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OVERVIEW OF FUNGAL PATHOGENS INVOLVED IN WHEAT LEAF SPOT COMPLEX - PREVALENCE, RELATIVE IMPORTANCE AND PLANT RESISTANCE

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Abstract

Under this general name are included the symptoms caused by several phytopathogenic fungi. The negative effect on wheat plants is mainly due to the reduced photosynthesizing area and the accelerated aging of the leaves, which leads to the poor nutrition of the grain, significant losses in yield and lowering the quality of the production. This determines the great economic importance of these diseases and the need for their in-depth study. The term wheat septoria refers to diseases caused by three anamorphic fungal pathogens of the genus Septoria. The fungal pathogens involved in the leaf spotting complex include the tan spot diseases caused by Pyrenophora tritici-repentis, Cochliobolus sativus, Monographella nivalis and several species of the genus Alternaria. An important component of the integrated control of septoriosis is genetic resistance. No complete resistance has been established in wheat to Zimoseptoria tritici, Parastagonospora nodorum and Parastagonospora avenae f. sp. triticea. Over 20 major septoria tritici blotch resistance genes have been mapped. Sources of quantitative resistance that are longer lasting under field conditions have also been identified.

Key words*: fungal pathogens, genus resistance, Septoria wheat, spred.*

INTRODUCTION

Wheat is a traditional crop in Bulgaria and it plays an essential part in terms of economic significance and distribution within the country's agricultural practices and crop rotation. A major problem affecting wheat production is the occurrence of pathogens causing diseases that lead to reduced yields and grain quality. In recent years, there has been an increasing occurrence of leaf spotting of wheat, possibly due to changes in agrotechnical practices (minimal processing, nitrogen fertilization, monoculture cultivation), the use of new, more susceptible varieties, and favorable climatic conditions. The distribution and relative importance of leaf pathogens in certain cultivation regions have changed drastically due to new market trends, which prompt changes in the agricultural practices and introduce new varieties. Monitoring these changes is crucial to implementing appropriate and timely measures to prevent the spread of diseases. Information on the most commonly encountered fungi causing leaf spotting of wheat will aid in prioritizing disease resistance in breeding programs.

Leaf spotting of wheat is a complex disease with a complex etiology. Depending on the varietal composition and specific meteorological conditions during the growing season, certain species may predominate in the complex. Under favorable conditions, leaf spots proliferate, merge, and form large areas of necrotic tissue. Heavily affected leaves dry up and die prematurely. Fungal pathogens in wheat significantly limit the obtained yields and deteriorate the quality of production. Diseases caused by them can lead to annual losses of up to 15-20%, with rusts, leaf spots, and spike blight contributing most significantly (Figueroa et al., 2018).

The aim of this study on the species composition of fungi in the leaf spotting complex was to provide a solid theoretical foundation for further phytopathological and genetic breeding research.

MATERIALS AND METHODS

Septoria diseases in wheat are important diseases with a significant impact on wheat production in many countries worldwide (Figueroa et al., 2018). Their growing economic importance is attributed to the introduction of high-yielding, low-stem, and susceptible wheat varieties; changes in the cultivation technology of the crop; increased use of nitrogen fertilizers; and falling behind in utilizing genetic resistance compared to the increased resistance to other foliar pathogens such as rusts and powdery mildew. Several reviews on Septoria diseases in wheat have been published (Shipton et al., 1971; King et al., 1983; Eyal, 1999; Cunfer & Ueng, 1999), as well as one monograph (Lucas et al., 1999).

The term "Septoria diseases in wheat" historically referred to diseases caused by three fungal pathogens with an anamorph of the genus *Septoria: S. tritici, S. nodorum*, and *S. avenae* f. sp. *triticea*. Further on, the last two species were renamed to *Stagonospora* based on the length-to-width ratio of their conidia. It is accepted that in the genus *Septoria* spp., unlike those in *Stagonospora* spp., they are 10 times longer than they are wide (Cunfer & Ueng, 1999). A few years ago, modern mycologists made taxonomic changes to *S. tritici*. A new genus, *Zymoseptoria* gen. nov., was introduced, and several *Septoria* species, including *S. tritici*, found on cereal hosts, were placed in it. Both *Stagonospora* species (*S. nodorum* and *S. avenae* f. sp. *triticea*) were assigned to a new genus, *Parastagonospora* (Quaedvlieg et al., 2013).

Zymoseptoria tritici

Z. tritici is one of the most important fungal pathogens in wheat in Europe and many regions in Africa, Asia, North and South America (Chartrain et al., 2004; Dean et al., 2012; O'Driscoll et al., 2014; Fones & Gurr, 2015; Figueroa et al., 2018). It causes a destructive leaf disease known globally as septoria tritici blotch (STB), and in our country, it is referred to as spring leaf spotting. In Europe, STB is the main threat to the production of this crop and leads to costs for EU producers estimated at 280-1,200 million euros annually, including direct losses and expenses for fungicides (Fones & Gurr, 2015).

Z. tritici is a polycyclic pathogen that appears at the beginning of the growing season and can go through up to 6 cycles by its end (Fones & Gurr, 2015). Typical symptoms of STB appear 14-21 days after infection and consist of irregular gray-brown spots where light to dark brown pycnidia develop. This necrotic damage reduces the photosynthetic capacity of the leaves. Damage to the infected leaf tissue induces complex changes in the plant's carbon metabolism and assimilate distribution. The disease causes significant losses (up to 50%) in wheat yield, usually associated with a substantial reduction in green area (Eyal et al., 1999).

Z. tritici is a hemibiotroph with a two-phase life cycle. The first infectious phase, often called biotrophic, is prolonged and occurs asymptomatically, while the second, known as necrotrophic, is characterized by the appearance and development of symptoms (Rudd et al., 2015).

The cellular aspects of the pathogenesis of *Z. tritici* in wheat varieties have been cytologically and histologically studied. The infection cycle of *Z. tritici* can be divided into three main stages: entry of fungal hyphae, colonization of the plant tissue, and the formation of fruiting bodies (Steinberg, 2015). After conidia germination and fungal entry through the stomata, the hyphae grow very slowly in the intercellular space between mesophyll cells (Kema et al., 1996). It is typical that the host's protective reaction is either absent or very weak (Rudd et al., 2015). The latent period passes without symptoms and has an unusually long duration, ranging from 7 to 28 days, depending on the specific combination between wheat genotype and fungal isolate (Lee et al., 2014). The prolonged asymptomatic phase is followed by rapid induction of cell death and loss of membrane permeability. The necrotrophic feeding phase is necessary for the fungal asexual reproduction – the formation of pycnidia (Kema et al., 1996; Dean et al., 2012). During the transition between the biotrophic and necrotrophic phases of the disease, the host's defense responses are strongly activated, resulting in a significant accumulation of fungal biomass (Rudd et al.,

2015). The activity of most cell wall degrading enzymes greatly increases during the necrotrophic phase (Brunner et al., 2013). Some differences in the response of susceptible, moderately susceptible, and resistant wheat genotypes to fungal invasion have been identified (Kema et al., 1996). In a compatible reaction, fungal hyphae colonize mesophyll intercellularly but are in close contact with cell walls. The initial and subsequent spread of the pathogen in tissues has a noticeable effect on the number, size, and shape of chloroplasts. They condense and, along with the nucleus, move toward the cell walls. After the asymptomatic latent period, rapid cellular collapse occurs, suggesting an active role of phytotoxic compounds. In moderately susceptible genotypes, in addition to changes in chloroplasts, starch granules are released, likely limiting further colonization. The incompatible response is characterized by a weaker spread of the pathogen in the host's tissues. Hyphae are observed occasionally between mesophyll cells, mainly near the substomatal cavities, with no visible effect on the cells (Kema et al., 1996). In recent years, researchers have focused on various aspects of compatible and incompatible interactions between the fungus and its host, including transcriptomic and metabolomic profiling (Brunner et al., 2013; Lee et al., 2014; Rudd et al., 2015; Steinberg, 2015; Palma-Guerrero et al., 2016; Orton et al., 2017).

Parastagonospora nodorum

Pa. nodorum is a necrotroph that affects leaves and spikes, causing the disease Stagonospora nodorum blotch (SNB), which holds significant economic importance, especially in regions with frequent rainfall. In Bulgaria, this disease is known as "glume spotting", as symptoms on spikes are most noticeable. Losses can reach up to 31% (Bhathal et al., 2003). Common hosts include common and durum wheat, triticale, and other cereal crops, as well as some wild grasses. The pathogen is frequently found in all the geographic regions where wheat is cultivated, including Europe, North America, and Australia (Solomon et al., 2006; Francki, 2013).

Unlike *Z. tritici*, the symptoms induced by *Pa. nodorum* on the leaves of susceptible wheat varieties develop rapidly, and the infection cycle can be completed within 7 days under favorable conditions (Solomon et al., 2006). Studies in recent years have shown that *Pa. nodorum* produces several selectively acting toxins (SnToxA, SnTox2, SnTox3, SnTox5, SnTox6) which interact with gene products of the corresponding dominant susceptibility genes in wheat (*Tsn1, Snn2, Snn3, Snn5, Snn6*) (Friesen et al., 2012; Tan et al., 2014; Gao et al., 2015; Ruud et al., 2017). These are regulatory (effector) proteins that serve as virulence factors and facilitate the fungus's growth in plant tissues (Friesen et al., 2009; Oliver et al., 2012). Each of these necrotrophic effectors induces programmed cell death in susceptible wheat genotypes, but the mechanisms through which they suppress the host's protective responses differ. The SnTox1 protein provides protection to *Pa. nodorum* from wheat chitinases induced as part of the defense response (Liu et al., 2017). SnTox3 and SnToxA interact with the wheat PR-1 proteins (Lu et al., 2014; Breen et al., 2016). Population analysis of *Pa. nodorum* shows that the SnTox5–*Snn5* interaction plays a crucial role in the development of SNB under field conditions. When the SnTox5–*Snn5* and SnToxA–*Tsn1* interactions occur together, the degree of infection significantly increases (Friesen et al., 2012).

Pa. nodorum appears with the lowest frequency (12%). A trend confirmed in many European countries indicates that *Pa. nodorum* was the predominant species until around 1970, and then it was replaced by *Z. tritici (= M. graminicola*) (Oliver et al., 2012). This displacement is due to a combination of factors, including fungicide use and cultivation of varieties sensitive to the pathogen (Arraiano et al., 2009).

Parastagonospora avenae* f. sp. *triticea

Pa. avenae f. sp. *triticea* causes a disease known in many countries as Stagonospora avenae blotch (SAB) (Duveiller et al., 2012). The pathogen attacks a wide range of hosts, including wheat, triticale, barley, rye, and some cereal grasses (Cunfer, 2000; Kiehr & Delhey, 2007).

The asexual morph of fungus *Pa. avenae* f. sp. *triticea* has long been known as *Septoria*

avenae f. sp. *triticea*. In 1994, a decision was made to assign it to the genus *Stagonospora*, and more recently, a new genus, *Parastagonospora*, has been introduced for some species found on cereal hosts, including *Pa. avenae* and *Pa. nodorum* (Quaedvlieg et al., 2013). During the studied period from 2010 to 2017, *Pa. avenae* f. sp. *triticea* was the most important pathogen in the *Septoria/ Stagonospora* complex on durum wheat. In Bulgaria, it forms pycnidia with conidia and pseudothecia with ascospores. The sexual morph of this fungus was first reported on wheat in Canada (Johnson, 1947) and later in the northern states of the USA (Hosford et al., 1987; Shearer & Calpouzos, 1973; Luz & Bergstrom, 1985), Brazil (Luz, 1982), Argentina (Kiehr & Delhey, 2007), and Europe (Mäkelä, 1975). In Bulgaria, both the asexual and sexual morphs of the pathogen have been reported by Rodeva (1989). The fungus has been isolated and characterized (Rodeva, 1989).

RESULTS AND DISCUSSIONS

Resistance of wheat to Septoria diseases

Complete resistance of wheat to *Z. tritici*, *Pa. nodorum*, and *Pa. avenae* f. sp. *triticea* has not been established, but varieties differ significantly in their response to each of these pathogens (necrosis and/or pycnidia). This variation can be utilized in resistance breeding. There are numerous reports in the literature on wheat resistance to *Z. tritici* (Rosielle, 1980; Krupinsky et al., 1984; Ruzgas et al., 2002; Adhikari et al., 2003; 2004a; 2004b; Chartrain et al., 2004; Simón, 2010; Francki, 2013; Arraiano et al., 2017) and to *Pa. nodorum* (Rosielle & Brown, 1980; Rufty et al., 1981; Ruzgas et al., 2002; Abeysekara et al., 2009; Friesen et al., 2009; Friesen & Farris, 2010; Phan et al., 2018). Sources of resistance to both pathogens have been identified among species of the genera *Aegilops* and *Agropyron* and, in some cases, also successfully transferred to the wheat genome (Murphy et al., 2000; Loughman et al., 2001).

Over 20 major STB resistance genes (Stb) have been mapped (Brown et al., 2015; McCartney et al., 2002; Brown et al., 2015). Sources of quantitative resistance, which is more durable under field conditions and often provides protection against different pathogen genotypes, have also been identified. A total of 167 genomic regions containing loci (QTLs) related to STB resistance have been reported (Brown et al., 2015). Phenotyping these loci demonstrates their involvement in various stages of the disease development – sporulation, necrosis, and latent period. Laboratory methods have been developed for testing resistance at an early age (Arraiano et al., 2017). According to Chartrain et al. (2004), pyramidization of several resistance genes is an effective long-term strategy for selection for STB resistance. Achieving satisfactory long-term resistance requires the use of genetically diverse material.

Progress in improving the STB resistance has been made through crossbreeding of lines from various European breeding programs (Brown et al., 2015).

In some cultivar-isolate combinations, the reaction of young wheat plants to *Z. tritici* can predict the response of adult plants (Eyal & Prescott, 1983). In most cases, the correlation between resistance in young and adult plants to *Pa. nodorum* is low and insufficient for use in resistance breeding (Arseniuk et al., 1991). Additionally, leaf and spike resistance are independent traits (Wicki et al., 1999). The formation of necroses and pycnidia by *Z. tritici* is controlled by different genes (Eyal & Prescott, 1983). Under field conditions, the resistance assessment is influenced by plant height and early maturity. These two traits are negatively correlated with resistance to *Z. tritici* (Van Beuningen & Kohli, 1990) and *Pa. nodorum* (Scott et al., 1982). The genotype of the host plays an important role in fighting SNB. Numerous loci (QTLs) controlling both qualitative and quantitative SNB resistance have been reported (Francki, 2013).

Differences in the species composition and frequency of occurrence of fungi included in the SLB complex, where *Z. tritici* is the predominant species, have been observed. This is one of the economically most important fungal pathogens on wheat in Europe (Eyal, 1999; Kema et al., 2008). Infections by *Z. tritici* in many European countries start from airborne ascospores of the sexual morph of the fungus and droplet infection by conidia formed

on residues from the previous growing season. In some countries (the Netherlands), the pathogen is able to complete several sexual cycles in one growing season (Kema et al., 1996), and the ascospores are an important source of primary inoculum. The sexual morph has not been found in Bulgaria so far. High humidity is required for all stages of SLB infection: spore germination, penetration into host tissues, mycelial development, and conidia formation (Shaw, 1990). In a previous study on foliar pathogens of common wheat, *Z. tritici* was the predominant pathogen (70%), followed by *Pa. avenae* f. sp. *triticea* (18%)

In Bulgaria, the response of common and durum wheat varieties and lines to *Z. tritici*, *Pa. nodorum*, and *Pa. avenae* f.sp. *triticea* has been investigated, and sources of resistance have been identified (Rodeva, 1989).

Yellow leaf spots on wheat - the tan spot (TS) disease caused by *Pyrenophora tritici-repentis (Ptr)* has been identified in most countries worldwide where wheat is grown, including Europe, North and South America, Asia, Africa, and Australia (Moreno & Perelló, 2010; Ali et al., 2010; Bankina & Priekule, 2011; Ciuffetti et al., 2010). It is typical for drier regions, as it thrives better under such conditions rather than other foliar diseases (Gilbert et al., 1998).

Its increased economic importance in affected areas is due to certain agrotechnical practices, mainly minimal soil tillage (Bockus & Claasen, 1992). The primary inoculum source is ascospores formed on overwintered residues of wheat straw (Bankina & Priekule, 2011). Pathogen transmission through seeds has also been proven (Schilder & Bergstrom, 1995).

Symptoms consist of formation of oval to elongated brown leaf spots surrounded by a chlorotic halo. The pathogenicity of *Ptr* is largely attributed to three necrotrophic effectors: ToxA, ToxB, and ToxC. The products of each of these genes interact by a reverse scheme in a gene-for-gene manner with sensitivity genes *Tsn1*, *Tsc2*, and *Tsc1* of the host (Ciuffetti et al., 2010; Liu et al., 2017). It is assumed that ToxA has been horizontally transferred from *Pa. nodorum* to *Ptr*, and the acquisition of this gene leads to increased pathogenicity of *Ptr* (Friesen et al., 2009). ToxB controls the secretion of a protein that induces chlorotic reaction in the presence of the sensitivity locus *Tsc2* (Abeysekara et al., 2010). Homologs of ToxB have also been found in other pathogens of genera *Bipolaris*, *Alternaria*, and *Pyrenophora* (Ciuffetti et al., 2010). The interaction of ToxC with the *Tsc1* gene in wheat manifests as a chlorotic phenotype (Effertz et al., 2002).

Based on the presence of ToxA, ToxB, and ToxC or a combination of them, 8 races of *Ptr* have been identified, differing in their ability to induce necroses and/or chloroses on a set of varieties - differentiators, as well as in the production of specific toxins (Lamari et al., 2003). ToxA is present in races 1, 2, 7, and 8; ToxB – in 5, 6, 7, and 8; and ToxC - in 1, 3, 6, and 8. Recognizing the race structure is crucial for resistance breeding. Sources of resistance in *T. aestivum* (Friesen, Faris, 2009; Chu et al., 2008; Faris et al., 2013; Kokhmetova et al., 2017) and *T. durum* (Chu et al., 2010) have been identified. Significant attention is paid to the genetic studies of resistance (Friesen & Faris, 2010). It has been reported that additive gene action was predominent (Sharma et al., 2004). Testing durum wheat genotypes revealed that different resistance mechanisms operate at different plant organs, and the resistance observed in adult plants is not manifested in young ones. This suggests that the response of both young and adult plants should be studied. The best method to determine the response in young plants is to record the type of spots, whereas in adult plants - the length of the spots (Fernandez et al., 1994). Tests on young plants conducted in greenhouse conditions can be used to study the response of a large number of wheat accessions to *Ptr*, from which perspective lines can then be evaluated in field experiments (Evans et al., 1999). In Bulgaria, the tan spot disease was reported for the first time in 2005-2006. The reaction of different common wheat varieties has been studied under field conditions and artificial infection, as well as against different pathogen isolates in the second leaf stage under greenhouse conditions.

Ptr sporulates daily, and the conidia spread through wind. A large number of conidia appear in the afternoon hours following prolonged humid periods (Francl, 1997). The spores of Septoria diseases are dispersed by

raindrops, and the infection moves from the lower to the upper part of the crop. *Z. tritici* has a latent period of 3-4 weeks (Shaw, 1990), whereas *Ptr* has a much shorter latent period of 5-8 days (Riaz et al., 1991), indicating greater competitiveness of *Ptr*.

The primary inoculum of *Ptr* consists of ascospores formed in pseudothecia on overwintered wheat straw. Ascospore release begins in spring but can continue throughout the entire growing season, contributing to new infections (Bankina & Priekule, 2011). Pseudothecia of *Ptr* form in large quantities under the climatic conditions of Bulgaria (Todorova, 2005). Other sources of inoculum, mainly in the form of conidia, include infected seeds, volunteer plants, and other cereal grasses (Schilder & Bergstrom, 1995). During periods of precipitation and high air humidity throughout the growing season, multiple cycles of conidia formation and release occur, leading to rapid propagation of the pathogen (Ronis & Semaškienė, 2006). It has been observed that during prolonged humid periods and optimal temperatures following inoculation, conidia germination, number of mycelial sprouts from one conidium, sprout length, and appressoria formation increase (Hosford et al., 1987).

The *Ptr* population has a complex racial structure, and at least 8 races have been described, denoted from race 1 to race 8 (Lamari et al., 2003; Ciuffetti et al., 2010). This fungus produces toxins with specific action (host-selective toxins - HSTs), which are crucial for the pathogen's compatibility with its host. Five HSTs have been identified: Ptr ToxA, B, and C, and two more grouped together as Ptr ToxD. The first three are well-characterized, and their role as pathogenic factors has been demonstrated (Singh et al., 2010; Faris et al., 2013; Virdi et al., 2016; Kariyawasam et al., 2016). Ptr ToxA induces necrotic symptoms, whereas the other two toxins - Ptr ToxB and Ptr ToxC cause chloroses, but on different varieties and lines of the host (Strelkov et al., 2002; Effertz et al., 2002; Ciuffetti et al., 2010). Currently, racial differences are explained by the formation of these three HSTs, i.e., each race differs in the expression of one or a combination of these toxins (Ciuffetti et al., 2010). The interaction of virulent *Ptr* races with host genotypes is highly specific. The formation of necroses or chloroses after infection with *Ptr* is controlled by independent genetic factors (Lamari & Bernier, 1991). Inoculation with individual pathogen isolates leads to differentiated development of the two types of symptoms (Lamari & Bernier, 1989).

It is caused by the fungal pathogen *Cochliobolus sativus*. The infection occurs through conidia, as the sexual form is extremely rare in natural conditions. *C. sativus* is a hemibiotroph. The biotrophic phase is short and involves the formation of appressoria, which facilitate the direct penetration of infectious hyphae through the cuticle (Kumar et al., 2001). During the necrotrophic phase, fungal invasion into mesophyll tissue occurs, leading to cell death in the affected plant parts. Pathogenicity is associated with the production of toxins (Bach & Kimati, 1999). It attacks a wide range of hosts, but mainly wheat and barley (Kumar et al., 2002). It causes several different diseases, with the most important being spot blotch and common root rot. On leaves, it manifests as brown necrotic spots. Significant losses in wheat, up to 50%, have been reported in countries with hot and humid climates in Africa (Kenya, Sudan, South Africa, Tanzania), South Asia (India, Indonesia, Thailand, Bangladesh, Nepal), South America (Argentina, Brazil), North America (Indiana, Kansas, Minnesota, Montana, North and South Dakota), Australia, and New Zealand (Kumar et al., 2002; Acharya et al., 2011). It is also found in Europe (Austria, Belgium, Germany, Italy). The optimal temperature for infection and disease development is 28°C.

After sequencing three isolates of *C. sativus* of Australian origin (McDonald et al. 2015) found that one of them contains a gene almost identical to ToxA, described in *Pa. nodorum* and *Ptr*. Further analysis reveals that ToxA is present in 30% of the Australian isolates. If this gene is prevalent in the population of *C. sativus*, in resistance breeding, the susceptible gene *Tsn1* should be eliminated from wheat varieties in the affected regions (Figueroa et al., 2018).

Sources of resistance have been identified (Kumar et al., 2010). Resistance to spot blotch is a quantitative trait controlled by the additive

effect of more than two genes (Joshi et al., 2004). Mapping of loci for resistance confirms the involvement of multiple genes in controlling this trait (Singh et al., 2016).

The fungal pathogen *Monographella nivalis* (previously often reported as *Fusarium nivale*) causes snow mold, a disease where leaves, and sometimes the crown node, can be destroyed under a snow cover. It also induces the formation of spots, which sometimes appear on the upper leaves of wheat, barley, and especially triticale (Rodeva & Mihova, 1989). Symptoms are observed on internodes and stem nodes as well. The infection weakens the stem, leading to lodging or bending at affected nodes (Jenkins et al., 1988). In its further development, the disease can reach the spike and affect the grain, resulting in seedborne infection in the next vegetation. The spread from lower to upper layers occurs through conidia via droplet infection or with airborne ascospores formed in pseudothecia, which appear predominantly in leaf sheaths.

The general term "alternariosis" is associated with symptoms of leaf blotch of wheat caused by several species of *Alternaria* (Perelló & Sisterna, 2006; Perelló, 2010). By morphological traits, species of this genus are divided into three groups: *A. infectoria*, *A. arborescens*, and *A. tenuissima*. Molecular studies show that species pathogenic to wheat, such as *A. infectoria*, *A. triticimaculans*, and *A. triticina*, genetically belong to the *infectoria* group, which consists of more than 30 species (Andersen et al., 2009). This is the only group within the genus *Alternaria* in which some species have a sexual morph, related to the genus *Lewia* (Perelló & Sisterna, 2008). An important morphological characteristic is the formation of small conidia (up to 70 µm in length) in branched chains with long, knee-like secondary conidiophores (up to 120 µm) between them. Species of this genus are the most well-known producers of toxic secondary metabolites (over 70 compounds with varying toxicity) (Tralamazza et al., 2018). Chemical analysis shows that the metabolic profile of the *infectoria* group is very different from the other two, producing few common metabolites with them (Andersen et al., 2002). New compounds have been isolated, which are specific only to fungi from this group and can be used as chemotaxonomic markers (Christensen et al., 2005).

The most frequently isolated among leaf spot species on wheat is *A. triticina* (Perelló & Sisterna, 2006), and from the grain - *A. alternata* and *A. triticina* (Logrieco et al., 1990). *A. triticina* was first described as a new species in India and later in Argentina (Perelló & Sisterna, 2006). Initially, the leaf spots are small, oval, but with the development of the disease, they expand and take an irregular shape, often with a chlorotic halo. Common and durum wheat are the main hosts, with the latter being more susceptible (Perelló, 1998). *A. triticina* is a quarantine species in many countries.

The species *A. infectoria* was first reported by Simmons (1986), and its sexual morph (*Lewia infectoria*) - by Perelló and Sisterna (2008). The presence of the sexual morph is important both for the long-distance spread of *A. infectoria* and for resistance breeding (Perelló & Sisterna, 2008).

A. triticimaculans was identified as a new pathogenic species on common wheat in Argentina (Perelló et al., 1998). At first, small individual chlorotic spots (1.5 mm in diameter) appear. Later, they turn greenish-brown, have an elliptical or oval shape, and are scattered or coalescent. Sometimes they have a yellow halo. With disease progression, the entire affected leaf dies. A scale for recording leaf spots caused by this pathogen has been developed (Perelló et al., 1998). So far, the fungus has not been reported in other parts of the world or on other hosts.

Resistance breeding

A comparative study of common and durum wheat for their response to leaf pathogens reveals significant variation among varieties but not within species groups. Among both wheat species, there are varieties with both low and high levels of attack from the leaf spotting complex. *Ptr* is isolated more frequently from durum wheat than from common wheat, while *Pa. nodorum* is much more common in common wheat (Fernandez et al., 1994). Wheat genotypes with complex resistance to STB, SNB, and *Ptr* have been identified (Šíp et al., 2005; Ali et al., 2008).

Selective breeding for combined disease resistance is a promising strategy in creating new varieties. The goal of modern resistance breeding is satisfactory resistance to all major diseases, not just high resistance to one disease. An important point is the elimination of highly susceptible lines, which, besides being heavily attacked, also provide inoculum for other varieties. In practice, resistance breeding is based on field selection and depends on the natural occurrence of the tested diseases. Pot tests in greenhouse conditions can expedite breeding, but their value is limited only to resistance manifested in both young and adult plants. Field trials remain the most useful breeding method, especially when natural infection is enhanced by artificially inoculated plants or using varieties that spread the infection.

CONCLUSIONS

The results obtained in this study reveal the biodiversity of fungal species involved in the leaf spotting complex of wheat in Bulgaria. Information about the most commonly encountered pathogens and their specific characteristics will serve as a scientific basis for disease control and resistance breeding. In addition to its fundamental importance, the results also have practical application. Accurate identification is important for several reasons. It is necessary when deciding on control methods, as several diseases may manifest similarly, and precise identification is essential. It is not always possible to distinguish fungal leaf diseases based solely on sight examination of the size and shape of the leaf spots. For precise diagnosis, it is necessary to observe the reproductive structures of the pathogens and isolations. Knowledge of important diseases in a given production area, their identification, modes of propagation, and response to environmental conditions is crucial when deciding on a profitable and ecologically sustainable wheat production. Since different diseases require suitable control strategies, their accurate diagnosis is of paramount importance. Information about the most commonly encountered fungi causing leaf spotting will alert regional breeders and phytopathologists to increase their efforts in combating the leaf spotting complex to avoid future epiphytotic diseases.

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