Research progress in sunflower diseases and their management

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ABSTRACT

Sunflower diseases are of major concern in the production of this crop worldwide. This is due to the regular and quite often severe attack by different pathogens. As a result considerable yield losses occur or the quality of product lessens. Though the number of pathogens known to attack sunflower is relatively high, only a handful to a dozen are considered important ones depending on region and cultivar. In this review, I am focusing particularly on these significant pathogens. The emphasis will be on new findings and results obtained by researchers related to pathogen biology, ecology, genetics, host resistance, and control. It was interesting to note a considerable shift in the relative dominance of diseases over the last four years as reflecting in the number of publications available. The scientists' efforts have resulted in better understanding of individual diseases and underline their significance in the improvement of sunflower management.

Key words: ecology – diseases – genetics – host resistance – pathogen biology

INTRODUCTION

Sunflower diseases are one of the major constraints in influencing production stability of this crop worldwide. Though there are more than a dozen of pathogens that may attack cultivated sunflower resulting yield losses, just a few are of concern in a particular country or region. In a literature survey of the past four years (2004 to 2007), I found a great number of publications regarding sunflower diseases. Similarly to Felicity Vear's findings reported at the 16th International Sunflower Conference in Fargo (Vear, 2004), there were significant differences in the number of papers dealing with individual pathogens and/or originating from different countries and regions. An overview of reference sources obtained from the Web of Science or kindly provided by individual scientists served as a basis of this present review. A total of 13 sunflower pathogens were the subject of papers available and there were major differences in the proportion of these references (Table 1). Most of the publications dealt with downy mildew (*Plasmopara halstedii*), and broomrape (*Orobanche cumana*), followed by white rot (*Sclerotinia sclerotiorum*), stem canker (*Diaporthe helianthi*), Alternaria blight (*Altermaria helianthi*, *A. helianthinficiens*), rust (*Puccinia helianthi*) and black stem (*Phoma macdonaldii*) in a decreasing sequence of order. Some diseases of local importance represented by several or just a few references are Verticillium wilt (*Verticillium dahliae*), white blister rust (*Albugo tragopogonis*), charcoal rot (*Macrophomina phaseolina*), Fusarium wilt (*Fusarium* spp.), Rhizopus head rot (*Rhizopus arrhizus*), and *sunflower chlorotic mottle virus* (SuCMoV). In addition, information on sunflower diseases is also available in a Progress Report by Masirevic (2005a) based on contributions of the sub-group leaders of the Working Group Sunflower Diseases, presented at the 10th FAO European Research Network on Sunflower Consultation Meeting in Novi Sad in 2005.

Table 1. A list of references concerning sunflower diseases for the period 2004-2007 available for the author

Disease	Pathogen	No. of records
Downy Mildew	*Plasmopara halstedii*	46
Broomrape	*Orobanche cumana*	31
White rot	*Sclerotinia sclerotiorum*	17
Stem canker	*Diaporthe helianthi*	15
Alternaria blight	*Alternaria helianthi*, *A. helianthinficiens*	15
Rust	*Puccinia helianthi*	12
Phoma black stem	*Phoma macdonaldii*	10
Virus	*Sunflower chlorotic mottle virus*	7
Verticillium wilt	*Verticillium dahliae*	5
Charcoal rot	*Macrophomina phaseolina*	4
White blister rust	*Albugo tragopogonis*	4
Fusarium wilt	*Fusarium* spp.	3
Rhizopus head rot	*Rhizopus* spp.	1

DISCUSSION

Downy mildew. Based on a literature survey for the years 2004-2007, most of the publications for this period have dealt with this devastating disease caused by *Plasmopara halstedii* (Farl.) Berl. et de Toni. It continued to occur in almost all parts of the world where sunflower was grown, except for Australia. The biology and ecology of this organism is well-known as are many aspects of its pathogenicity, host – pathogen interaction and genetic and chemical control. Its capacity to diverse, both in virulence and fungicide, however, is very high, giving a continuous challenge to scientists.

The most detailed and up-to-date list of global distribution of *P. halstedii* pathotypes has been compiled by Gulya (2007) in a paper presented at the 2nd International Downy Mildew Symposium, Olomouc, Czech Republic. In this accurate overview he comprised as many as 35 pathotypes (races), an unbelievably high number considering the fact that in most sunflower producing countries from just a few to 12 well-distinguished virulence phenotypes exist. In Europe, France, Germany and Spain reported the highest numbers but the pathogen is rather diverse in the USA, Canada, and in South Africa as well. Furthermore, there are five *P. halstedii* pathotypes (300, 330, 710, 730, 770) that are universally distributed globally, recorded from North and South America, Europe and Africa. Apart from the quantitative aspect of virulence, it is interesting to consider the dynamics of diversity as well, i.e. the changes in a given region over time. In this respect, France leads with the highest number of new pathotypes arisen in the last 6-7 years (Vear et al., 2006). Considering population changes for virulence, a good example has recently be found in the USA by Gulya (unpublished), where 3 out of 11 pathotypes (710, 730, 770) were recorded from North and South Dakota and Minnesota in each year during the 1998 to 2007 period whereas two others (300, 772) appeared in one year and a third one (300) in two years only. Recently, Delmotte et al. (2008) analyzed the possible origin of *P. halstedii* populations existing in France using different molecular methods. Based on single nucleotide polymorphisms they assumed a multiple introduction into France of the pathogen populations exhibiting differences in virulence phenotype.

Like other biotrophic obligate parasites, *P. halstedii* has a narrow host range. In other words, though it has originally been described to occur on a number of Composites and was found to attack a few wild *Helianthus* species as well, until recently no much attention has been paid to any alternate host as potential infection sources. Recently, however, two records of the natural occurrence of this Oomycete on wild asteraceous plants appeared, on velvetleaf (*Abutilon theophrasti*) by Masirevic (2005b) in Serbia and on *Rudbeckia fulgida* by Hong (2006) in Virginia. With these records, a total of five asteraceous wild plant species (the other three being *Xanthium strumarium*, *Ambrosia artemisiifolia* and *Iva xanthifolia*) are known to be as alternative hosts of *P. halstedii*. The natural host state in each case has been proved with successful reinoculation to cultivated sunflower.

With the rapid improvement of molecular techniques and their use in plant pathology, new developments have opened new insight into research on fungal biology, detection technology, and genetics and host - pathogen interactions. For example, Hammer et al. (2007) in Germany, and Ioss et al. (2007) in France, using different approaches, were successful in detecting fungal structures from sunflower host tissues.

Furthermore, it became possible to study the genetic recombination in *P. halstedii* through parasexual events using DNA fingerprinting (Spring and Zipper, 2006). In attempts to characterize the molecular structure of this Oomycete, Thines and co-workers (2005) detected and characterized an exceptional length of ITS that was due to multiple repetitions in the ITS-2 region. Further, ITS sequence data were also used to detect possible differences between isolates differing in virulence and/or in geographic origin.

Recently, two papers dealt with the isozyme analysis of *P. halstedii* isolates in order to find out intraspecific polymorphism in sub-populations of this organism. Guchetl et al. (2007) studied eight different isoenzymes. While interracial differences on esterase were inessential, the other seven isoenzymes appeared to be monomorphous for all pathotypes studied (330, 700, 710 and 730) suggesting that downy mildew populations in the Krasnodar region had low intraspecific variability for these traits. Komjáti et al. (2008) in Hungary used cellulose-acetate gel electrophoresis to analyze sixteen isozyme systems of 10 field isolates and 35 single-spore lines representing 10 different virulence phenotypes. Apart from sunflower, isolates were also from cocklebur (*Xanthium strumarium*) and from *Helianthus x laetiflorus*. Three isozymes, isocitrate dehydrogenase (IDH), malate dehydrogenase (MDH) and phosphoglucomutase (PGM) revealed some polymorphism among the isolates. PGM differentiated two groups among the isolates from cultivated sunflower, while the other enzymes were polymorphic between isolates from different hosts. However, polymorphisms did not relate to virulence phenotype.

To develop and release sunflower cultivars resistant to different pathotypes is of extreme importance to growers. Therefore, breeders are continuosly searching for new genes or gene clusters conferring resistance to *P. halstedii* (Sreten et al., 2007), and selecting for such genes using molecular markers. In the recent years, Radwan et al. (2004) in France and Dussle et al. (2004) in Germany achieved considerable results with PCR markers for the *Pl5*/*Pl8* locus from complete CC-NBS-LRR sequences and, with the localization of the Pl_{arg} gene using SSR markers, respectively. Furthermore, in inheritance studies of resistance to the 703 pathotype Pankovic et al. (2007) used both traditional segregation tests and PCR markers and obtained identical results related to the *Pl6* gene conferring resistance to 730.

At the Fargo conference, Felicity Vear presented an outstanding review of recent breeding work for resistance to sunflower pathogens (Vear, 2004). At that time she outlined the importance of durable resistance to combat the increasing number of new *P. halstedii* pathotypes overcoming the *Pl* gene-mediated resistance. Since then the Clermont-Ferrand group has proceeded with this work and some of their results have already been published (Tourvieille et al., 2005; Vear et al., 2006). Durable resistance was found to be independent of major gene resistance so they proposed for the future to combine both types of resistance in new cultivars for more effective and long-lasting genetic control. Partial resistance to *P. halstedii* in high oleic sunflower hybrids have been reported by Baldini et al. (2006) as well in Italy.

The hypersensitive reaction (HR), a well-known phenomenon among plant pathologists, has been the subject of investigations by Radwan et al. (2005) to characterize this mechanism in the sunflower downy mildew system. RT-PCR analysis showed that resistance was associated with the activation of a hsr203J-like gene, a molecular marker of HR in tobacco. Activation of this gene was specifically observed during the incompatible interaction and coincided with cell collapse in hypocotyls. No such HR or a significant activation of the hsr203J-like gene were observed during the compatible combination suggesting that HR failed to halt the parasite, rather it trigged a systemically-aquired resistance (SAR) taking place in the upper part of the hypocotyl and this might arrest pathogen growth.

Apart from durable resistance, induced resistance might be useful for improving downy mildew management. In the recent years, Hungarian and Spanish laboratories conducted studies to better understand this type of host defense. The plant activator benzothiadiazole (BTH) significantly depressed disease symptom appearance and pathogen growth in susceptible sunflowers treated and inoculated at the germling stage (Körösi et al., 2007). Furthermore, microscopical observations revealed a high similarity between genetic (*Pl*-gene mediated) and induced resistance responses in compatible combinations. Roldan Serrano et al. (2007) recently published a paper about chitinase and peroxidase activities in BTH-treated sunflower inoculated with *P. halstedii*. They found an increased level of activity for both enzymes in susceptible but not in resistant seedlings. In our laboratory, we compared susceptible, partially resistant and resistant interactions for various enzyme activities (unpublished) and also tested the glutathion S-transferase (GST), defensin (PDF) and catalase (CAT) gene expression. Preliminary results of this work will be presented by Körösi et al. in this conference.

Resistance or tolerance to metalaxyl has already been noted in France and the USA. Quite recent records, however, came from Spain (Molinero-Ruiz et al., 2005) and Germany (Spring et al., 2006). It is interesting to note that, as an alternative, Fernández-Ocaña et al. (2004) conducted experiments with the essential oil of *Bupluerum gibraltarium*. They found this oil acting as a host defense activator rather than directly inhibiting sporulation.

Broomrape. *Orobanche cumana* Wallr. is a parasitic plant that infects sunflower causing considerable damage. In Spain, parasitized plants exhibited lower shoot dry weight, and they were shorter (due to reduction in internode length) with smaller head diameter as compared to healthy ones (Alcántara et al., 2006). In addition, a significant decrease in the mineral composition of the leaves of affected plants could be detected.

Different pathogenic races of *O. cumana* are known to exist in various regions of Europe and in the southeastern Mediterranean where the climate is favorable for this parasite. Due to this genetic diversity a new pathogenic form, race F appeared recently in Spain (Pérez-Vich et al., 2004), in Russia (Goncharov et al., 2004), in Turkey (Kaya et al., 2004), in Israel (Eizenberg et al., 2004), and in Bulgaria (Shindrova, 2006), with the highest diversity existing in Turkey. More expanded field surveys and subsequent identification processes are required to get a better view of the incidence and distribution of pathogenic variants of this parasite. In this respect, Román et al. (2007) succeeded in developing a detection method by using cpDNA diagnostic markers and they proposed this molecular protocol for use in identification work.

In a study on the mechanism of broomrape parasitism in sunflower, Slavov et al. (2004) pointed out that seed germination of the parasite was triggered by a germination stimulant secreted by the host-plant

roots. Further, they quantified indole-3-acetic acid as early as 24 h after the seeds were exposed to the germination stimulant, suggesting the role of IAA in the germination process. When comparing different populations of this parasite for their virulence on different sunflower genotypes, Veronesi et al. (2005) found that before attachment, *Orobanche* seedlings released cell-wall-degrading enzymes such as pectin methylesterase and polygalacturonase. These enzymes' activity were very high in the most virulent, recently discovered race F. Eizenberg et al. (2005) developed a new methodology that allowed them to facilitate the in-situ study of major aspects of the host - parasite interaction.

Broomrape resistance is poorly understood and new races of the parasite evolve rapidly to overcome the resistance of newly introduced sunflowers. Labrousse et al. (2004) screened a number of recombinant inbred lines derived from interspecific crossings. A considerable variation in the characters tested showed that polygenic resistance could occur in some lines. In another experimental system Echevarría-Zomeno et al. (2006) investigated the histology of host – parasite interface. Suberization and protein cross-linking at the cell wall were seen in the resistant sunflower cells in contact with the parasite and confocal laser microscopy revealed accumulation of phenolic compounds during the incompatible reaction. Letousey et al. (2007) carried out molecular analysis of the resistance mechanism. RT-PCR and cDNA blot experiments revealed that the *Orobanche* resistant genotype exhibited a stronger overall defense response against *O. cumana* than the susceptible one. The SA-responsive gene, *def.* (defensin), appeared to be characteristic of LR1 sunflower resistance. Ha-DEF1 (a sunflower defensin) was found to induce cell death in the parasitic plants appearing as a brown symptom at the radicle apex of the parasite (de Zélicourt et al., 2007). The resistance phenomenon to broomrape in sunflower was also the subject of studies to map and characterize quantitative trait loci for resistance to race E and race F by Pérez-Vich and co-workers (2004). Their results suggested that resistance to broomrape in sunflower is controlled by a combination of qualitative, race-specific resistance affecting the presence or absence of broomrape and a quantitative, non-race specific resistance affecting their number.

In inheritance studies on the sunflower line J1 to *Orobanche* race F, Velasco et al. (2007) detected incomplete dominance of the *Or6* alleles and subsequent segregation ratios suggested the presence of a second gene, *Or7*, the expression of which was influenced by the environment. Meanwhile Spanish breeders were successful in finding sunflower germplasm resistant to race F of broomrape (Fernandez-Martinez et al., 2004) and those with quantitative resistance to the same race (Pérez-Vich et al., 2006). In addition, sunflower hybrids resistant to race F have been released in Spain (Pérez-Vich et al., 2004), in Russia (Goncharov et al., 2004), and in Turkey (Kaya et al., 2004).

For future broomrape management it might be of interest to consider to use host defense system as an alternative to genetic resistance. Buschmann et al. (2005) reported about positive results with BTH against *O. cumana* infestation, and later on Fan et al. (2007a), from the same laboratory, evaluated the efficacy of prohexadione-calcium against this parasite. Neither of these plant activators had a direct effect on the parasite, but rather induced host defense only.

An additional way of broomrape control could be by using biological antagonists. One of the candidates is *Fusarium oxysporum* f. sp. *orthoceras*, which was the subject of investigations by Dor et al. (2007). They studied the pathogenicity and toxin production of this fungus. Two main toxic metabolites caused mortality of germinating broomrape seeds and these were identified as fusaric acid and 9,10-dehydrofusaric acid. Müller-Stöver et al. (2004) also found *F. oxysporum* f. sp. *orthoceras* (Fo) as a potential of biocontrol agent and they were successful developing two granular formulations under laboratory conditions. In an other experiment Müller-Stöver and co-workers (2005) were able to increase control of *O. cumana* through integration of Fo with BTH-treatment. Under laboratory conditions no enhancing effect of BTH on virulence and growth of the fungus was observed. Fan et al. (2007b) achieved similar results when they combined the application of Fo and acibenzolar-S-methyl (ASM). The interaction between ASM and Fo was highly significant on *O. cumana* number and dry matter. ASM soil drenches combined with Fo were more effective than ASM foliar spray with Fo.

White rot. Sunflower stalk and head rot incited by *Sclerotinia sclerotiorum* (Lib.) de Bary is considered the most important disease of this crop in many parts of the world. Since cultural practices or fungicides are insufficient to control the disease, efforts are being made by breeders to develop resistant or tolerant cultivars. This may explain the dominance of publications dealing with various aspects of resistance.

Disease incidence of white rot may vary with location and season, as well as with sunflower genotype, and the symptoms appearing on sunflower stem or head are also diverse. For example, in the United States a three-year field survey (2005-2007) was made by Tom Gulya and co-workers (2008) in the main sunflower growing regions (North and South Dakota, Minnesota) regarding *Sclerotinia* stalk rot

occurrence. Both, the percentage of fields with stalk rot and the severity of affected fields varied between 16-30 %, and 4.4-6.3 %, respectively.

In the recent years, several reports have dealt with the evaluation methods of sunflower genotypes for resistance to *S. sclerotiorum*. Thus, Baldini et al. (2004) in Italy compared host reactions to basal stem and head inoculation, Pedraza et al. (2004) in Argentina examined the suitability of the length of susceptible period as a measurement of partial resistance, van Becealere (2004) in the USA described an improved screening method for assessing head rot resistance, and Castaño et al. (2005) compared the reaction of sunflower accessions to both *S. sclerotiorum* and *Albugo tragopogonis*. By looking for resistance sources among the wild *Helianthus* species, Cáceres et al. (2006) found differences in lesion length of leaves following inoculation but it was not the case with stem inoculation.

In a breeding program in France, Felicity Vear and co-workers (2007) aimed at improving the *S. sclerotiorum* head rot resistance using recurrent selection of a restorer population. In 4 cycles an 80 % reduction in diseased area was obtained and thereafter the population remained stable and homogenous for this character.

Maringolo et al. (2007) in Argentina successfully studied quantitative trait loci for sunflower capitulum resistance to head rot.

Stem canker. *Diaporthe helianthi* Muntanola-Cvetkovic, Mihaljcevic et Petrov (anamorph: *Phomopsis helianthi* Muntanola-Cvetkovic, Mihaljcevic et Petrov) has become a serious threat in sunflower production in the early 1980s in Europe and subsequently in other parts of the world, e.g. in North and South America. Relatively soon after its appearance, it became one of the most limiting factor of sunflower production in many parts of Europe, including the former Yugoslavia, Romania, Hungary, and France. However, following several years of epidemics in these countries, the disease occurrence lessened probably due to unfavorable weather conditions (dry and hot). In contrast, Huguet (2005) reported about a severe attack of this pathogen from a region of Uruguay close to the Argentinian border having an average incidence of 39%.

Walcz and Nébli (2006) investigated the persistence of this pathogen in infected stems and achenes. They found that *D. helianthi* perithecia even disposed to outdoor conditions for 3 years produced viable ascospores, as well as a few pycnidia (the latter occurring most on achenes). The fact that *D. helianthi* can be distributed with seed underlines the importance of phytosanitary measures in seed production and commerce.

Molecular studies on the intraspecific diversity of this fungus using intergenic spacer sequence analysis revealed a high homology among French/Yugoslavian and among Italian isolates (Pecchia et al. 2004). The phylogenetic tree obtained from the aligned data revealed three separate groups. The analysis also showed that all isolates originating from countries with regular and severe outbreaks of the disease (e.g. France, Yugoslavia) formed a well-defined taxon with relatively low variability compared to isolates from Italy where the disease is much seldom to occur. In another paper, Rekab et al. (2004) pointed out a polyphyletic nature of this fungus.

Besides traditional methods of resistance testing (Walser et al., 2005), Quaglia and Zazzerini (2007) reported about an *in vitro* screening for sunflower calli to *D. helianthi* fungal culture filtrate. Looking for recent publications regarding resistance breeding programs, only two reports were available. A collaborative work between Bulgaria and Germany (Encheva et al., 2004) evaluated somaclonal variation, and a study from Hungary (Csikász et al., 2006) in which selection of elite lines was described for specific resistance alleles.

Alternaria blight. The disease can be incited by two fungi *Alternaria helianthi* (Hansf.) Tubaki et Nishihara and *A. helianthinficiens* Simmons, Walcz et Roberts, but Gonorazky et al. (2005) described *A. alternata* as well as one of the seed infecting species found in Argentina. Calvet et al. (2005) determined the average decrease in the photosysnthetic rate in diseases leaves, and Leite et al. (2006) showed that disease severity could be used as an independent variable in a sunflower – *Alternaria* leaf spot management system by providing recommendations for resistance breeding or for studies on sowing date.

Madhavi et al. (2005a) compared six wild *Helianthus* species for resistance to *Alternaria* blight: *H. occidentalis* and *H. tuberosus* were found highly resistant, and *H. hirsutus* moderately resistant. Furthermore, on growth media supplemented with leaf extracts of these plant species the inhibition of fungal growth corresponded to *in vivo* responses of the particular species to inoculation. Further, the resistant *Helianthus* species possessed higher levels of constitutive as well as induced total phenols and total sugars as compared with susceptible sunflowers (Madhavi et al., 2005b).

Resistance breeding was the subject of several papers appeared in the recent years. De Oliveira et al. (2004) reported about mutation breeding from Brazil, and Murthy et al. (2005) assessed heritability of resistance using molecular markers. In India, ploidy manipulation and introgression of resistance to *A. helianthi* using wild *Helianthus* species as resistance source (Sujatha and Prabakaran, 2006), sporophytic and gametophytic recurrent selection for improving partial resistance (Rani and Ravikumar, 2006; 2007), and the description of transcripts during the necrotrophic interaction with *A. helianthi* (Anjana et al., 2007) reflected to the relative dominance of this disease in this country.

Rust. *Puccinia helianthi* Schwein. has a world-wide distribution but it has been considered as a severe pathogen causing considerable yield losses mainly in Australia and Argentina (Huguet et al. 2007). However, Zazzerini et al. (2005) reported about a considerable occurrence and spread of this disease from Mozambique as well.

The diversity of the sunflower – *P. helianthi* pathosystem has got a special attention by Sendall and co-workers (2006) describing a rapid and frequent virulence changes in the rust fungus population. Virulence data accumulated over 25 years coupled with studies on genotypic diversity and sexual reproduction permitted them to conclude that *P. helianthi* may evolve in wild sunflower populations providing a continuum of genetically heterogenous hosts on which this fungus can potentially complete its sexual cycle.

In Spain, Prats et al. (2007) carried out experiments to characterize the mechanism of resistance. Microscopical observations revealed that rust development depended on host genotype, i.e. impairment of rust spore germination and of appressorium formation associated with different excretion of coumarin on leaf surface. Mohase et al. (2006) in South Africa investigated the effect of rust infection on intercellular beta- 1,3-glucanase and chitinase activities, PAL acitivity and total salicylic acid content in relation to susceptibility vs. resistance interactions. Rust infection selectively increased the activity of pathogenesis-related proteins and other parameters studied. Treatment of susceptible plants with BTH induced intercellular glucanase activity and reduced susceptibility to rust. Induced resistance was also the subject of another paper by Amzalek and Cohen (2007) from Israel. Besides BTH, they used other inductors as DL-3-amino-n-butanoic acid (BABA), 2,6-dichloroisonicotinic acid (INA), and two enantiomers of BABA as well.

Phoma black stem. The disease is caused by *Phoma macdonaldii* Boerema (teleomorph: *Leptosphaeria lindquistii* Frezzi) appearing as black spots on stems and seldom on leaves of affected plants. Though its occurrence is quite common in several European countries, the disease is extremely severe in France where basal stem lesions often result in lodging. This could be the reason of the absolute dominance of French publications that appeared in the recent years. Darvishzadeh et al. (2007a) undertook experiments to determine the partial resistance of sunflower genotypes to seven isolates and highly significant differences were observed among genotypes, isolates and their interactions. Two genotypes exhibited specific resistance with a wide range of isolate-nonspecific partial resistance appearing as well. In addition, QTLs were also found associated with isolate specific and non-specific partial resistance (Darvishzadeh et al., 2007b). Alignan et al. (2006) developed a 1000-element cDNA microarray containing genes putatively involved in primary metabolic pathways in order to identify genes responsible for partial resistance. They were successful in identifying 38 genes differently expressed among genotypes, treatments and times. Comparative genetic analysis for the characterization of QTL involved in resistance of sunflower to *Ph. macdonaldii* has been made by Bert et al. (2004), and QTL mapping of partial resistance to stem and root necrosis in sunflower as well as inheritance studies were the subject of investigation by Abou Al Fadil et al. (2006, 2007).

Verticillium wilt (*Verticillium dahliae* Kleb.) is considered an important disease affecting sunflower in most production areas in Argentina (de la Vega et al., 2007), and is of concern also in Canada and the Unites States. Estimation of yield losses is difficult because of the absence of highly efficient chemical control (Creus et al., 2007). Therefore, host resistance is a major concern of breeders in these countries. Resistant breeding is in progress in Argentina (Maranesi and Mancuso, 2007) and in the United States (Radi and Gulya, 2007).

Charcoal rot (*Macrophomina phaseolina* [Tassi] Goidanich) may cause premature death of sunflowers grown on light, sandy soil under hot and dry climate. The disease is well-known in the Southern part of Europe, but the first occurrence in Slovakia was unexpected and probably due to the extremely warm and

dry seasons at that time (Bokor, 2007). In Hungary, Walcz and Piszker (2004) have developed an inoculation method for screening sunflower lines for resistance to this pathogen.

White blister rust (*Albugo tragopogonis*) is know to occur as a pathogen of significance only in South Africa, but it was recorded recently from Germany (Thines et al., 2006a) where the percentage of affected plants varied between 20 and 80 %. Another record is known from Belgium (Crepe et al., 2006). Thines et al. (2006b) studied the fatty acid profile, ultra structural characteristics and ITS sequencing of this fungus. Castaño et al. (2005) evaluated the reaction of a number of sunflower accessions originating from the North Central Regional Plant Introduction Office, Ames, Iowa, USA. Statistical analysis showed differential responses to white rust severity, incidence and relative incubation period among the accessions tested.

Fusarium wilt (*Fusarium* spp.) has been reported as a pathogen of concern only from Russia (Antonova, 2004) where it appeared to be harmful for sunflower production. Based on the extent of necrosis incited by the fungus on the main root and the root – hypocotyl transition zone of sunflower seedlings, some tolerance to pathogen attack could be detected among the genotypes (Antonova et al., 2005). In a breeding program a number of new breeding lines were developed exhibiting relatively good field tolerance (Goncharov et al., 2006).

Rhizopus head rot (*Rhizopus* spp.) exists under warm climatical conditions, like in North Africa, Australia and India. However, with the global warming it seems to occupy new areas, such as the Mediterraneum and southern Hungary. The most typical disease symptoms include rotting of sunflower head with a loose cover of grayish fungal spore mass. In Hungary Walcz et al. (2004) examined the effect of *Rhizopus* head rot on the oil content, oil quality and the germinability of seeds. All these parameters showed a negative tendency in the affected seeds as compared to healthy ones.

Virus (*Sunflower chlorotic mottle virus*, SuCMoV) has recently been detected in commercial hybrids and wild sunflowers in Argentina (Lenardon et al., 2005; Lenardon and Gioletti, 2007). More than two hundred lines were screened for resistance using artificial inoculations under greenhouse conditions and only three lines showed partial resistance in which virus replication was delayed and morphological traits (plant height, leaf width) were less affected. Arias et al. (2005) studied the mechanism of oxidative damage in SuCMoV infected sunflowers. Jan and Gulya (2006) in the United States reported about the registration of three virus resistant sunflower genetic stocks.

CONCLUSIONS

In this review I tried to outline the recent progress in research and development of sunflower diseases involving various aspects of each, in most cases depending on the amount of information available. Unfortunately there was no means to include all relevant experimental data, due to the page limitation for plenary papers. Similarly, I did not try to make any statistics in relation to diseases, topics or countries. Instead, I want to express my hope and feeling that the great number of references discussed in this review will give the reader an up-to-date summary of the most recent pathology research, the impact of diseases on sunflower production, and challenges remain for researchers.

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