms on the developmental stage of the pathogen on leaves. One study demonstrated the potential protection of rapeseed (*B. napus*) leaves against *A. brassicae* by seed treatment with *Bacillus amyloliquefaciens*, indicating that the induction of plant defence responses [191].

Induction of systemic defence reactions of plants by Trichoderma spp. was associated with the protection of tomatoes from *Alternaria solani*, which causes early blight of this crop [192], and may be useful for *A. brassicicola* research.

There are a number of biological control agents that are registered for foliar use in organic production and listed as control agents, but the effectiveness of these products in reducing disease intensity is not known and this area needs more attention in the future.

The strong cross incompatibility, polygenic background of resistance (additive and dominant gene interactions), and differences in ploidy between cruciferous species of interest make it difficult to transfer resistance to Alternaria from wild species to cultivars. In addition, this often involves the use of in vitro hybridisation techniques, including somatic hybridisation, embryo and ovary preservation or protoplast fusion of cruciferous species,

as the transgenic approach has been unsuccessful. Thanks to our growing understanding of pathogen-host interactions, identification of sources of resistance and evaluation of resistance trait inheritance, cruciferous crop breeding programmes for alternaria resistance mode of inheritance can be improved.

This is of particular importance, as in recent years there has been a dynamic development of ecological and integrated crop production with an emphasis on plant resistance to biotic stresses. highly resistant genetic resources have been reported among cultivated cruciferous species, although some varieties differ in terms of resistance and susceptibility.

This means a significant economic impact of the disease: In 2020, Polish production of cabbage and other cabbage crops ranked 7th, and cauliflower and broccoli 8th in the world (1141200 tonnes and 252325 tonnes, respectively) with a net production value of USD 171 million and USD 60,5 million, respectively (FAOSTAT: http://faostat.fao.org). Methods of preventing and controlling Alternaria include a combination of good agricultural practices and chemical protection. An important method of disease prevention is the production of healthy seeds obtained from crops with intensive fungicide protection. In a 2-year period of cruciferous seed production, fungicides containing iprodione as an active ingredient had a good effect of protection against Alternaria infections during the 1st year of growth [193]. In Poland, the only product containing it is the T 75DS WS Seed Treatment (Zaprawa Nasienna). As both pathogens survive on crop residues, seeds and in association with weeds[752], Crop residue management (e.g. through crop rotation and deep tillage), as well as the use of clean seeds and good weed control should reduce the disease. Once symptoms appear, infection can be limited by repeated spraying with fungicides containing strobilurins as active ingredients (Amistar 250 SC, Signum 33 WG, Zato 50 WG) and iprodione-based fungicides (Rovral FLO 255 SC) [194]. This method, however, is not economically viable and may not be effective under favourable weather conditions, especially on seed crops that favour the spread of pathogenic infection. An alternative method of protection is the use of antagonist fungi; the deployment of *Aureobasidium pullulans* and Epicoccum nigrum on cruciferous leaves reduced infection rates under controlled conditions [195].

It is noted that chemical control of Alternaria on seeds and in the field remains the most common method of controlling this disease worldwide.

Chlorothalonil, a broad-spectrum fungicide with multiple sites of action, has also been widely used as a foliar spray to control Alternaria [196], and there are several new fungicides that have become widely used. The most important of these new fungicides is azoxystrobin, a quinone external inhibitor with a very specific mode of action that exerts very high selection pressure on resistant isolates in pathogen populations.

At the same time, resistance of *A. brassicicola* to several major fungicides has been reported elsewhere and in other plant pathogenic Alternaria spp [197-198]. Field isolates of *A. brassicicola* with resistance to iprodione, the most widely used chemical for controlling late blight, were first identified in the mid-2000s. Cross-resistance to both dicarboximides (including iprodione) and phenylpyrrols was also present in these populations. Resistance to azoxystrobin has not been demonstrated for A. brassicicola or A. brassicae, but its development in other Alternaria species (as well as in many other plant pathogenic fungi) indicates that resistance management procedures should be carefully followed.

Research into the biological control of seed infection includes the use of antagonistic fungi and bacteria, most commonly *Nectria inventa* spp., *Trichoderma* spp., *Gliocladium* spp., *Penicillium* spp., *Periconia* spp., *Chaetomium globosum* i *Streptomyces griseoviridis* [199–200]. Although many of these biological control agents have been shown to be capable of controlling disease incidence, none of those studied to date have been as effective in controlling internal seed infections as fungicides. Many biological control antagonists also have less protective effect in the field due to narrow growth requirements or weak competition in the rhizosphere, and as a result, fungicides are still the most widely used control method worldwide.

Some studies have found that [201–202], that pre-sowing treatment of seeds with Protect, CS (3,5 l/t); Kruiser Rapeseed, SC (11.0 and 15.0 l/t); Modesto Plus, CS, (16.0 l/t) and Agrovital Plus, CS, (5.0 l/t) contributed to a reduction in seed infection by 84,5–100%, an increase in laboratory germination by 15.2–19.3%, field germination by 6,2–7,6% and inhibition of Alternaria development on leaves by 35.0–82.0%. On pods, the effect of the treatments on reducing the development of Alternaria ranged from 3.3 to 36.6%.

During the years of research [203] Kruiser Rapeseed, SC at a consumption rate of 1,5 l/t provided high laboratory germination rate of

92.0–99.0% and field germination rate of 72.7–75.0% compared to other treatments. Also in this variant, the effect of restraining the development of Alternaria on pods was noted – 14.7–36.6 %, which resulted in saving 3.3–6.2 centner/ha of yield. In general, seed treatment made it possible to save from 1.3 to 8.2 centner centner/ha, depending on the preparation and the year of research.

Studies have shown that the duration of the protective effect of seed treatment in inhibiting the development of Alternaria is limited. Therefore, to protect the crop in the second half of the growing season, fungicides are required. Out of 43 fungicides approved in different countries for use on winter rape, 32 products, or 74.0%, are 2-component.

The choice of preparations is determined by the active substances most often used to protect rapeseed from diseases – Mirador Forte, CE (azoxystrobin, 60 g/l + tebuconazole, 100 g/l) – 2,0 l/t and Custodia, KS (azoxystrobin, 120 g/l + tebuconazole, 200 g/l) – 1,2 l/t.

It is noted that long-term studies of the dynamics of the disease development indicate a variation in the degree of leaf damage within 1.2–16.2%, which led to the preservation and accumulation of infection to affect the pods. The surveys showed that the development of Alternaria on pods increases with the degree of leaf damage. It has been established that repeated use of the same products can lead to a decrease in the effectiveness of fungicides due to the emergence of highly resistant forms of pathogens in the population.

In the absence of resistant varieties, fungicides are the most reliable means of disease control [204]. In order to achieve economic yields and acceptable quality on infected crops, multiple applications of fungicides are required. Thus, Khan et al. in his research, he used the systemic fungicides Thiophanate methyl, Ridomil MZ (Mancozeb, 64% + Metalaxyl, 8%) and Carbendazim separately and in combination with four non-systemic fungicides Kaptan, Mancozeb, Zineb and Tiram at a rate of 0.2% a.i. in the field. Ridomil MZ was the most effective, followed by the combination of Carbendazim + Kaptan.

Reported [205], that three consecutive sprays of Mancozeb resulted in maximum control of leaf blight intensity, followed by two consecutive sprays of Mancozeb (0.2%) and the third spray of Rodomil MZ (0.25%). Foliar spraying with Mancozeb proved to be the most effective in controlling the disease [206–207].

It is effective to supplement fungicides with plant extracts to control Alternaria. The use of various plant extracts and natural products is encouraged as they do not pose a health hazard and do not pollute the environment. Extracts *Canna indica, Convolvulus arvensis, Ipomoea palmata, Cenchrus catharticus, Mentha piperita, Prosopsis spicigera, Allium cepa, A. sativum, Lawsonia inermis, Argemone mexicana, Datura stramonium* і *Clerodendron inerme* completely suppressed the germination of *A. brassicae* spores isolated from cauliflower leaves [208].

The use of eucalyptus leaf extracts significantly reduced the number of leaf spots, the minimum spot size, the minimum disease index and the highest yield, followed by spraying with calotropis, ocimum and polyanthesis extracts [209]. Foliar spraying with aqueous extracts of *Allium sativum* (garlic) and Eucalyptus globulus (eucalyptus) has been reported to be effective in controlling Alternaria on leaves and pods and may be an environmentally safe substitute for the chemical fungicide mancozeb in the control of mustard diseases [210].

Some research results indicate the possibility of biological control of Alternaria. Foliar application of *T. harzianum* and *P. fluorescens* isolates proved to be effective in controlling Alternaria [211].

Resistance of susceptible mustard cultivar PR-15 to highly and moderately virulent isolates of *A. brassicae* was induced using an avirulent isolate of A. brassicae. The induction of resistance by an avirulent isolate against a highly virulent and moderately virulent isolate of *A. brassicae* resulted in a significant reduction in disease severity.

Due to increased awareness of the risks associated with fungicide use, considerable attention is being paid to an integrated approach to pathogen management. Burning the previous year's crop residues, timely sowing, using healthy certified seeds, timely weeding, using balanced doses of nutrients, maintaining optimal plant numbers, and avoiding irrigation at sensitive stages of crop development (45 and 75 days after germination) can help minimise the incidence. Potassium application at a dose of 40 kg/ha [212–213], as well as the addition of minerals such as sulphur, borax, potassium and zinc to the soil have proven to be effective in the fight against mustard Alternaria. These minerals were found to increase plant resistance.

The disease was also found to develop at a minimum degree at a row spacing of 45 cm compared to the spread seeding method and in early crops with low weed levels.

Spraying with iprodione was effective in controlling the infection on pods caused by A. brassicae. Both a reduction in disease incidence and an increase in seed yield and weight were observed with the use of iprodione [214], and its residues in edible parts of plants were lower than the maximum residue level, which indicated the safety of this fungicide at the recommended rate. A higher number (3–4) of sprays with iprodione had a significant reduction effect on the number of spots on siliques. Currently, there is a need for new fungicide molecules to control this pathogen, given their fungicide resistance (Table 2.24). Mycelial growth, conidial germination and germ tube elongation revealed the existence of A. brassicicola isolates with high resistance (EC_{50} > 100 mg/l) to both dicarboxymides (e.g. iprodione and procyamidone) and phenylpyrroles (e.g. fludioxonil).

The use of fungicides on seeds reduced the content of two Alternaria toxins, namely Alternaria and Alternaria methyl ester [215]. Two consecutive foliar sprays with Mancozeb 75 WP (0.2%) followed by spraying with metalaxyl + Mancozeb (Ridomil MZ 72: 0.25%) provided high seed yield and 1000 seed weight [216].

Tiram (75%) was the most effective fungicide at 5000 ppm, while complete suppression of Alternaria was observed at 10.000 ppm in the case of Tiram (TMTD 80%) and Arasan 50% [217]. All fungicides showed significant efficacy in reducing disease severity and increasing seed yields. The time of the first spray, interval and number of sprays depended on the type of crop.

On *B. rapa* and *B. juncea* crops in India, significant disease control was achieved if the first spray was applied 60–75 days after sowing; 2–4 more sprays at 10–15 days intervals, depending on crop maturity, increased control [218–220].

It should also be noted that the literature contains information on the low fungicidal effect of azoxystrobin against fungi of the genus Alternaria, on the other hand, high efficiency is noted when it is used in field experiments. In this regard, studies were conducted to investigate the population structure of Alternaria spp. fungi isolated from leaves and pods of winter rape plants. The analyses showed that 32.8% of the fungal isolates on the leaves were *A. brassicicola*, 35.9% – *A. tenuissima*, 9,4% – *A. alternata*, 21.9% –

A. arborescens, and the share of *A. brassicicola* on the affected pods was 25,0%, *A. tenuissima* – 50.0%, *A. alternata* – 0.0% and *A. arborescens* – 25.0%.

To determine the susceptibility of Alternaria spp. fungi, a drug screening was performed. The fungi *A. alternata, A. tenuissima, A. brassicicola, A. arborescens* were included as test objects. It was found that [221], that the Alternaria spp. fungal population is heterogeneous in terms of susceptibility of isolates to fungicide active ingredients when evaluated in vitro. High susceptibility was observed to tebucanazole (EC_{50} less than 10 μg/ml) in *A. alternata, A. tenuissima* and *A. brassicicola*, and to azoxystrobin in *A. arborescens* and *A. brassicicola*. In the populations of these fungi, there are also isolates with medium sensitivity (EC_{50} from 10,1 to 40 μg/ml) and resistant, 60.0% of isolates of *A. alternata*, 10.0% of *A. tenuissima* and 20.0% of *A. brassicicola* to azoxysitrobin, and 10.0% of *A. arborescens* to tebucanazole. Therefore, in the case of dominance of *A. brassicicola* fungus on the leaves, the pathogenicity of which is 100%, the effectiveness of fungicides can be high, since 80.0% of isolates are sensitive to azoxystrobin and 90.0% to tebucanazole.

In the years of moderate and depressed development of Alternaria in field experiments [222] the effectiveness of Custodia, KS is higher in protecting the leaf than Mirador Forte, CE, while the effectiveness of pods is higher than Mirador Forte, CE. During the years of epiphytic development of the disease on leaves and pods, the effectiveness of the preparations is relatively the same, but in the protection of the leaf apparatus is higher – 83.7 and 80.8%, than in the protection of pods – 46.7–38.9%, which is due not only to the timing of the use of fungicides against Alternaria, but also to the structure of the fungi of the pathogenic complex. Fungicides were applied at the threshold of disease development, in these variants, 1.8–6.2 centner/ha were saved. Evaluation of the effectiveness of fungicides against Alternaria has been studied quite intensively and is still being studied in Ukraine. Thus, studies to determine the effectiveness of the use of disinfectants and fungicides for spraying during the growing season were conducted at the Agronomic Research Station of NUBIP of Ukraine, the Research Centre 'Experimental Field' of Lviv National Agrarian University and the experimental field of Bila Tserkva National Agrarian University [223] (Table 4.9–4.11).

The range of oilseed rape protectants includes 12 products, of which 6 are two-component, 5 are three-component and 1 is one-component.

The effectiveness of Cruiser OSR 322 FS t.c.s. (15 l/t), TMTD c.c.s. (5 kg/t), Vitavax 200 FF c.c.s. (Z kg/t), Maxim XL 035 FS t.c.s. (5 l/t) and the biological preparation Phytodoctor – Bacillus subtilis bacterium (2 g/kg) was studied on the Siesta F1 hybrid. All the studied preparations reduced the spread and development of Alternaria compared to the control variant (without treatment) (Table 4.9), but did not affect the yield of the variants.

The effectiveness of the use of fungicides Tilmor c.e. (0.6 l/ha), Faraday v.g. (0.5 kg/ha), and Pictor c.s. (0.5 l/ha) alone and with pre-treatment with the growth-regulating preparation Karamba v.r. (1 l/ha) was investigated. The survey carried out in the phase of economic ripeness of rapeseed showed that the spread of the disease in the variant without the use of fungicides in the conditions of Lviv region reached 90.3%, its development – 54.3%. Spraying the plants with the studied preparations made it possible to reduce these indicators to 45.7–50.3% of the disease spread and 10,6–21,6% of the disease development and provided an increase in the yield of fungicide-treated varieties from 0.27 to 0.42 t/ha.

Karamba is both a fungicide and a rapeseed growth regulator. Its regulatory effect is to strengthen the root system, increase branching, uniformity of flowering, stronger and shorter stems. In production, it is recommended to use the following spring rape protection system, which involves treatment with Karamba at a plant height of 20–25 cm and with Pictor fungicide during flowering.

This treatment scheme provided the most effective protection of plants – the development of the disease was 10.6% with 45.7% prevalence. Treatment with Pictor without preliminary application of Karamba was less effective – the development of Alternaria reached 12.7%, and the spread was 48.0%. The ability of plants to resist alternaria after spraying with Tilmore and Faraday was slightly lower compared to Karamba and Pictor, and significantly higher compared to the control variant (Table 4.11).

Based on the research, the authors concluded that varieties with high resistance to Alternaria can reduce the number of fungicide treatments, which helps to obtain environmentally friendly products. These varieties can be used in further breeding work as donors of resistance to Alternaria pathogen. At the same time, the use of plant protection products and growth regulators significantly reduces yield losses in the face of high infection rates and favourable conditions for the disease.

Table 4.8

Chemicals and fungicides tested against Alternaria species on cruciferous plant species [224]

Acetone	Phenyl acetate	
Actidion	Ferbam	Silite
Agrosan	Fermat	Tebuconazole
Alar	Folicur (tebuconazole)	Tetrahydropyrimidine
Anthracol	Folpet	Thiabendazole
Arasan	Flutriafol	Thiophanate-methyl
Azoxystrobin (Amistar)	Formaldehyde	Tiovit
Azadirachtin (Nimarin)	Granosan	Tiram
Baffin	Guazatin	Tilex
BAS 480 F	Halogen derivatives	Topsin M
Baykor	Imazalil	Triademephon
Bayleton	Indophyll M-45	Triapentanol
Bavistin	Indophyll Z-78	Triarymol
Benlate	Iprodion (Rovral)	Tri-basic copper sulphate
Benz (1,2) isoxadoles	White urea	Trisonic copper salts
Biokvin	Kavach	Tridemorph
Blitox	Lunasan	Trifloxystrobin
Bordeaux liquid	Malic acid	Trimiltox forte
Boric acid	Mancoceb	Vinclosoline (Ronilan)
Boscalid	Maneb	Water-soluble sulphur
Brassicol	Manzate	Zato 50 WG
Brestan	Merpan	Zincop
Bromosan	Metalaxyl	Cineb
Calixin	Metiram	Zinc sulphate
Kaptaf	Miltox	Ziram
Kaptafol	4-Nitrosopyrazole	Antibiotics
Kaptan	Nurimol	Griseofulvin
Carbendazim	Ozone	Mycostatin
Carboxyne (Vitavax)	Panogen	Mycotricin
Chlorothalonil (Daconil)	Penconazole (Topaz)	Polyoxine B and D
Copper oxychloride (Blitox)	Pentachlorophenol	Kipermethrin
Copper sulphate	Phygon	Deltamethrin
Kuman L	P-Methoxytetrachlorophenol	Dimecron
Cupravite	Profloraz	Fenvalerate
Copper acetate	Propiconazole	Flucitrinate
Cuprox	Propineb	Methoxytox
Cyclohexamide	Pyraclostrobin	Methyldemethon
Delan C	Pyrene compounds	Permethrin
Dichlorofuanide	z-Cunolate	Phosphomedon
Diphosphonates	Cunon	Rogar
Difenoconazole (Skor)	Ridomil MC	Humates
Dithane D-14	Ronilan	N, P, K, Ca, Cacl2
Dithane M-45 (Mancozeb)	Semesan	CuSO4, CO(NO3)2, Fe EDTA,
Dithane Z-78	Signum 334WG	Mn SO4, Na2 BO7, Zn SO4
Duter	Sistan	Thiourea
Edifenofos	Sodium fluoride	Borax
Euparen	Spergon	S, SO2
Fenarimol	C-triazine	Zn
Fenpropimorph	Sumilex (Procimidone)	

Table 4.9

Spread and development of Alternaria in spring oilseed rape variety Siesta F_1 under the use of seed treatment [223]

Варіант	LNAU				BNAU			
	Prevalence, %		Development, %		Prevalence, %		Development, %	
	Phase outlets	Ripeness	Phase outlets	Ripeness	Phase outlets	Ripeness	Phase outlets	Ripeness
Without dressing	4.7	90.5	1.1	19.8	4.3	77.5	0.8	15.4
Cruiser OSR 322 FS, hp	4.1	90.2	0.9	20.2	2.7	75.9	0.5	15.2
TMTD, in.s.c.	4.3	90.8	0.9	20.1	2.9	75.7	0.5	15.1
Vitavax 200 FF, b.s.c.	3.7	89.7	0.8	18.6	3.3	76.5	0.6	15.3
Maxim XL 035 FS, hp	3.9	91.1	0.8	19.4	3.0	77.1	0.6	15.4
Phyto doctor	4.3	90.6	0.9	20.1	3.1	76.4	0.6	15.3
SSD_{05}	0.33	1.1	0.1	1.6	0.5	1.5	0.2	0.4

Notes: LNAU – Lviv National Agrarian University, BNAU – Bila Tserkva National Agrarian University.

Table 4.10

Spread and development of Alternaria in the variety Siesta F_1 of spring rape under the use of different fungicides (LNAU) [223]

Variant	Disease prevalence, %	Disease progression, %	Yield, t/ha
Without spraying	90.3	54.3	2.00
Karamba, v.r. + Pictor, h.p.	45.7	10.6	2.42
Pictor, h.p.	48.0	12.7	2.40
Tilmore, k.e.	47.0	18.1	2.29
Faraday, v.g.	50.3	21.6	2.27
SSD_{05}	3.2	2.4	0.05

The use of alternaria-resistant varieties in production, combined with the application of rational protection systems, reduces the cost of production and helps to increase the profitability of production. Seed treatment helps to protect plants from pathogens that may be present in the seeds and the environment in the early stages of growth, but does not have a significant

impact on the susceptibility to Alternaria during the most dangerous period – pod formation and seed maturation.

Table 4.11

Spread and development of Alternaria on spring oilseed rape variety Kalinovsky under different fungicides (NUBIP) [223]

Variant	Disease prevalence, %	Disease progression, %	Yield, t/ha
Without spraying	81.3	16.1	2.46
Karamba, v.r.	64.6	14.5	2.58
Pictor, h.p.	54.2	10.9	2.63
Tilmore, k.e.	52.1	12.5	2.62
Faraday, v.g.	68.8	14.2	2.60
Ridomil Gold MC, v.d.g.	72.9	14.0	2.55
SSD_{05}	5.6	2.2	0.10

Thus, the fungicide Pictor is highly effective in controlling the spread and development of Alternaria pathogen on spring rape crops, while Tilmore, Faraday, and Ridomil Gold are somewhat less effective.

It should be borne in mind that an individual approach to the selection of a specific fungicide for controlling Alternaria is advisable, taking into account the soil and climatic characteristics of the region, varietal composition and prevalence of the pathogen itself.

List of references for the subheading 4.1

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4.2. Integrated systems and methods for controlling sclerotinia in cruciferous crop agrocenoses

Controlling sclerotinia is challenging due to the wide range of hosts and the long survival of the pathogen. Since the disease is transmitted through sclerotia with crop residues, and plant waste stimulates the formation of sclerotia, it is advisable to collect and harvest all infected residues to destroy sclerotia. Clean seeds free of sclerotia should be used for sowing. Due to the airborne transmission of ascospores and the wide range of hosts (about 408 species), crop rotation seems to be a less successful method of controlling the disease. However, deep summer ploughing and crop rotation with non-susceptible hosts (rice, maize), using only recommended doses of nitrogen fertiliser, irrigation and maintaining plant populations within recommended rates, and flooding the soil if possible, will minimise the population of sclerotia in the soil, which may later prove useful in controlling soil inoculum disease [1]. Avoiding thickening in the row to minimise contact between plants through the roots and stem, to reduce the spread of the disease by mycelial pathways, is also an effective measure. Control of broadleaf weeds such as Chenopodium spp. is important in disease management.

The occurrence of sclerotinia can be reduced or avoided by sowing rapeseed late [2]. Late sowing can be beneficial as it shortens the period between phenological susceptibility and maximum ascospore load. Sowing dates had a significant impact on the incidence of sclerotinia rot and yield [3]. Correlation analysis showed that there is a significant positive correlation between maximum and minimum temperature on the occurrence of the disease. The data showed that stem rot infection was negatively and significantly affected by maximum temperature (r= -0.697*) and number of sunny hours (r= -0.855**), while maximum relative humidity (r= 0.883**) and minimum relative humidity (r= 0.871**) showed high reliability and positive correlation with Sclerotinia sclerotiorum infection.

Application of compost to the soil inhibited the carpogenic germination of S. sclerotiorum and reduced sclerotinia infection [4]. The extracts of five organic additives, namely sunflower cake, safflower cake, mustard cake, neem cake and manure, significantly reduced the growth of *S. sclerotiorum* mycelium [5].

The combined effect of trace elements, namely: B at a dose of 1 g/l, Mo at a dose of 1 g/l, S at a dose of 2 g/l and Zn at a dose of 2 g/l, on reducing the incidence of sclerotinia rot and increasing the yield of rapeseed [6].

Currently, there are a number of disease control measures aimed at different stages of the S. sclerotiorum infection cycle (Figure 4.13). Some of these are theoretical and have only been demonstrated experimentally, while others are already being applied by growers. The different types of control measures include cultural control, fungicides, biological control, selection for genetic resistance and genetic modification (which is currently theoretical).

Current methods of controlling the disease are based primarily on cultural control and fungicide use. Most cultural control methods are aimed at reducing the level of *S. sclerotiorum* inoculum in the soil or creating a local environment unfavourable for the pathogen.

Fungicides are commonly used to prevent the development of S. sclerotiorum embryos on rapeseed root tissue in order to break the disease cycle. However, the effectiveness of these fungicides depends on the ability to predict when the fungal ascospores first appear on rapeseed tissue. Failure to apply fungicides on time can lead to economic losses.

Cultural control methods contribute to the creation of local conditions unfavourable for the survival of pathogens and the development of diseases [7]. In order for cultural control methods to be effective, an understanding of the basic biology of the target pathogen is essential. The control of sclerotinia in cruciferous crops is largely dependent on cultural control practices (Figure 4.7).

Crop rotation is a popular way to control the number of sclerotia in oilseed rape fields. Crop rotation with crops that are not susceptible to *S. sclerotiorum* can disrupt the annual life cycle of *S. sclerotiorum*, reducing the annual number of sclerotia entering the soil 'sclerotia bank'.

Currently, canola producers are advised to rotate their crops with other crops such as wheat or barley once every 4 years [9]. However, this advice should only serve as a guide, as it has been suggested that individual *S. sclerotiorum* sclerotia can remain viable in the field for at least 7 years [10–11]. A study conducted in Canada showed that sclerotia isolated from a canola field that had been reseeded with barley for three consecutive years remained viable and were able to germinate and develop apothecia [12]. The accumulation of sclerotia in the soil is further enhanced when the

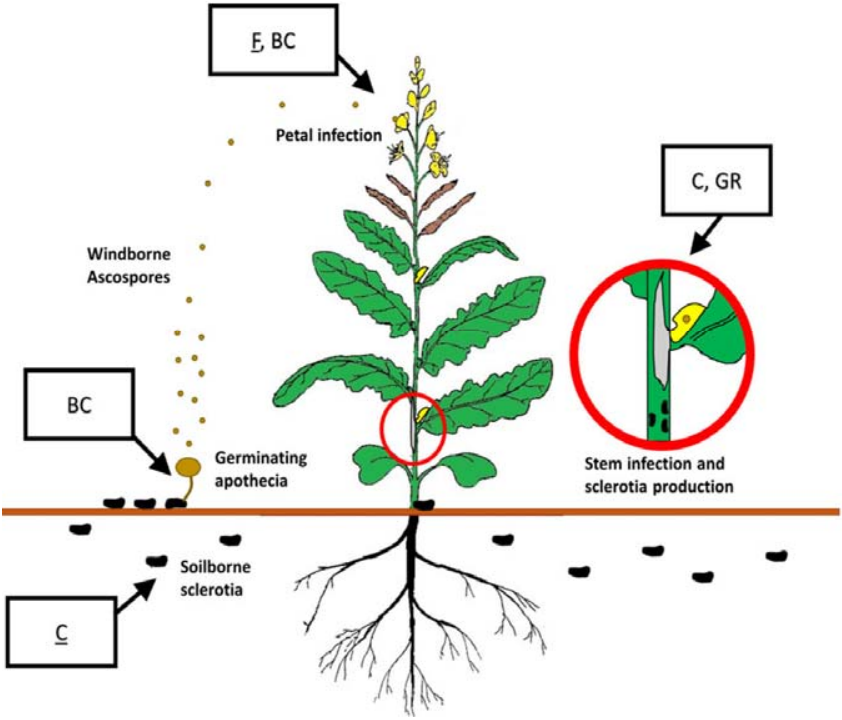

Figure 4.7 – The infection cycle of Sclerotinia sclerotiorum and potential points of intervention. Ascospores are released from the germinating apothecia and are carried by the wind to the aboveground tissues. The ascospores germinate and primarily infect the aging tissues of the petals. The infected petals fall off and lie in the leaf axils, allowing S. sclerotiorum to penetrate the stems and infect them, causing sclerotinia. After infection, sclerotia are formed and can persist in the soil for several years. At different stages of the infection cycle, different control measures can be used to combat this pathogen: cultural control (CC), biocontrol (BC), fungicides (F) and genetic resistance (GR) [8]

host crop is grown for several consecutive years without crop rotation. Crop rotation duration is a key component of cultural control that must be carefully considered when trying to limit the incidence of sclerotinia.

Crop rotation is only effective when the life cycle of the target pathogen is interrupted. One of the challenges associated with *S. sclerotiorum* is that it can complete its life cycle on more than 400 different host species, including common weeds such as wild radish (*Raphanus raphanistrum*), shepherd's purse (*Capsella bursa-pastoris*), field thistle (*Cirsium arvense*), yellow thistle (*Sonchus arvensis*) and medicinal dandelion (*Taraxacum officinale*) [13]. For crop rotation to be effective against S. sclerotiorum, it must be combined with an effective weed management programme that minimises the likelihood of a "green bridge" that allows S. sclerotiorum to persist in fields where oilseed rape is not grown in the rotation.

The impact of tillage on the survival of sclerotia in soil has been widely studied. Tillage practices reduce the ability of sclerotia to form apothecia by burying them in the soil.

Reported [14], that *S. sclerotiorum sclerotia* function only in the top 2–3 cm of soil, as the apothecia are unable to grow longer than 3 cm. It was confirmed that ploughing sclerotia to a depth of 6–15 cm reduces germination and the formation of apothecia the following year. However, the survival of *S. sclerotiorum sclerotia* increases when the sclerotia are buried deep into the soil [15]. Subsequent years of tillage will only bring the previously buried sclerotia back to the soil surface. Therefore, this method is more effective if the sclerotia are buried and remain embedded in the soil until the viability of the sclerotia is reduced.

Another method used to reduce the viability of sclerotia in the soil is to manipulate the water content of the soil. It has been shown that high soil moisture negatively affects the survival of sclerotia [16–17]. In extreme cases, when soaking lasted for more than 24 consecutive days, the viability of sclerotia was seriously reduced [18]. On the other hand, limiting the availability of water in the top 5 cm of soil can also affect the viability of sclerotia. Manipulation of soil water content to control sclerotinia will be limited to cultivation systems that use controlled irrigation schemes. In addition, these control methods will need to be applied between growing seasons, as the described water and soil environment is likely to have a negative impact on the viability of the crops themselves.

Chemical soil treatments have also been shown to be effective in reducing the carpogenic initiation of sclerotia. In particular, the commercially available fertiliser calcium cyanamide has been shown to significantly reduce the number of aptocia in four field trials – by up to 87% [19].

It is noted that the susceptibility of plants to sclerotinia rot was higher when using ammonium sulphate, while spraying with thiourea showed a lower intensity of the disease development [18].

Some cultural control methods focus on modulating the local field environment so that conditions are unfavourable for the development of sclerotinia. Relative humidity is considered to be a key factor influencing the epidemiology of *S. sclerotiorum* in *B. napus* crops. The analysis of data collected at local weather stations showed that relative humidity values exceeding 80% correlate well with the development of sclerotinia in culture [20].

High relative humidity is particularly problematic during flowering, as senescent flower petals are the main site of *S. sclerotiorum* ascospore accumulation. Relative humidity in crops can be reduced by using appropriate row spacing and seeding rates at sowing time to ensure air exchange between individual plants.

Few genetic sources of resistance to the pathogen are available to breeders, and this has led to fungicide applications becoming the main method of controlling sclerotinia. Among the fungicides that suppress sclerotinia are Benomyl, thiophanate-methyl, vinclozolin and tebuconazole. Ditan M-45 (Mancozeb), Ditan Z-78 (Zineb), Ziram, Difolatan-80, Blitox-50, Benlat; Iprodion, fenpropimorph; Tiram [21–23]. However, the number of flowers under the canopy and the density of plants prevent from achieving good spray coverage, and an effective forecasting tool is needed for timely fungicide spraying [24]. Even more importantly, the development of fungicide resistance threatens the continued effectiveness of this method of disease control. In addition to routine chemical control, various forecasting systems are used, including the petal test method for the prediction of cruciferous sclerotinia. This forecasting method was developed based on the relationship between disease incidence and the level of pathogen infection of petals at the beginning of flowering. A risk score table was developed, but the prediction based on the risk map was not as accurate for different fields. The use of both petal infestation and enzyme-linked immunosorbent assay

(ELISA) can reduce some of the shortcomings of each prediction method as tools for stem rot risk assessment in oilseed rape. Risk prediction maps for *S. sclerotiorum* in rapeseed are currently available [25].

Globally, several different classes of fungicides have proven effective against S. sclerotiorum, including anilinopyrimides, benzimidazoles, dicarboxamides [26], demethylation inhibitors [27], external quinone inhibitors (otherwise known as strobilurins) [28–29] and succinate dehydrogenase inhibitors [30–31]. Others have been tested in vitro for activity against *S. sclerotiorum*, but have not yet been used in the field, such as phenylpyrroles. These classes of fungicides demonstrate different modes of action: anilinopyrimidines inhibit methionine biosynthesis [32], and dicarboxamides are thought to inhibit osmotic signal transduction [33], although the exact molecular mechanisms of this activity are unknown. Benzimidazoles bind tubulin, which disrupts microtubule formation [34]. Finally, similar to dicarboxamides, phenylpyrroles inhibit osmotic signal transduction by inhibiting a specific protein kinase [35]. Thus, there is a wide range of fungicides that can be used to prevent sclerotinia in oilseed rape, although not all of them are registered for use in oilseed rape in all growing regions. As most of the above classes of fungicides are active only in certain areas, the likelihood of fungal resistance developing is relatively high. Indeed, for each of these classes of fungicides, resistant strains of pathogenic fungi are known [36–42], Thus, there is a wide range of fungicides that can be used to prevent sclerotinia in oilseed rape, although not all of them are registered for use in oilseed rape in all growing regions. As most of the above classes of fungicides are active only in certain areas, the likelihood of fungal resistance developing is relatively high. Indeed, for each of these classes of fungicides, resistant strains of pathogenic fungi are known [43–44]. However, resistance of *S. sclerotiorum* has been reported worldwide only to benzimidazoles and dicarboxamides [45–46], and in both cases, resistant strains emerged more than ten years after the introduction of these fungicides.

The reason for the low tendency of *S. sclerotiorum* to develop resistance to fungicides may be the monocyclic cycle of infection. By definition, this type of infection originates from a single source of inoculum during the growing season [47]. This effectively reduces the population size and genetic potential, as secondary spread of the disease occurs through the expansion

of multinucleate mycelium by mitotic division. Indeed, a predictive model has demonstrated that the strategy of monocyclic or polycyclic infection is an important determinant of the risk of fungicide resistance in plant pathogens [48]. This effectively reduces the population size and genetic potential, as secondary spread of the disease occurs through the expansion of multinucleate mycelium by mitotic division. Indeed, a predictive model has demonstrated that the strategy of monocyclic or polycyclic infection is an important determinant of the risk of fungicide resistance in plant pathogens [49].

In summary, the data suggest that fungicides are a useful long-term method of controlling S. sclerotiorum as it is unlikely to develop resistance quickly, especially if fungicides with different mechanisms of action are used. However, there is still a need to test and register a more diverse set of fungicides in regions where chemicals with a limited spectrum of action are used. This is because no matter how small the risk of resistance development, it still exists and can pose a potential threat to future crop yields if fungicides are not managed properly [50]. In addition, selective pressure caused by the overuse of a small number of registered fungicides on cruciferous crops can create resistance in other species that infect these crops, such as Alternaria brassicicola, which has already developed resistance to several classes of fungicides [51].

In addition, in some cases, unregistered fungicides have shown greater potential than those registered for sclerotinosis. One example is the phenylpyrrole compound fludioxonil, which has demonstrated greater activity against Chinese *S. sclerotiorum* isolates than iprodione, which is a widely used fungicide in China [52]. In the future, testing of alternative fungicides for the prevention of sclerotinia may be important for controlling the disease in regions where the availability of registered fungicides is limited.

Due to environmental constraints on fungal growth and plant infection, outbreaks of sclerotinia are difficult to predict. Consequently, deciding on the need and timing of fungicide spraying to control the disease is a difficult task. If fungicides are sprayed prophylactically, regardless of the potential outcome of the disease, years in which the incidence of sclerotoniosis would have been low or non-existent will suffer economic losses due to unnecessary fungicide consumption.

As a result, a large number of studies have been conducted to develop mathematical models and sampling methods that can be used to predict sclerotinia outbreaks. Numerous quantitative variables have been described that affect the degree of damage to different crops, including the number and spatial distribution of apothecia, the amount of airborne infection, the frequency of petal infection, soil moisture content and relative humidity [53–56]. These variables are inextricably linked to each other and to other qualitative variables such as weather forecasts and crop history. Several attempts have been made to use these variables individually or in combination to predict sclerotinia outbreaks in cruciferous oilseed rape.

A checklist has been developed to optimise fungicide spraying decisions in oilseed rape based on factors thought to influence the incidence of sclerotinia [57]. A forecasting scheme based on controlled germination of sclerotia was developed. It involved collecting sclerotia from infected rapeseed fields, then priming them for germination and sowing them in designated locations. The percentage of germinated sclerotia in a particular repository was then used to predict outbreaks of sclerotinia in rapeseed crops within a 15 km radius [58]. However, this approach has a number of drawbacks, including its insufficient predictive power for individual fields with different historical cultivation regimes and, consequently, sclerotial numbers, its inability to account for ascospore formation and dispersal, and its subjectivity to local environmental conditions. To improve the predictive power of this scheme, attempts have also been made to measure the release of ascospores from sclerotial depots [59].

Another approach is to use the number of infected rape petals to predict outbreaks of sclerotinia. To do this, rapeseed petals were incubated on agar to determine whether *S. sclerotiorum* would grow from them, which allowed us to indirectly assess the presence/absence of the disease. Using this method, we were able to show that the percentage of petals infected with S. sclerotiorum could be used to accurately predict the severity of sclerotinia in 74% of cases, based on the results of several consecutive field trials. It is likely that these studies led to the development of petal testing kits that became commercially available to Canadian canola growers. They consisted of plates containing agar medium suitable for *S. sclerotiorum* growth and instructions for use [60].

Similar petal testing kits have been commercialised in Australia, although they were sold with the caveat that they were only useful for determining the presence or absence of *S. sclerotiorum* in a canola crop, not for predicting disease severity.

Although petal testing has proved useful in predicting sclerotoniosis, it loses its predictive power when environmental conditions are not favourable for S. sclerotiorum infection. Attempts to counteract this have been made [61] based on the inclusion of environmental variables such as light, leaf area index and crop height in prediction systems based on petal infection. This study also showed that the amount of petal infection varied between early, full and late flowering, with a general trend of increasing infection as the flowers developed. However, the authors of the study raised questions about the ease of measuring environmental variables and the practicality for growers of continuously sampling petals covering different stages of flowering.

Another attempt to combine environmental variables with petal infection to predict sclerotinia was made [62]. A simpler prediction model has been developed that is based on both the level of petal population and relative soil moisture. However, another problem with the petal test method is that the assessment of the level of petal population may itself be dependent on environmental variables. For example, it has been shown that collecting wet petals or collecting petals just before heavy rain can lead to an underestimation of petal infestation [63], as well as picking petals in the morning rather than in the afternoon.

Perhaps because of their ease of implementation, models based on simpler factors such as crop history, disease incidence in previous years and weather forecasts are currently the most widely used by growers to make decisions about fungicide use against sclerotinia.

Developed [64] a forecasting model based on a risk score table using the factors "number of years since the last rapeseed sowing", "incidence of the last *S. sclerotiorum* host crop", "sowing density", "rain in the last 2 weeks before sowing", "weather forecast" and "regional risk of aphotic development". With a given threshold score, the model provided accurate spraying recommendations for 75% of the fields that needed fungicide application and 84% of the fields that did not.

Although oilseed rape growers have applied various S. sclerotiorum risk assessment schemes based on the information from the above studies,

a centralised assessment scheme for oilseed rape growers in a particular region was not developed until 2007 in Germany. This model, called SkleroPro, worked by creating a regional disease risk assessment based on environmental variables obtained from weather stations, such as relative humidity and rainfall, which could then be used to determine the risk for a particular field based on parameters such as cropping history, expected yield, product price and spraying cost. Spraying recommendations could be automatically determined by entering data on these parameters through an online platform. This model could provide accurate recommendations in 70-81% of cases, based on historical data and field trials. However, the authors acknowledged that the model does not take into account external levels of inoculation and is therefore most suitable for regions where *S. sclerotiorum* inoculation is consistently present at high levels.

An alternative to assessing petal infestation to predict the severity of oilseed rape sclerotinia is to assess inoculum levels detected in air samples adjacent to field areas, as previously done with Burkard spore traps. Traditional methods of quantification, i.e. microscopic analysis of trapped spores, have been replaced by PCR-based detection [65]. This method can be used to determine the presence of *S. sclerotiorum* DNA in a spore trap, which is used as an indirect measure of its presence/absence in the field. The method itself has been improved with the development of a quantitative real-time PCR-based assay [66]. This new method was sensitive enough to detect as little as 0,5 pg of *S. sclerotiorum* DNA, which is equivalent to approximately 1,5 ascospores. This is well suited for deploying widely scattered spore traps to detect *S. sclerotiorum* in large regions rather than in individual fields, which may be useful for more coordinated efforts to reduce the impact of the disease on cruciferous plant species.

An improvement on PCR-based detection systems is the SYield sensor system, commercially available from Syngenta. This system is able to automatically detect and quantify the amount of *S. sclerotiorum* inoculum in the air by monitoring the level of oxalic acid in the culture medium. Information from the spore traps can be transmitted wirelessly to alert farmers to high levels of infection, which can help in the timing of fungicide spraying [67–68]. The above models were developed in temperate regions.

The detection of the pathogen on mustard (*B. juncea*) plants affected by sclerotinia rot was made possible by the use of a remote sensing method

that can help in multi-stage disease tracking and forecasting [69–70]. Developed [71] a stepwise multiple linear regression model for sclerotinia rot of white mustard. A multiple linear regression model was described as part of an epidemiological study of sclerotinia rot and 10 independent weather variables. The equation of the fitted model is as follows: Incidence of sclerotinia rot = -11.2351 + 0.9529 x (Sum of temperatures) + 4.93924 x (Evenness of rainfall distribution) + 3.83308 x pH (Acidity) + 0.60885 x RF (Rainfall) – 0.406458 x RH1 (Relative humidity) + 0.524095 x RH2 (Soil moisture) + 0.17386 x Soil moisture (%) – 0.30461 x Tmax (Maximum temperature) – 0.677744 x Tmin (Minimum temperature) – 2.19556 x WS (Wind speed). The ScleroPro system is easy to use and fully computerised, and based on weather and field condition data, this programme has been available since 2006.

Biological control has also been explored over the past few decades due to growing concerns about the use of chemical pesticides [72]. Many biological agents are potential means of controlling this disease. The most studied biocontrol agents are mycoparasitic fungi, hypovirulent strains of *S. sclerotiorum*, bacteria and insects [73]. The use of organic and inorganic materials or developed compounds has also been shown to inhibit *S. Sclerotiorum* [74]. For biological control, it is recommended [75]; *Coniothyrium minitans* і *Talaromyces flavus* (Klocker) Stolk and Samson. The use of an actinomycete fungus was effective *Streptomyces arabicus* [76–77].

Over the past 20 years, significant efforts have been made to identify biological control agents (BCAs) for *S. sclerotiorum*. Despite the rapid rate of discovery of potential BCAs, only a small number of them have been commercialised [78]. Most of the reported BACs belong to the fungal and bacterial kingdoms, but it has recently been shown that viral particles can also be used to disrupt growth of *S. sclerotiorum* [79]. Many organisms identified as parasitic to S. sclerotiorum are also fungi. *Trichoderma harzianum* parasitises both the sclerotic and hyphal stages of growth *S. sclerotiorum* [80-81]. Analysis of gene expression during these parasitisation events indicates that *T. harzianum* actively produces enzymes that destroy the fungal cell wall. Functional studies have demonstrated the importance of the endogenous chitinase Chit42 in the destruction of the *T. harzianum* cell wall. Trichoderma harzianum isolates transformed with

a constitutively expressed Chit42 transgene had higher levels of chitinase activity than wild-type isolates, making them more capable of inhibiting the growth of *S. sclerotiorum* [82]. It was shown that the fungus Ulocladium atrum has an antagonistic effect on germinating ascospores of *S. sclerotiorum* [83]. Joint inoculation of rape petals with *U. atrum* and *S. sclerotiorum* reduced the amount of pathogen development compared to inoculation with *S. sclerotiorum* alone.

The most studied mycoparasite for biocontrol of *S. sclerotiorum* is *Coniothyrium minitans*. Like *T. harzianum*, *C. minitans* parasitises sclerotia and mycelium of *S. sclerotiorum* [84-86]. The key to the success of *C. minitans* as a BAC is its ability to persist and spread in the soil.

Coniothyrium minitans is relatively resistant to annual fluctuations in soil temperature and moisture. In non-irrigated soil with ambient temperatures ranging from 10 to 39 °C *C. minitans* was able to survive for 750 days [87]. Conidia of *C. minitans* can easily spread through the soil by free water. It has been shown that active spread of C. minitans at the stage of rapeseed seedlings can reduce the number of carpogenic germination of *S. sclerotiorum* later during the growing season [88]. The parasitisation of *C. minitans* by *S. sclerotiorum* is likely to be associated with the breakdown of oxalic acid, a known pathogenicity factor of *S. sclerotiorum*. Research [89] showed that *C. minitans* actively breaks down oxalic acid, which leads to local pH changes. It is assumed that the induced change in pH of the infected tissue stimulates the production of enzymes that destroy cell walls. The role of oxalic acid in the mycoparasitism of *C. minitans* on *S. sclerotiorum* was further confirmed by studying defective mutants of *C. minitans* oxalate decarboxylase that lost the ability to break down oxalic acid. When coincubated with *S. sclerotiorum*, the Cmoxdc1 mutant had a reduced ability to infect *S. sclerotiorum* mycelium compared to wild-type *C. minitans* [90]. In addition to its ability to break down oxalic acid, *C. minitans* is also thought to secrete antifungal compounds. It has been shown [91], that substances previously added to the culture filtrate of *C. minitans* were active against mycelial growth and germination of *S. sclerotiorum* ascospores.

In addition to parasitic fungi, many bacterial mycoparasites have been identified as potential enemies *S. sclerotiorum* – *Streptomyces platensis* [92], *Streptomyces lydicus*, *Bacillus subtilis* [93–99], *Bacillus*

amyloliq-uefaciens [100], *Bacillus megaterium*, *Pseudomonas fluorescens* [101–102], *Pseudomonas chlororaphis* [103–105] and *Serratia plymuthica* [106]. Unlike the fungal BACs described above, most of the bacterial mycoparasites described above target ascospores and growing hyphae *S. sclerotiorum* [107]. Usually, broth cultures or cell suspensions of potential bacterial BAC are applied to the aerial parts of rapeseed plants by spray inoculation.

Scanning electron microscopy (SEM) images of rapeseed leaves pre-inoculated with *S. sclerotiorum* and *B. subtilis* EDR4, showed *S. sclerotiorum* hyphae with abnormal growth, cytoplasmic leakage and fewer infection cushions compared to the negative control. It was also shown that the mycelia of *Sclerotinia sclerotiorum* acquire an irregular shape when incubated with *P. fluorescens*. As a result, the growth of *S. sclerotiorum* was reduced by 84,4% [108].

It is reported [109], that *T. harzianum* and *P. fluorescens* did not differ significantly in terms of pathogen control efficiency. Similar results were obtained with the use of garlic bulb extract, *T. harzianum* as a seed treatment in combination with *P. fluorescens* spraying significantly outperformed chemical fungicides in the control of sclerotinia rot. Foliar spraying with garlic bulb extract significantly increased seed yield compared to the control. It was reported that the combination used in this study significantly reduced the incidence of sclerotinia rot and was as effective as the combination of seed treatment with *Trichoderma harzianum* and foliar spraying with *Pseudomonas fluorescens* and *T. harzianum*. The economic profitability was higher when using biological products (*A. sativum, T. harzianum, P fluorescens* bulb extract) compared to chemical fungicides.

Combination of seed treatment with T. harzianum and its further use in the form of foliar spraying [110], as well as a similar combination of seed treatment and foliar spraying with bulb extract of *A. sativum* [111], has led to a higher profit-to-cost ratio.

They found [112], that isolates Trichoderma harzianum-3 and T. Harzianum-4 were significantly better and most potent in reducing the growth of *S. sclerotiorum*. Similarly, the antagonists T. Harzianum-4, T. Harzianum-3 and T. virens antagonists reduced the formation of sclerotia.

It is reported [113], that the tested fungicides and garlic extract significantly reduced the incidence of sclerotinia, and [114] reported that

preventive foliar spraying with carbendazim twice at 45 and 60 days after sowing was the most effective for controlling white mustard stem rot in the field.

Trichoderma harzianum was tested against *Sclerotinia sclerotiorum*. The highest reduction of sclerotinia rot (69,0%) was achieved by *T. harzianum* GR isolate compared to the control, followed by soil application of *T. harzianum* SI-02 isolate with manure (60.8%) and foliar spraying with aqueous garlic extract (60,8%). Independent studies on spray inoculation with *B. amyloliquefaciens* [115] and *B. subtilis* Em^7 [116] led to a decrease in the incidence of sclerotoniosis by 83.3% and 50–70%, respectively. The levels of *S. sclerotiorum* control achieved in these experiments are comparable to those achieved with synthetic fungicides [117].

As an alternative to spraying methods, *S. sclerotiorum* bacterial ABA can also be applied as seed treatment granules. These protective structures increase the viability of the BAC over time and may also facilitate colonisation of roots and the rhizosphere [118]. Some of the bacterial ABAs used for seed treatment had the additional effect of increasing plant growth and yield. *Bacillus megaterium* A6, *B. subtilis* Tu-100 and *P. fluorescens* PS demonstrate plant growth stimulation effects combined with the ability to reduce the incidence of sclerotinia [119–121].

Three of the above-mentioned biological control agents have been commercialised for use against *S. sclerotiorum*. The fungal preparations *C. minitans* and *T. harzianum* were developed for sclerotia control, while the bacterial preparation *B. subtilis* was developed for controlling fungal growth in the phyllosphere [122].

A study comparing the ability of each of the three commercially available ABAs to inhibit *S. sclerotiorum* germination showed that C. minitans was the most effective, reducing the viability of S. sclerotiorum in soil by 95.3% and reducing the overall disease severity by 68.5%. In an independent study, the use of Contans WG in field trials on rapeseed resulted in a significant reduction in the incidence of slough disease compared to untreated controls [123]. At the same time, the ability of the spraying equipment to maintain effective concentration on the field is often questionable and largely depends on environmental conditions.

However, BACs are often seen as environmentally friendly alternatives to conventional fungicides [124]. Contans WG is considered to be relatively

cheap compared to other biological control agents and remains the most promising non-cultivated control option for the eradication of *Sclerotium sclerotiorum*.

Integration of seed treatment with foliar sprays contributed to better disease reduction [125–126]. *Trichoderma atroviride* demonstrated the formation and penetration of pathogen hyphae [127]. Soil application of *T. harzianum* at a dose of 15 g/kg soil at the same time or 7 days before the pathogen emergence resulted in low intensity of the disease development [128]. It is reported [129], that the W-1 strain of Caseobacter spp. can control the pathogen.

Carpogenic germination of pathogenic sclerotia can be reduced with a bioagent *Gliocladium virens* [130].

It was observed [131] antifungal activity of strain 11-3-1 of *Streptomyces longisporoflavus npomu S. sclerotiorum.*

Pseudomonas fluorescens P13, released from the soil of a rapeseed field produced hydrogen cyanide [132] and *Pseudomonas chlororaphis* PA-23 induced rapeseed plants to produce more hydrolytic enzymes, namely chitinase and beta-1,3-glucanase [133], in response to *S. sclerotiorum* infection, thus being effective against the pathogen. However, the control of the pathogen by Psuedomonas DF41 strain depends on the production of lipopeptides and the presence of a functional Gac system in the bioagent [134]. The chitinase activity of different *B. napus* genotypes significantly correlates with the sclerotinia rot damage, suggesting that chitinase can be used in breeding programmes to increase the disease resistance of rapeseed.

Treatment of seeds with a bacterial strain, namely Mesorhizobium loti MP6, isolated from the root nodules of Mimosa pudica, led to improved seed germination, early vegetative growth and seed yield with a sharp decrease in the incidence of sclerotinia rot [135]. Strains Y1 [136], NJ-18 [137], YS45 [138], Tu-100 [139–140] та EDR2 [141] *B. subtilis* and BS6 *Bacillus amyloliquefaciens* [142] have shown promise against this disease in oilseed rape. A new antifungal protein produced by Bacillus licheniformis W10 could be used as a biofungicide to combat this disease [143].

Bioagent, namely Coniothyrium minitans, which destroys hyphae [144] and sclerotia [145–146] *S. sclerotiorum*, was used to control the disease. This bioagent decomposes oxalic acid, negating its pH effect. In addition, it can stimulate the production of beta-1,3-glucanase by the bioagent and

can improve the mycoparasitism of the agent on *S. sclerotiorum*, which will lead to the protection of plants from infection by the pathogen [147].

Water application of C. minitans during rapeseed sowing was effective in suppressing carpogenic germination of the pathogen [148]. Soil treatment with this bioagent proved to be effective in reducing the production of ascospores by the pathogen [149]. To increase long-term effectiveness, infested areas can be regularly inoculated with deep soil spores of *C. minitans* before or after sowing crops. Reported [150], that the aerial application of this bioagent is an effective method of inhibiting the growth of pathogen mycelium on petals. Tautomycin, produced by Streptomyces spiroverticillatus, and other related compounds, namely 2,3-dimethylmaleic anhydride, diphenylmaleic anhydride and dimethylmaleate, have significant potential against the pathogen [151].

P. fluorescens PS1 biological product based on sawdust caused morphological changes by perforating hyphae, which allowed to control the disease [152].

The use of microviruses to control *S. sclerotiorum* is an exciting new development in biological control research [153]. It has been demonstrated that viral particles of the hypovirulent DNA virus *Sclerotinia sclerotiorum*-1 (SsHADV-1) are able to limit the growth of *S. sclerotiorum* when applied to infected plants of Arabidopsis thaliana. In addition, when a fragmented suspension of virus-infected *S. sclerotiorum* hyphae was applied to the aerial parts of B. napus, the incidence and severity of sclerotoniosis were increased. Despite the limitations on the spread of these viral particles through host cells, the data indicate that they can be transmitted between fungi regardless of vegetative compatibility.

The suitability of these viruses as potential BACs is also supported by the fact that they have a limited host range [154]. For example, SsHADV-1 can infect species in the genus Sclerotinia, but is unable to infect Botrytis cinerea, a close relative of the Sclerotiniaceae. The advent of high-throughput sequencing has facilitated virus detection and contributed to the discovery of additional hypovirulent S. sclerotiorum viruses that could be developed as potential BACs [155–156]. Although the cost-effectiveness of viral BACs has not yet been determined, they open up new possibilities for the future control of rape sclerotinia. Thus, there are a number of potential and already commercialised options for biocontrol of sclerotinia

in oilseed rape. Reported [157], that *C. minitans* reduces the number of sclerotia in the soil by 95.3%, in a similar study by the same authors [158] *C. minitans* to reduce the number of sclerotia by only 50%. It has also been shown that different strains of *S. sclerotiorum*, which produce different amounts of oxalate, are differently sensitive to *C. minitans*. In addition, the study [159] application of *T. harzianum* did not inhibit germination of sclerotia compared to untreated controls, despite several reports of its effectiveness [160].

Another factor influencing the potential use of BACs for disease control is their cost-benefit ratio compared to traditional methods. For products such as Contans WG, which may require multiple seasonal applications to achieve full effect, it is likely that their use by growers is strongly influenced by this factor [161]. Despite these obstacles, the fact that several biocontrol agents exist and are registered for plant protection in different countries suggests that there is at least a limited level of use by producers.

Overexpression of proteins involved in plant defence mechanisms against diseases is one of the strategies proposed to increase plant resistance to fungal pathogens. A hybrid endochitinase gene under a constitutive promoter was introduced by Agrobacterium-mediated transformation into an inbred line of winter rape (*B. napus* var. *oleifera*). When the progeny of transformed plants are exposed to pathogens, the plants show increased disease tolerance compared to non-transgenic parental plants [162].

It is noted that the control of cruciferous sclerotinia by means of cultural and chemical control is often very tedious and not very effective due to its complex mode of infection and its long survival (up to 10 years in the soil in the absence of a host) in the form of a dormant structure called a sclerotium [163]. In addition, the use of fungicides poses a serious threat to the climate and increases the cost of growing crops. Therefore, genetic host resistance is the most convenient, economical and environmentally friendly approach to effectively control this devastating pathogen [164-165]. Previous attempts to identify sources of resistance to this disease in white mustard have been complicated because all tested genotypes of *B. juncea* were susceptible to sclerotinia stem rot, and all identified sources of resistance belonged to other cruciferous crops and their wild relatives, such as *Brassica napus*, *B. fruticulosa*, *B. rupestris*, *B. incana*, *B. insularis, B. villosa, Erucastrum cardaminoides, E. abyssinicum, Sinapis alba* та *Diplotaxis tenuisiliqua*

[166–178] However, there were no reports of resistance in B. juncea, which is an important oil cruciferous crop. However, in the last few years, more and more attention has been paid to this issue, which eventually led to the identification of several mustard genotypes resistant to this pathogen [179–180].

Heritability of sclerotinia resistance in *B. napus* is high, controlled by nuclear genes and is not associated with the low erucic acid content trait. The petal mutant of *B. napus* is almost free from stem rot compared to the normal petal mutant [181].

Inheritance of resistance to *S. sclerotiorum* in *B. napus* is partially dominant [182]. Genetic analysis of resistance to *S. sclerotiorum* in *B. napus* 15 days after inoculation on petals is controlled by major genes with additive dominant epistatic effects and polygenes with additive dominant epistatic effects [183].

Genetic analysis of resistance to *S. sclerotiorum* in *B. napus* 15 days after inoculation on petals is controlled by major genes with additive dominant epistatic effects and polygenes with additive dominant epistatic effects (Figs. 4.8–4.9).

In India, rapeseed genotypes Cutlon, ZYR-6, PSM 169, PDM 169, Wester, PYM 7, Parland, Tobin, PCR 10, Candle, Wester, Cutlass and Torch and mustard genotypes PCR 10, RW 8410, RW 9401, Hyola 401,

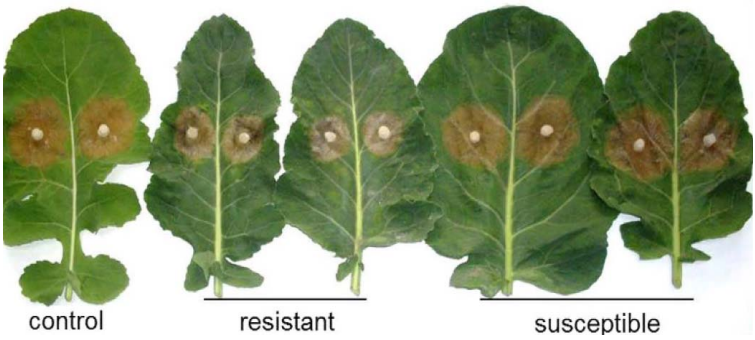

Figure 4.8 – Appearance of *Brassica oleracea* leaves infected with Sclerotinia sclerotiorum. (A) Symptoms of resistant and susceptible *Brassica oleracea* plants 3 days after inoculation with *S. sclerotiorum*

PBN 9501, PWR 9541, Kiran, RH 9401, RH 492, RW 8410, PAB 9511 and RGN 8006 were found to be resistant to S. sclerotiorum. In Japan, the rapeseed varieties Koganenatane, Aburamasari and Kizakinonatane have low incidence rates [185].

Notified [186], that the oilseed rape lines OKEG 8, 94, POH 285 and H 243/33 are the most resistant to *S. sclerotiorum*. In Poland, the winter rape (B. napus) varieties BOH 2600, BOH 1592, BOH 1693, MAH 1391 and MAH 1592, Bermuda, Capio and Mohllan are resistant to Sclerotinia. The strains PNG 2170, MA 1615-1, MZL 236, BK 2466/93, MA 1649-1 are the most resistant. A high degree of resistance was observed in the Isuzu rapeseed variety.

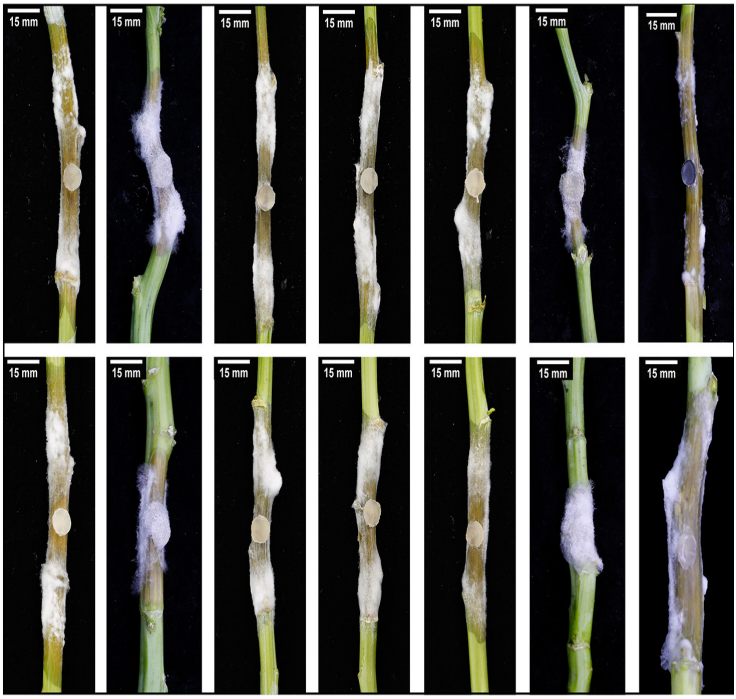

Figure 4.9 – Stems of rape varieties and lines with different resistance to *S. sclerotiorum* 120 h after inoculation with *S. sclerotiorum.* Scale bar 15 mm [184]

Due to the wide range of hosts and the lack of tissue specificity, the development of sclerotinia-resistant varieties has been less successful. However, differences in the general growth pattern and morphological traits of plants may be important characteristics for disease tolerance. Another trait, i.e. plant stem diameter, may be a useful parameter for pathogen tolerance. It has also been reported that lines with high glucosinolate content are more susceptible to *S. sclerotiorum* than lines with low glucosinolate content [187]. Several genotypes of mustard oilseed rape have been tested for sclerotinia rot caused by *S. sclerotiorum* using different methods under natural and artificial conditions [188–189]. The response of some genotypes was relatively consistent, regardless of the pathogen isolates, while some other genotypes showed highly variable responses to the same isolates. Although complete resistance was not found in rapeseed, partial field resistance to sclerotinia rot was found in Chinese cultivars Zhongyou 821 [190] and Zhongshuang № 9 [191].

Four B. napus cultivars, namely BOH 2600, Bermuda, Capio and Mohican, were found to be resistant to *S. sclerotiorum* after a 3-year study. During three consecutive growing seasons, eight genotypes, viz: Hyola-401, PBN-9501, PWR-9541, Kiran, RH-9401, RH-492, RW-8410 and PAB-9511, were found to be resistant to S. sclerotiorum.

In India, white rot of mustard can be effectively controlled by three foliar sprays of benomyl at 0.025% followed by zirram (0.156%). Significant reductions in disease development and lesion size were observed with benlate and topsin-M sprays on B. juncea. In Europe, spraying with prothioconazole 250 EC (Proline) at 175 g a.i./ha controls *S. sclerotiorum* in spring and winter oilseed rape [192].

In Canada, a single spray of Benomyl is available at the early flowering stage in disease-prone regions. The simultaneous use of Benomyl and vinclozolin fungicides effectively controls rape stem sclerotinia when applied at 25% flowering stage. Control is less stable when using Iprodion. Application of Benomyl at a dose of 1.0 kg and Iprodione at a dose of 0.5 kg e.i./ha by air in large-scale trials provides control equivalent to comparable treatments in small-scale trials [193].

Aerial application of benomyl to Altex and Kendle rape seeds reduces the level of sclerotinia stem rot from 44 to 8% with an increase in yield, and

this application is economically feasible when the yield increases by at least 180 kg/ha [194].

In Canada and the United States, for effective control of *S. sclerotiorum*, it is recommended to spray canola with protioconazole 480 EC at a rate of 150–200 g a.i./ha. It is also recommended [195], that such fungicides as azoxystrobin, benomyl, boscalid, iprodione, protioconazole, tebuconazole, thiophanate-methyl, trifloxystrobin and vinclozolin consistently reduce the incidence of sclerotinia stem rot in rapeseed.

In Poland, Contans WG (*C. minitans*) applied before sowing and Alert 375 SC (flusilazole + carbendazim) applied during flowering of rapeseed reduced S. sclerotiorum infection and increased yields [196].

The use of vinclozolin at a rate of 1.5 kg/ha at the stage of full flowering proved to be very effective in controlling white rot of the rapeseed stem with an increase in yield of up to 8% [197].

Suggested [198] use spore traps to optimise the timing of vinclozolin application to achieve maximum disease control.

The use of Iprodion, Prochloraz + carbendazim and tebuconazole (Flikur) on rapeseed gives good disease control with increased yields [199]. The optimal period for chemical control of rape sclerotinia is from 100% flowering stems to 80% flowering branches. The second spraying should be carried out five to seven days after the first, when all branches are in bloom [200].

In France, the fungicides Benomil (1000 g a.i./ha), Procimidone, Winclozolin, Iprodion WP and Iprodion Flow at 750 g a.i./ha were found to be effective against rape sclerotinia. However, the most effective were vinclozolin and procyamidone [201].

Flutriafol (117.5 g a.i./ha) + carbendazim (250 g a.i./ha) provides good control of rape stem rot with yield increases in the UK and France [202]. However, they found [203], that guazathine is an effective fungicide against *S. sclerotiorum* in oilseed rape. Among all five fungicides, it was found that carbendazim completely (100%) inhibited the growth of the pathogen mycelium [204].

In Switzerland, the best control of *S. sclerotiorum* is achieved by fungicide spraying during full flowering. The germination of sclerotia is reduced by calcium cyanamide, which is applied in early spring when new shoots are 5–10 cm long. Preventive measures include the use of

less susceptible crops in crop rotation, careful ploughing of affected plant residues, control of cabbage stem weevil, control of intercrops of rape and cruciferous crops, and cruciferous weeds, including shepherd's purse and field mustard [205].

In China, the agricultural antibiotic 2–16 (in 100-fold and 150-fold dilution) reduces the level of sclerotinia rot in rapeseed by 82.6 and 78.1 per cent, respectively [206].

The list of fungicides recommended in the world practice of cruciferous sclerotinia control is given in Table 4.12.

For most fungicides, the recommended application period is between 20 and 50 per cent flowering, and the optimal time is usually around 30 per cent flowering. To estimate the flowering stage, take a few plants in the field and count the number of open flowers. One method is to identify the main stem, remove the secondary branches, and count only the open flowers on the main stem. Generally, it takes two to four days from the first flower to 10 per cent flowering (Figure 4.35).

The purpose of applying fungicide is to cover as many petals as possible, while ensuring that a certain amount of chemicals also penetrate the canopy to protect potential infection sites (e.g. axils and leaf bases). However, the fungicide only works on the petals present at the time of spraying and does not protect petals that appear after spraying. Most fungicides are contact fungicides with limited systemic function. In addition, the therapeutic properties of most fungicides are limited on existing sclerotinia stem rot lesions, especially those that are large or have penetrated the stem tissue. Therefore, it is important to apply the fungicide before significant petal drop when environmental conditions are favourable for sclerotinia infection.

Seeding density can influence the risk of sclerotinia under favourable environmental conditions The density of the agrocenosis can be changed by seeding rate, row spacing and fertility, which is the main factor that influences the sowing density.

Generally, high numbers of fast-growing plants result in faster canopy closure and the formation of a thick, dense canopy. While these dense agrocenoses often have the highest yield potential, they tend to maintain high soil moisture levels in the stem, which increases sclerotia germination. Soil moisture for approximately 10 days or more encourages sclerotia to form apothecia and subsequently release spores. Wet conditions in the

lower part of the stem also increase the likelihood of infection when petals carrying spores fall on the lower leaves or in the leaf axils. In growing areas with a short season, *B. rapa* cultivars tend to have a lighter top cover and generally lower infection rates.

There are currently two biological products available for the control of sclerotinia stem rot: Contance and Serenade OPTI [207]. Kontans is a biological control agent registered for use in oilseed rape, soybean, dry edible beans, sunflower and safflower. The active ingredient is Coniothyrium minitans, a fungus that colonises and slowly destroys sclerotia on contact with them. The product is a pre-emergence biofungicide that takes several months for the fungus to destroy the viability of sclerotia.

Table 4.12

List of fungicides recommended for control in cruciferous crops [206]

Product	Company	Active ingredient	Rate per acre	Phase of application
1	2	3	4	5
Acapela	Corteva Agriscience	250 g/l picoxystrobin	325–485 ml	20–50% of flowering plants
Azoshy250 SC	Sharda Crop Chem Canada	250 g/l azoxystrobin	280–400 ml	up to 30% flowering
Contans WG	Bayer	5.0% strain Coniothyrium minitans	400–800 g	Pre-sowing and post-harvest processing
Cotegra	BASF Canada	250 g/l boscalid and 150 g/l prothioconazole	240–280 ml	20–50% of flowering
Dyax	BASF Canada	250 g/l of fluxapiroxide and 250 g/l of pyraclostrobin	120–160 ml	20–50% of flowering
Evito 480	UPL AgroSolutions Canada Inc.	480 g/l of fluoxastrobin	59–118 ml	20–50% of flowering
Holdfast	WinField United	480 g/l of prothioconazole	125–150 ml	20–50% of flowering
Lance AG	BASF Canada	70% boscalide; 250 g/l pyraclostrobin	132 ml; 140 g	20–50% of flowering

(End of Table 4.12)

1	2	3	4	5
Lance WDG	BASF Canada	70% boscalide; 250 g/l pyraclostrobin	140 g	20–50% of flowering
Miravis Bold	Syngenta Canada Inc.	200 g/l podiflumethophen	405 ml	20–50% of flowering
Overall 240SC	ADAMA	240 g/l of iprodione	0,85–1,25 ml	20–50% of flowering
Priaxor	BASF Canada	167 g/l of fluxapiroxide and 333 g/l of pyraclostrobin	180 ml	20–50% of flowering
Prodex	Sharda Crop Chem	240 g/l of iprodione	0.85–1.25 ml	20–50% of flowering
Proline 480 SC	Bayer	480 g/l of prothioconazole	125–150 ml	20–50% of flowering
Proline Gold	Bayer	200 g/l fluopyram and 200 g/l prothioconazole	253 ml	20–50% of flowering
Quadris	Syngenta Canada	250 g/l azoxystrobin	280–400 ml	20–30% of flowering
Quash	Valent Canada distributed by Nufarm Agriculture Inc.	50,0% metronazole	57–115 g	20–50% of flowering
Quash SC	Valent Canada.	480 g/l metronazole	59–118 ml	20–50% of flowering
Quasi	AgraCity Crop & Nutrition Ltd.	250 g/l azoxystrobin	280–400 ml	20–50% of flowering
Rovral Flo	FMC Corporation	240 g/l of iprodione	0.85–1.25 ml	20–50% of flowering
Serenade OPTI	Bayer	1,31 x 10 x 10 CFU/g Bacillus subtilis (strain QST 713)	0,1–0,4 кг	20–50% of flowering
Serenade SOIL	Bayer	1.31 x 10 x 10 CFU/g Bacillus subtilis (strain QST 713)	0,4–1,6 ml	Start applying at 20–30% of flowering
Soratel	ADAMA	250 g/l prothioconazole	240–280 ml	20–50% of flowering

Serenade OPTI is a bacterial preparation based on *Bacillus subtillis* that is applied foliarly at the 20 to 30 per cent flowering stage of rapeseed, similar to how fungicides are applied. *B. subtillis* is an antagonist of fragile ascospores and developing hyphae.

Crop rotation is not always effective in controlling sclerotinia stem rot, as *S. sclerotiorum* can be a host on many plants (including many popular crops) [208]. In addition, ascospores can be transported by air from neighbouring fields, where they are released by apothecia germinating from sclerotia left over from previous broadleaf crops. Nevertheless, the likelihood of high levels of sclerotinia stem rot increases with successive broadleaf crops due to the increased potential for spore formation within the field.

Cereals and grasses are not susceptible to the pathogen and may contribute to the reduction of viable sclerotia in the soil through decay and germination in the absence of susceptible hosts. Therefore, it is best to avoid sowing rape next to a field that was heavily infested the previous year. Controlling susceptible weeds and reservoir plants in cereal crops also helps to avoid replenishing the level of viable sclerotia.

It is recommended to use clean varietal seeds free of sclerotia. Since sowing density is an important factor, the recommended sowing rates should be observed. Studies conducted at the University of Manitoba have shown that increasing the seeding rate by two to three times the normal seeding rate can lead to lodging, which can increase sclerotinia infection [209].

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that increasing the seeding rate by two to three times the normal seeding rate can lead to lodging, which can increase sclerotinia infection [210].

Unfortunately, this disease can progress rapidly in the swath in wet years, especially in winter oilseed rape (*B. napus*) varieties. It is recommended not to swath rapeseed crops with a high infection rate if rain is forecast, especially if the crop is immature (green) at the time of mowing. In wet, compacted swaths, especially on corners, sclerotinia rot can progress rapidly. The disease can be detected by the smell of rotten eggs coming from the swaths. Obviously, this problem is more prevalent in humid regions. The heavier and more compact the swath, the more likely it is that sclerotinia will rot in the swath before it is threshed.

In addition to seed infestation, viable sclerotinia pose a potential quarantine risk when exporting seed. Viable sclerotia in infected cruciferous seeds can be destroyed by fumigation. In order to check the secondary spread of the disease, the possibility of controlling the disease by foliar spraying with chemicals has been investigated. Some chemicals, such as quintosene, phenyl acetate and calcium cyanamide, have been found to be effective in inhibiting apothecial development of the fungus. Ridomil MZ (mancoceb + metalaxyl) as a seed treatment provided the highest germination and post-emergence mortality of *S. sclerotiorum* [211].

Seed treatment at sowing and foliar spraying at first budding-flowering with 0.2% benomyl proved to be the best option. The use of carbendazim at 0.25% w/w for foliar spraying can be effective in controlling this disease.

Application of fungicides in the full flowering phase [212] using venturi nozzle technology was effective in reducing pathogen infection. Foliar spraying with zinc pyrithione inhibited the development of the pathogen.

As noted earlier, no single method can effectively control *S. sclerotiorum*, and the best approach to controlling the pathogen is to integrate various environmentally sound measures. In recent years, the growing awareness of pesticide pollution and the development of fungicide-resistant strains of *S. sclerotiorum* [213] encourages plant pathologists to search for environmentally friendly means to combat sclerotinia rot.

Boscalid (trade name Cantus in China) is a new broad-spectrum fungicide belonging to the carboxamide class. It inhibits the enzyme succinate ubiquinone reductase (Complex II), also known as succinate dehydrogenase, in the mitochondrial electron transport chain [214–215].

A new fungicide, namely manganese prochloride chloride, proved to be effective in delaying both mycelial and carpogenic germination of *S. sclerotiorum*; thus, it has both a protective and therapeutic effect on the disease [216].

A combination of cultural and chemical means can be used to control S. sclerotiorum. Once again, crop rotation is an important practice to reduce the sclerotia population in the soil. Due to the long survival of sclerotia, the use of deep ploughing is questionable. It has also been found that drip irrigation can dramatically reduce the incidence of sclerotinia.

It should be noted that the degree of stem rot damage in cruciferous crops is assessed on a scale of 0–4 as follows [217]:

0 = No visible lesion;
1 = 0.1–2 cm lesions on the stem;
2 = 2.1–4 cm lesions on the stem;
3 = 4.1–6 cm lesion length on the stem and
4 = > 6 cm lesion length on the stem (completely dried plant).

Inoculum of S. *sclerotiorum* can be detected in field air samples (using the Burkard spore trap) and on petals by PCR analysis of nuclear ribosomal sequences.

The presence of S. sclerotiorum on plants can be detected by an immunological detection method, namely, dimeric single-chain variable fragments (scFv) of antibodies with affinity for the pathogen [218] and enzyme-linked immunosorbent assay based on polyclonal antibodies [219].

S. sclerotiorum infestation of petals can be rapidly detected using real-time and nested PCR methods [220]. Although the detection of *S. sclerotiorum* ascospores can be performed using a passive trap, bulk trapping and PCR methods can also be used to quantify ascospores [221–223].

Resistant cultivars with combined resistance to different isolates of white rust can be potential donors for further improvement programmes in oilseed rape [224]. Transfer of resistance to white rust in oilseed rape and mustard from *B. carinata* to *B. juncea* may be partially successful by growing disease-free plants under high disease pressure and then re-crossing them with *B. juncea* [225]. White rust resistance in oilseed rape and mustard is dominant, controlled by one or two genes with dominant-recessive epistasis or complete dominance in both gene pairs, but either gene, when dominant, is epistatic to the other. These genes can be located at

the same locus or at different loci [226]. Disease resistance at the true leaf stage and susceptibility at the cotyledonous leaf stage of the same genotype of *B. juncea* genotype appear to be regulated by two independent genes. Therefore, screening for white rust resistance at the cotyledon leaf stage is possible. Interspecific crosses between *B. juncea* and *B. napus* showed that resistance to *A. candida* is controlled by a single dominant gene.

In research [227] for of three interspecific crosses between *B. juncea* and *B. napus* revealed a digeneic control with epistatic interaction for white rust resistance trait and a close association between parental species and different degrees of leaf coverage.

Using diallel crosses between two white rust-resistant Canadian *B. juncea* cvs. Domo and Cutlass and two susceptible Indian *B. juncea* cvs. Kranti and Varuna, reported that the F_1 hybrids, except for susceptible × susceptible, were resistant; segregation for resistance in F_2 and test crosses was controlled by a single dominant gene in Domo and Cutlass, and that a recessive susceptibility gene was present in Kranti and Varuna.

Reported [228] on monogenic inheritance, which showed complete dominance in four crosses and no dominance in seven crosses between *B. juncea* and resistance sources from different species. Resistance was reported to be dominant in all crosses except susceptible × susceptible, where it was recessive. Under controlled conditions, inoculation with three different races of *A. candida* of the F_2 population from resistant × resistant crosses showed that resistance genes can be located at the same locus or at different loci. The partial resistance of *B. napus* to *A. candida* is controlled by a single recessive gene, designated wpr, with variable expression [229].

Resistance to white rust in *B. juncea* [230–232] is controlled by a single dominant gene. These studies have shown that only one resistance allele is sufficient to cause an incompatible response in this pathosystem.

Resistance genes were mapped and identified on *B. juncea* chromosomes, namely:Acr [233], AC-21 [234], AC-2, ACB1-A4.1 та ACB1-a5.1 [235]; *B. rapa*, namely ACA1 [236]; *B. napus*, namely ACA1 [237] та AC 2V1; та *A. thaliana*, namely RAC-1, RAC-2, RAC-3 та RAC-4 [238], effective against one or more than one race *A. candida.* One gene (Acr) responsible for resistance to *A. candida* was identified in a densely populated population of *B. juncea*.

Two closely related RFLP markers (X42 and X83) were identified at a distance of 2.3 and 4 cM from the Acr locus, respectively. To date, the following have been processed [239] linkage mapping of genes controlling white rust resistance in B. napus. A polymerase chain reaction (PCR)-based split amplified polymorphic sequence (CAPS) marker was developed for the tightly linked randomly amplified polymorphic DNA (RAPD) marker OPB061000.

The data obtained from 94 recombinant inbred lines showed that the CAPS marker for OPBO61000 and the AFLP marker E-AAC/M-CAA350 flank the Ac2(t) gene at a distance of 3.8 and 6.7 cM, respectively. Validation of the CAPS marker in two different F_2 populations of Varuna × BEC-144 and Varuna × BEC-286 crosses showed its usefulness in marker assisted selection for white rust resistance.

Crop rotation helps to manage this pathogen. Disposal of plant residues affected by the disease, especially carrion, helps to minimise the accumulation of the inoculum in the soil. Excessive irrigation of crops should be avoided, which helps to reduce the damage. Clean, healthy and certified seeds should be used to avoid seed-borne white rust disease.

Since *A. candida* spores require free water to germinate, not just high humidity, minimising the appearance and duration of leaf wetness, as the use of a drip irrigation system can reduce infections.

As the disease is more prevalent in areas with high humidity or in moderately humid climates, planting crops during dry seasons can reduce infection and the spread of the pathogen.

Removing symptomatic weeds of disease reservoirs can also help control the prevalence of the pathogen, as the pathogen infects more than 240 different plant species [240].

The application of K to the soil as the main fertiliser at a dose of 40 kg/ha resulted in a significant ($P < 0.05$) reduction in the amount of white rust on leaves and pods. Early sowing dates can help reduce the prevalence of the disease and increase seed yields [241–243]. An appropriate sowing date must be determined based on location and other epidemiological considerations to ensure that disease can be avoided in different locations.

In recent years, the growing awareness of the problem of environmental pollution by pesticides and the development of fungicide-resistant

strains of plant pathogens has prompted plant pathologists to search for environmentally friendly tools in disease control.

Aqueous extract of *A. sativum* bulbs 1% (w/v), isolate of *T. viride* as a seed treatment and in combination with appropriate foliar sprayers was statistically not inferior to manoczeb, combination of metalaxyl 35 ES 6 ml/kg seed treatment + 0.2 g/l spray of combination metalaxyl + manoczeb in terms of the degree of rust development on leaves and the amount of pathogen on the plant [244].

The inhibition of oospore development in *A. candida* by the natural bioagent *Psuedomonas syringe* in the field has been reported. Spraying with leaf extract of Eucalyptus spp. [245] can also effectively control the disease.

The first work on chemical control of white rust focused on the use of copper-based fungicides to control the developmental stage of the disease on leaves. In this case, it is reported [246–247], that this cruciferous disease can be controlled by frequent spraying with copper-based fungicides.

It is recommended to use Bordeaux mixture or Perox to control white rust of cabbage and other diseases. With the development of dithiocarbamate, control of white rust was achieved by repeated applications of protective fungicides. However, these fungicides did not provide sufficient protection against the sporulation phase of the disease. Acylalanines, which are specifically active against peronosporous fungi, now allow for control of both the leaf and cotyledon stages of white rust by seed treatment or soil impregnation and fewer foliar sprays. Three sprays of Polyram M 0.2% at 15-day intervals were most effective in controlling white rust of B. campestris in Pakistan.

Many fungicides have been evaluated for their effectiveness against white rust of cruciferous crops in India. Benlat 0.1%, Calixin 0.1%, Difolatan 0.2%, Dithan Z-78 0.2%, Miltox 0.3%, Tiovit 0.3%, Mancozeb 0.2%, Mancozeb + Metalaxyl (Ridomil MZ 0.05%) and Ridomil 0.2% were effective in controlling both foliar and heading stages of the disease and increasing crop yields [249–255]. Application of Metalaxyl, metalaxyl + manoczeb (ridomil MZ) [256–258], aluminium tris, and the combination of metalaxyl 35 ES 6 ml/kg seed treatment + 0.2 g/l spray of metalaxyl + mancozeb combination are reported to be able to control the disease. Three foliar treatments with the fungicide at an interval of 15 days after the disease emergence or on the 40-45th day of crop growth provide effective and cost-

efficient disease control [259]. For maximum disease control and high seed yield, it is recommended to treat seeds with metalaxyl (6 g Apron 35 SD/kg of seeds) followed by three sprayings with Ditan M-45 or metalaxyl [260].

Table 4.13

Effective fungicides used worldwide against white rust in cruciferous vegetables [248]

Name of the fungicide	Active ingredient	Pharmace-utical form	Crops for which it is recommended for use
Ridomil Gold® SL	Metalaxyl-M	Emulsion concentrate	All types of cruciferous plants
Serenade® MAX	*Bacillus subtilis* strain QST 713	Water-soluble powder	All types of cruciferous plants in organic cultivation systems
Abound® FF	Azoxystrobin	Emulsion concentrate	All types of cruciferous plants
Чемпіон® WG	Copper hydroxide	Wetting powder	All types of cruciferous plants
Earth-tone® GF	Copper salts	The solution is ready to use	All types of cruciferous plants
Regalia® BC (organic)	*Reynoutria sachalinensis*	Emulsion concentrate	All types of cruciferous plants in organic cultivation systems

Seed treatment with Apron 35 SD protects the crop from white rust for at least 60 days [261]. To control the phase of entering the tube, foliar spraying with Mancozeb, Metalaxyl or a mixture of Mancozeb + Metalaxyl is necessary [262]. Adequate leaf cover and a single spray coinciding with the start of flowering are essential to prevent secondary spread of the disease and infection of flower buds.

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4.3. Integrated systems and methods for downy mildew and powdery mildew control in cruciferous crops

There is no single method or approach to control downy mildew in cruciferous crops that is appropriate, effective, environmentally friendly and cost-effective. It is always necessary to integrate available disease management methods.

The cultural control of downy mildew in cruciferous vegetables is largely a matter of sanitation and manipulation of the environment to the benefit of the host and the detriment of the pathogen. Since the pathogen survives as oospores in the tissues of the host plant, it has been suggested that infected plant residues should be removed, destroyed and burned together with weeds to limit the source of the primary inoculum.

In addition, the use of clean, well-drained soils with a two-year crop rotation using non-cruciferous crops is recommended. Measures to reduce the relative humidity around the plants through adequate aeration and avoiding dense sowing and weed control also contributed to the reduction of the disease incidence. Avoiding continuous rapeseed crops in the same field or next to a field sown with rapeseed in the previous year also helped to reduce *P. parasitica* infection. Widespread cultivation of one or only a few varieties of the same species can contribute to the spread of the disease.

In India, late crops of canola and mustard were reported to have a higher incidence of downy mildew than early or timely crops [1]. Fungicide seed treatment followed by foliar spraying is a common practice for the control of downy mildew in cruciferous vegetables.

In the period from the mid-1940s to the mid-1990s, downy mildew control in cruciferous crops was based on the frequent use of sprays or fungicides such as chloranil (spergon), copper-based products and cyneb. These products were subsequently replaced by other non-systemic fungicides such as captafol, daconil, dichlorofluanide, propineb, Bordeaux mixture, copper oxychloride, mancozeb, ziram, chlorothalonil and fentin hydroxide. Captafol, mancozeb, difolate, copper oxychloride, dichlorofluanide, propineb and metalaxyl were better than other fungicides on large numbers of cruciferous plants in several locations. The timing of fungicide applications, number and interval of sprays depend on the duration and type of crop grown.

Biological control. Garlic juice or aqueous extracts of garlic have been reported to be toxic to *P. parasitica*, which causes downy mildew of radish [2].

Lepidium graminifolium – bacteria that have been observed on the mycelium, conidia and conidia of P. parasitica to reduce conidial germination. Many sources of resistance to downy mildew in cruciferous plants have been identified in major host species from different parts of the world. Information is also available on the genetic interaction between the parasite and the host. Efforts are underway to develop downy mildew-resistant varieties of various cruciferous crops using traditional and biotechnological methods.

In the quadrangle of integrated management (chemical-cultural-biological-host resistance) of downy mildew in cruciferous vegetables, biological control has not been used in the field Breeding for resistance has been successful for only some cruciferous vegetables. Chemical control of this disease may not always be reliable, as P. parasitica has developed resistance to metalaxyl, which at one stage proved to be extremely effective in controlling downy mildew [3].

Thus, there is a clear need to develop sources of resistance that can withstand pathogenic variation. It is also possible that differentiated sources of host resistance may be useful in integrated control programmes if used in conjunction with fungicides; this would potentially prolong the effectiveness of both control procedures [4]. Other methods include sanitation, field practices such as sowing time, plant density, and judicious use of nutrition and irrigation to prevent inoculum levels from building up [3].

Figure 4.10 – Monitoring signs of downy mildew in winter oilseed rape [5

The level of infection with downy mildew depends on the resistance of varieties and hybrids, the amount of infection in the field and protection systems. An additional incentive for the spread of the disease is the fact that recently a lot of agricultural land has been planted with rapeseed. The difficulty of controlling downy mildew is due to its biological characteristics. The use of ineffective products leads to the accumulation of pathogens in the soil. Peronosporosis is difficult to control because it is a fake fungus, and most of the products on the market are designed to control real fungi.

As a result, the main measures for controlling downy mildew on cruciferous crops are:

1. Growing resistant varieties and hybrids.
2. Sowing with treated seeds.
3. Destruction of carrion, which is a reservoir of infection. Complete clearing of the field from the residues of the previous harvest
4. Observance of crop rotation with the return of cruciferous crops to the same field no more than once every 3–4 years.
5. Adhere to agrotechnology, especially in the context of thickened crops.
6. Maintain spatial isolation (at least 1 km) of cruciferous crops from the fields where they were grown in previous years.
7. Use of balanced mineral nutrition of the crop. Magnesium and Boron increase the resistance of plants to downy mildew.
8. Use of fungicides during the growing season according to the "List of Pesticides and Agrochemicals Permitted for Use in Ukraine" based on active substances such as carbendazim, azoxystrobin, cymoxanil, etc.

Preparations based on a mixture of carboxyl substances with thiram or the active ingredient carbendazim are also effective. Preparations based on iprodione or potassium dithiocarbamate and a mixture of fludioxanil and metalaxyl will also be effective. The first spraying should be done after the vegetation has resumed on winter cruciferous crops before the branching phase on spring and winter forms, and the second spraying should be done before the budding and flowering phase.

Application of fungicides at the end of flowering and on pods – systemic fungicide Arbalet, CS – 0.6–1.0 l/ha or systemic and contact fungicide Junker, WP – 2,5 kg/ha. Defined [6] controlling powdery mildew through exclusion, eradication, protection, immunisation and therapy. Of these methods, chemicals and genetic manipulation are probably the most

important at the moment, given the nature and mechanisms by which the pathogen reaches epidemic levels within a very short period of time after disease onset. Future projections of climate change worldwide, with rising temperatures and dry periods, may make the situation worse for powdery mildew to develop into epidemic forms. Under such conditions, chemical control is the best option to combat the disease from the initial stage. However, the cultivation of resistant varieties is prioritised wherever they are developed or available to protect against environmental contamination and to save cultivation costs that may be imposed in the form of chemical and labour costs. Limited sources of powdery mildew resistance have been identified in *B. alba, B. alboglabra, B. rapa* var. *brown sarson*, *B. chinensis, B. japonica* and *E. sativa.* Transgenic *B. napus* plants expressing bacterial catalase in the chloroplast can inhibit the growth of E. cruciferarum. These plants showed constitutive expression of catalase and polyphenol oxidase enzymes and high levels of free polyamines such as putrescine, spermidine and spermine [7]. Alternatives, such as cultural control based on the time of sowing to avoid a favourable period for the pathogen to multiply, have also been recommended and effective. Certain micronutrients and biological control agents also help to control cruciferous powdery mildew. Selecting appropriate sowing dates according to the area is a promising method of controlling the disease. Scheduling irrigation only at the 50% stem branching stage in cruciferous crops can control the disease [8].

Powdery mildew hyperparasites can be used as a means of biological control. Cultural practices such as mixed cropping and intercropping can also be useful in application. Several chemicals have been tested against cruciferous powdery mildew. Some of them have been shown to be effective in controlling cruciferous disease and preserving crop yields. For effective control of the disease, fungicides should be applied immediately after the disease appears, as it spreads very quickly after its occurrence. The effective active ingredients of fungicides studied are presented in the table below 4.14–4.18.

General cultural practices, such as the use of healthy seeds of improved varieties, weed and crop residue control, sowing at the recommended time, long crop rotation, maintaining optimal plant density, and a rational fertilisation system are effective in controlling the development of powdery mildew in cruciferous crops by preventing primary sources of inoculum and secondary spread of the disease in the field.

Table 4.14

Fungicides and chemical compounds tested for effectiveness against powdery mildew [8]

Fungicide, chemical compound*	Reference to the source of study
1	2
Acrix	[7]
Actidion	[9]
Ammonium copper carbonate	[10]
Ammonium sulphide	[11]
Eipron 35 SD	[12]
Arsenat	[13]
Bavistin	[14]
Bakor Bayleton Baytan Benomil Blitox-50	[15]
Boora	[16]
Bordeaux liquid	[17]
Burgundy liquid	[18]
$CaCo_3$	[19]
Calixin	[20–25]
Carbendazim	[26]
Chloranil	[27]
Chlorothalonil	[28]
Copper-lime dust	
Copper sulphate	[29]
Cycloheximide	
Dichloronaphthoquinone	[30]
Diphenoconazole	[31]
Dinitrocapril phenyl crotonate	
Dinocap	[32–34]
Elgethol	[35]
Elosal	
Fermat	[36]
Flusilazole	
Formalin	[37]
Hexaconazole	

(End of Table 4.14)

1	2
Iprodion	[38]
Karatan	[39–41]
Liquid lime sulphur	[42–43]
Lithium carbonate	[44-45]
Malachite green	[46]
Mancozeb	
Maneb	[47]
Manzat	[48]
Metalaxyl	
Morestan	[49]
Morosid	
Nimbicidin	[50]
Penconazole	[51]
Potassium permanganate	[52]
Propiconazole (tilt)	
Ridomil MS-72	[53-54]
Rizoleks	
Salicylic acid	[55]
Sodium bicarbonate	[56]
Sodium chloride	[57]
Sodium thiosulfate	[58]
Spergen	[59]
Sulfex	[60-62]
Sulphuric acid	[63]
Sulphur dust	[64]
Colloidal sulphur	[65–69]
Tebuconazole (Folicur 48 EC)	
Tiovit	
Topsin	[70–72]
Triadimephon	[73–74]
Tridemorph	[75]
Vegetable oils	[76]
Vulcanised rubber	[77]
Zinc sulphate	[78]

*The name of the chemical corresponds to the original publication.

Plant extracts and some biological control agents of fungal and bacterial origin have been shown to have antagonistic effects against powdery mildew of cruciferous plants (Tables 4.19–4.20).

Breeding resistant cruciferous varieties is also important in terms of pathogen control. Sources of resistance to the main host genes were identified in *B. juncea, B. rapa, B. napus, B. carinata, B. alba, A. thaliana* and *R. sativus* from different countries.

Table 4.15

Effect of time and number of fungicide sprays on the development of mustard powdery mildew [77]

Fungicide	Concentration (%)	Disease intensity (%) (T_1)	Reduction in morbidity (%)	Disease intensity (%) (T_1)	Reduction in morbidity (%)	Disease intensity (%) (T_1)	Reduction in morbidity (%)
Sufflex	0.20	32.0	65.2	70.0	30.0	7.5	92.5
	0.30	30.0	67.3	70.0	30.0	5.2	94.8
Topsin M	0.05	63.0	31.5	82.0	18.0	14.5	85.5
	0.10	62.5	32.0	80.5	19.5	12.5	87.5
Karatane	0.05	26.6	71.0	63.4	36.6	6.2	93.8
	0.10	24.7	73.1	60.2	39.8	4.3	95.7
Calixin	0.05	36.1	60.7	72.3	27.7	8.4	91.6
	0.10	35.0	61.9	70.4	39.6	5.2	94.8

T1 = One spray at the time of disease onset; T2 = One spray 10 days after disease onset; T3 = Two sprays, the first at the time of disease onset and the second 10 days after the first.

These sources are used for breeding varieties resistant to powdery mildew using conventional and biotechnological methods. It is emphasised [79] that, that the most effective, cost-efficient and environmentally friendly management of powdery mildew disease can be achieved through the use of resistant varieties that can be easily developed using resistance sources by transferring cloned resistant R genes into agronomically susceptible varieties (Figure 4.11).

Table 4.16

Influence of variants of powdery mildew control agents application on the formation of white mustard seed quality indicators [79]

Variant of the active substance	Concentration (%)	PDI	Number of abnormal seeds per 100 seeds	Percentage reduction to control	Seed germination (%)	Length of seedling, cm	Growth strength index
Hexanazole	0.05	35.00	18.66	45.65	92.75	12.86	1192.8
Colloidal sulphur	0.20	38.67	18.33	46.61	91.75	12.53	1149.6
Tebuconazole	0.05	35.33	19.00	44.65	90.75	12.74	1156.2
Tridemorph	0.04	23.33	18.00	47.57	92.75	12.81	1188.1
Diphenoconazole	0.05	51.67	19.66	42.73	90.50	12.70	1149.4
Onion extract *(Allium cepa* L.)	5.00	78.33	32.66	4.86	86.00	12.50	1075.0
Neem leaf extract *(Azadirachta indica* A.)	5.00	74.00	30.66	10.69	85.25	11.99	1022.1
Eucalyptus leaf extract *(Eucalyptus globulus* L.)	5.00	74.67	29.00	15.53	85.75	12.36	1059.9
Karan leaf extract *(Nerium indicum* L.)	5.00	73.33	30.00	12.61	86.00	12.25	1053.5
Karanja leaf extract *(Pongamia pinnata* L.)	5.00	76.33	33.00	3.87	84.75	12.45	1055.1
Culture of *Trichoderma viride*	3.00	65.67	23.33	32.04	87.75	12.51	1097.8

Table 4.17

Effect of different fungicides on the development of powdery mildew on white mustard [77–79]

Variant	Concentration, %.	Percentage of damage (%)	Technical efficacy of the product (%)
Galixin	0,2	33,4	62,88
Hexanazole	0,2	30,6	66,0
Bovistin	0,1	46,5	48,3
Colloidal sulphur	0,2	40,3	55,2
Topsin M	0,2	63,7	29,2
Blitox-50	0,2	49,9	44,5

Table 4.18

Effect of different treatments on the development of powdery mildew on white mustard [77–79]

Variant	Pathogen infestation after application (%)
Garlic extract	50.6
Apron 35 SD	22.0
Carbendazim	16.0
Apron 35 SD + Carbendazim	13.0
Apron 35 SD + Ridomil MC	29.0
Mankotseb	59.6
Carbendazim + Ridomil MC	34.0

Table 4.19

Bio-agents tested against powdery mildew of cruciferous plants

Bioagents	References
Allium sativum (bulb extract, 1%)	[81–82]
Azadirachta indica (leaf extract, 2%)	[83]
Datura stramonium (leaf extract, 2%)	
Eucalyptus (leaf extract, 2%)	[84]
Trichoderma harzianum (10 g/kg)	[85]
Trichoderma harzianum (10^8CFU/кг)	[86]
T. viride (suspension)	
Ampelomyces quisqualis (10^8CFU/ml)	[87]
Nerium indicum L. (Karan) (leaf extract, 5%)	
Pseudomonas fluorescens (oil (10 g/l water))	[88]
Pongamia pinnata L. (Karanj) (leaf extract, 5%)	
Pseudomonas fluorescens (10^8 cells/ml)	[89]

Plants have developed a well-organised defence system against fungal attack by the powdery mildew pathogen. For example, cuticle wax acts as an important physical barrier to prevent fungi from entering the host cell [90], особливо для патогена, який росте поверхнево на рослинах [91]. Moreover, the host organism demonstrates a number of immune responses, including the accumulation of reactive reactions (Figs. 4.13–4.17).

Figure 4.11 – Rapeseed pods and stems infected with Erysiphe cruciferarum in fungicide-treated plants (left) and untreated plants (right) [79]

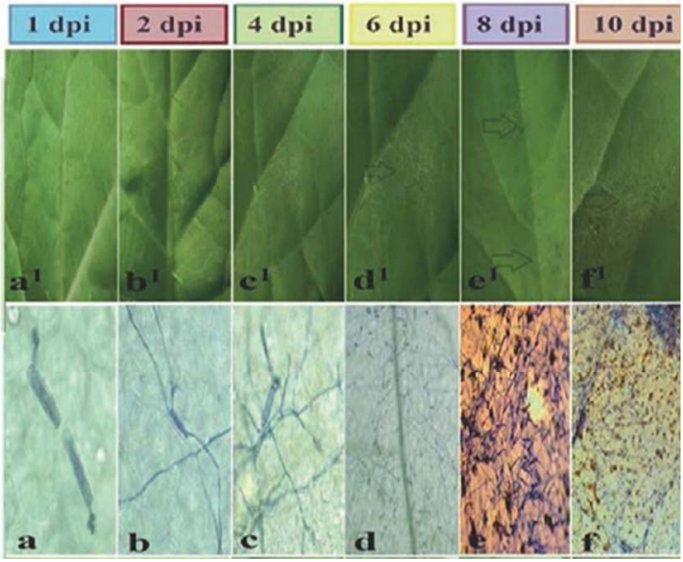

Figure 4.12 – Symptoms of *Erysiphe cruciferarum* on leaves infected with *B. napus* (pathogen resistant form) at six time points of recording. Symptom images were (a1, b1, c1, d1, e1, f1) and light micrographs were (a, b, c, d, e, f) at 1, 2, 4, 6, 8 and 10 days (dpi) after infection, respectively. Scale bars for light micrographs are 25 μm [89]

Some cruciferous species, such as S. *alba, E. sativa* and *R. sativus*, showed complete resistance to powdery mildew in both years of research. Varietal resistance provides an opportunity for effective management of powdery mildew in oilseed rape [92]. Since host resistance plays an important role in the spread of the disease, genetic engineering is a promising tool for reducing infection rates. There are promising results of increased resistance to *E. cruciferarum* in transgenic lines of *B. juncea*. The content of glucosinolates or the release of their volatile metabolites or other nitrogen- and sulphur-containing phytochemicals, such as phytoalexins and glutathione, which are synthesised after pathogen damage, may be one of the reasons for the different stress resistance of cruciferous plants [93–95]. In addition, daily temperature and light intensity, not to mention soil pH, influence stress tolerance, as the combined effect of UV-B radiation intensity and temperature was probably the reason why *B. juncea, B. napus* and *E. sativa* grown in the field contained four times more glucosinolates than those grown in the greenhouse [96]. Finally, the higher resistance of *S. alba, R. sativus* and *E. sativa* to powdery mildew compared to *B. juncea, B. napus* and *B. nigra* may also be related to morphological features of the plants, such as the density of non-glandular trichomes on the plants [97]. For example, a sparse distribution of trichomes is characteristic of *B. napus* leaves, while *B. villosa* leaves are much more densely and evenly covered with trichomes [98].

In general, a large number of non-glandular trichomes increases the reflectivity of the leaf surface and thus increases the resistance of plants to drought stress [99]. However, they also capture airborne particles, including fungal spores, and thus a higher density of trichomes can increase the risk of fungal infections [100]. Non-glandular trichomes are an excellent habitat for pathogens if they do not contain an antifungal hydrolase, which inhibits or reduces fungal infection by hydrolysing the fungal cell walls. Thus, based on the knowledge of glucosinolates and trichomes, attempts have been made to increase stress tolerance in, for example, B. napus through genetic engineering by transferring the tissue-specific lipid transfer protein from B. rapa to B. napus to increase the number of trichomes and glucosinolates in the leaves of plants [101].

Currently, there is a limited range of alternative cruciferous and non-cruciferous oilseeds for cultivation in the northern regions, and there

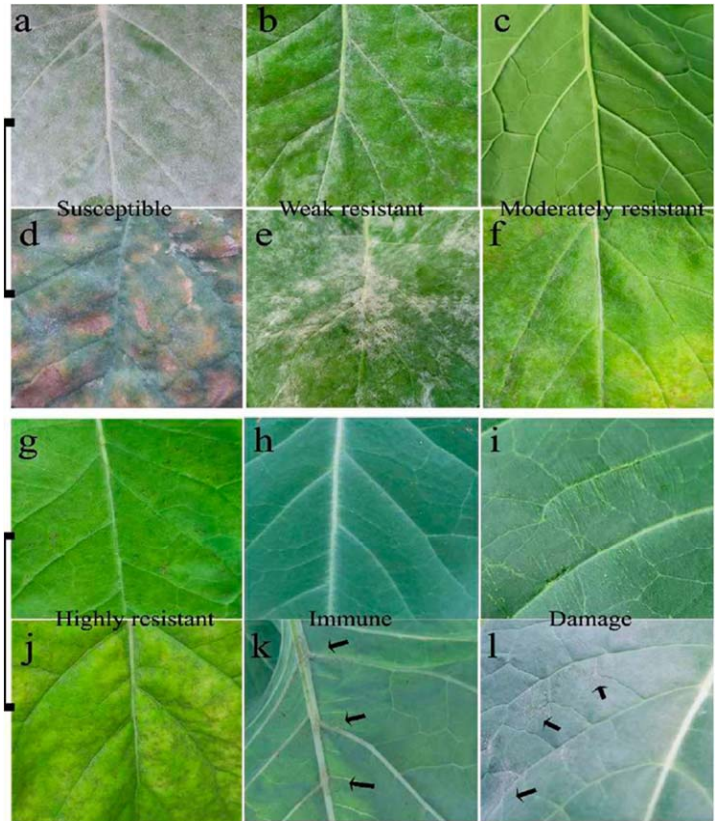

Figure 4.13 – Phenotypic differences in leaves of different plant samples after inoculation with powdery mildew pathogen. The susceptible variety (a) and the low-resistant plant (b) showed more hyphae and mycelium than the medium-resistant plant (c) and the highly resistant plant (g). Necrotic lesions on old leaves (d-f, j) of the above plants. No hyphae were found on the leaves of the immune plant (h), but several brown dots were observed on the old leaf (k, arrows). Spores failed to penetrate the leaves of the immune plant where surface damage was caused by sandpaper friction (i), but light infection (l, arrows) was observed on unhealthy plants with lateral damage [102]

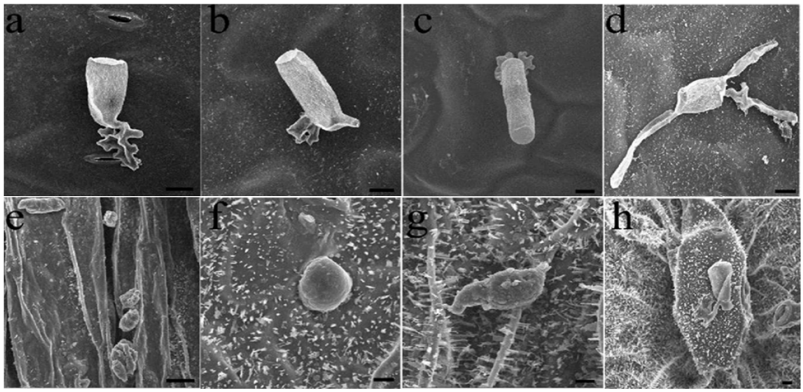

Figure 4.14 – Scanning electron micrographs of *E. cruciferarum* developing on rapeseed leaves (top row, non-susceptible plant, bottom row, resistant plant). Penetration into the host cell and formation of an appressoria on the stomatal cell (a), stomatal cell (b) or furrow between cells (c). Secondary hyphal growth after cell penetration (d). Dead cones on the leaf epidermis of a moderately resistant plant (e), a high-temperature resistant plant (f) and a pathogen-penetrating plant (g) after powdery mildew infection. Scabs formed as a result of shrinkage and condensation of tissue around the infection site (h). Scale bar 50 µm [99]

is a significant demand for new oilseeds from local producers [103]. Alternative oilseeds, such as *S. alba*, *E. sativa* and *R. sativus*, even under favourable conditions for powdery mildew infection, have great potential for chemical-free cultivation in different parts of the world. Since powdery mildew is a significant threat to oilseeds, it is essential to use available resistant species and resistant varieties, especially in organic farming systems. This knowledge is also important for the production of cover crops and green manure to suppress pathogens [104–105], and therefore for the selection of species and varieties that are less susceptible to the spread of the disease. White mustard (*S. alba*) may be particularly interesting as it is very suitable as a cover crop due to its rapid initial growth, high glucosinolate levels and low maintenance [106–107] (Table 4.20).

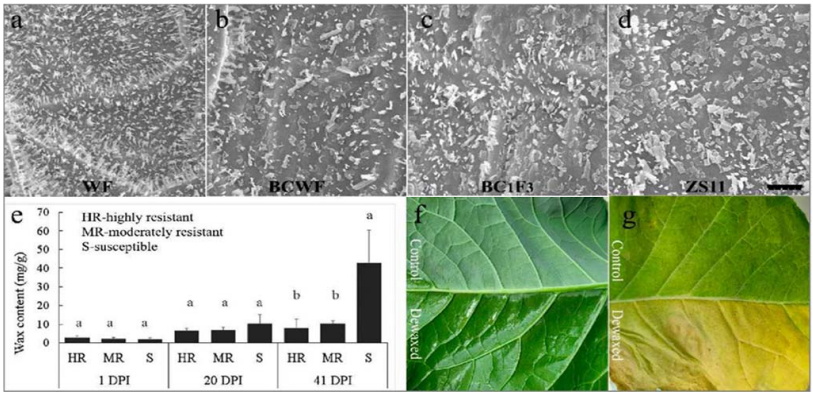

Figure 4.15 – Comparison of the shape and content of wax plaque on different plants and the effect of partial washing of surface wax. Samples in (a-d) are a resistant 'WF' and a moderately resistant 'BCWF' plant in the BC1F3 and 'ZS11' populations, respectively. The cuticle wax of 'BCWF' and 'WF' is needle-shaped. The wax crystals of the resistant 'WF' and 'BCWF' plants are tubular or short and tubular, whereas those of 'ZS11' are mostly flaky. Cuticle wax content in leaves of plants with different levels of resistance (e). Different letters indicate significant differences ($p < 0.05$). Removal of surface wax from half of the blade of a young leaf of 'WF' (f) leads to early senescence but not to changes in powdery mildew susceptibility (g) [99]

Table 4.20

Priorities for cruciferous powdery mildew management strategies [107]

Priorities	Order of importance
Resistant varieties	1
Application of fungicides	2
Seed production	3
Sanitation	4
Crop rotation	5
Mixed crops	6
Density of standing	7
Date of sowing	2
Balanced nutrition	4

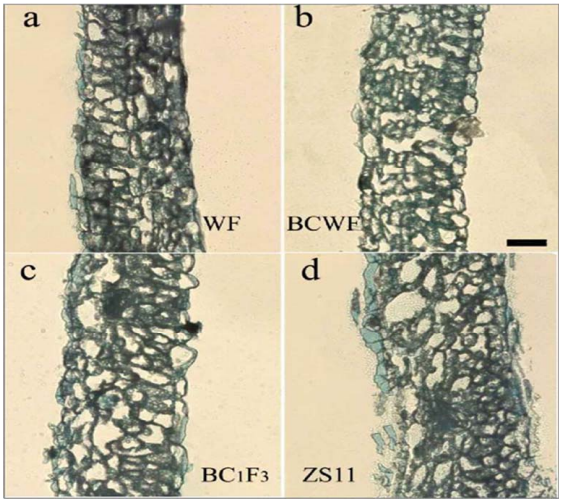

Figure 4.16 – Cross-sections of leaves of four plant samples: 'WF' (a), 'BCWF' (b), 'BC1F3' (c) and 'ZS11' (d). Images have the same magnification and scale bar = 100 µm3068 (resistant 'WF' and moderately resistant 'BCWF' forms to powdery mildew)

It is noted that [107] the need to prioritise powdery mildew control strategies according to the emergence of the disease in an area and the extent and intensity of the disease. The order of prioritisation in the table can be manipulated depending on the timing of disease emergence by using different combinations of control strategies for which the underlying pathogenicity data will be useful in making the appropriate technological decision.

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4.4. Integrated systems and methods for phomosis control in cruciferous crop agrocenoses

Global rapeseed crop losses from phomosis are estimated at more than $900 million per growing season [1–2].

Phomosis is one of the most damaging diseases of rapeseed *Brassica napus* L. ssp. *oleifera* (Metzger) Sinsk. The growing risk of epiphytic development of this disease is associated with the expansion of the area under rapeseed, shorter terms of returning the crop to its original place, low genetic diversity of varieties, a relatively wide range of pathogens, and increased marketing exchange of seeds between countries. The disease is of great economic importance in the main rapeseed growing areas [3]. Yield losses are usually at least 10%, but in some years with high intensity of the disease, they can reach 30–50% [4–6]. (рис. 4.17–4.19).

Phomosis is a very significant problem – the shortfall in rapeseed production due to this disease is 5–20% [7], 50% [8–11] and 75–90% [12–14].

Phomosis is an oilseed disease of international importance. In areas where cruciferous oilseeds are grown (especially in Australia, North America and Europe), this disease can cause significant yield losses [15]. The disease is associated with two closely related fungi, *Plenodomus lingam* and *P. Biglobosus* [16]. These fungi have been classified as Leptosphaeria, but recent studies have shown that they should be classified as Plenodomus [17–18]. The coexistence of these two pathogens has been reported in various European countries, including Poland and Ukraine [19], Lithuania [20] and the Czech Republic [21].

The name ‘rapeseed phomosis’ covers not one, but at least four different diseases, each with its own unique characteristics. The most common causative agent of phomosis in Ukraine is the fungus *Phoma lingam* (Tode: Fr.) Desm. (marsupial stage of *Leptosphaeria maculans* (Desm.) Ces. et De Not.). Due to its ability to produce the nonspecific toxin sirodesmin PL, this fungus causes the greatest damage to rapeseed plants.

The pathogen causes the appearance of large (up to 3 cm in diameter) necrotic spots on the leaves and stem, as well as in the root collar area. The density of the affected tissues gradually decreases, cancerous ulcers and signs of dry rot are observed. During strong gusts of wind, the stems can break. Numerous black fungal pycnidia with conidia are formed on

necrotic rapeseed organs, which contribute to the spread of the disease during the growing season. The marsupial sporulation formed on overwintered plant residues is one of the main sources of infection in spring. Rapeseed disease caused by the fungus *Leptosphaeria macu*lans is often referred to in the old phytopathological literature as A-form of phomosis. Another causative agent of phomosis is the fungus *Leptosphaeria biglobosa* Shoemaker et H. Brun.

The main difference between this species and the previous one is the inability to produce the toxin syrodesmin PL, so the signs of phomosis during the development of this pathogen are not so pronounced.

Necrotic spots on leaves and stems are much smaller (1–3 mm) and dark in colour. The number of pycnidia with conidia on the plants is also significantly lower. This manifestation of phomosis is known as B-form of phomosis.

This form dominates in Asian countries, is quite common in France and the Netherlands, and is probably present in Ukraine. However, its role in the occurrence of rapeseed phomosis in our country remains unexplored. The third pathogen is the fungus *Phoma sublingam* Boerema with the pouch stage of *Leptosphaeria submaculans* L. Holm. This species is distributed only in European countries and has not yet been recorded in other regions of the world. *Leptosphaeria submaculans* can infect various

Figure 4.17 – Leaf spot of phomosis, (upper position – the beginning of infection, lower position – partial death of leaves at the later stages of pathogenesis [22]

Figure 4.18 – Phomosis development in a pathogen-resistant winter oilseed rape variety

Figure 4.19 – Classic symptoms of stem canker are a sign of phomosis infection in rapeseed [23–25]

members of the cruciferous family, so rapeseed is not the main substrate of this species. It is usually found in large numbers on weeds such as dry ribweed (Sisymbrium), hiccup (Berteroa) and yellow brome (Erysimum). The intensive development of this form of phomosis is observed in the case of weed infestation of fields and roadsides. The fourth causative agent is the fungus *Didymella macropodii* Petr., which parasitises plants in the asexual sporulation stage of *Phoma nigrificans* (P. Karst.) Boerema, Loer. et Wittern. An important ecological feature of *Phoma nigrificans* is its high tolerance

to cold. It develops massively during the cold season, so its substrates are mainly perennial or winter cabbage plants: horseradish (*Armoracia rusticana* Gaertn., Mey et Scherb.), winter rape (*Brassica napus* L. var. *oleifera* Metzger), and sometimes field thistle (*Thlapsi arvense* L.). Until recently, this species was recorded only in Northern Europe, but now it is known in Ukraine. This form of phomosis can infect various plant organs, but the most dangerous is root collar rot in winter rape crops.

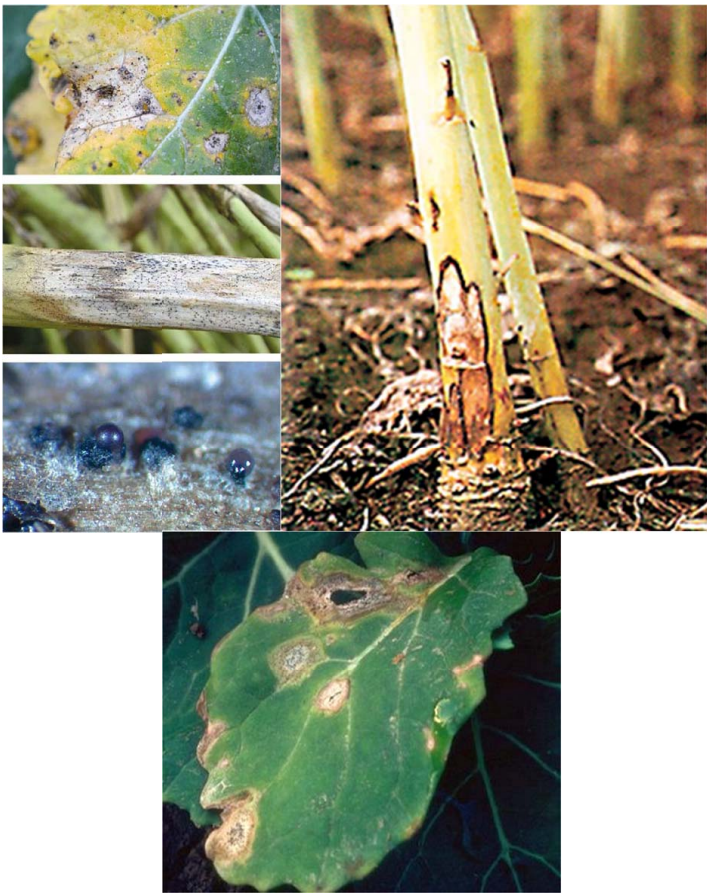

Figure 4.20 – Signs of rape phomosis [26–29]

Figure 4.21 – Both photos show phomosis damage to rapeseed leaves. Note the dead area in the centre, surrounded by a yellow zone. The small black dots in the centre of the lesion are asexual fruiting bodies of the fungus. Spores are formed inside the fruiting bodies and can be spread by rain and spray [30]

A number of studies have additionally reported that Leptosphaeria maculans (Desm.) Ces. & De Not. (anamorphic stage of Phoma lingam (Tode: Fr.) Desm.) and *Leptosphaeria biglobosa* R.A. Shoemaker & H. Brun are the causative agents of rape phomosis. Until recently, *L. maculans* was considered a complex species, within which two groups of isolates were distinguished. These groups were named by different researchers as highly virulent (HV) and weakly virulent (WV) [31], virulent and avirulent [32], aggressive and non-aggressive [33], Tox+ i Tox^0, pathotypes A and NA [35], Group A and Group B [36]. Groups differ in the morphology of colonies on nutrient media [37]. The significant difference between isolates of groups A and B in a number of features allowed us to hypothesise that they are different species [38] (Figs. 4.22–4.23).

They found [41] also morphological differences in the structure of pseudothecia of isolates of these groups and isolates of group B into an independent species Leptosphaeria biglobosa. In the life cycle of rapeseed phomosis pathogens, there are marsupial and conidial stages. Pycnidia develop on spots on leaves, stems and pods, as well as on dead

Figure 4.22 – Cotyledons of winter rape infected with: left: *Plenodomus lingam* and right: *Plenodomus biglobosus* [39]

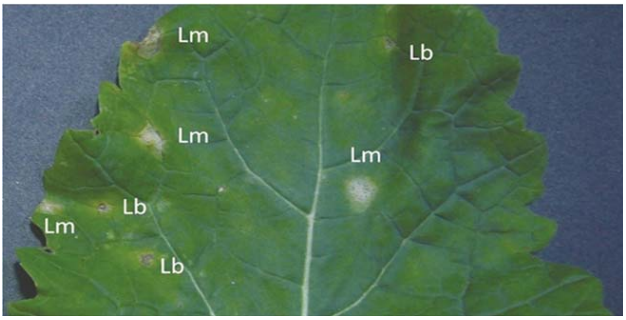

Figure 4.23 – Two closely related pathogens, *Leptosphaeria maculans* (Lm) and *L. biglobosa* (Lb), cause phomose leaf spot and stem cancer [40]

plant parts (Figs. 4.24–4.26). Pseudothecia are formed on dead lignified parts of infected plants. Pycnidia of both species practically do not differ in morphology. The pseudothecia of *L. biglobosa* differ from those of *L. maculans* by the presence of proboscis swollen at the top [42–45]. They are thought to have evolved from a common ancestor, with L. biglobosa being the older species [46–47].

*L. maculans (*anamorpha *Phoma lingam* (Tode: Fr.) Desm.) is an economically important pathogen because it causes stem cancer [48].

The causative agent of phomosis was first described by Tode [49] in 1791. The pathogen has a wide range of host plants within the Brassicaceae family, including wild and cultivated species, including the model species for genetics, *Arabidopsis thaliana* (L.) Heynh [50]. In 2001, weakly virulent isolates of this species were assigned to the species *L. biglobosa* [51].

It is believed that the prevalence of *L. maculans* and *L. biglobosa* in different countries of the world is due to seed transmission of *B. oleracea* L., *B. napus* L., *B. rapa* L. and other Brassica [53]. Previously, only *L. biglobosa* was found in North America and Eastern Europe, but then *L. maculans* was discovered [54]. Until the mid-1990s, the incidence of phomosis in Poland was usually also associated with *L. biglobosa* [55–56].

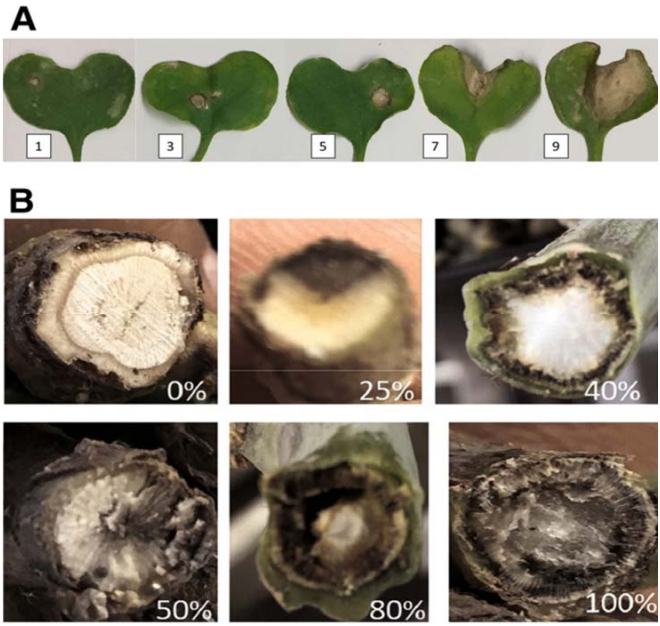

Figure 4.24 – Phomosis severity scale (black leg) used to assess damage (A) at the seedling stage on cotyledon leaves and (B) at the adult stage in percentage of internal tissues in the stem apex area of spring and winter rape, mustard species [50]

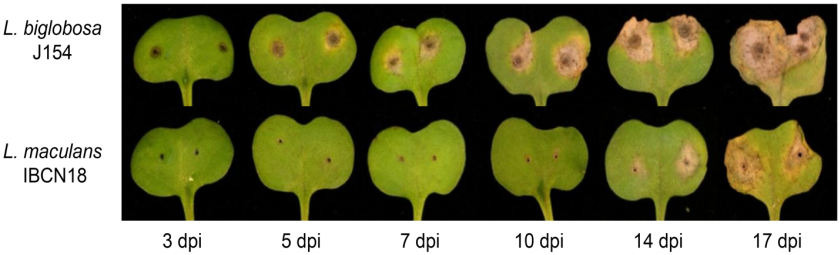

Figure 4.25 – Symptoms on cotyledons of *B. napus* cv. Westar infected with L. maculans brassicae or *L. biglobosa canadensis*. Cotyledons of *B. napus* cv. Westar were wounded and inoculated with spores of *L. maculans brassicae* or *L. biglobosa* canadensis. The disease developed within 17 days after inoculation (dpi). Cotyledons were harvested and photographed at 3, 5, 7, 10, 14 and 17 days (dpi) after inoculation to monitor the development of the lesion by the two pathogens [52]

A recent study of Leptosphaeria spp. on rapeseed leaves in Poland showed an increase in the number of L. maculans isolates compared to their prevalence ten years ago [57]. Changes in the relative frequency of these two pathogens were also found in the Czech Republic and Hungary [58]. The study of the distribution of species in Lithuania showed a variation in their proportion depending on environmental conditions, but in general, 70.3% of *L. maculans* and 29.7% of *L. biglobosa* isolates were detected [59]. These results indicate a west-to-east spread of *L. maculans* in Europe. Currently, *L. maculans* is a threat to rapeseed in Asia. *L. maculans* is present in many countries (except China) where cruciferous crops are widely grown [60]. In China, only *L. biglobosa* was found on rapeseed [61]. In Europe, yield losses are not attributed to *L. biglobosa*, as the pathogen, which affects leaves and the upper part of the stem, usually does not lead to plant death [62].

However, in countries with high summer temperatures, such as Poland and Ukraine, this species can cause significant losses in rapeseed yields [63].

It was believed that the dominance of *L. maculans* over *L. biglobosa* was due to the low aggressiveness of the latter, as *L. biglobosa* develops

only on aging plants at the end of the season and does not lead to significant phomosis damage. However, recently it has been found that this pathogen can cause significant damage not only to the upper part of the stem, but also to its base, leading to large yield losses. Under conditions of high relative humidity, *L. biglobosa* isolates become highly aggressive in cotyledon damage, as less lignin accumulates in the plant cell walls [64].

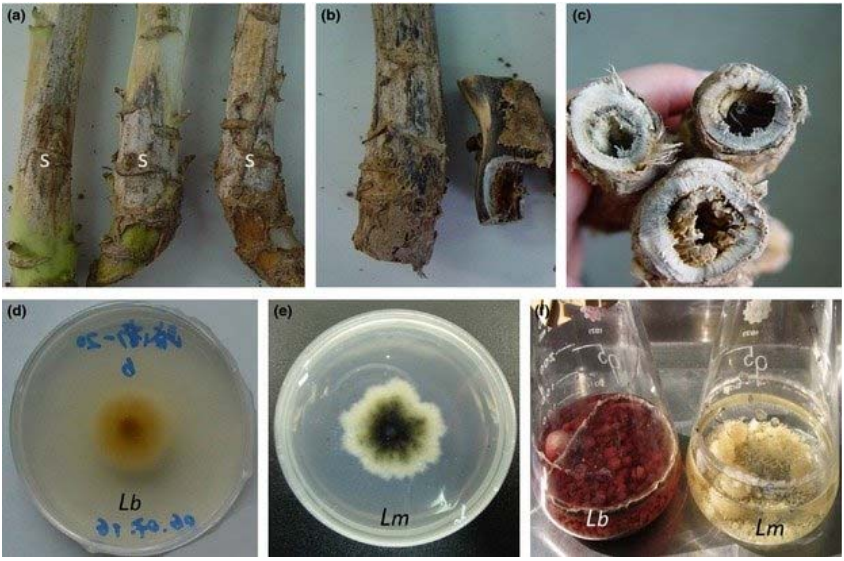

Figure 4.26 – Phomosis stem blight in oilseed rape and identification of Leptosphaeria species causing it. External cancer symptoms (s) observed on the upper stems (a) and stem bases (b) of winter oilseed rape sampled before harvest (black dots are *Leptosphaeria biglobosa* pycnidia), with cross sections showing internal necrosis (c). Potato dextrose agar tests showing the presence of *L. biglobosa* (Lb) (d) but not *Leptosphaeria maculans* (Lm) (e), and Chapeka-Dox broth tests (f) showing the presence of *L. biglobosa* but not *L. maculans* were used to confirm the presence of *L. maculans* or *L. biglobosa* in seeds imported from Canada, Australia and Ukraine [64]

Both pathogens are hemibiotrophs and have a complex life cycle. The fungus can survive as a saprophyte on stem and stubble residues for many years. Once it gets on the leaves, it becomes a necrotroph, producing spots on the leaves. Then, as a biotroph, the pathogen colonises the intercellular spaces, while the endophytic latent stage, where the fungus lives dormant, is asymptomatic. After colonising the intercellular spaces, L. maculans reaches the vascular system of winter rape and spreads down the xylem to the base of the stem within 9 months (in Europe). As a result, the stem bark is destroyed and stem cancer develops [65], that is, the second necrotrophic stage begins at the end of the plant growing season.

The life cycle of the fungus occurs over a wide temperature range. It takes 5 days at 20 °C and 2 weeks at 8 °C for lesions to appear on leaves inoculated with ascospores [66], and the time between the manifestation of lesions on the leaves and their manifestation on the stems is 77 days at 18 °C and 175 days at 3 °C [67].

At high temperatures, the severity of symptoms on cotyledons, leaves and stems increases, so the threat of phomosis increases with climate warming. It was noted that incompatible reactions (small necrotic lesions) on cotyledons introduced at 18 °C by avirulent isolates change to fully compatible reactions at 27 °C [68], which indicates the temperature sensitivity of resistance genes, so, for example, the disease develops faster at 24 °C than at 14 °C. High summer temperatures can lead to serious phomosis epidemics. Pseudothecia ripening depends on humidity and temperature (optimum 14–15 °C) [69].

Pseudothecia usually form on the stubble 9–10 months after harvesting, as temperatures below 0 °C in winter delay their maturation [70]. Залежно від умов навколишнього середовища період вивільнення аскоспор може тривати 3–4 місяці і більше, а пік спостерігається зазвичай через 1–2 місяці після його початку [71]. For germination of ascospores after their release, at least 8 hours of humidity at 4–28 °C (optimum 15–20 °C and 48 hours of humidity) are required [72]. The survival of *L. maculans* on stubble plays an important role in the epidemic, as ascospores and pycnidiospora from infected residues can serve as primary inoculum for infection of rapeseed in spring. The preservation of the fungus on infected residues is influenced by weather conditions and agricultural practices. The rate of degradation of residues depends on soil moisture and temperature,

and dry summers and cold winters are favourable for the pathogen's survival. In Western Canada, for example, ascospores are released from phomosis-infected stubble on the soil surface within 3–5 years because winters are very cold and summers are dry and hot [73].

Survival of the fungus on rapeseed stubble for 3–5 years exceeds the duration of the crop rotation (3 years on average). In Western Australia, L. maculans can survive for up to 4 years because infected canola residues do not decompose in hot, dry summers [74]. In the UK, where the climate is mild and humid, oilseed rape residues take 2 years to decompose. Therefore, proper crop rotation and stubble removal are effective farming practices for controlling phomosis, as this reduces the amount of inoculum available for overwintering [75].

In the generative stage (teleomorphs of *L. maculans, L. biglobosa*), the fungus sexually forms asci with ascospores developing in pseudothecia on lignified remnants of rapeseed roots or stems. In the vegetative stage (anamorph of *Ph. lingam*), the fungus forms almost superficial spherical black pycnidia with a thick sclerotial membrane, which produce colourless pycnidiospora. With sufficient moisture, pink-purple mucus containing vegetative spores is released from the pycnidia. Phomosis pathogens overwinter in the form of pycnidia and pseudothecia on stubble [76] in the form of mycelium in infected plants of winter rape and on the affected residues of cruciferous crops, in the form of mycelium or pycnidia on the seed coat [77–78]. The disease development cycle begins with the airborne spread of ascospores, which are released from pseudothecia (in Europe, for example, both in autumn and spring) and, when germinating, inoculate plants through stomata or in wounds (primary infection) [79]. Ascospores from the primary source of infection can be transported 1–2 km, up to a maximum distance of 10 km [80]. Recently, it was reported that in Canada, the potential for windborne ascospores of the pathogen is 17 km per year, and in China, 47 km per year in spring rapeseed and 70 km per year in winter rapeseed [81]. Shortly after leaf infection, pycnidia with pycnidiospora form on the leaves, which are carried by rain over short distances of up to 1 m and usually cause a less severe secondary infection. However, it has been noted that, for example, in Western Canada, pycnidiospora can also cause primary infection. Secondary cycles produced on affected plants by pycnidiospora do not lead to significant yield reduction, so the pathogen is considered a monocyclic pathogen [82–83].

Symptoms of the disease are observed on the hypocotyl and cotyledons, leaves, stems, pods and roots [84–86]. When infected seeds are sown, watery spots of various shapes appear on the hypocotyl and cotyledons of young plants, which, when dry, become grey with black dots (pycnidia) on the surface. Later on, rounded or elongated, slightly depressed light brown or greyish spots or ulcers covered with black pycnidia appear on the stems near the petioles of the lower leaves. As they grow, the spots and sores cover the stem all around. This form of the disease is called stem cancer. When the base of the stem is affected (root neck cancer, neck necrosis), *L. maculans* often spreads to the root system, causing dry root rot, which leads to lodging and death of plants. Sometimes phomosis is observed in the form of necrotic grey spots with dark pycnidia on the internodes. On the leaves and pods, the disease manifests itself in the form of grey, dry oval spots with concentric zonation and pycnidia. Affected pods crack and have small, wrinkled, tentacled seeds. In Australia, a close correlation was found between the frequency of cotyledon damage and the subsequent development of stem cancer [87]. In winter oilseed rape, the most damaging damage to the stem base is usually associated with phomose leaf spot if it has developed before the rapid elongation of the stem.

The global spread of rapeseed phomosis can cause serious yield losses in Europe, Australia and North America [88–89]. The harmfulness of phomosis is manifested in a decrease in germination of infected seeds, death of young affected shoots in autumn, loss of diseased plants during wintering, death of adult plants due to cancer of the stem base, reduction of the assimilation surface due to premature death of affected leaves, reduction of fodder qualities of green mass, significant reduction in the weight of 1000 seeds, deterioration of technological properties of seeds.

Certain differences in the morphometry of both types of phomosis pathogens of cruciferous plant species were also determined [89]

*Leptosphaeria maculans (*Desm.) Ces & De: Pseudothecia on stems initially recessed, then superficial, scattered, globose to pear-shaped, flattened at the base, 300–400 (500) μ in diameter, firm, smooth, glabrous or with few brown mycelial strands; when artificially inoculated with numerous smoky brown, septate, curved hyphae, 2–3 μ wide, sometimes covered with dark brown globules. Proboscis central, truncate-conical, papillary 90(100)×100 μ with 5–8 layers of scleroplectenchymal cells

3–5(10) μ in diameter. Stomata 60-100 μ wide, initially filled with hyaline pseudoparenchymal cells 8-10 μ in diameter, later open. The surface of the pseudothecia is globular to prismatic in texture, built of brown thick-walled cells, 8–12 μ in diameter. Lateral wall 30–70–100–150 μ thick, 3 zones are distinguished in it: outer zone consists of 2–3 (5) layers of isodiameter brown, scleroplectenchymal cells, 4–7 μ in diameter, central zone consists of 4–6 layers of prismatic brown scleroplectenchymal cells, 8–15 × 4–6 μ, inner zone consists of 2–7 layers of prismatic hyaline to yellowish pseudoparenchymal cells, 8–15 × 4–9 μ. Pseudoparaphyses numerous, 2–3 μ wide, septate, with anastomoses. Pouches numerous, bitunicate, cylindrical, to almost club-shaped, rounded at the top, on short stalks, 100–120(150) × 12(18)–21(22) μ with 8 ascospores. Ascospores rolling, elongate spindle-shaped, straight or slightly curved, (45)50–60(68) × 6–7 μ, with 5 septa, central cells largest, yellowish, with 1–2 drops per cell, smooth, with conoid to globose terminal appendages, 5–6 μ in diameter [90].

Leptosphaeria biglobosa R.A.Shoemaker & H.Brun: Pseudothecia on stems scattered, subepidermal, later breaking through, globose to pear-shaped, flattened at the base, 280–350 μ in diameter, hard, brittle, smooth, covered with a layer of loose hyaline mycelium. Upper part of the pseudothecia and proboscis with numerous smoky-brown septate, curved hyphae, 2–3 (5) μ wide; hyphae sometimes bearing dark brown globules. Proboscis central, almost cylindrical, pear-shaped in reverse, 200–400 μ long, 200–300 μ wide, with 8–10 (15) layers of polygonal scleroplectenchymal cells, 5–8 μ in diameter, with occasional scattered large cells 25–30 μ in diameter. Stomata 60–100 μ wide, filled with pseudoparenchymal cells 8–10 μ in diameter, sometimes with hyaline periphyses, 10–20 × 5–6 μ. Swelling of the proboscis in the upper part is often noted. The surface of the pseudothecium shell has a globular texture, composed of thick-walled brownish cells 8–10 μ in diameter. The wall of the pseudothecium in the lateral part is 50–75 (100) μ wide, consisting of 4–7 (10) layers of prismatic to isodiameter cells 10–15 × 8–12 μ. The outer several layers are dark brown, scleroplectenchymous, the inner layers are pale brown, scleroplectenchymous at the base of the proboscis and in its wall, the main cells are thin-walled. The innermost layers are composed of spherical, hyaline cells. Pseudoparaphyses are numerous, 2 μ wide,

20–25 μ long, septate, with droplets. Bags few, in the basal hymenium, bitunicate, rounded at the top, cylindrical to almost club-shaped, on a short stem, 100–140 × 12–16 (20) μ with 8 ascospores. Ascospores rolling, elongate spindle-shaped, straight to slightly curved, 42–48 (60) × 6–7 μ, with 3–5 septa; central cells largest, yellowish, with 1–2 drops per cell, smooth, with conoid to globose terminal appendages, 5–6 μ in diameter. Pycnidia scattered, spherical, up to 200–700 μ, smooth, glabrous, with a central cylindrical straight papilla, 150–200 × 100 μ, papilla wall 15–20 μ thick, with 6–8 layers of hyaline (except for the outermost brownish layer) polygonal cells, 2–4 μ in diameter. Stomata oblong, 80 μ. Pycnidial wall 18–24 μ thick, consisting of 3–5 layers of polygonal pseudoparenchymal cells, 4–6 μ in diameter. Conidia are unicellular, cylindrical, straight, 4–5 × 1.5–2 μ, hyaline, with 1 drop at each end, smooth. In pycnidia on lignified stems, the walls become scleroplectenchymous [90].

The host range of L. maculans is limited to species of the family Brassicaceae. The micromycete has been recorded on Brassica, Raphanus, *Sinapis alba*, *Tlaspi arvense, Camelina sativa* [91–92]. The source of infection is infected plant residues of rapeseed and other cruciferous plants, as well as infected seeds [93]. Airborne ascospores are the primary inoculum for infection of rape seedlings [94–95]. Airborne ascospores are the primary inoculum for infection of rape seedlings [96] or abundant dew [97].

Typically, spots begin to appear on infected leaves after at least 20 days of rain in August and early September. Each of the pathogens that cause phoma has its own characteristic leaf spot symptoms.

L. maculans: Usually brown spots with dark specks (asexual fruiting bodies – pycnidia)

L. biglobosa: Usually dark spots with a small number of dark specks (pycnidia).

The spots develop on the upper side of the leaf, the underside is free of fungal growth (in contrast to the white fungal growth associated with downy mildew). Some spots may have a yellow halo and cause browning of the leaf veins.

Leaf spot has a minimal impact on crop growth and yield, except when severe cotyledon infection leads to seedling death.

From the leaf, the pathogen grows along the petiole to the stem, penetrating and killing plant tissue cells. The classic symptoms of cancer

often form around the leaves at the base of the stem. They develop further and wrap around the stem, restricting the transport of water and nutrients. This can lead to premature aging. In extreme cases, the stem can break, the crop can lie down and the plants can die. The earliest infections are associated with the most dangerous consequences.

L. maculans: Usually causes relatively severe phomosis of the stem base

L. biglobosa: Usually causes damage to the upper part of the stem.

Symptoms of infection can also occur on flowers, buds and pods. The latter may show brown pod lesions with pycnidia and a black edge.

After harvest, the pathogen continues to develop on the stubble. However, the symptoms gradually disappear as the fungus enters the sexual stage on the stems and roots.

Leaf spot infestation can start from September when the seedlings emerge. The initial symptoms are white or fawn round lesions that become dotted with small black fruiting bodies. These leaf lesions are green underneath. They sometimes cause partial leaf death before winter, but usually have minimal effect on growth until spring. These large leaf lesions are caused by *Leptosphaeria maculans* (Phoma A). The second species, *Leptosphaeria biglobosa* (Phoma B), causes small, dark lesions with few fruiting bodies. L. biglobosa can spread to the stems, but has less impact on yield than *L. maculans*.

Over time, the fungus grows from a leaf spot to the stem through the petiole. The rate of this growth varies widely: from 5 mm per day at 20°C to 1 mm per day at 5°C. In summer, these stem cankers cause lodging and premature ripening. Symptoms of deep brown stem canker appear about six months after the initial infection. They gradually increase, encircle the stem and weaken it, which leads to premature ripening, lodging and death of the plant. The disease can spread to the pods, which develop brown lesions with a black border, potentially leading to premature maturation and infection of seeds, which can be a secondary source of infection to new crops.

At a temperature of 15–20°C, the first signs of the disease will be observed in 3–4 days. The presence of wounds (pest damage) greatly facilitates the process of infection of plants with phomosis.

The period of ascospore emergence depends on climatic conditions and is usually timed to coincide with the presence of young, sensitive rapeseed plants. For example, in Australia, ascospore emergence begins in May after

winter rains, which are essential for seedling development [98]. In Canada (Ontario), ascospores begin to emerge in September-November, which is the time when they can infect winter rape seedlings [99]. In western Canada, ascospores emerge from May to August and infect the leaves of young spring rape plants [100]. In Western Europe, ascospores emerge from the end of September during the autumn-winter period, with the time of maximum ascospore flight varying from year to year [101–102]. In Eastern Europe, ascospores emerge in September-November and spring [103]. Ascospores can remain viable for up to 6 weeks and can be spread by wind for several kilometres [104–106].

Ascospores and conidia germinate in a moist environment as infectious hyphae. The infectious hyphae enter the plant through stomata and wounds [107–108]. The minimum dew period required for infection of rapeseed plants by ascospores of both species is 8 hours. The maximum number of leaf lesions is observed after a 48-hour dew period at 20°C. For *L. maculans* isolates, the incubation period is 5 days at 20°C and 13 days at 8°C; for *L. biglobosa* isolates, respectively, 2 and 7 days [109].

The incubation period varies depending on the variety and age of the leaves [110]. The first six leaves of rapeseed plants were found to be more susceptible to infection caused by *L. maculans* isolates.

Inoculation of plants after the formation of six leaves leads to too late development of stem canker, which does not cause significant yield losses [111]. Proved [112] that symptoms develop faster on the sixth leaf than on the second or fourth leaf. However, when the pathogen was isolated from leaves without symptoms, the fungus was isolated from the second leaf 2 days after inoculation, from the fourth leaf 6 days later and from the sixth leaf 14 days later. Symptoms of the disease caused by *L. biglobosa* were more severe on senescent tissues. In order to initiate the development of damage at optimum temperature and humidity, 1–2 ascospores are sufficient [113]. Conidia can infect only damaged leaves, stems and petioles. It was noted [114] that the conidia could only cause infection of intact leaves if very high concentrations of inoculum were used to infect old leaves.

Conidia emerge from the pycnidia immersed in a sticky matrix and are carried by rain drops to other leaves and plants. Conidia spread more successfully in light rains with wind. Secondary infection caused by conidia is rare in Europe and Canada, but is more common in western Australia,

although it has little impact on yield. Symptoms of the disease caused by isolates of the two species are quite similar, although there are some differences. Damage caused by *L. maculans* on leaves initially appears as pale green spots that increase to 1–2 cm in diameter, often turning pale brown with numerous black pycnidia. Sometimes the centre of the spot may crack and fall out. *L. biglobosa* causes smaller brown spots with fewer or no pycnidia [115].

Despite these differences, changes in the nature of spots with age make it difficult or impossible to visually identify these species in the field [116]. The fungus grows from leaf and cotyledon spots biotrophically in the leaf blade and leaf petiole and penetrates the hypocotyl and stem [117].

Defined [118] that the natural infection goes through 5 phases: latent leaf infection, symptoms on the leaves, asymptomatic growth in the petiole, latent stem infection, and development of symptoms on the stem. After colonising the intercellular space in the spongy mesophyll of the leaf blade, the fungus reaches the vascular system and spreads down the petiole, mainly through xylem vessels or intercellular spaces of the xylem, parenchyma and cortex. During this phase, the fungus is biotrophic.

Direct infection of petioles and stems is possible only if they are damaged. In case of hypocotyl infection, symptoms similar to blackleg develop. Above the soil level and below the level of the first pair of leaves, watery spots form, which then dry up and turn grey, often with a stretch mark at the site of the lesion. Pycnidia develop on the affected tissue. This form of the disease is harmful in Australia and Canada [119]. The death of seedlings from the black leg in some fields can reach 70% [120]. In older plants, elongated, oval, depressed, beige spots are formed at the base of the stem, often surrounded by a clear dark brown or purple border, with numerous pycnidia in the centre of the spot. This type of damage is caused by the spread of pathogen hyphae from leaf spots that developed early in the growing season (e.g. in Europe in autumn). During pod development and seed ripening, these spots can enlarge and merge, completely covering the stem, dry rot develops in the root part of the stem, the stem is often bent, and the plant gradually dries up. Stem bases may break at the site of damage. This stage of the disease is called root neck cancer and is the most harmful. Stem damage at the soil level often spreads to the root system, causing root ulcers and root dry rot [121]. From foliage infected later in the season

(e.g. late winter or spring in Europe), the pathogen spreads along the petiole and produces oval, beige spots with a dark brown or purple border in the upper (> 5 cm from the root collar) part of the stem. This damage occurs at relatively early stages of development, such as during flowering, and can cause yield losses, Canada [122] and Europe. Damage to the stem in the upper part, as well as at its base, can increase in size, encircling the stem, and cause premature pod ripening due to impaired water transport in the plant [123]. In severe cases, the stems break. Leptosphaeria species differ in pathogenicity. *L. maculans* isolates are highly aggressive and usually cause symptoms of stem base canker. *L. biglobosa* is considered to be less aggressive and is mainly associated with damage to the upper part of the stem [124]. *L. biglobosa* isolates penetrate the stem core and can cause browning (which is only detected when the stem is cut longitudinally), but rarely lead to external symptoms [125]. However, as the stems age, isolates of both species develop numerous pycnidia on their surface.

The spots on the pods are elongated, slightly depressed, brown or grey, sometimes with a dark brown border. The pods are infected by conidia developing in pycnidia on spots of leaves and twigs. Pods are rarely affected, but can cause premature ripening and cracking [126]. The infection from the flaps can spread to the seeds, which become shrivelled and dull [127]. The pathogen is found in seeds as a dormant mycelium in the seed coat or cotyledons, and rarely in the embryo. The frequency of occurrence of rapeseed infected with the phomosis pathogen is low, for example, in Canada – about 5% [128], in Western Australia – 0.1–0.2%. Seed infection can be important for the spread of the disease to new areas. Infected seeds of other cruciferous plants, such as mustard, can contribute to the spread of the disease [129]. After harvesting, the aging stem tissue is rapidly colonised by pathogens that form numerous pycnidia. Conidia can colonise plant residues saprotrophically, which can increase the level of inoculum and, consequently, the number of pseudothecia. Pseudothecia are formed on plant debris. The maturation of pseudothecia depends on temperature and humidity, with an optimum at 14–15°C [130]. In Western Australia, pseudothecia ripen in the autumn and winter, as their maturation is slowed by hot, dry summer weather. In North America and Europe, pseudothecia are formed by harvest time, although due to dry weather in summer, their maturation may be delayed [131]. In Canada (Ontario), pseudothecia form

in September, and ascospores begin to emerge by the end of September. In western Canada, pseudothecia form within 9–10 months after harvest [132]; Ascospores emerge from pseudothecia that developed on plant debris from the previous year in late June and August, when plants are in flowering or at later stages of development. The following year, mature pseudothecia may release ascospores earlier (in May-June), which can lead to severe damage to seedlings.

The intensity of disease development on cotyledons, leaves and stems increases at higher temperatures. On plants inoculated at the cotyledon stage, stem base cancer was more intense at temperatures above 12°C [133–134]. The resistance genes of young plants can be heat-sensitive, which determines the intensive development of the disease at 24°C than at 14°C [135]. On plants inoculated at the bud stage, the intensity of disease development was higher on stems at 18°C than at 12°C. The most severe epidemics are associated with climatic conditions, for which temperatures of 25–30°C are typical during the development of stem canker. Such epidemics can also occur in Canada and Eastern Europe, where summer temperatures are high. In China, despite the high temperatures, epidemics of this magnitude are not typical, which is determined by the absence of group A isolates [136]. The quantitative ratio of rapeseed phomosis pathogen isolates is an important factor that determines the disease severity in different regions. In most rapeseed growing areas of the world, *L. maculans* isolates are considered economically important. In most countries, where epidemics are often very severe, populations are represented by this species [137].

The ratio between *L. maculans* and *L. biglobosa* isolates changes during the growing season. In the UK, pseudothecia develop earlier at the base of the stem and ascospores mature earlier in them than in pseudothecia on lesions of the upper part of the stem. Studies conducted in France, the UK and Germany have shown that isolates of L. maculans are isolated from the base of the stem, and isolates of both species are isolated from the upper part of the stem in equal proportions [138]. Differences in the maturation of pseudothecia determine the following facts: 95% of ascospores in early spring in Canada belong to *L. maculans*. In the UK, leaf lesions caused by *L. biglobosa* isolates appear later than those caused by L. maculans isolates; in infected seeds from the UK, the proportion of *L. biglobosa* isolates infecting seeds later in the season is higher [139].

As seed infection is dominated by *L. biglobosa, L. maculans* is less likely to be introduced into new areas. As the upper parts of the stalks, which have a higher proportion of *L. biglobosa*, are harvested at harvest time, the proportion of *L. maculans* increases during this period [140].

Two different forms of growth of the phomosis pathogen on agarified media were found. In culture, colony types were identified that differ in morphology, growth rate, spore production, degree of medium pigmentation, and toxin excretion into the liquid medium [141].

L. maculans grows slowly on nutrient media – on plum-lactose-yeast agar, the growth rate is 0.4-1.5 mm/day, forms irregular (with unevenly lobed edges), colonies that age rapidly (after 21 days, growth almost stops, and colonies rarely reach the edge of the cup).

Isolates of *L. biglobosa* grow rapidly (1.9–3.1 mm/day) and usually reach the rim of the cup by day 14. Saltants (in the form of sectors with pycnidia of different sizes) are often found in cultures. Sometimes isolates of L. biglobosa also grow slowly (growth rate of 0.7 mm/day), but in this case the colony edge is flat and no rapid aging is observed. Conidia of L. biglobosa form longer and more branched growth tubes during incubation on water agar for 40–44 hours than conidia of *L. maculans* [142]. This method allows for visual identification of the species of isolates without measuring the length of the growth tubes, and it allows conidia to be used directly from the pycnidia without isolating the pathogen into pure culture.

The external manifestations of phomosis can be very diverse: stem and root collar cancer, dry rot, leaf spot, and fruit damage. In the case of fruit damage by phomosis, seeds can form sick, weakened seedlings (black leg) or even completely lose germination.

In Ukraine, the main source of initial infection is airborne ascospores from stubble or residues of previous rapeseed crops near the new crop during warm, humid weather. They are dispersed by air currents and land on the leaves and sometimes on the root collar of young plants, where the disease develops. Early epidemics are associated with above-average rainfall in August and September. If cotyledons are infected, seedlings can die in autumn. The causative agents of cruciferous phomosis have several alternative sources of infection, which makes the disease difficult to control. During the growing season, the fungus spreads mainly by asexual spores – conidia, which are formed in necrotic areas of rapeseed and other plant

species from the cruciferous family. Under favourable conditions, several generations of conidia are formed, which are found mainly in the mucilage and are usually spread by rain over a short distance. The fungus overwinters in the form of mycelium in unmineralised plant debris and in affected seeds. The first mature ascospores are formed on the remains of winter rape in autumn, but the most massive sporulation usually begins in spring. The period of ascospore formation is quite long and reaches several months. Ascospores are spread by air currents and, unlike conidia, can travel long distances. Affected seeds produce affected seedlings. It is important that even a slight infestation of seeds with phomosis (< 0.1%) in the absence of control measures can initiate a significant outbreak of the disease in the field. For successful infection of plants with spores, drip moisture is required (at least 4 hours). The temperature requirements of different types of phomosis pathogens vary significantly, with Phoma nigrificans being the most cold-tolerant. At low temperatures (5–10°C), phomosis pathogens develop asymptomatically for a long period (up to 2 weeks).

Phomosis is one of the most dangerous diseases of rapeseed. At one time, it was phomosis in the form of rapeseed root collar cancer that severely limited the spread of this crop. And the fact that rapeseed is now grown on 6.5 million hectares in Europe was a significant impetus for the fact that breeders defeated this disease. New hybrids are usually relatively resistant to phomosis, but over time, new, more aggressive races of its pathogens appear, so successful rapeseed cultivation is impossible without the use of fungicides.

Control measures:

– Strict observance of crop rotation. Spatial isolation of rapeseed and other Brassica crops.
– Accelerate mineralisation of plant residues (shredding, ploughing, etc.).
– Control of weeds from the Cabbage family. Destruction of carrion.
– Seed quality control. Use of effective disinfectants.
– Timely use of fungicides during the growing season.
– Pest control (rapeseed flea beetle, covert borer, etc.).
– Selecting more resistant varieties and hybrids.
– Phomosis is a widespread disease of rapeseed worldwide.

The disease has become increasingly important due to the intensification of rapeseed production and climate warming. Effective phomosis control includes both agronomic practices and genetic protection of varieties.

In recent years, phomosis incidence has increased due to intensification of rapeseed production, climate warming and lack of phomosis management strategies. This has led to an urgent need for effective control of the disease, where both agronomic practices and the use of genetic resistance play an important role.

To control this disease, agronomic practices are used: crop rotation, soil cultivation, optimal sowing dates, seeding rates, fertiliser doses, and pesticide treatment. Depending on the conditions in the growing areas, it is recommended to return this crop to the same field in 3–5 years, and in Ukraine – in 4–5 years.

The spread of the disease can be contained by quarantine measures. For example, in Alberta (Canada) in 1984 (a year after phomosis was detected in the area), sowing and transporting infected seeds was banned, and farmers were also prohibited from sowing rapeseed for 4 years in fields where the disease was detected. Although the disease has now spread to most of the province, its spread has been significantly slowed.

Crop rotation is one of the oldest and most effective control strategies, which reduces fungal populations to a level where they are not economically important if the crop is re-seeded. The principle of crop rotation is to plan the order of placement of crops on the same field in such a way that the infected stubble decomposes. In this case, the ability of the pathogen to produce inoculum as a source of disease is reduced [143]. A 3–5 year crop rotation is recommended for rapeseed. Ploughing increases soil aeration and temperature, which leads to rapid decomposition of stubble, which is the source of primary infection, so ploughing crop residues into the autumn is a good way to control phomosis [144]. No tillage, for example, in Western Canada, leads to the persistence of infection on infected crop residues.

A good method of controlling phomosis is to plough the crop residues into the ground until autumn. In China and India, whole plants are harvested from the field (for use as fuel) and then flooded for rice, which in warm climates leads to rapid destruction of the inoculum.

The possibility of using the fungi Cyathus striatus and C. olla, which usually live in bird nests, to accelerate the destruction of rapeseed residues is being studied [145].

Minimal tillage is practiced, which in a hot, dry climate contributes to the accumulation of infected residues on the soil surface and their preservation

as a source of inoculum for 4 years. The increase in acreage in Australia has led to a reduction in the time it takes for canola to return to the same site, which also increases the amount of inoculum. Cold winters and dry, hot summers help to preserve plant residues for several years [146].

Moderate, humid climates cause rapid decomposition of residues within 2 years (Europe, south-eastern Australia), and deep ploughing accelerates the process of decomposition.

Phomosis is favoured by early sowing of winter rape and late sowing of spring rape [147]. Thickened rapeseed crops with thin stems are more severely affected. The severity of phomosis increases as a result of mechanical damage to plants by insects (cruciferous flea beetle, rapeseed flower beetle, etc.), as well as during plant care measures using machinery.

In France, with early sowing, rapeseed has time to develop a sufficient number of leaves before ascospores fly, which allows it to avoid infection at the most susceptible stages [148].

To reduce the infection of phomosis pathogens, optimal seeding rates and sowing dates are also important, taking into account environmental factors [149].

As for the influence of the fertilisation system, the effect of different fertiliser rates on the development of phomosis was investigated. The most effective of all studied variants of fertiliser rates was $N_{60}P_{60}K_{60}$, which reduced the spread of phomosis compared to the control by 32%, and the development by 9.3%. In terms of seed yield, this variant exceeded the control by 1.5 t/ha (Table 4.21).

As *Leptosphaeria maculans* overcomes major resistance genes, the disease can be controlled by diversifying cultivars in terms of resistance genes and their placement. Rotation of varieties containing different genes or combinations of resistance genes is recommended every four years. Regular monitoring of virulent gene frequencies is important to determine the effectiveness of known major resistance genes. One strategy to increase resistance to forms of phomosis is to use individual core genes or different combinations of genes in a genetic background with non-specific resistance. Long-term resistance can be increased by using a combination of species or varieties with different resistance genes in the same field and inter-field diversification. It is also necessary to protect rapeseed from *L. biglobosa*, which can cause significant damage, especially to varieties with effective core resistance genes for *L. maculans*.

Table 4.21

Effect of mineral fertilisers on spring rape resistance to phomosis [150]

Fertiliser rates	Phomosis infestation, %					
	2010		2011		Average value	
	Distribution	Development	Distribution	Development	Distribution	Development
Control	64	12.8	56	11.8	60	12.3
$N_{30}P_{60}K_{60}$	53	7.7	47	8.5	50	8.1
$N_{60}P_{60}K_{60}$	33	3.7	23	2.3	28	3.0
$N_{90}P_{60}K_{60}$	60	10.6	52	9.8	56	10.2
$N_{120}P_{60}K_{60}$	71	13.6	59	12.4	65	13
SSD_{05}	1.3	2.2	3.5	2.8		

Thus, the best way to manage phomosis is to grow resistant varieties [151]. At the same time, it is important to regularly monitor the frequency of virulence genes to determine the effectiveness of known major resistance genes. One strategy to increase the duration of resistance to phomosis is to use single major genes or their various combinations in a genetic background with non-specific resistance. The duration of resistance can be increased by using mixtures of varieties or species with different resistance genes in the same field and inter-field diversification. It is also necessary to protect rapeseed from *L. biglobosa*, which can cause significant damage, especially to varieties with effective main resistance genes against *L. maculans*. In addition, chemical protection of rapeseed against L. biglobosa requires higher doses of triazoles than for control of *L. maculans*, so there is a great need for breeding resistant rapeseed varieties to both *L. maculans* and *L. biglobosa*.

Avirulence genetics of *Leptosphaeria maculans* (*L. maculans*) is a haploid fungus with a small genome size of 45.12 Mb, encoding probably 10,000–13,000 genes within 17–18 chromosomes (some chromosomes are optional, i.e. B-type), the genome of *L. biglobosa* is smaller (30–40 Mb) [152].

The genome was created in 2004 by Genoscope (CEA) (http://www.genoscope.cns.fr), and in 2011 the sequencing of the *L. maculans* genome was completed. The genome sequence is publicly available (http://urgi.versailles.inra.fr/index.php/urgi/Species/Leptosphaeria) [153].

The genetic diversity of the *L. manculans* population is mainly due to sexual recombination, mutation, large population size and high gene flow due to large-scale distribution of ascospores. Sequencing of the *L. maculans* genome revealed the presence of many transposons (about 30% of the genome) [154]. This degenerate, retrotransposon-rich part of the genome is thought to contribute to the rapid evolution of virulence in *L. maculans* isolates through multiple whole-gene deletions, mutations, and re-induced point mutations in avirulence alleles (AvrLm) [155–157]. Probably, due to RIP mutations and localisation of Avr genes within repetitive regions, the fungus adapts under the pressure of selection by resistance genes [158].

In L. maculans, 14 avirulence genes were identified, and 8 of them are genetically clustered in two different regions. The first cluster contains AvrLm^{1}, AvrLm^{2}, AvrLm^{6} [159], the second – AvrLm^{3}, AvrLm^{4}, AvrLm^{7}, AvrLm^{9} and AvrLepR1 [160–162]. These clusters can be hundreds of kilobases in size due to the absence of meiotic recombination in such regions. To date, 7 avirulence genes of L. maculans have been cloned – AvrLm^{1} [163], AvrLm^{2} [164], AvrLm^{3} [165], AvrLm^{4}-7, AvrLm^{6}, AvrLm11 [166], AvrLmJ1 [167]. All of the activity genes, with the exception of AvrLm^{1}, which is localised to the heterochromatin region, encode small, cysteine-rich, secreted proteins that are highly expressed in the early stages of pathogenesis [168]. The AvrLm^{4}-7 gene encodes a protein of 143 amino acid residues. A single base mutation leading to the replacement of glycine with arginine results in the loss of the ability to recognise the Rlm^{4} gene, while recognition of Rlm^{7} is preserved (AvrLm^{7} specificity is not changed). The point mutation is the main event leading to the loss of Rlm^{4}-mediated resistance. After cloning AvrLm^{7}, it was found that AvrLm^{4} and AvrLm^{7} are two different alleles of the same gene (renamed AvrLm^{4}-7).

Investigation of the population structure of *L. maculans* on the basis of avirulence (virulence) in Poland, Sweden, Germany, England and France [169] showed a high frequency of the virulence genes avrLm^{2}, avrLm^{3}, avrLm^{9} and avrLm^{5}. The avrLml and avrLm^{4} genes were detected in a small number of isolates (less than 10%). In England, in 2012–2013, single isolates with avrLm^{7} were detected, and all of them had avrLm^{4} [170]. In Canada, less than 5% of isolates with AvrLml and AvrLm^{3} genes were detected in 2012 [171]. However, according to the [172], in 2012, in Canada (Manitoba), 22.0% of isolates with AvrLml were found in the fungus

population, 2.7% with AvrLm^3, 3.3% with AvrLm^9, 10.7% with AvrLepR2, 39.1% with AvrLepR1, 64.3% with AvrLm^2, 65.3% with AvrLm11, and 66.0% with AvrLm^6. The proportion of isolates with the avirulence genes AvrLm^4, AvrLm^5 and AvrLm^7 was high – 77.1, 80.7 and 89.2%, respectively. In Germany, an analysis of fungal populations in 2011–2014 revealed a high frequency of virulence genes for the resistance genes Rlm^1, Rlm^2, Rlm^3, Rlm^4 and Rlm^9 (more than 80% of isolates) and a low frequency for the Rlm^7 gene (less than 5%) [173].

Two types of disease resistance have been identified in species of the genus Brassica, including rapeseed: qualitative (race-specific, juvenile, mono- and oligogenic) and quantitative (non-race-specific, age-specific, usually polygenic) [174–183]. Juvenile resistance, which is expressed from the seedling (cotyledon) stage, depends on the presence of the R-gene for resistance in the plant genotype and the presence of the corresponding Avr-gene in the pathogen isolate. This is a very effective resistance, which works through the activity of the R-gene – the pathogen enters the cotyledons or leaves, resulting in a hypersensitivity reaction that prevents the further spread of *L. maculans* infection to the entire plant, although the impact of the pathogen can continue throughout the growing season [184]. That is, effective master genes of race-specific resistance to L. maculans act when ascospores or pycnidiospora infect cotyledons or leaves, preventing further spread of infection to the stem [185]. On the contrary, polygenic resistance is a partial resistance, and the small genes that cause it interact with each other in a complex way to form the plant's response to the pathogen. Each of these genes usually does not have a large phenotypic effect, so there is no strong selection pressure for certain fungal pathotypes [186]. Quantitative resistance is especially important for field crop protection, as the greatest damage to yield and product quality is caused by damage to adult plants [187–188]. The type of resistance can be determined only by the presence (or absence) of race-specific resistance genes in the tested genotype of B. napus genotype under test and avirulence alleles in *L. maculans* isolates used for inoculation in controlled environments or field experiments. Field (age) resistance can be caused not only by small genes, but also by race-specific master genes. It can be controlled by a major gene to which the field population of *L. maculans* carries avirulent isolates or by many genes with small effects. There are usually no differences in the symptoms of phomose

spot development on the leaves of young plants of varieties with and without polygenic resistance to L. maculans, but at the end of the season, varieties with quantitative resistance do not develop stem ulcers or are less severe than varieties without this resistance [189].

All the major race-specific phomosis resistance genes identified so far were found in the A-genome of *B. napus* and none were found in the C-genome [190]. To date, several such genes have been identified and genetically mapped in B. napus, several such genes have been identified and genetically mapped (Table 4.22).

It is not yet clear whether the Rlml, Rlm^3, Rlm^4, Rlm^7, Rlm^9 genes are a cluster of closely related factors [227]. It is believed that Rlml is different from Rlm^3 because, while present in the same variety, they are genetically mapped at different positions. The Rlml and Rlm^4 genes are linked to each other, are not allergic and can be present in the same variety as the Rlml and Rlm^3 genes. At the same time, the Rlm^3 and Rlm^4 genes, which occur in many rapeseed varieties, are rarely present together in the same genotype and may be allelic forms of the same gene. No varieties have been found that contain both Rlm^7 and Rlm^9 genes. It is also not clear whether Rlm^4 and Rlm^7 are different genes or allelic forms of the same resistance gene. The LEM1, LmRl, cRlmm and cRlmrb genes present in different B. napus cultivars are mapped to chromosome A7 [228–229]. It was found that the LEM1 gene of juvenile resistance to isolate c Avrl-2-4-7 [230] is localised in the region of a large tandem duplication. The LEM1, LmRl, cRlmm and cRlmrb genes may be identical to the Rlm^4 gene [231]. French varieties Major, Jet Neuf and Australian varieties Maluka, Dunkeld, Skipton carry the Rlm^4 gene [232].

The race-specific genes Rlm^8, Rlmll, LepR1-LepR4 were found in the A-genome of *B. rapa* (Table 4.22). The LepR3 gene, which was introduced into rapeseed from *B. rapa* ssp. *sylvestris*, is the first cloned gene for rapeseed resistance to phomosis. It belongs to the family of receptor-like proteins. The recessive resistance gene LepR4, which causes a wide range of resistance and is mapped to chromosome A6, is represented by two different alleles – LepR4a and LepR4b [233]. In addition, B. rapa may contain genes previously identified in B. napus, – Rlm^1, Rlm^2, Rlm^4, Rlm^7, which are located in the same position in *B. rapa* and *B. napus* [234]. For example, using fine mapping, the localisation of the LepR3 and Rlm^2 genes was found to coincide [235].

Table 4.22

Main resistance genes of some cruciferous crops to Leptosphaeria maculans (Desm.) Ces. et de Not. [191]

Resistance gene	The source of the gene	Localisation in a chromosome *B. napus*	References
Rlm^{1}	*B. napus* (AACC, 2n = 38)*	A7	[192–196]
Rlm^{2}	*B. napus**	A10	[197–201]
Rlm^{3}	*B. napus*	A7	[202]
Rlm^{4} = *LEM1*	*B. napus**	A7	[203–207]
Rlm^{5}	*B. juncea* (L.) Czern. (AABB, 2n = 36) Sareptic (Indian, brown (brown sarason) mustard)	A8	
Rlm^{6} = *Jlm*^{1}	*B. juncea*	A8	[208–211]
Rlm^{7}	*B. napus**	A7	[212–214]
Rlm^{8}	*B. rapa L.* (AA, 2n = 20)	–	
Rlm^{9}	*B. napus*	A7	[215–216]
Rlm10	*B. nigra* (L.) W. D. J. Koch (BB, 2n = 16) black (French or real) mustard	A7	[217]
Rlm11	*B. rapa*	Dispensoma	[205]
LepR1	*B. rapa* ssp. *sylvestris* Janch.	*Al*	[218–219]
LepR2	*B. rapa* ssp. *sylvestris*	A10	
LepR3	*B. rapa* ssp. *sylvestris*	A10	[220–221]
LepR4	*B. rapa* ssp. *sylvestris*	A6	[222]
rjlm^{2}	*B. juncea*	–	[223–224]
LmFr1	*B. napus*	A7	[225]
cRlmj	*B. napus*	A7	
aRlmj	*B. napus*	A7	[226]

Note * – the gene is also present in B. rapa, "-" – not determined.

In the species B. nigra with a B-genome, two resistance genes, Rlm^{1} and Rlm10, were identified [236]. The Rlm10 gene introduced into rapeseed is localised in B. napus in chromosome A7 [237]. Rlml rapeseed plants have better resistance to L. maculans in the cotyledon phase and at the adult stage of development, when the gene is overexpressed in B. napus. It is important to note that the Rlm^{1} gene belongs to the family of serine/threonine kinases [238]. It was pointed out that it controls a large proportion of age-related

resistance (about 70% of phenotypic variation). It was noted that the age resistance of Maxol was mainly due to the presence of the Rlm^1 gene, which is effective when the population is dominated by fungal isolates with $AvrLm^1$ [239]. The resistance gene Rlm^2 is also associated with age-related resistance to phomosis, either as having a residual effect on age-related resistance or as being linked to other genes located in this QTL and causing a part of the variation in age-related resistance [240].

The resistance to L. maculans of the amphiploid species B. juncea is mediated by two genes, named Rlm^5 and Rlm^6 [241]. In addition, the recessive gene $rjlm^2$ was identified in *B. napus* hybrids, which originates from B. juncea and is very effective against a wide range of *L. maculans* isolates at the cotyledon stage. Based on resistance gene analogues (RGAs), a SCAR marker was developed that is closely linked to the $rjlm^2$ resistance locus in *B. napus, B. rapa* and *B. oleracea*. Sequence analysis of this gene showed significant homology of two putative R-genes in the resistance gene cluster in chromosome 5 of *Arabidopsis thaliana* [242]. Introgression of Rlm^6 resistance genes [243] and $rjlm^2$ [244] in B. napus led to its effective resistance to *L. maculans* isolates at the seedling stage.

In the presence of major resistance genes in plant genotypes and complementary avirulence genes in fungal isolates, a typical gene-for-gene interaction between Brassica and L. maculans was observed, first established in the study of the flax rust phytopathosystem [245]. It involves the direct or indirect recognition by a protein encoding a plant resistance gene of an effector controlled by a specific pathogen avirulence gene [246–248]. When the R-gene corresponds to the complementary Avr-gene, the spread is stable (race-specific resistance) [249–251]. The *B. napus* genome has also been sequenced [252].

Thus, all known major genes of oilseed rape resistance to *L. maculans* are localised in the A-genome. Some genes are introduced into the genome of rapeseed from other species (*B. rapa, B. juncea, B. nigra*). In the Brassica – *L. maculans* phytopathosystem, pathogen avirulence genes interact with complementary plant resistance genes in a gene-for-gene manner [253–259]. Some major genes contribute to field resistance. For example, Rlm^1 can control a larger proportion of age-related resistance (about 70% of phenotypic variation) if the pathogen population has a low frequency of isolates virulent to this gene [260]. The Rlm^2 gene contributes

to age-related resistance either through a residual effect or through linkage with other genes localised in the same region. In addition to the main juvenile resistance genes, small genes of quantitative (partial, field) resistance of rapeseed to phomosis were identified, and using association mapping, resistance loci were identified not only in the A- but also in the C-genome of rapeseed [261–267]. It should be noted that the genetics of rapeseed resistance to L. biglobosa is practically not studied

So, once again, the best way to manage phomosis is to grow resistant varieties and lines, but due to the selection pressure exerted by race-specific genes on fungal populations with a high potential for virulence, varieties with major resistance genes quickly become susceptible. Studies conducted in Europe and Australia have shown that *L. maculans* populations using the same major resistance gene in many varieties grown over large areas quickly overcome this resistance [268]. A study of *L. maculans* isolates collected around the world showed that many of the known major resistance genes have already been overcome [269]. For example, as a result of crossing B. napus and B. rapa ssp. sylvestris, a phomosis-resistant variety Surpass 400 was developed [270], probably with the resistance genes LepRl, LepR2 and LepR3 introduced from *B. rapa* [271–272]. Surpass 400 was released commercially and had high resistance to phomosis in the field. However, after 3 years, this resistance became ineffective due to the rapid increase in local fungal populations of isolates with virulence to the LepR3 gene [273]. In Australia, phomosis incidence in sylvestris-resistant varieties was higher than in polygenic varieties that lack effective major resistance genes [274]. There is evidence of phomosis resistance breaking down in varieties in France [275–276], where, after the introduction of rapeseed varieties with the Rlml gene, it was overcome within 3 years. In France in 2000 [277] and in Australia in 2003, the breakdown of variety resistance was the result of a rapid change in the frequency of A-virulence genes, which depends on the resistance genes present in the cultivars grown. For example, in France, an increase in the area under Rlml rapeseed led to a large fungal population size and a high concentration of pathogen isolates with the avrLml gene, and subsequent sexual recombination and changes in agricultural practices (shorter crop rotations and minimal tillage) contributed to an increase in the frequency of virulent alleles [278]. There is also evidence of the gradual eradication of the major resistance genes

Rlm^9, Rlm^2 and Rlm^4 in France, and the Rlm^4 gene in Australia, following widespread use of varieties containing these race-specific genes [279]. It is known that rotation of varieties protected by different resistance genes can reduce the frequency of virulent isolates to a particular resistance gene by reducing the selection pressure of this gene on the fungal population, so phomosis can be controlled by using the appropriate R-genes. For example, the Rlm^4 gene was present in 53% of Australian varieties until 2002, and now this number has dropped to 29%. In Canada, the Rlm^2 and Rlm^4 genes are effective for breeding [280]. In addition, other useful genes can be introduced into Canadian rapeseed varieties – Rlm^5, Rlm^6, Rlm^7 and Rlm11 [281]. High efficiency of Rlm^6 and Rlm^7 resistance genes noted in Europe [282]. However, field experience using the Rlm^6 gene introduced into *B. napus* from *B. júncea* showed rapid overcoming of resistance [283]. In Australia, the Rlm^6 gene is known to have already lost its effectiveness, and an increase in the frequency of virulence to this gene occurred in its absence in cultivars [284] through the clutch AvrLml and $AvrLm^6$ [285–286]. In France, after the introduction of varieties with the Rlm^7 gene, 36% of virulent isolates were found to be resistant to this gene in 3 years. Thus, within a few years after the introduction of varieties containing the main race-specific resistance genes, the effectiveness of some of them decreases, which limits the usefulness of these genes for phomosis control when used in varieties grown on large areas [287–289].

In order to determine the effectiveness of known major resistance genes for their use in breeding, it is necessary to regularly monitor the frequency of avirulence/virulence genes in the population of the phomosis pathogen. It is important to rotate resistance genes in time and space. For example, in Australia, in 2003, the development of phomosis increased 3 years after the commercial release of varieties with Sylvestris resistance. However, as the area under alternative resistance varieties increased, the frequency of virulent *L. maculans* isolates on Sylvestris-resistant varieties decreased by 2005 [290–291], In other words, there was a rise and fall in the development of the disease in varieties with Sylvestris resistance. In Canada, the incidence of phomosis in rapeseed crops increased from 2005 to 2012, which correlated with an increase in the frequency of virulent isolates of the Rlm^3 resistance gene. This was due to the pressure of selection of the Rlm^3 gene on the pathogen population due to the increase in the

cultivated area under varieties with Rlm^3 during this period. The increase in damage from phomosis was also caused by a 2-year crop rotation instead of a 4-year one due to the intensification of oilseed rape production [292]. A strategy similar to the one used in Australia for sylvestris resistance could be used to change the situation in this country.

Rotation of different resistance genes may be an acceptable approach to minimise selection pressure on avirulent isolates towards rapid virulence evolution and overcome resistance genes, which will increase the duration of resistance in varieties. The rotation of genes is also aimed at reducing the spread of phomosis by reducing the inoculum. To increase the duration of resistance, it is necessary to identify many different resistance genes to diversify their use in the varieties being developed in order to determine a strategy for managing phomosis through genotype placement [293]. The duration of the effectiveness of race-specific resistance genes depends on the size of the *L. maculans* population, which is directly related to the amount of affected rapeseed residues, and therefore appropriate agronomic practices, such as less intensive rapeseed rotation, should be applied to maintain resistance.

Thus, successful rapeseed production and phomosis management requires control strategies such as variety selection or rotation of major resistance genes over time, combined with good quantitative resistance and best agricultural practices. Rotation of cultivars containing different genes or combinations of resistance genes is recommended every 4 years to minimise selection pressure on the fungal population and increase the duration of resistance [294]. However, not all resistance genes can be used in crop rotation. For example, varieties with Rlm^1 should not be replaced with varieties with Rlm^6, because selection of fungal isolates with virulence to the Rlm^1 gene also leads to an increase in the frequency of virulence to Rlm^6, because the $AvrLm^1$ and $AvrLm^6$ genes are closely linked in the pathogen genome. Thus, an understanding of the genetic interaction between resistance genes and avirulence genes is required to develop a strategy for rotating race-specific resistance genes.

Many plant breeders and phytopathologists believe that one of the important goals is to develop varieties with long-term resistance, but this is not an easy task [295]. The resistance of a variety is considered to be long-lasting if it persists for a long time when grown on large areas under

favourable conditions for the development of the disease. It is believed that non-race-specific resistance should be more durable, as a wide range of pathotypes is reproduced on varieties with such resistance without much selection pressure to increase virulence. Thus, polygenic resistance is usually long-lasting. However, over time, in some B. napus cultivars, polygenic resistance also becomes less effective due to changes in virulence and aggressiveness (ability to cause severe disease) in the L. maculans population. This is because polygenic resistance is also characterised by variety x isolate interactions, as it may include a race-specific component [296].

One of the effective strategies to increase long-term resistance to phomosis is to breed rapeseed varieties by combining juvenile and age-related genes. Juvenile and age-related resistance play an important role in phomosis control. The interaction of specific R genes and their corresponding avirulence genes at the seedling stage usually results in a very low disease severity at the adult stage [297]. In addition, the combination of the main resistance genes with polygenes increases the duration of resistance by slowing down the adaptation and reproduction of virulent isolates to race-specific genes, i.e., rape samples with juvenile and age-related resistance genes may be characterised by longer R-gene resistance [298–301]. This points to the need to identify and combine both existing and new genes for qualitative and quantitative resistance to phomosis. In order to effectively use race-specific resistance genes alone or in conjunction with quantitative resistance, the pathogen must be monitored at regular intervals to identify fungal isolates with virulence to the resistance genes used. Thus, one of the strategies for breeding for phomosis resistance can be the use of single major genes or their combinations in different combinations (gene pyramiding) on a genetic background with a high level of non-race-specific resistance. This is possible with MAS using markers that are closely linked to race-specific resistance genes and QTLs associated with non-race-specific resistance. The use of specific master genes will depend on the virulence structure of the pathogen population. The presence of a genetic background with non-race-specific resistance will prevent large yield losses of varieties in the event that the race-specific resistance genes present in them are overcome. The duration of resistance can be increased by using not only pyramids of major resistance genes introduced into a single variety, but

also mixtures of varieties or species with different resistance genes at the level of one field, inter-field diversification, and, of course, agronomic practices [302].

However, the diversification of varieties by resistance genes and their rotation in time and space makes it possible to manage phomosis [303]. Rotation of cultivars containing different genes or combinations of resistance genes is recommended every 4 years [304]. All this points to the need to identify and combine both existing and new genes for qualitative and quantitative resistance to phomosis. The current approach is genome-wide association mapping, which allows identifying markers for known and new resistance loci in the collection of parental forms and promising rapeseed breeding material for further use in marker-assisted selection in a particular zone [305–306].

Protection of rapeseed against *L. biglobosa*, which can cause significant damage, especially to varieties with effective main resistance genes against *L. maculans*, should also be considered [307–308].

To combat blackleg, different countries treat seeds and spray soil and plants with fungicides. Pesticides (seed treatment, fungicides for spraying soil and plants) are used to combat phomosis. For example, in Canada, seed treatment, which began in 1978, is recommended even if phomosis is not detected in seed lots. In the field, it is necessary to spray plants with the product at an earlier time, because when the fungus infects the stem, the disease is no longer controlled by the fungicide. Thus, in Western Europe, fungicide spraying for chemical control of phomosis is used in autumn/winter when spots appear on the leaves [309–310]. Various combinations of seed treatments, soil and foliar fungicides are used to control rapeseed phomosis. In Canada, carbatin, thiram and iprodione are used for seed treatment, in Europe, thiram and iprodione, and in Australia, iprodione.

Flutriafol is used as part of fertility pellets and provides long-term protection for seedlings. The use of foliar fungicides is economically justified when crop yields are high, inoculum levels are high and the variety is low in resistance. In Canada, propiconazole is sometimes used, but it does not provide complete control [311]. In western Europe, where high crop yields economically justify the use of foliar fungicides, treatments with difenoconazole, difenoconazole in a mixture with carbendazim, flusilazole in a mixture with carbendazim are used to control phomosis [312].

The optimal time to apply fungicides in Western Europe is autumn, 6 months before symptoms appear on the stems.

Fungicides are effective for a limited time due to their degradation and the emergence of new untreated leaves. The timing of fungicide applications is based on the presence of spots on the leaves of young plants. However, damage may not develop on young leaves for a long time, despite the presence of infection. The fungus reaches the stem before leaf damage appears and becomes insensitive to fungicides.

For the past 5 years, Bayer has been offering an innovative solution in Ukraine and other countries of the world – Tilmore fungicide – to protect both winter and spring rape from a range of diseases, as well as an effective growth-regulating effect to increase winter hardiness of winter rape. The combination of two active substances – protioconazole from the triazolinthion class and tebuconazole from the triazole class – in an effective formulation has a protective and therapeutic effect on the pathogens of the main diseases of rapeseed (phomosis, Alternaria, cylindrosporium, etc.), destroys both existing and latent forms of infections, which significantly increases the wintering of winter rapeseed.

The use of fungicides after a certain stage of plant development is not necessary, as new leaf lesions do not lead to a strong degree of stem cancer development and do not cause yield losses [313]. This stage depends on climatic conditions and parasite-host relationships. In the UK, the most widely used forecast for rape phomosis is based on the presence of leaf spots in autumn. Such a forecast does not always indicate the optimal timing of fungicide application, as the fungus can reach the stem and escape the fungicide. There are prospects for developing a more accurate forecast based on the interaction of weather factors and ascospore maturation, as well as for using immunological or molecular methods to detect ascospores in the air and asymptomatic infection in leaves. France has developed a system for predicting the timing of fungicide treatments based on epidemic risk and agronomic factors [314]. The risk of infection is determined by weather and biological factors (7 days with rain after sowing, pseudothecia maturity or the first detection of more than 20 ascospores in the air per day). If the risk of infection is high, the decision to use fungicides is based on agronomic factors such as variety resistance, soil type, and the stage and degree of plant development.

In addition, higher doses of triazoles are required for chemical protection of rapeseed against *L. biglobosa* than for control of *L. maculans* [315]. Since many of the most effective fungicides are not allowed under the new EU legislation, there is a great need for breeding rapeseed varieties with resistance to both *L. maculans* and *L. biglobosa* [316].

For the conditions of Ukraine, the systemic disease identification and control card has the following technological sequence:

Manifestations of the disease: It occurs on cotyledons, leaves, stems and pods. The causative agent Leptosphaeriamaculans (sexual stage) produces ascospores, Phomalingam (asexual stage) – picnics; Leaf damage takes the form of irregularly shaped spots of greyish-white or ashy colour, often covered with black specks;

The stem is affected in the root part or in the places of leaf attachment;

Phomosis on the stem is manifested in the form of dry ulcers with black edges that encircle the base of the stem and lead to lodging of the plant;

Seeds grown in an infected field may contain the infection and can help to prevent the disease from spreading.

Control measures for phomosis

Agronomic measures: observance of crop rotation; ploughing and destruction of plant residues; control of carrion and weeds.

Chemical measures: seed treatment to control infection of seeds with the phomosis pathogen; foliar treatments (growth regulators are helpful).

Use of tolerant hybrids.

Favourable conditions for the development of the disease:

Warm, humid conditions increase the risk of phomosis and the spread of the disease; Rain drops help ascospores to spread from the stubble; splashing drops scatter pycnidospores; Wind carries ascospores over long distances (up to several kilometres); High humidity promotes spore germination and initiates fungal growth;

Moisture in the early stages of plant development (up to the 6-leaf stage) plays an important role in the emergence of this disease.

The rows and conditions in which the early phase takes place have a significant impact on the final outcome of a phomosis outbreak;

The impact of the disease is more severe as temperatures rise towards the end of the growing season.

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4.5. Integrated systems and methods for clubroot control in cruciferous crop agrocenoses

Clubroot, caused by the soil-biotrophic obligate pathogen Plasmodiophora brassicae Woronin, is one of the most damaging diseases of cruciferous crops worldwide [1–3]. In many regions, clubroot causes serious damage to a number of cruciferous vegetable crops. Like Plasmodiophoridae, the pathogen belongs to the Rhizaria group of protists [4–5]. Together with the phyla Stramenopiles (also known as Heterokonta) and Alveolata, Rhizaria represent the eukaryotic supergroup SAR, which is a highly diverse group of eukaryotic organisms [6]. Clubroot causes root tumours that lead to impaired water and nutrient absorption. As a result, the infection can lead to wilting and stunted growth (Figs 4.27–4.28). Oilseed rape yield losses can range from a 10% reduction in yield to complete yield loss, including plant loss and reduced seed count per pod [7]. Disease development and cellular changes in host plants after infection have recently been described in detail [8–9].

Successful management of the disease is difficult because chemical control of clubroot is not permitted or has not been successful. In the soil, the pathogen survives as extremely tough, thick-walled dormant spores. These spores are easily transported from field to field through contaminated soil by machinery, animals, water or wind [10]. Thus, the disease can spread rapidly over large areas. The spread of the pathogen was recorded as early as the 18th century. In the 19th century, clubroot was first described in Ukraine [11]. It is assumed that the clubroot arrived with immigrants, the colonisation movement and the first settlers from Europe to North America [12], South America [13] and Australia [14], who probably transmitted the pathogen with contaminated food and fodder, or with contaminated soil.

Currently, foot-and-mouth disease is recorded on all continents (except Antarctica) and in more than 75 countries [15]. It is very likely that the disease is spread throughout the world where cruciferous crops are grown or cruciferous plants are present. The regions where the pathogen occurs with a high population density are predominantly humid temperate regions [16]. Expansion of sown areas and intermediate crop rotation contribute to an increase in the damage caused by this pathogen.

Figure 4.27 – Modelling the type of clubroot on rapeseed roots (upper position – the beginning of the formation and decomposition of the affected root with the release of spores, lower position – fully formed signs of clubroot) [17]

Figure 4.28 – Winter oilseed rape plants affected (left) and unaffected by clubroot [7]

An important factor in the control of clubroot is the implementation of effective host plant resistance. Not all known R-genes are active against all races, and resistance can be undermined by new races of pathogens. Crop management practices are widely used to control the disease. However, control measures such as crop rotation and increasing soil pH are not sufficient to reduce the spread of the disease. In order to improve control strategies, research into biological control measures has played an increasingly important role in recent years. In addition, the strengthening of plants with growth stimulants is becoming increasingly important.

If the host plants are repeatedly grown in the field, dormant spores of the pathogen can accumulate in the soil [18]. These spores are persistent and can remain in the soil for many years, leading to infection of cruciferous plants even after several years of cultivation [19]. Estimated [20], that the infection level in the field decreases below the level of detection only after 17.3 years. However, more than a 2-year break in cultivation and a diverse crop rotation scheme can reduce the number of dormant spores in the soil [21–22].

In order to control the P. brassicae pathogen, it is essential to know the optimal environmental conditions that lead to an outbreak of the disease. The causative agent of clubroot was described in 1878 [23], and more than 50 years later, its life cycle was shown for the first time [24]. Since then, the environmental conditions that are optimal for the development of the disease and contribute to its spread have been studied in detail [25–26].

Soil temperature and moisture play an important role in the development of the disease. The highest infection rates and symptom severity are observed at temperatures of 21–25 °C. At temperatures well below 20 °C, the damage is significantly reduced [27–29] For successful disease development, high soil moisture is required, especially during the first two weeks after inoculation or during the first and second infection [30]. KIn addition, soil moisture promotes the spread of mobile zoospores [31]. Soil type has little effect on the intensity of infection. However, sandy soils and soils with a low humus content have been shown to inhibit the development of the disease [32] and that the damage was lower on light sandy loam soils than on loamy soils or clay [33–35].

Another important factor for the development of clubroot is soil pH. A low pH value (pH 5 to < 7) in the soil promotes spore germination [36–37]

and usually leads to a stronger infestation. As the pH value increases, starting at around 7.2, new infestations hardly occur in most cases [38–41].

Thus, the most favourable conditions for clubroot development are high summer temperatures combined with light acidic soils and good soil moisture in the first weeks after sowing. It can be assumed that this will have a significant regional and interannual impact on the spread of the disease.

To date, the most thorough summary of edaphic and environmental factors that are crucial for the development of clubroot on cruciferous plant species was made in the study by R.G. Dixon [1]. Below, we provide a literary summary of his work, leaving a list of literary citations and formulations by this author.

Plasmodiophora brassicae Wor., the microbe that causes plant cankers in the Brassicaceae family, is very well adapted to live successfully for three reasons. Firstly, it is robust, well protected and apparently long-lived dormant soil-borne spores allow this organism to withstand adverse conditions, and yet these dormant structures seem to be able to react quickly as soon as a suitable host plant appears. Secondly, when this host is available, the primary zoospores that emerge from perennial spores have efficient means of movement, penetration and invasion. These features allow *P. brassicae* to make the best use of the soil environment, its profile, and the rhizosphere. Thirdly, after entering the host environment, *P. brassicae* reproductive cycles are protected from adverse external conditions, which allows the production of numerous new dormant spores that eventually restore the potential of soil pathogenic material [42–43]. During this phase, the pathogen has the ability to change the host's metabolic activity to its advantage. Only for a short time and at short distances in the soil are the primary zoospores exposed to unfavourable conditions. While in this phase of the soil, the fragile and vulnerable single-layered zoospores, equipped with double flagella, float in the soil due to the film of soil moisture from germinated dormant spores to the outer surfaces of root hairs. This is the most vulnerable part of the entire P. brassicae life cycle. However, this phase has not received the scientific attention it deserves, possibly because the tools necessary for such a study are either absent or too imperfect. There is some evidence to suggest that soil chemical and physical components affect *P. brassicae* itself, mostly gathered as a result of attempts to create extremely unfavourable conditions and thus stop the host from invading, thereby controlling the disease. Little

is known about how *P. brassicae* interacts with its biological environment, except for a few studies of microbes that, by analogy with the developmental cycle of *P. brassicae*, may offer elements of strategies for controlling the pathogen.

The dormant spores of *P. brassicae* are an obvious place to start when considering the ecological interactions of the pathogen. These robust spores are designed to ensure the long-term survival and reproduction of *P. brassicae*, and have evolved to remain viable in the soil despite exposure to adverse weather conditions for many seasons. Field studies show that their half-life is at least 3.6 years, and some spores can survive for at least 18 years in the absence of suitable hosts before spore populations decline to levels that are undetectable from the point of view of potential infection of cruciferous plant species [44].

Temperature, moisture content and position in the soil profile affect spore life expectancy [45–46]. The pH level of the soil obviously affects the rate of formation of primary zoospores, the number of which increases in acidic soils compared to alkaline soils [47], but without significant changes in overall germination. Spore dormancy and the need for external stimulants are elements of the initial relationship between P. brassicae and the environment [48]. Only a few spores from plant root debris in the soil germinate immediately [49]. However, certain external stimuli may be required to initiate the infection process [50–51]. The germination readiness of spores released from the roots of the host plant has been studied by a number of researchers [52–56]. In general, they concluded that bacteria and other organisms destroy diseased tissue in the host plant and "prepare" the spores for more efficient germination. But these secondary microbes are not essential for the germination process itself. Unknown mechanisms present inside the dormant spore initiate germination and control its speed. It turns out that these mechanisms in some spores act quite separately from other spores, as not all spores germinate synchronously. Rainwater and floodwater spread *P. brassicae* over quite long distances, especially on slopes. Wind carries spores collected with light, dry, dusty soil particles over even greater distances. Earthworms [57] and possibly moles, root nematodes and insects can be potential vectors [58–59] *P. brassicae* in soil. Spores spread with the manure [60] and on the farm animals themselves, as they are able to withstand the animal gut environment. The farm animals

and their food supplies that arrived with European colonists in the New World and Australasia are likely to have been vectors of *P. brassicae* in virgin areas.

Dirty machinery, wheels, crates and piles are all potential vehicles for the spread of *P. brassicae*. Wild and weedy Brassicaceae, as well as infected crop seedlings, harbour and spread the pathogen. Once established in the soil profile, further distribution is related to soil textural and structural properties, as well as the frequency and intensity of agricultural operations. Soil compaction and loosening by rotational tillage reduces the movement of spores into the subsoil, as does a strong humus-accumulative horizon of the soil profile with an active rhizosphere. The dormant spore population density decreases with increasing soil depth, with more than 97% of the total *P. brassicae* inoculum present in the surface soil layer (0–5 cm depth) and only a small number of dormant spores found below 40 cm [61].

Since the density of dormant spores is influenced by soil type, pH and host susceptibility, the combination of these factors determines the intensity of the inoculation potential at a particular site. It follows that after germination in a particular environment, the inoculatory potential of *P. brassicae* creates dose-response curves [62] unique to that particular location. During germination, the volume of dormant spores increases as the vacuoles enlarge and the walls thicken and become more transparent [63–64].

One zoospore is released from each dormant spore, leaving behind cytoplasmic residues. Germination is characterised by the loss of refractory globules, which are characteristic of the reserves in dormant spores, probably indicating enzymatic mobilisation of these resources. Immersion of spores in water can stimulate germination [65]. Ayers [66] germinated within 1 to 10 days using tap water, with the germination rate depending on the maturity of the spores. The absolute need for a stimulating environment may be questionable, as Honig (1931) [67] induced germination at temperatures below 21 ^{0}C in the absence of seedling roots. The optimum temperature for spore germination at rest is 24 ^{o}C and pH 6.0–6.7, with an upper lethal temperature of 45 ^{o}C and visible light inhibiting germination. Spores can be stored as dense suspensions at 3–4 ^{o}C for 3 years without loss of viability [68], apparently withstand anaerobic conditions and do not die at -20 ^{o}C for 3 days. It is standard practice to store galls at -20 ^{o}C

for several years as a seeding material [69]. These few pieces of information are sufficient to define dormant P. brassicae spores as very robust, able to withstand very unfavourable conditions. Comparative experiments aimed at determining the effect of temperature on dormant spore germination, motility and host infection require that spore maturity, age and hydrogen ion concentration in the immediate vicinity of the host-microbe interaction be known, standardised and reproducible. Evidence for the influence of the host on spore germination at rest is provided by Niwa and others (2008) [70] who reported a significant increase in the percentage of germinated spores (without nucleus) in rhizospheres where the host *B. rapa* var. perviridis was present.

The participation of root exudates as stimulants of spore germination at rest was investigated and subsequently confirmed [71–75]. Concluded [76], that the germination stimulating effect is nonspecific and can come from exudates of many species, not only from *P. brassicae* host exudates. This is supported by the data that root exudates of both calabash and perennial ryegrass stimulate spore germination. It was found that [77–78] that most germination (75%) is induced by root exudates of susceptible cruciferous host plants. It was also found that [79] that an abiotic stimulant may be present in root exudates, especially in exudates of susceptible and resistant Chinese cabbage varieties. Complex carbohydrate compounds found in cabbage exudates stimulated germination of pathogen spores [80]. It is possible that several factors can consistently influence germination [81]. Thus, it was found that the release of calcium ions from spores induces their germination. The exudate of the host plant stimulated the germination of the spores, which in turn released a second stimulating factor that encouraged further activity. The environment in which the host plant grows affects the composition of the exudate, for example, drought promotes the release of amino acids. Identified [82] calcium as a factor in the inhibition of *P. brassicae* in the soil and, consequently, a negative impact on germination, but it is recognised that this element does not act in isolation from the influence of soil microbial flora. Similar conclusions were drawn by another researcher [83] which used a comparable range of calcium sources. The number of dormant spores was adversely affected by the introduction of high-calcium BOF slag into the soil [84]. Direct evidence that the inhibition of spore germination is the main reason for the inhibition of pathogens at

neutral pH is provided by another study [85]. The number of germinated dormant spores in the soil correlates with the level of root hair infestation. When the calcium content of the soil decreased, the number of germinated spores and the level of root hair infestation also decreased. Therefore, not only does the host exudate affect germination, but also the number of spores available for germination is in some way related to the presence of calcium in the soil. Potentially, calcium and pH can affect the lifespan and viability of dormant spores in situ in the soil. Calcium-rich compost or calcium carbonate, which changes the soil pH from 6.0 to 6.9 and from 6.2 to 7.1, respectively, significantly reduces the percentage of germinated spores in the rhizosphere and the number of root hair infections. This study provides direct evidence that spore germination and subsequent colonisation of root hairs is slowed by calcium and alkaline pH values. Previously, was founded [86] that the introduction of a large amount of organic matter over 15 years increased the concentration of calcium in the soil, changed the pH to alkaline values, as a result of which the soil, which was previously favourable for the development of the clubroot, became unfavourable for their development.

Organic matter inhibited *P. brassicae* infection, and fine particle size fractions (< 5 mm) were most effective at changing the pH. Calcium hydroxide, calcium carbonate and potassium hydroxide also inhibited infection, with potassium hydroxide being the least effective [87]. The addition of sulphuric acid facilitated the development of infection by acidifying the soil.

It is concluded that soil pH has a significant impact on infection processes, and that calcium makes a separate contribution to these effects, with both factors acting in unison. Dormant spores from "non-natural" sources, such as callus crops, are less capable of germination than spores from whole plant galls [88]. This may be due to the fact that such spores are physiologically different from naturally grown spores, possibly due to the callus culture system.

The number of dormant spores per diseased plant increased at low disease severity values, but then remained almost constant for plants with symptoms of category "3" and above [89–92]. The average number of dormant spores per diseased plant ranged from 93 to 109, regardless of the disease index value, apparently crossing the saturation threshold. When the

load of dormant spores in the soil reaches even moderate concentrations, the severity of the disease increases [93].

Direct knowledge about the movement of zoospores is very limited, as it concerns behaviour after release from dormancy until incubation on the root surface in soil (Figs. 4.29–4.30).

Since the first studies of *P. brassicae*, soil moisture has been empirically considered as the medium through which host contact is achieved. In practice, of course, the influence of seasonal water supply varies, so although clubroot is considered a disease of wet soils, there are

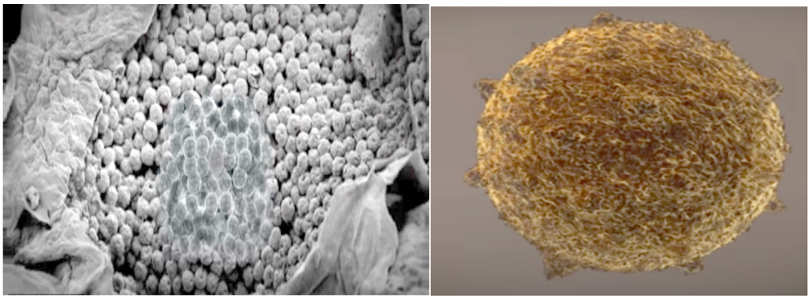

Figure 4.29 – Spores of *Plasmodiophora brassicae* [94]

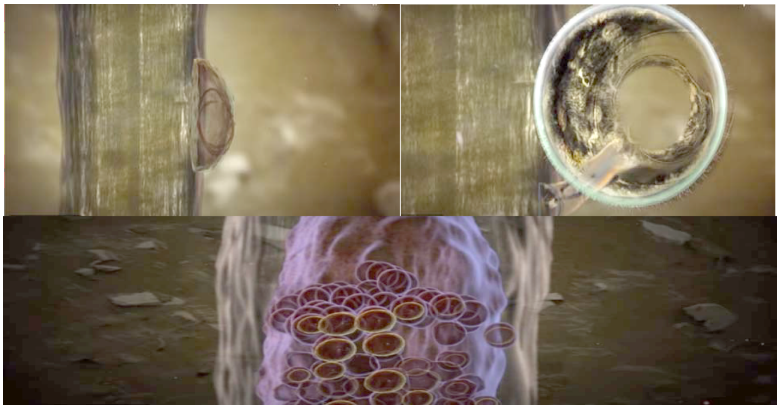

Figure 4.30 – The process of clubroot infection through the rhizosphere root hair system of rapeseed [94]

many reports of its intensity increasing in dry seasons or in dry areas. This probably reflects the loss of productive root systems, which makes the leaves very sensitive to water stress during periods of moisture deficit. It is believed that clubroot is associated with hydromorphic, poorly drained soils, and the disease is most severe after prolonged wet weather. For this reason, soil moisture is classified as the dominant environmental factor in the interaction with *P. brassicae*.

When soil moisture exceeds 50% of the moisture capacity, the disease develops very rapidly, indirectly demonstrating the speed at which primary zoospores move. Differences in the effect of soil moisture content may reflect differences in soil texture used by different researchers. Texture may influence the motility of P. brassicae zoospores, as suggested [95] because in their experiments, sand and soil mixtures caused the highest levels of infection. The infection developed at a moisture content of 9% in mineral soils, while organic soils require a moisture content of 60% [96]. Where soil moisture increased from 50% of the maximum water-holding capacity to saturation, disease severity increased. Underlined [97]. The dependence of zoospore microbes on free water existing between soil particles for the movement of zoospores. Free water is critical for the formation, release and dispersal of zoospores and can influence the processes of incursion and penetration at the root hair surface. The distances travelled by soil-borne zoospores are relatively short, probably between 10–20 mm, based on information for *Olpidium brassicae* or *Synchitrium endobioticum*, which are related to *P. brassicae*. Root hair infection occurred up to 75 mm from the source of *P. brassicae* infection in soils where free water movement was minimised [98].

Mathematical modelling [99], demonstrated a link between soil moisture and host invasion. Temperature was considered to be less important than soil moisture as a factor influencing the successful movement and invasion of *P. brassicae* than soil moisture. It has been studied with contradictory results in the same way as soil moisture and for similar reasons. A strong infection developed on acidic soils at an air temperature of 16.6 ^{0}C, while on alkaline soils the disease manifestation was less pronounced. The development of the disease was more favourable when working with alkaline soils at an air temperature of 23 ^{0}C [100]. As with soil moisture, the results [101] showed that the creation of conditions in which one of the main environmental

factors was very favourable to the pathogen allowed the disease to develop, despite other seemingly unfavourable factors. Previously, it was believed that temperatures below 20 °C were a barrier to the development of the disease [102–103]. But it showed [104-105] that the symptoms of the pathogen develop in the range from 9 to 30 ^{0}C.

The minimum temperature for the infection of root hairs of certain cruciferous plant species was also determined to be 12–14 ^{0}C. Research [106] have shown that in the early stages of root hair infection, temperatures of > 22.5 °C are required immediately after inoculation. Once root hair infection is complete, lower temperatures are sufficient to support symptom formation.

Growth analysis research [107], showed that temperature is the most important regulatory factor in the second week after inoculation, when root hair colonisation reaches its peak and zoosporangia are formed. It is possible that the predominance of individual environmental factors varies depending on the stage of the pathogen's life cycle.

The following assumptions were made [108]: "when one factor limits the manifestation of a disease (e.g. pH), another can significantly modulate its level (e.g. temperature)". This means that one factor sets the actual limit, while the other interacts with it to determine the frequency or intensity. It is also assumed [109] that pathogen can develop at 7 °C with daily temperature fluctuations and an increase in day length from 8 to 12 hours.

Light, sandy, humus-rich and clayey soils are thought to be the most favourable for the disease. As with the interaction between clubroot and acidity or alkalinity, there is a lack of rigorously tested scientific evidence on disease development and soil characteristics. Soil type affects the ability of physiological races of P. brassicae to pathogenise the disease. Clay and loamy soils are prone to compaction and have a pronounced neutral reaction of the environment, which leads to an interaction between favourable and inhibitory effects [110].

The conditions in which dormant spores are stored affect their viability [111] while germination is stimulated and increased by the presence of host root exudate. Interacting factors, such as humidity, temperature, pH, light intensity, as well as intrinsic factors including spore size, age and nutritional status, influenced the overall outcome of the host-parasite interaction.

The conclusion was made [112] that under environmentally unconstrained conditions and below the threshold level of infection required for maximum disease severity, canker severity is proportional to the increase in inoculum concentration and total root hair infection. Above this threshold, an increase in spore concentration may result in a higher level of root hair infection, but is not associated with an increase in disease severity. The Webster's threshold is actually a saturation point beyond which the physiological and biochemical processes that regulate the development of symptoms in the plant cannot be disrupted by the inoculum load. Saturation itself is not fixed and unchanging, as Webster's work supported the idea that only a small percentage of spores in an inoculum can cause a successful infection or invasion at any given time. The saturation of the root hair space in the root area under study, the distribution of spores around susceptible root hairs and the distance they can travel in the soil are all factors that influence the likelihood that an additional spore will be able to cause infection and cause a clubroot.

At the same time, the two-stage life cycle of *P. brassicae* inevitably means that only a limited number of invasive zoospores can ultimately cause symptoms. In addition, there may be competition for rhizosphere space between different physiological races of *P. Brassicae* [113]. Both antagonistic and synergistic relationships between *P. brassicae* races can affect the relationships between physiological forms [114–117]. In any population of *P. brassicae* spores, there may be a range of viability or infectivity, so that some spores, once infected, progress through the life cycle more quickly than others.

It is reported [118], that more than one physiological race of *P. brassicae* can occur in a population or in a suspension of spores obtained from a single spore formation.

Host resistance can be seen as an environmental component that influences the success of *P. brassicae*. From this point of view, it becomes an additional sink for the energy expended on spore penetration. The process of overcoming host resistance may be a function of the biological fitness of successive waves of zoospore infestations, both primary and secondary, as well as the reduction of overall host resistance. Ultimately, successful infections develop in the root hairs of resistant cultivars.

It turns out [119] that specific resistance is expressed against the secondary phase of the *P. brassicae* life cycle. Therefore, during this phase, *P. brassicae* can expend more energy, to no avail in the presence of resistance, but more successfully in the absence of such resistance. Given the highly polygenic nature of some forms of *P. brassicae* resistance, especially in *B. oleracea*, these events may go some way to explaining the time gaps between infections that result in less advanced infection states on assessment days when plants are exposed to lower inoculum concentrations. As a result, the number of infections falls below the observed threshold and is not, in practice, taken into account in the analyses used to determine the value of resistant genotypes. These phenomena have led to much debate as to what constitutes visible or phenotypic resistance to *P. Brassicae* [120].

Perhaps the most controversial issue related to clubroot is the calcium content of the soil and the associated hydrogen ion content (pH). As noted earlier, calcium is a fundamental factor in the life cycles of both *P. brassicae* and its hosts. Detailed long-term experiments have confirmed this [121–127]. It is clear that calcium has the greatest impact when it is present between spore germination and post-penetration to root hairs. The latter period seems to be the time when root hair infection has the greatest impact on the subsequent formation of plasmodia. A very important finding is that high calcium concentrations at pH 6.2 or 7.2 reduce the total number of root infections and the rate of maturation through the plasmodial, sporangial and zoosporangial stages compared to the control. Elevated calcium concentrations completely suppress the late stages of *P. brassicae* development in the root hair, even when high doses of inoculum are applied [128].

Also demonstrated [129] that the effect of pH is independent of calcium concentration, and found that alkaline pH reduces the total number of infected root hairs and slows the maturation of plasmodia, sporangia and zoosporangia. The effect of pH on the maturation of root-hair infections is activated by alkaline pH within 3 days of penetration. Prolonged exposure for more than 3 days has no additional effect. There may be a double effect, as alkaline pH increases the sensitivity of the host and P. brassicae to calcium and also increases the efficiency of calcium uptake. The effects of pH and calcium are remarkably similar, but this does not necessarily mean that they are identical, as some researchers have suggested.

They can regulate the pathogenic potential of the seed quite separately. Since pH regulates the response to calcium, intracellular function can be further modified. The high concentration of H^+ ions in plant tissues is potentially antagonistic to calcium. The permeability of membranes decreases both at alkaline pH and at high calcium content. Such an environment can affect the growth and reproduction of P. brassicae, as it proliferates in epidermal cells, root hairs and epidermis, or in the bark cells of the host plant. The alkaline environment can affect primary and secondary invasion, bark migration and cell hypertrophy.

It was also demonstrated that in the absence of boron, the inhibitory effect of calcium on root hair infection is suppressed, and it was suggested that lime may not be able to reduce the development of canker in soils deficient in boron. The following results were achieved [130–136] a significant reduction in the disease index when sodium tetraborate was applied to acidic granite soils during three consecutive years of field research. Later studies have shown that an environment with an increased concentration of boron has a significant effect on both the root-hair and cortical phases of P. brassicae. At all stages of the life cycle of P. brassicae in planta, boron affects the microbe. There is also a correlation with the amount of boron in the plant, which depends on the uptake in time and space, determined by the size of the plant root system and its ability to absorb boron.

Little is known about the relationships between *P. brassicae* and macro- and microflora and fauna in the soil. Free-floating *P. brassicae* zoospores are undoubtedly a threat to other soil biota. Cases of "disease suppression" may well be associated with the presence of such organisms, which can increase in undetermined quantities either naturally or as a result of agricultural activities. The addition of organic or inorganic fertilisers that stimulate the microflora has a significant impact on the survival of P. brassicae. Bacteria such as Bacillus spp. and fluorescent Pseudomonas spp. are recognised to have an impact on P. brassicae growth [137]. Since spore dormancy walls contain chitin, it is likely that chitinolytic bacteria may be the main antagonists of *P. brassicae*, reducing the potential of the inoculum [138]. Antibiosis induced by microbial sources has generally been viewed as a means of biological control of *P. brassicae* rather than as an advancement in understanding the ecological relationships between organisms. Extensive studies of soil suppressive capacity against *P. brassicae* were conducted by

researchers in the Fukushima area of northern Honshu, Japan. It was found that the haplic andozoic soils were more favourable to *P. brassicae* than the low humus andozoic soils, even though the latter had high concentrations of spores. It has been suggested that the suppressive effect of low-humic andosols is associated with the presence of biological antagonists. Biological inhibition of *P. brassicae* in the presence of Chinese cabbage (*B. raopa*) host plants is reported to be the result of the presence of the soil endophytic fungus *Heteroconium chaetospira* [139].

Crop rotations, especially those containing maize (*Zea mays*), suppressed the activity of *P. Brassicae* [140]. This may be due to ecological interactions and biological control. Primary plasmodia were found in the root cultures of both susceptible and resistant varieties, but secondary plasmodia multiplied only in the cultures of susceptible hosts. It was concluded that the reason for this difference was the alkalisation of the root culture of resistant varieties.

The fungus *Heteroconium chaetospira* suppressed the activity of *P. brassicae* even where the physical soil conditions (moisture and pH) were favourable [141]. Other representatives of soil microflora, such as Bacillus spp., Pseudomonas spp. and Trichoderma spp. reduce the activity of *P. Brassicae* [142] and *Streptomyces* [143–144]. Soil environments created by host and non-host plants, such as leeks (*Allium porrum*), winter rye (*Secale cereale*) and perennial ryegrass (*Lolium perenne*), tended to reduce *P. brassicae* growth in greenhouse studies, but in the field, these effects were less effective.

Thus, clubroot is potentially the most serious disease of cruciferous crops, especially cabbage and closely related crops. It is caused by the parasitic phytopathogen *Plasmodiophora brassicae* Woronin, which is soil-borne and causes economic losses in many regions of the world [145]. In 1878, Mikhail S. Voronin was the first to recognise a plasmodiophoric organism as a causative agent of clubroot and named it *Plasmodiophora brassicae* [146]. Later, this disease was registered in many countries on different continents.

The disease can progress significantly without showing any visible above-ground symptoms. The earliest above-ground symptoms are stunted plant development, flag-like leaves and wilting of the entire plant on hot sunny days, as if the plant is suffering from a water deficit. It looks like wilting when there is enough moisture in the soil. However, the above-

ground symptoms are not enough to diagnose a clubroot infestation. You need to dig up the roots. When such plants are dug up, you can see a hypertrophied root system. The infected root forms "mace-like" nodules on the main and lateral parts of the root system, depending on the type of host plant and the nature of the infection. There are several types of clubbing: (a) clubbing of the entire main and lateral root systems, as in cabbage, (b) clubbing of the main root only, while the lateral roots are free, (c) clubbing of the lateral roots only, while the main root is free, (d) clubbing in the form of a tumour, as in radish, (e) dark decomposed several spots in the root system.

The life cycle of Plasmodiophora brassicae Wor. has three stages: survival in the soil as dormant spores, infection of root hairs and infection of the bark. The life cycle of P. brassicae Wor. begins with spore dormancy. The pathogen persists as dormant spores in the soil or on plant debris. The dormant spore has the ability to survive for a considerable period of time in the soil in the absence of a host crop (Figure 4.31).

Dormant spores in the soil can germinate even after 17 years (Figure 4.32). They release primary zoospores that swim to the surface of root hairs, where they penetrate the cell wall. Primary zoospores are pyriform and bivalve [148], with unequal bifid flagella: one short with a blunt end and the other longer with a pointed end. This stage is known as the root hair infection stage [149]. The pathogens then form primary plasmodia within the root hair. The plasmodia undergo a series of nuclear divisions in synchronisation; eventually, the plasmodia turn into zoosporangia. Each zoosporangium gives rise to 4–16 secondary zoospores, which are released

Figure 4.31 – Cruciferous species that can be hosts of clubrooted cowpea [147]

into the soil, ready to penetrate the cortical tissue of the main root system, a process called cortical infection [150]. Later, the pathogen develops into secondary plasmodia, which are associated with cellular hypertrophy, leading to the formation of galls in tissues. Finally, these plasmodia develop into a new generation of dormant spores that are released into the soil as overwintering structures [151].

In the absence of a host, P. brassicae survives as haploid dormant spores 3–5 μm in diameter. The dormant spore is a very stable structure. Its cell wall (including the membrane) consists of approximately 25% chitin, 2.5% other carbohydrates, 34% protein and 18% lipids [152].

Germination of dormant spores leads to the release of a biflagellate zoospore (primary zoospore). The germination rate increases with the maturity of the spores, increases with increasing humidity and temperature, decreases with alkaline pH and varies depending on the content of certain inorganic ions in the soil [153–154]. In contrast to thick-walled dormant

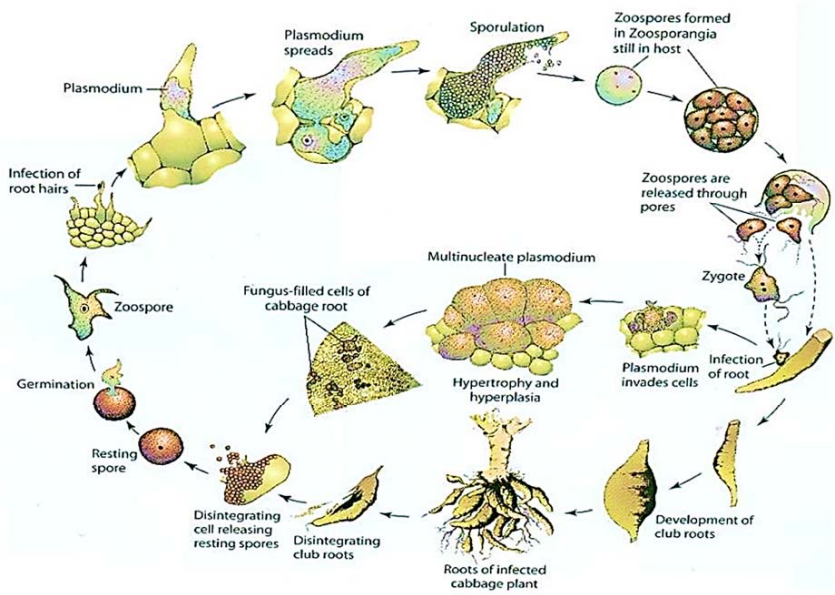

Figure 4.32 – Developmental cycle of cruciferous disease caused by *Plasmodiophora brassicae* [156]

spores, zoospores are sensitive to various types of environmental stress. Without access to the host plant, zoospores are thought to survive only for short periods of time [155].

The abundance and virulence of the pathogen, the susceptibility of the host plant and the suitability of environmental factors influence the severity of the disease. When a plant is infected with a pathogen, environmental factors affect the severity of the disease and the growth, development and yield of the plant.

Clubroot causes huge losses in cabbage yields. In addition, it is almost impossible to eradicate the pathogen from the field once it has been infected. Once a field is infected, management practices to reduce the frequency and severity of the disease include increasing the pH by liming [157], use of decoy plants to reduce the load of pathogen spores in the soil [158], inhibition of germination of dormant spores by means of a neutral pH [159], use of biological products and biocontrol agents [160], postponement of the harvest date for cruciferous vegetable crops to October, when the average air temperature before harvesting is appropriate [161], reducing the density of spores in the soil through crop rotation, steam and nutrient application [162].

Cultural practices, such as crop rotation and improved drainage conditions, can protect crops from disease to a certain extent; however, when infestations are severe, disease control is usually not satisfactory [163]. Some plants, such as lettuce (Lactucasativa), spinach (Spinaceaoleracea) and Italian ryegrass (*Lolium multiflorum*), can be used to reduce root hair infection and clubroot damage. The statement that prevention is much better than cure is the best fit for this disease – once a field is infected, it is very difficult to make it free of the pathogen again. Prevention can be achieved by applying lime to fields with an acidic soil reaction [164]. In addition, a neutral pH level (pH = 7) inhibits the germination of dormant spores [165].

Additionally, it is reported that due to high market prices for rapeseed, the area under rapeseed has increased and crop rotation has narrowed [166]. This has led to an increase in problems with the pathogen and, therefore, another important factor in clubroot control is to maintain crop rotation. A break of more than 2 years or diverse crop rotations can reduce severe clubroot epidemics [167]. In addition, the previous crop can also have an impact on clubroot infection. A recent study compared different preceding

crops before sowing rapeseed. The results showed a 40 and 50% reduction in disease index and incidence, respectively, when soybeans were sown before rapeseed [168]. In addition, microbiological analysis of the soil showed that more bacteria and fungi with known biocontrol functions were found in soybean-rape soil than in soil where corn or other crops were grown [169].

Soil and crop management practices, such as crop rotation, fallow and nutrient application, can reduce the density of pathogen spores in the soil [170]. Recently it has been shown that dormant spores are very sensitive to ultraviolet light [171]. Therefore, tillage practices that bring dormant spores to the soil surface to be exposed to sunlight can be an effective way to support clubroot management.

During one growing cycle of a susceptible variety, the number of dormant spores in the soil increased to 2 x 108/g soil, and that of a resistant variety to 1.7 x 107/g soil, compared to fallow soil.

In addition, sanitation is an important aspect of clubroot prevention. Preventing the movement of dormant spores to a pathogen-free field. As spores live in the soil and move through the soil mass, anything that can move them into pathogen-free soil is a source of the pathogen, such as farm machinery, tractor tyres, boots, tools, livestock and containers. Soil can also be transported with running water. Soil from an infested field can be transported to water sources and other clean fields, which is often problematic during floods. Soil moisture management, especially in the root zone, plays a key role in clubroot control. High soil moisture means that there is a high probability that the bivalve zoospores can infect healthy plants. Over-irrigation and waterlogging should therefore be strictly avoided. Soil has the ability to hold, move and infiltrate a certain amount of water. It is important to improve the physical properties of the soil to accelerate the infiltration of excess soil water into the root zone. To do this, the soil needs organic fertilisers, less tillage and less compaction.

Crop residues, especially root parts, are a source of inoculum or a reservoir of pathogens. Therefore, they should be removed from the field and destroyed after harvesting the economically useful parts of plants. This practice gradually reduces the spore load in the soil.

Boron in the form of borax (boric acid) can suppress clubroot. Boron inhibits both the primary and secondary stages of infection [172].

An aspect that is often overlooked by practitioners is that dormant spores are very easily carried with soil particles, such as machinery, footwear, surface water or animals, thus spreading the disease. In addition, dormant spores have also been shown to be carried by wind-blown dust or soil erosion from field to field [173]. Therefore, measures that can reduce the transfer of spores from field to field, such as sanitising agricultural machinery or measures that help prevent soil erosion, prevent initial infection of fields [174–175].

As soon as the spores enter the field, they are spread not only by cruciferous crops, but also by cruciferous weeds, intercrops and rapeseed residues, which serve as alternative host plants [176], that can be infected by the carpet at any time and therefore should be removed in a timely manner [177].

Sowing resistant varieties is an effective way to suppress the disease. Several resistant loci have been identified by quantitative trait loci mapping in *Brassica napus* and *B. rapa* [178–180] The most widely used resistance loci originate from *B. rapa*; however, these loci do not confer resistance to all *P. brassicae* pathotypes or pathotypes can overcome resistance. Therefore, breeding for resistance remains an important tool for *P. brassicae* control [181].

In addition, pH regulation by liming is an effective way to control clubroot of vegetable cruciferous plants. Although liming is traditionally used as a control measure, it has certain caveats [182-184]. One reason is that the term "liming" refers to the use of different lime compositions. Most commonly, lime is used with different proportions of calcium carbonate (often mixed with Mg^{2+}) [185], cyanamide [186]. In addition, a combination of calcium carbonate and calcium sulphate or calcium hydroxide was used [187]. Calcium oxide, also known as burnt lime or quicklime, is only occasionally used [188]. In addition to the calcium concentration, other factors such as the amount of lime, the date of application and the pH of the soil can affect the development of clubroot. However [189] it is not the effect of calcium that is decisive, but rather the pH of the soil. The germination of dormant spores is drastically reduced at neutral soil pH. Due to the variety of substances and differences in soils, the comparisons of most field trials or greenhouse studies on the effect of lime on clubroot development are rather controversial.

The lack of effective control measures against *P. brassicae* makes it necessary to explore other, new control options. In particular, the use of biological control measures can help reduce the number of soil-borne pathogens. However, the complex life cycle of P. brassicae makes it difficult to apply biological control mechanisms against this pathogen. At least three phases can be used for control: (i) germination of dormant spores or secondary spores that initiate (ii) primary infection of root hairs and secondary infection of root cortex; (iii) antagonism or competition with the pathogen developing in the root tissue of the host plant. In addition, biological control options may include induction of resistance in host plants and changes in microbial communities in the soil rhizosphere [190].

The biological control agents that have been investigated are bacteria or fungi, including oomycetes. The mechanisms are mostly parasitism, antagonism of secondary toxic metabolites or competition. Many studies have illustrated the potential of biological control against soil fungi in the strict sense of the word. This refers to a direct antagonistic or inhibitory effect on the pathogen, rather than indirect effects such as plant growth stimulation or induction of plant resistance [191–192].

Organisms such as, for example, Trichoderma spp. and *Bacillus subtilis sensulato* are commercially used in many products to control various groups of plant pathogens [193–197]. There are numerous examples that illustrate that excellent control results can be achieved in trials *in vitro* [198]. Whereas in field trials, these successful control results often cannot be confirmed [199]. Therefore, for successful control, it is necessary to select proven control measures for clubroot.

The bacteria of the *Bacillus subtilis* species complex are well studied in terms of their biocontrol activity against plant pathogens. These bacteria are capable of producing many hydrolytic enzymes and various secondary metabolites with antimicrobial properties [200]. One of the very well characterised biological control agents patented in China is the *B. subtilis* XF-1 strain. Like other bacillus strains, it produces fungicins, which are a group of non-ribosomal lipopeptides. These metabolites have fungicidal activity and are involved in the biocontrol of many Bacillus species [201–202]. Dormant P. *brassicae* spores directly treated with fungicides were destroyed and the cell contents leaked out [203]. Nevertheless, the mode of action of fengicin was demonstrated on *B. subtilis* NCD-2, which

showed a decreasing effect on the development of clubroot; whereas, when using fungicin, the defective mutants did not show any effect against the clubroot pathogen [204].

Recently, in addition to the new strain *B. amyloliquefaciens*, another member of this genus, *B. velezensis*, has recently been described as a biocontrol agent against *P. brassicae*.

Several species of bacteria in the genus Lysobacter are known for their activity against soil pathogens. These bacteria synthesise many hydrolytic enzymes and antimicrobial compounds, and there are several commercial products against soil fungal pathogens [205]. By screening bacterial strains from vegetable rhizosphere soil, *Lysobacter antibioticus* strains were isolated, and their culture filtrates reduced the incidence of nodule fungi on Chinese cabbage after application as a soil application or seed treatment.

Another strain of Streptomyces, *S. platensis* 3–10, was used to optimise the culture medium and achieved up to 80% inhibition of dormant clubroot spore germination [206]. Recently, a strain of *Bacillus cereus*, MZ-12, isolated from the rhizosphere soil of asymptomatic *B. campestris* (pak choi), showed an inhibitory effect on the germination of dormant spores. Joint inoculation of pak choi plants with *P. brassicae* and MZ-12 spores led to a 64% reduction in the formation of nodule galls [207].

In addition to free-living microorganisms in the rhizosphere or epiphytic microorganisms, endophytic microorganisms can also contribute to biological control. In most cases, endophytic bacteria derived from the rhizosphere enter the plant and colonise its tissues without any negative impact on the plant [208–209]. In many cases, this form of bacterial colonisation contributes to plant growth through various mechanisms [210]. However, the antagonistic activity of endophytic actinobacteria against colic has also been reported [211]. They isolated 81 strains of actinobacteria from surface-sterilised Chinese cabbage root tissue. Among them, they selected three strains that showed in vivo biocontrol activity against *P. brassicae*. Two of these *strains were identified as Microbispora rosea, and the third as Streptomyces* olivochromogenes.

It also tested [212] 63 strains of actinobacteria isolated from the rhizosphere of Chinese cabbage were evaluated for inhibition of germination of dormant P. brassicae spores. As a result, six strains were isolated and used in greenhouse and field trials against clubroot. Strain A316 showed

high control values of 73.69% in the greenhouse experiment and 65.91% in the field experiment.

In general, chemical control of soil-borne diseases is difficult and expensive, and is banned in many countries due to environmental impacts. Of the chemicals registered globally, the oomycete fungicides fluazinam and cyazofamide reduce clubroot damage. They are registered for the treatment of cabbage in some countries, but are not authorised in the EU for the control of clubroot. The use of these products is complicated and expensive, as they need to be applied to the soil to be effective against this pathogen. Therefore, appropriate crop management practices are important measures to control clubroot.

A number of chemicals are fungicidal against fungal plant pathogens, but the term "protozoic" is more appropriate for controlling *Plasmodiophora brassicae* Wor. as this pathogen is not a true fungus. Chemical pesticides should be used as a last resort when all other methods fail. Some chemical pesticides may be banned in one country but not in another. Fluazinam and thiazofamide are quite effective in controlling nodule rot – in advanced disease they are more effective than drugs to fight infection. Some of the most commonly used chemicals against *Plasmodiophora brassicae* Wor. are as follows: Nebizin (Flusulfamide) Among the flusulfamide (2',4-dichloro-a,a,a-trifluoro-4'-nitro-m-toluenesulfonanilide) (trade name: MTF651, Nebizin) dust and suspension formulations, the suspension formulation did not show significant differences. On the contrary, 2.4 kg/ha of flusulfamide was found to significantly reduce clubroot symptoms, including the incidence and severity of the pathogen [213]. Synthetic fungicides are promising for controlling clubroot, and mercury-based fungicides are the most effective, although they are toxic to the environment. Chemicals such as thiazophamide, fluazinam, flusulfamide, procyamidone, and calcium prohexadione reduce the intensity of corm damage by reducing pathogen inoculum density and population composition, but results have not been consistent.

After the identification and evaluation of pentachloronitrobenzene (PCNB), it was reported that chlorinated nitrobenzene could provide significant control in fields that are not heavily infested with the pathogen. In field trials conducted in Alberta, soil impregnation with PCBs (Terraclor 75% WP) reduced clubroot blight and seedling mortality and

increased plant height and assimilation surface area of canola. In greenhouse trials of chemicals, soil applications of benomyl, methyl thiophanate and NF 48 were found to have the potential to control clubroot [214].

Hydrogen peroxide (H_2O_2) is a by-product of cellular metabolism and is mainly produced in mitochondria, chloroplasts, peroxisomes, plasma membrane and cell wall [215]. It induces the expression of the PR 1 gene and systemic acquired resistance (SAR) to pathogens [216]. The CHC is a whole-plant resistance response following a previous localised exposure to a pathogen. In plant-pathogen interactions, hydrogen peroxide can limit pathogen infection by directly inhibiting the pathogen or inducing defence genes in plant cells. In addition, H_2O_2 plays a key role in the regulation of plant growth and development. H_2O_2 mediates stomatal closure induced by abscisic acid (ABA), which is an endogenous antitranspirant that reduces water loss from the leaf surface, preventing wilting of plants [217].

A number of chemicals related to alkylene bisdithiocarbamates are active against clubroot. Among them, mancozeb (manganese ethylene bisdithiocarbamate) is one of the most effective. Similarly, another chemical related to alkylene bisdithiocarbamates, Zineb (zinc ethylene bisdithiocarbamate), has been used as an alternative to benomyl, but this substance has been shown to be phytotoxic to cauliflower [218].

Carbendazim is a broad-spectrum benzimidazole. It is a metabolite of benomyl that is used for the treatment of grafts against Plasmodiophora brassicae Woronin. Carbendazim (methyl lH-benzimidazol-2-ylcarbamate) has proven effective for clubroot control only when applied at very high rates of 80–100 kg/ha, but this makes it expensive and contaminates the soil [219].

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