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Net blotch (*Pyrenophora teres* Drechsler): An increasingly significant threat to barley production

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Abstract: *Pyrenophora teres* is a pathogen causing a net blotch disease in cultivated barley, which is present worldwide and can thus significantly reduce barley yields. This fungus also infects wild barley and other plants of the *Hordeum* genus, as well as barley grass, wheat, oats and plants from various genera, including *Agropyron*, *Bromus*, *Elymus*, *Hordelymus* and *Stipa*. Based on the symptoms it causes on the infected barley plants, the pathogen can be divided into two forms: *P. teres* f. *teres,* which causes net-like symptoms, and *P. teres* f. *maculata,* which causes blotchy symptoms. Infected seeds, stubble and plant debris, and volunteer and weed plants represent primary sources of pathogen inoculum. During the growing season, the pathogen enters a sexual stage, developing pseudothecia with asci and ascospores. This is followed by an asexual stage, during which conidiophores with conidia are formed. The conidial (anamorphic) stage is much more common, whereby conidia is a source of inoculum for secondary infection during the barley growing season. The first symptoms appear at the end of winter and the beginning of spring, often during the tilling phase. The most characteristic symptoms form on barley leaves. Frequently, symptoms of the net form can be mistaken for other diseases occurring on barley, making molecular analysis essential for accurate detection of *P. teres*, its forms, mating types and hybrids. Current net blotch control measures are based on the combined application of cultural, chemical and biological control methods and the selection of resistant varieties.

Keywords: *Pyrenophora teres*; net blotch of barley; taxonomy; disease cycle; toxin production; control

Barley (*Hordeum vulgare* L.) is a plant species cultivated across all continents that holds great significance in the beer industry, spirits production, livestock feed production and human nutrition (McLean et al. 2010; Verstegen et al. 2014). However, barley is susceptible to numerous plant pathogens, the most significant being the foliar pathogen *Pyrenophora teres* Drechsler [anamorph

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Drechslera teres (Sacc.) Shoemaker] causing net blotch (Backes et al. 2021). Within the *P. teres* species, the *teres* and *maculata* forms are particularly widespread (Smedegård-Petersen 1971).

Significant economic losses under favourable conditions for net blotch emergence are inevitable, as extensive research on this topic indicates. For example, Jordan et al. (1985) found that, under laboratory conditions, artificial pathogen inoculation reduces root size, dry matter percentage and healthy leaf size with minimal impact on yield. However, repeated inoculations resulted in up to 48% yield reduction, while also adversely affecting the number of spikes, kernels per spike and straw volume. According to Jordan (1981), in England, yield losses caused by the net form of the pathogen can reach 22%, while Plessl et al. (2005) found that yields in Germany can be reduced by up to 40% in years with extreme rainfall levels. Grain yield and absolute grain weight were reduced by 9–11.1% and 7.8–8.7%, respectively, in investigations conducted in Denmark (Smedegård-Petersen 1974). In Kenya (Were et al. 2016) and Algeria (Lhadj et al. 2022), barley grain yield under conditions conducive to infection can be reduced by as much as 15–40% in the absence of proper protection. Other authors have reported similar figures, ranging from 10% to 44% (Khan 1989; Jayasena et al. 2002, 2007; Ma et al. 2004; Afanasenko et al. 2015; Mair et al. 2016; Rozanova et al. 2019; McLean & Hollaway 2019; Taibi et al. 2016; Abebe 2021). On the other hand, Murray and Brennan (2010) estimated that, over a ten-year period, the average yields in different barley-producing regions of Australia could potentially be reduced by 4.5–9.9% due to net blotch infection, resulting in the projected losses of 192 USD × 10^6 for the spot and 117 USD × 10^6 for the net form of the pathogen. According to Martin et al. (2021), yield losses in favourable conditions for an epidemic on the sensitive Maritime variety in South Australia can reach up to 70%, while other authors are of the view that, in areas where very sensitive barley varieties are grown, and the crop is heavily affected by the disease, 100% yield losses can occur (Oğuz et al. 2019; Abebe 2021). Yield losses are often accompanied by significant reductions in 1 000 grain weight, number of spikes per m^2, number of kernels per spike, grain size and malt quality (Sutton & Steele 1983; Khan 1987; Murray & Brennan 2010; McLean & Hollaway 2019; Abebe 2021; Oğuz & Karakaya 2021; Tini et al. 2022).

These findings have motivated this overview, which will cover the most important characteristics of the pathogen *P. teres*, including its geographic distribution, host range, nomenclature and pathogen systematics, symptoms, developmental cycle and epidemiology, morphological traits, molecular markers for detection, as well as the range of crucial disease control measures against this pathogen.

TAXONOMY

The genus *Helminthosporium* was first established in 1809 (Alcorn 1988), initially as *Helmisporium* but was later corrected to *Helminthosporium* (Manamgoda et al. 2014). It is divided into two subgenera based on the mode and form of conidial germination: *Eu-* or *Fuso-Helminthosporium* (species with fusoid conidia, often curved, germinating at either end) and *Cylindro-Helminthosporium* (species with cylindrical conidia germinating from any cell) (Ito & Kuribayashi 1931; Shoemaker 1959; Alcorn 1988). As the name *Cylindro-Helminthosporium* was deemed too long, a proposal was made to replace it with *Drechslera* in honour of Dr. C. Drechsler, who significantly contributed to the knowledge of the genus *Helminthosporium* (Ito 1930; Ito & Kuribayashi 1931). The new subgenus *Drechslera* would include members associated with the teleomorph *Pyrenophora,* characterized by cylindrical, non-curved conidia germinating from every cell (Ito 1930; Alcorn 1988). Likewise, a genus *Bipolaris* was proposed to replace the earlier subgenus *Eu-* or *Fuso-Helminthosporium* for species with bipolar conidial germination (Shoemaker 1959). Further, a new genus *Exerohiulum* was proposed for species with distinctly protruded hilum, previously included in the genus *Bipolaris* (Leonard & Suggs 1974). Today, the genus *Helminthosporium* is divided into the anamorphic genera *Drechslera*, *Bipolaris* and *Exerohilum*, which correspond respectively to their teleomorphs *Pyrenophora*, *Cochliobolus* and *Setosphaeria* (Shoemaker 1959, 1962; Sivanesan 1987; Alcorn 1988; Zhang & Berbee 2001; O'Brien 2005). The genus *Pyrenophora* was initially placed in the family *Pleosporaceae*, but after nearly three decades, it was moved to the family *Pyrenophoraceae* along with *Cochliobolus* and *Setosphaeria* (Ariyawansa et al. 2014). Some scientists disagreed with this change, arguing that

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the original classification was correct, which was later confirmed by molecular analyses, placing the genus *Pyrenophora* in the family *Pleosporaceae* (Berbee 1996; Zhang et al. 2012; Ariyawansa et al. 2014; Marin-Felix et al. 2019), which belongs to the suborder *Pleosporineae* within the order *Pleosporales* (Zhang et al. 2012).

Pathogen *P. teres* was first described by Saccardo in 1886 under the name *Helminthosporium teres*. In 1891, the fungus was redescribed by Eidam under a different name, *H. hordei* (Index Fungorum 2023), but subsequent confirmations established that the pathogen is indeed *P. teres*. Initially, as stated, the fungus was classified in the genus *Helminthosporium*, but it was later moved to the genus *Drechslera* (formerly *Cylindro-Helminthosporium*) due to the absence of curved conidia and the ability to germinate from any cell (Shoemaker 1959, 1962; Alcorn 1988; Kinzer 2015). The teleomorphic stage of the fungus was first described by Drechsler in 1923 and was given the name *Pyrenophora teres* (Webster 1951). It was subsequently determined by Smedegård-Petersen (1971) that this is a form within the species *P. teres*, which is now divided into f. *teres* and f. *maculata*. The species *P. teres* was initially placed in the genus *Pleospora* and was subsequently reclassified into the genus *Pyrenophora* based on the presence of seta in the perithecium (Ito & Kuribayashi 1931), a classification that still stands today.

The genus *Pyrenophora* belongs to the following systematic classification: *Dothideomycetes*, *Pleosporomycetidae*, *Pleosporales* and *Pleosporaceae* (Marin-Felix et al. 2019) and has 212 currently recognized members, according to Index Fungorum (2023).

The current taxonomic position of *P. teres* is as follows: Kingdom: *Fungi*, Phylum: *Ascomycota*, Subphylum: *Pezizomycotina*, Class: *Dothideomycetes*, Order: *Pleosporales*, Family: *Pleosporaceae*, Genus: *Pyrenophora*, Species: *teres* (Liu et al. 2011; Kinzer 2015; Backes et al. 2021). This classification is also supported by Marin-Felix et al. (2019), who additionally mention the sub-class *Pleosporomycetidae* to which the genus *Pyrenophora* belongs.

GEOGRAPHICAL DISTRIBUTION

Net blotch is widely distributed and is one of the economically most significant diseases in all regions of the world where barley (*H. vulgare*) is commercially cultivated (Williams et al. 2001; Frisen et al. 2006; Tuohy et al. 2006; Liu et al. 2012; Adawy et al. 2013; Novakazi et al. 2019; Backes et al. 2021). The disease is present in many European countries, including Serbia (Tančić-Živanov et al. 2017) and other countries that previously formed Yugoslavia such as Bosnia and Herzegovina (Stojčić & Trkulja 2001; Tomić unpublished data), Russia (Afanasenko et al. 2007; Mikhailova et al. 2010; Volkova et al. 2020), Italy (Aragona et al. 2000; Rau et al. 2003), France (Arabi et al. 1992; Youcef-Benkada et al. 1994; Toubia-Rahme et al. 1995), Hungary (Tóth et al. 2008), Poland (Baturo-Ciesniewska et al. 2012), Finland (Peltonen et al. 1996; Robinson & Jalli 1996; Serenius et al. 2005; Tuohy et al. 2006), Slovakia (Tuohy et al. 2006), Ukraine (Retman et al. 2022), Lithuania (Statkeviciute et al. 2010, 2012), Estonia (Sooväli & Koppel 2010), Norway (Tuohy et al. 2006; Wonneberger et al. 2017), Sweden (Jonsson et al. 1997; Tuohy et al. 2006), Denmark (Smedegård-Petersen 1971; Tuohy et al. 2006), Germany (Vatter et al. 2017), Czech Republic (Minarikova & Polisenska 1999; Tuohy et al. 2006), Bulgaria (Vasileva et al. 2022), Great Britain (Abebe 2021), Turkey (Oğuz et al. 2019), Austria, Baltic States, Cyprus, Faroe Islands, Greece, Ireland, Malta, Moldova, Netherlands, and Romania. In the USA, its presence was recorded in North Dakota and Montana (Liu et al. 2012; Lartey et al. 2013), California and Minnesota (Steffenson & Webster 1992), and Pennsylvania (Delserone & Cole 1987), while in Canada, net blotch was found in Alberta, Saskatchewan and Manitoba (Tekauz & Mills 1974; Tekauz 1990; Van den Berg & Rossnagel 1990; Akhavan et al. 2015, 2016). It was also reported in Mexico (Romero-Cortes et al. 2021), Argentina, Brazil, Uruguay, Colombia and Peru (Garozi et al. 2020; Uranga et al. 2020; Abebe 2021; Gamba et al. 2021), as well as in Asian countries such as Iran (Dokhanchi et al. 2021; Vasighzadeh et al. 2022), Israel (Ronen et al. 2019), Syria (Bouajila et al. 2011), Uzbekistan (Abebe 2021), Afghanistan, Armenia, China, India, Iraq, Japan, Korea, Kyrgyzstan, Myanmar, Nepal, Pakistan, and Turkmenistan. It is also widespread across Africa, with reports for Tunisia, Ethiopia, Kenya, Algeria, Morocco, Egypt, Libya, South Africa, Saint Helena, Tanzania, and Zambia (Scott 1992; Louw et al. 1996; Douiyssi et al. 1998; El Yousfi & Ezzahiri 2001, 2002; Campbell et al. 2002; Jebbouj & El Yousfi 2010; Bouajila et al. 2011; Owino et al. 2013; Taibi et al. 2016; Were et al. 2016; Lammari et al. 2020a; Abebe 2021; Mohammed et al. 2021; Lhadj et al. 2022). Likewise, net

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blotch has been identified in several studies conducted in Australia (Wallwork et al. 1992, 2016; Williams et al. 2001; Jayasena et al. 2007; Murray & Brennan 2010; Fowler et al. 2017; El-Mor et al. 2018; Ellwood et al. 2019; McLean & Hollaway 2019) and New Zealand (Hampton 1980; Cromey & Parkes 2003).

SYMPTOMS

As its name, "net blotch", suggests, the disease leads to symptoms resembling a network pattern, mainly on the leaves (Figure 1) but also on other parts of barley, such as leaf sheaths and stems, as well as on flowers and grains (McLean et al. 2009; Liu et al. 2011; Fowler et al. 2017; Backes et al. 2021). The initial symptoms are primarily observed on the leaves of young plants shortly after the winter dormancy period and even during the tilling phase (author's observation). Indeed, leaf symptoms aid in visualising the pathogen forms, which were previously not recognized but later identified and described in detail (Smedegård-Petersen 1971).

The symptoms of the net form of the pathogen were first observed in 1920 (Atanasoff & Johnson 1920; Liu et al. 2011; Poudel 2018). On the other hand, the symptoms of the spot form were first noticed in 1963 during the study of net blotch but were considered mutants of the *P. teres* species (McDonald 1963, 1967; El-Mor 2016; Poudel 2018).

Smedegård-Petersen (1971) provided a detailed description of net blotch symptoms as well as identified that the same pathogen causes two distinct types of symptoms on barley leaves. Based on these symptoms, the author classified the pathogens into two forms: *P. teres* f. *teres* (or the net form), which causes net-like symptoms (Figure 1A and 1B), and *P. teres* f. *maculata* (or the spot form), which causes symptoms in the form of dark spots (Figure 1C and 1D). Lightfoot and Able (2010) similarly noted that the different pathogen forms have distinct pathophysi-

Figure 1. *Pyrenophora teres*. Symptoms of the net form on the barley leaves (A, B). Tipical symptoms of the spot form caused by *P. teres* f. *maculata* (C, D) (photo A. Tomić)

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Table 1. *Pyrenophora teres*: list of host from Poaceae family (Shipton et al. 1973; Brown et al. 1993; Toth et al. 2008; Mikhailova et al. 2010; Ficsor et al. 2010, 2014; El-More 2016; Poudel 2018; Ronen et al. 2019; Agostinetto et al. 2020; Garozi et al. 2020; Uranga et al. 2020)

No.	Latin name	No.	Latin name	No.	Latin name
1	*Aegilops cylindrica* Host	33	*Bromus sterilis* L.	64	*Hordeum murinum* subsp. *leporinum* (Link) Arcang
2	*Aegilops juvenalis* (Thell.) Eig	34	*Bromus unioloides* Kunth	65	*Hordeum muticum* Presl
3	*Aegilops ovata* L.	35	*Cynodon dactylon* (L.) Pers.	66	*Hordeum parodii* Covas
4	*Aegilops searsii* Feldman & Kisler	36	*Decshampsia caespitosa* (L.) P. Beauv.	67	*Hordeum patagonicum* (Haum.) Covas
5	*Aegilops triuncialis* L.	37	*Elymys angustus* Trin.	68	*Hordeum patagonicum* subsp. *magellanicum* (Parodi & Nicora) Bothm et al.
6	*Aegilops ventricosa* Tausch	38	*Elymys canadensis* L.	69	*Hordeum patagonicum* subsp. *santacrucense* Parodi & Nicora) Bothm et al.
7	*Agropyron bonaepartis* (Spreng.) Dur & Schinz	39	*Elymys dahuricus* subsp. *excelsus* (Turcz. Ex. Griseb.) Tzvelev	70	*Hordeum patagonicum* subsp. *setifolium* Parodi & Nicora) Bothm et al.
8	*Agropyron ciliare* (Trin.) Franch.	40	*Elymys dahuricus* Turcz. Ex. Griseb.	71	*Hordeum roshevitzii* Bowd.
9	*Agropyron cristatum* (L.) Gaertn.	41	*Elymys giganteus* Vahl	72	*Hordeum stenostachys* Godr.
10	*Agropyron elongatum* (Host) P. Beauv.	42	*Elymys glaucus* Buckley	73	*Hordeum vulgare* L.
11	*Agropyron fibrosum* (Schrenk) Cand.	43	*Elymys mollis* Trin in Spreng.	74	*Hordeum vulgare* subsp. *spontaneum* (C. Koch) Thell.
12	*Agropyron intermedium* (Host) P. Beauv.	44	*Elymys sibiricus* L.	75	*Hordeum vulgare* var. *deficiens* Steud.
13	*Agropyron intermedium* var. *trichophorum* (Link) Halac.	45	*Elymys virginicus* L.	76	*Loilum multiflorum* Lam.
14	*Agropyron repens* (L.) P. Beauv.	46	*Festuca eliator* L.	77	*Phalaris arundinacea* L.
15	*Agropyron spicatum* (Pursh) Lams.-Scribn. & J. G. Sm.	47	*Festuca idahoensis* Elmer	78	*Secale cereale* L.
16	*Agropyron trachycaulum* (Link) Malte ex H. Lewis	48	*Festuca megalura* Nutt.	79	*Secale montanum* Guss.
17	*Agropyron violaceum* (Horn.) Lang	49	*H. murinum* ssp. *glaucum* (Steud.) Tzvelev	80	*Secale montanum* subsp. *antolicum* (Guss.) Tzvelev
18	*Avena fatua* L.	50	*H. vulgare* ssp. *spontaneum* (C. Koch) Thell.	81	*Secale montanum* subsp. *kuprijanovii* (Grossh.) Tzvelev
19	*Avena sativa* L.	51	*Hordelymus europaeus* (L.) Harz	82	*Secale vavilovii* Grossh.
20	*Brachypodium distachyon* (L.) P. Beauv.	52.	*Hordeum arizonicum* Covas	83	*Stipa pulchra* Hitchc.
21	*Brachypodium phoenicoides* (L.) Roem. & Schult.	53	*Hordeum bogdanii* Wil.	84	*Triticum aestivum* L.
22	*Brachypodium pinnatum* (L.) P. Beauv.	54	*Hordeum brachyantherum* Nevski	85	*Triticum aestivum* subsp. *sphaerococcum* L. em Thell.
23	*Brachypodium sylvaticum* (Huds.) P. Beauv.	55	*Hordeum brachyantherum* subsp. *californicum* (Covas & Stebb.) Bothm et al.	86	*Triticum militinae* Zhuk. & Migush.
24	*Bromus auleticus* Trin. ex Nees	56	*Hordeum brevisubulatum (*Trin.) Link	87	*Triticum monococcum* L.
25	*Bromus bromoideus* (Lej.)Crepin	57	*Hordeum brevisubulatum* subsp. *violaceum* (Boiss. & Hohen.) Tzvelev	88	*Triticum monococcum* subsp. *baeoticum* (Boiss.) C. Yen
26	*Bromus diandrus* Roth.	58	*Hordeum bulbosum* L.	89	*Triticum monococcum* subsp. *urartu* (Thum.) Löve
27	*Bromus erectus* Huds.	59	*Hordeum chilense* Roem. & Schult.	90	*Triticum turgidum* L.
28	*Bromus inermis* Leyss.	60	*Hordeum jubatum* L.	91	*Triticum turgidum* subsp. *turanicum* (Jacubz.) Löve
29	*Bromus inermis* subsp. *pumpellianus* (Scribn.) Wagnon	61	*Hordeum leporinum* Link	92	*Triticum turgidum* subsp. *dicoccoides* L. em Thell
30	*Bromus marginatus* Nees ex Steud.	62	*Hordeum marinum* Huds.	93	*Triticum turgidum* subsp. *dicoccum* L. em Thell.
31	*Bromus rubens* L.	63	*Hordeum marinum* subsp. *gussoneanum* (Parl.) Thell.	94	*Triticum turgidum* subsp. *durum* L. em Desf.
32	*Bromus sterilis* L.				

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ologies (resulting in different lesion symptoms). According to these authors, net-form symptoms initially appear as small spots or streaks, which later enlarge, elongate and form narrow, dark-brown, necrotic longitudinal bands resembling a net-like pattern. However, in highly resistant varieties, the development of the net-like pattern may not be observed (Liu et al. 2011; Tini et al. 2022; Vasileva et al. 2022). The affected leaf tissue becomes brown, while the surrounding tissue becomes bordered by a characteristic chlorotic zone (Shipton et al. 1973). Sometimes, initial lesions develop as large, greenish, watery areas that later become distinct dark-brown networks (net-like patterns). In some cases, the initial lesions of the dark-brown net-like form can be observed in tissue without any chlorosis. Still, later, a yellowish chlorotic halo appears around the lesions, which can spread to the entire leaf and cause wilting from the tip to the base of the leaf. Rehfus (2018) is of the view that elongated lesions with necrotic areas along the leaf veins and occasional transverse barriers are the characteristic symptoms of the net form of the disease.

The spot form, or the spotted symptoms, appears on leaf tips and sheaths. It can cause various types of dark-brown round to elliptical lesions measuring about 3 × 6 mm, surrounded by a chlorotic zone of varying width. This chlorotic zone can expand to cover the entire leaf, causing it to dry out (Smedegård-Petersen 1971; McLean et al. 2009; El-Mor 2016; Vasileva et al. 2022). Large dark-brown lesions may also exhibit slight necrosis, while small dark-brown lesions with wide chlorotic halos can merge, causing rapid leaf drying from the tip toward the base (Smedegård-Petersen 1971). Isolates of lower virulence result in the formation of smaller necrotic lesions or lesions without chlorotic borders (Liu et al. 2011). In resistant varieties, the spot form symptoms, as described by McLean et al. (2009), consist of small, dark-brown necrotic lesions that do not increase in size but may form a small chlorotic halo depending on the variety's resistance.

HOST PLANTS

The primary host of the causal agent of net blotch is cultivated barley (*H. vulgare*). Net blotch, in addition to infecting cultivated (commercial) barley, also affects its wild relatives (*H. vulgare* ssp. *spontaneum* and *H. murinum* ssp. *glaucum*), which are considered to be the main hosts of net blotch forms alongside cultivated barley (Shipton et al. 1973; Ficsor et al. 2010, 2014; Ronen et al. 2019; Agostinetto et al. 2020).

As shown in Table 1, the pathogen can also infect other plants such as barley grass (*Hordeum leporinum* Link), wheat (*Triticum aestivum* L.), oats (*Avena sativa* L.), as well as plants from various genera like *Agropyron*, *Bromus*, *Elymus*, *Hordelymus*, *Stipa* and other *Hordeum* species (Shipton et al. 1973; Brown et al. 1993; El-Mor 2016; Poudel 2018; Uranga et al. 2020). The presence of *P. teres* on wheat has been confirmed during the field surveys conducted by Toth et al. (2008), Mikhailova et al. (2010) and Garozi et al. (2020). Hosts of net blotch found in naturally infected fields include *Avena* sp., *Bromus diandrus* Roth, *Hordeum brachyantherum* Nevski, *H. distichon* L., *H. leporinum*, *H. hystrix* Roth, *H. marinum* Huds., *H. murinum* L., *H. spontaneum* K. Koch and *Triticum* sp. (Shipton et al. 1973). In California, 65 plant species serve as hosts for *P. teres* f. *teres* (Brown et al. 1993).

DISEASE CYCLE

P. teres is a hemibiotrophic pathogen that maintains a saprophytic lifestyle between two vegetation seasons (Suciu et al. 2021; Tini et al. 2022). It is transmitted through infected plant residues and persists in infected stubble, plant debris and barley seeds (Figure 2). This serves as one of the most important primary sources of inoculum for the upcoming vegetation, including volunteer plants and weed species (Hampton 1980; Jordan 1981; McLean et al. 2009; Liu et al. 2011; Kinzer 2015; Martin et al. 2021; Tini et al. 2022; Vasighzadeh et al. 2022).

The different forms of *P. teres* have almost identical developmental cycles (McLean et al. 2009; Poudel 2018) and are primarily transmitted through seeds, which can thus be a major source of pathogen introduction into new areas (Arnst et al. 1978; Martin 1985; Liu et al. 2011; Abebe 2021). However, there are conflicting reports on the transmissibility of seeds of the two fungal forms. According to Jordan (1981) and McLean et al. (2009), *P. teres* f. *teres* is transmitted through seeds, while this has not been established in natural conditions for *P. teres* f. *maculata*. On the other hand, experiments with artificial inoculation during different growth stages have shown that both pathogen forms can be trans-

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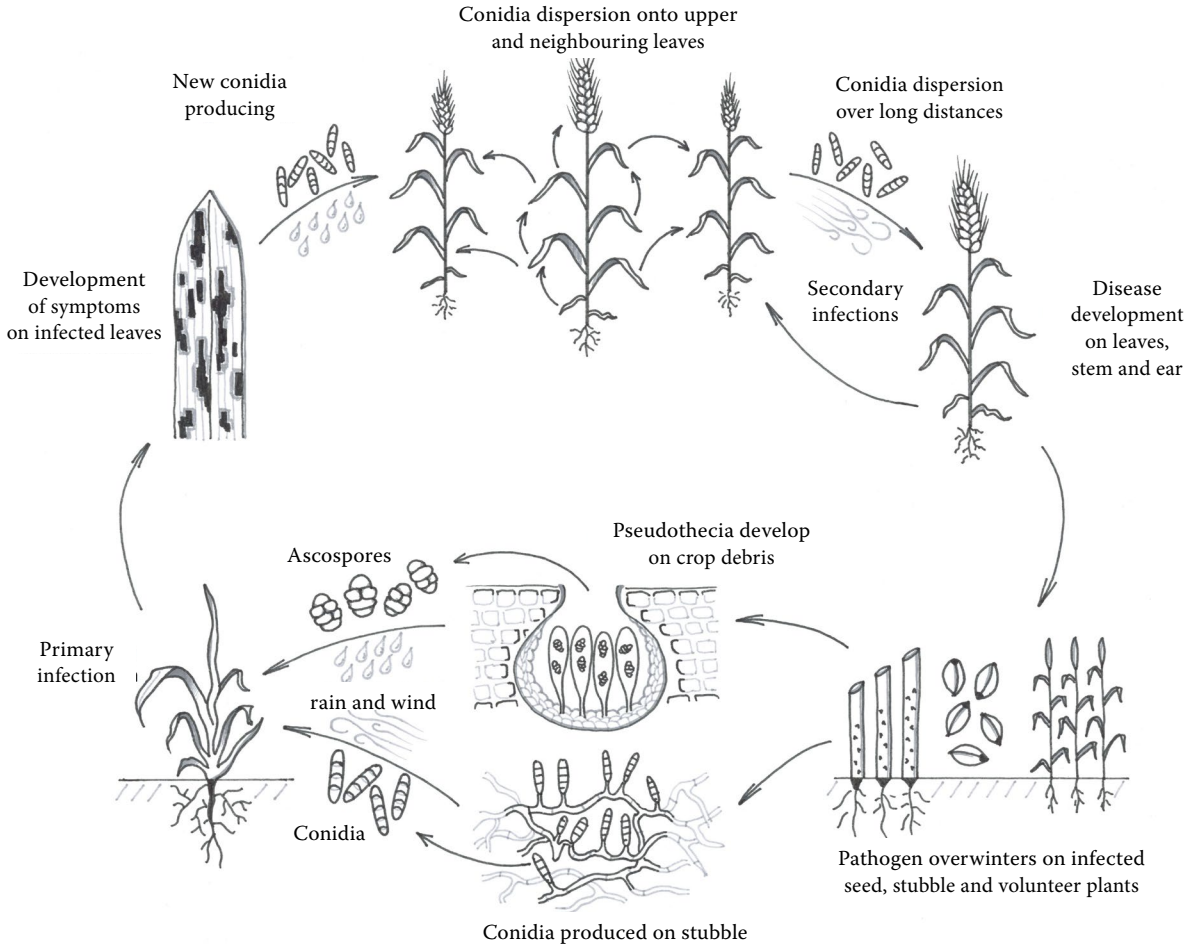

Figure 2. *Pyrenophora teres*. Disease cycle (original illustration by R. Iličić)

mitted through seeds (Youcef-Benkada et al. 1994). Contaminated seed parts where the pathogen is maintained include the pericarp, endosperm, embryo and caryopsis (Youcef-Benkada et al. 1994). Accordingly, seed transmission of the pathogen is a significant source of primary infection. During the growing season, the pathogen can cause several secondary infections, leading to severe damage in susceptible varieties, especially under favourable environmental conditions (Liu et al. 2011).

During its lifecycle, the pathogen forms both sexual and asexual stages (Figure 2). The sexual stage involves the formation of pseudothecia with asci and ascospores, while in the asexual stage, the pathogen forms conidiophores with conidia. Both stages can serve as primary inoculum sources for new infections (Suciu et al. 2021; Vasighzadeh et al. 2022). To produce a larger amount of primary inoculum, the pathogen requires cooler and more humid environmental conditions (McLean et al. 2009). The pathogen's mycelium can colonize stubble and lead to the formation of pseudothecia at the end of the growing season in the form of black dots, allowing the pathogen to survive in the field during unfavourable conditions and serving as a primary source of inoculum (McLean et al. 2009; Liu et al. 2011; Ronen et al. 2019). Pseudothecia, as a source of inoculum, can persist from one season to the next on infected stubble, where the formation and maturation of asci with 3–8 ascospores occur under cold temperatures and persistent soil moisture and are accompanied by conidiophores with conidia (Shipton et al. 1973; McLean et al. 2009).

The ascospores are released during late autumn, spring and summer and sometimes even toward the end of winter (Smedegård-Petersen 1972). Microscopic examinations indicate that ascospores can be released onto the surface of a water film, allowing them to be transmitted further by raindrops (rain splashes). It has also been observed that ascospores and conidia can be individually released into still air, while wind facilitates their dispersion over varying

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distances (Shipton et al. 1973; Jordan 1981). However, to initiate an infection, ascospores require high relative humidity (RH = 95–100%) (McLean et al. 2009).

Conidia are produced due to asexual reproduction and typically form on necrotic lesions previously developed on leaves (Shipton et al. 1973; Kinzer 2015). The asexual phase occurs on barley residues during summer and causes infections in young plants during autumn (Backes et al. 2021). Under humid conditions, perithecia (often immature) become densely covered with conidia found on short conidiophores at the tips of setae or on conidiophores emerging between setae (Smedegård-Petersen 1972). On necrotic leaves, conidiophores bearing conidia are formed individually or in groups (Smedegård-Petersen 1971; Arnst et al. 1978). Spores produced on infected stubble serve as primary inoculum, while those produced on lesions during the growing season serve as secondary inoculum (Poudel 2018). After initial colonization, the fungus produces a large number of conidia, which act as secondary inoculum (Liu et al. 2011). However, secondary inoculum can also result from primary infection by ascospores (McLean et al. 2009). After primary infection, conidia takes 14–20 days to develop and spread, leading to secondary infections and increasing the disease severity (McLean et al. 2009; Poudel 2018). Under windy and rainy conditions, conidia can spread across considerable distances from the infection site (Shipton et al. 1973; Deadman & Cooke 1989; Kinzer 2015; Vasighzadeh et al. 2022).

In addition to ascospores and conidia, the fungus also forms pycnidia, another spore type. Pycnidia typically develop on infected straw residues (stubble), dead leaves or their fragments and seeds, but they can also be formed in culture (Smedegård-Petersen 1972; Shipton et al. 1973; Jordan 1981). Artificial inoculation of barley leaves with pycnidiospores as a part of the studies conducted by Smedegård-Petersen (1972) was unsuccessful, and this finding was later corroborated by Jordan (1981). However, Smedegård-Petersen (1972) did establish that mature pycnidiospores are released at the top of the pycnidium in a slimy matrix that appears as a greyish-white drop. Although pycnidiospores can germinate and produce mycelium, their role in the fungus's lifecycle is still unknown (Liu et al. 2011).

EPIDEMIOLOGY

Plant residues are one of the most important sources of primary inoculum for the next growing season, significantly affecting disease severity under favourable environmental conditions. However, as infected seeds also represent an important means of pathogen spread, it is essential to note that temperatures in the 10–15 °C range are most conducive to disease development in young barley plants arising from mycelium-infected seeds (Abebe 2021).

The amount of inoculum (potential sporulation) that will develop on plant residues and lead to infection primarily depends on the environmental conditions. Van den Berg and Rossangel (1991) examined sampled plant residues. They found potential sporulation of 200–400 conidia per cm of stem and 700–900 conidia per leaf, whereby sporulation occurred on leaves that were at least 50% dry and were located only on the chlorotic and necrotic areas surrounding dark brown spots. When investigating the impact of environmental factors (temperature, relative humidity and leaf surface moisture) on the presence of conidia of the spot form in the air, Van den Berg and Rossangel (1991) determined that the number of airborne conidia decreased as the temperature, relative humidity and leaf surface moisture declined (Van den Berg & Rossangel 1991). The highest number of airborne conidia was observed during the later stages of vegetation, particularly during upper leaves' ageing (drying – senescence), exceeding 1 000 conidia per day.

Additionally, over 10 000 conidia were produced per leaf in the first two weeks after drying (Van den Berg & Rossangel 1991). In investigations of the quantity of *P. teres* spores released into the air as a potential source of infection, Deadman and Cook (1989) noted that the daily number of captured spores in Ireland was positively correlated with the rainfall intensity. Similarly, Jordan (1981) found that the highest number of conidia was captured on days with rainfall, during or shortly before the rain. In a subsequent study conducted by Jordan and Allen (1984), the number of released *P. teres* f. *teres* spores ranged up to 250 000 per m^2 of ground surface stubble. On the other hand, Martin and Clough (1984) reported that abundant spore release was preceded by continuous periods of leaf wetness (lasting more than 16 h) when relative humidity exceeded 85%. During periods of no leaf wetness, the extreme temperatures for spore release ranged from 15–32 °C. However, Van den Berg & Rossangel (1991) stated that conidial release can occur within a broader temperature range, i.e., 1–35 °C.

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Nonetheless, based on the available evidence, under longer-lasting cooler and humid conditions, the process of infection typically begins with the germination of mature conidia and ascospores on the surface of green barley plant parts (Liu et al. 2011; Martin et al. 2021; Suciu et al. 2021). A suitable wet period of 5–30 hours or even longer is necessary for successful infection, followed by appropriate temperatures. The germ tube can originate from conidia and ascospores (Webster 1951; Shipton et al. 1973; Van den Berg & Rossangel 1990). Ascospores typically produce germ tubes from central cells, occasionally from terminal cells, which are septate and branched (Webster 1951). The germination of conidia and the formation of germ tubes usually originate from terminal cells and, in some cases, simultaneously from up to four cells (Caeseele & Grumbles 1979). Recently, Backes et al. (2021) stated that only conidia with more than two septa can germinate and successfully cause infection, while Shaw (1986) previously found that the germination of *P. teres* conidia is only possible in the presence of liquid water and temperatures above 2 °C. Still, there is a prevalent view that the optimal temperature for conidial germination falls within the 20–25 °C range for spot blotch forms, whereas 25 °C is needed for net blotch forms. According to Van den Berg and Rossangel (1990), the percentage of germinating conidia increases with the length of the leaf wetness period, ranging from 77% to 98% when this period exceeds six hours. The authors noted that the conidia can germinate even when the leaf wetness period is much shorter (one hour) but requires temperature regimes of 10–25 °C. Still, they cautioned that while infection by the pathogen is possible at all temperatures within the 10–25 °C range, the required infection period significantly decreases with increasing temperature (at 20 °C, four hours are sufficient). In a later study, Liu et al. (2011) noted that spores can germinate within a few hours at an optimal temperature of 15–25 °C and a relative humidity approaching 100%, whereby the infection rate is higher if wet conditions persist for 10–30 hours or even longer.

During the process of infection, the pathogen undergoes two developmental phases. The first phase is biotrophic and asymptomatic and is characterized by penetration into epidermal cells and spread within the mesophyll. The second phase occurs after a few days and manifests as the appearance of chlorosis and necrosis, marking the transition of the pathogen into a necrotrophic stage (Suciu et al. 2021). The necrotrophic stage is dominant, and the newly formed germ tubes are often highly branched (Jørgensen et al. 1998). After germination, germ tubes and hyphae can grow to varying lengths (sometimes extending up to 0.5 cm) before forming club-shaped appressoria, which serve to penetrate the fungus into the host tissue (Caeseele & Grumbles 1979). The number of appressoria that can form on the germ tube varies, and while they usually emerge at the tip of the germ tube, they can also develop along its length (Jørgensen et al. 1998). The spot form produces more appressoria (which are also larger) than the net form (Lightfoot & Able 2010). Caeseele and Grumbles (1979) indicated that the fungus directly penetrates through the cuticle into epidermal cells. However, Lightfoot and Able (2010) noted that apart from direct penetration through the epidermis and between epidermal cells, the pathogen can also occasionally penetrate through stomata, although this mode of entry is less common. The authors also highlighted differences in fungus penetration between the spot and net forms. In addition to penetrating through the epidermis and between epidermal cells, the spot form has a higher percentage of penetration through stomata compared to the net form, which primarily penetrates between epidermal cells. They also observed that the growth of the infective hyphae on the leaf surface before the formation of appressoria was significantly less pronounced for the spot form compared to the net form. As a part of their investigations, Keon and Hargreaves (1983) and Jørgensen et al. (1998) observed that, after penetration, the fungus creates intracellular primary vesicles within the epidermal cell, which contain dense cytoplasm and numerous organelles. This is followed by the formation of secondary vesicles from which infective hyphae develop during or after penetration into the lower layers of the epidermis. The spot form tends to form a greater number of intracellular vesicles, while the net form forms them less frequently (Lightfoot & Able 2010). Moreover, the net form infects and feeds as a necrotroph throughout the infection process, growing only intercellularly. After successful penetration and the formation of invasive hyphae with spherical infective vesicles at the top of the epidermis, the development of one or more dense, short-cell intracellular hyphae occurs, extending from the vesicle (Jørgensen et al. 1998). Further growth of hyphae within the mesophyll tissue is restricted to intercellular spaces only. Once the pathogen penetrates the plant, lesions appear, causing disruption and disorganization of plant cells (Keon & Hargreaves 1983).

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P. teres f. *maculata*, regardless of its mode of penetration, develops sub-epidermally within the plant tissue (between epidermal and mesophyll cells). It then develops extracellularly through the mesophyll without forming intracellular vesicles but with attempts to penetrate cells. On the other hand, after penetration, *P. teres* f. *teres* develops sub-epidermally throughout the entire mesophyll, with more intensive and extensive growth compared to *P. teres* f. *maculata*, which experiences limited growth (Lightfoot & Able 2010). *P. teres* f. *maculata* causes cell death in the immediate vicinity of the fungal mycelium, while *P. teres* f. *teres* can induce disturbances in cells that are not even in direct contact with the fungus and may be distant from the fungal mycelium (Lightfoot & Able 2010). The above observations suggest that *P. teres* f. *maculata* initially grows as a biotroph (or hemibiotroph), transitioning to necrotrophic growth, while *P. teres* f. *teres* spends its entire lifecycle as a necrotroph, avoiding the biotrophic phase (Lightfoot & Able 2010; Moolhuijzen et al. 2021).

TOXIN PRODUCTION

The rapid onset of symptoms, such as necrosis and chlorosis of infected tissue within 24–48 hours after inoculation, is often attributed to the action of toxins secreted by the pathogen (Weiergang et al. 2002; Sarpeleh et al. 2007; Mikhailova et al. 2010; Liu et al. 2011). Three toxins have been isolated from the culture of the fungus *P. teres* to date: toxin A, toxin B and toxin C. Besides these toxins, other compounds have been isolated, such as protein metabolites and phytotoxic low molecular weight compounds (LMWCs), which can induce necrosis or chlorosis of infected tissue (Serpeleh et al. 2008a). The isolated toxic compounds belong to different chemical classes, including marasmicins, pyrenolides, pyrenolines and three peptide alkaloids: aspergilomarasmin A (toxin C) and its derivatives, toxin A and toxin B (Smedegård-Petersen 1977; Bach et al. 1979; Nukina et al. 1980a, 1980b; Coval et al. 1990; Friis et al. 1991; Nukina & Hirota 1992; Weiergang et al. 2002; Serpeleh et al. 2007, 2008a; Muria-Gonzalez et al. 2020).

The initial findings of toxin secretion by the fungus *P. teres* and its forms were reported by Smedegård-Petersen (1977), who isolated two similar chemical phytotoxic compounds. Initially, they were named toxin A and toxin B. The appearance of necrosis was followed by the rapid development of watery chlorosis and water-soaking, in which the presence of the pathogen's hyphae was not detected, indicating that such symptom appearance and cell death were a result of toxin action (aspergillosamine-derived toxins) (Smedegård-Petersen 1977). Soon after, another new toxin, denoted as toxin C, was confirmed (Bach et al. 1979). Their chemical structures were determined by purifying toxins A, B, and C. Toxin A is N-(2-amino-2-carboxyethyl) aspartic acid, a compound not previously isolated from natural sources. Toxin B is 1-(2-amino-2-carboxyethyl)-6-carboxy-3-carboxymethyl-2-piperazinone, named anhydroaspergillomarasmine A, while toxin C is aspergillomarasmine A. Toxin C is the main toxin secreted in the culture of the *P. teres* fungus (Friis et al. 1991). Non-enzymatic acid-catalyzed conversion converts the same toxin to toxin B (anhydroaspergillomarasmine A) at low pH values. Biosynthetic experiments using radioisotopes suggest that the chemical structure of toxin A indicates that it is a direct precursor to toxin C (aspergillomarasmine A) (Friis et al. 1991). The phytotoxicity of the identified toxins on detached leaves of sensitive and more resistant barley cultivars was investigated by Weiergang et al. (2002). The authors observed that 0.75 mM of toxin A was sufficient to induce the appearance of dark yellow chlorosis in sensitive barley cultivars after 46–72 h, whereby mild necrosis developed after 120 h on leaf tips. Highly resistant cultivars did not exhibit chlorosis or showed very limited chlorosis. Toxin C induced necrosis and light yellow chlorosis at the lowest concentrations (0.25 mM) in sensitive and resistant cultivars. Necrosis and chlorosis symptoms appeared earlier (48 and 72 h, respectively) in sensitive cultivars compared to resistant ones, with necrosis occurring before chlorosis in all experiments. Toxin B did not cause any symptoms at any of the tested concentrations. Further research on barley confirms the higher toxicity of toxins A and C compared to toxin B (Smedegård-Petersen 1977; Bach et al. 1979; Friis et al. 1991).

In addition to toxins, Nukina et al. (1980a, 1980b), Coval et al. (1990), and Nukina and Hirota (1992) identified pirenolides A, B, C and D as bioactive metabolites secreted by *P. teres.* Pirenolides A and B exhibited non-selectivity towards different hosts, with pirenolide A being more toxic than pirenolide B at the same concentrations (Coval et al. 1990). Pirenolide

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A displayed inhibitory effects on mycelial growth and exhibited morphogenetic activity against various treated fungi (Nukina et al. 1980a), while pirenolides B and C showed inhibitory effects and led to the formation of swollen hyphae in the phytopathogenic fungus *Cochliobolous lunata* (Nukina et al. 1980b). Among other metabolites, *P. teres* was found to produce catenarin, a reddish pigment on the substrate, which does not appear to induce pathological changes in plants (Engström et al. 1993).

Sarpeleh et al. (2007) determined the content of phytotoxic LMWCs and proteinaceous metabolites in the culture filtrate of both forms of *P. teres* (with sizes ranging from 10 to 100 kDa), which were responsible for symptom development. Proteinaceous metabolites caused only brown necrotic spots or lesions after 72 h, while LMWCs induced general chlorosis (yellowing and water soaking) after 240 h. Moreover, proteinaceous metabolites were effective only when injected into attached leaves but not detached leaves, indicating potential uptake failure or loss of activity before uptake. In the case of LMWCs, chlorosis symptoms were similar regardless of whether the treatment was applied via uptake on detached leaves or injection into attached barley leaves. Extracted LMWCs exhibited characteristics similar to the previously described aspergillomarasmine A (toxin C) and anhydroaspergillomarasmine A (toxin B), such as electrophoretic properties, staining, thermal stability and host spectrum (Sarpeleh et al. 2007).

Further research on the phytotoxicity of LMWCs proteins isolated from *P. teres* culture revealed their thermal stability and dependency on light and temperature. No symptoms were observed when inoculated plants were kept in the dark or at temperatures below 4 °C, indicating the importance of these factors. Moreover, the partially purified LMWCs exhibited non-selective phytotoxicity towards various plant species, including wheat, barley, beans and tobacco (Serpeleh et al. 2008a, 2008b).

CHARACTERIZATION

Morphological characterization. Numerous species within the genus *Drechslera* are morphologically very similar, making their differentiation challenging at the morphological level due to overlapping characteristics, which are similar to those observed in the genera *Bipolaris*, *Curvularia* and *Exerohiulum* (Shoemaker 1962; Sivanesan 1987; Zhang et al. 2012). Therefore, to facilitate proper identification of the causative agent, several criteria should be considered, including symptoms, hosts and morphological characteristics of the pathogen, and additional diagnostic methods, such as modern molecular methods for diagnosing plant

Table 2. Criteria for the morphological characterization of the genera *Drechslera*, *Bipolaris* and *Exerohiulum* (Shoemaker 1966; Sivanesan 1987; Alcorn 1988)

Characteristic	Genus (anamorph)		
	Drechslera	*Bipolaris*	*Exerohiulum*
Conidial shape	Cylindrical to broadly cylindrical, mostly straight, septate, brown	Spindle-shaped, ovoid-spindle (swollen at the base and spindle-shaped at the top), rarely cylindrical, straight or curved	Spindle-shaped, ovoid-spindle, or cylindrical, straight or curved
Hilum	Rounded, not protruding, with a well-defined interhilum cavity	Slightly raised and shortened to inconspicuous	Highly protruding, shortened with a sheath
Germination	From middle and/or polar cells	From polar cells of one or both, very rarely from middle cells	Mainly from one or both polar cells, rarely from middle cells
Basal germ tube	Lateral, in the middle between hilum and basal septum, rarely subaxial, close to the hilum	Semi-axial, near the hilum and very rarely lateral	Semi-axial, near the hilum, rarely lateral
Formation of septa	First – delimits the basal septum; second – middle; third – distal;	First – mid to submedian; second – delimits the basal cell; third – distal	First – submedian; second – submedian; third – median or variable
Conidiogenous node	Smooth	Smooth to verrucose (with protuberances)	Smooth to verrucose

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Table 3. Criteria for the morphological characterization of the genera *Pyrenophora*, *Cochliobolus* and *Setosphaeria* (Sivanesan 1987; Zhang et al. 2012; Ariyawansa et al. 2014; Marin-Felix et al. 2019)

Characteristic	Genus (teleomorph)		
	Pyrenophora	*Cochliobolus*	*Setosphaeria*
Ascocarp (perithecium)	Sunken but becoming almost superficial, scattered or clustered, globose to subglobose, broadly or narrowly cupulate, smooth-walled with a papilla that is covered with brown to reddish-brown setae, the base of which is dark; perithecium wall composed of 2–4 layers of thick-walled brown cells, textura angularis	Spherical with usually long cylindrical neck, with filamentous hyphae and conidiophores on the spherical part, less frequent or absent on the neck	Spherical to elliptical, usually with setae on the upper half and a short neck
Ascus	Broadly curved with distant apical nasses and a ring, 8-spored, bitunicate, fusiform to cylindrical, with a short and broad pedicel, surrounded by a distinct ocular chamber bordered by a large apical ring	Mainly narrowly cylindrical, cylindrically expanded at the base to rounded, without an apical ring, atrophied bitunicate	Broadly curved with apical nasse but not with a ring
Ascospores	2–3-seriate (arranged in one or more series), large, with horizontal and vertical septa, constricted at the septa, smooth-walled, with a thin mucilaginous sheath, initially colorless, later becoming pale brown to brown, rarely slightly rough	Usually coiled in a spiral with transverse multisepta, often surrounded by a thin mucilaginous sheath, smooth, colorless to pale brown	Large, with transverse and longitudinal septa, surrounded by a thin mucilaginous sheath that can extend beyond the ends of the ascospores, colorless at the beginning, later fading to brown, smooth, rarely slightly rough

pathogens, should be applied. Differences between *P. teres* as the causal agent of net blotch and other species within the *Pyrenophora* genus are based on the symptoms that the pathogens induce on barley plants and the morphological characteristics of the anamorph and teleomorph stages.

Several authors have provided a description of the most important morphological characteristics of the anamorphs of genera *Drechslera*, *Bipolaris* and *Exerohiulum* as well as the key morphological criteria for their differentiation (Shoemaker 1966; Sivanesan 1987; Alcorn 1988). These criteria include the shape of conidia, hilum morphology, conidial germination mode, growth and position of the basal germ tube in relation to the conidial axis, conidial maturation or ontogeny and formation of septa, as summarized in Table 2.

In addition to the morphological characteristics of the anamorph, the features of the teleomorph stage of the aforementioned genera were provided by Sivanesan (1987), Zhang et al. (2012), Ariyawansa et al. (2014) and Marin-Felix et al. (2019). Taxonomic characteristics of the teleomorphs are presented in Table 3.

As previously mentioned, the causal agent of net blotch *P. teres* is a pathogen that exhibits two morphologically very similar forms, making their determination difficult (Smedegård-Petersen 1971). Accurate and reliable differentiation between these forms is based on molecular analyses and the symptoms the pathogen induces in infected plants. The pathogen goes through teleomorphic and anamorphic phases (stages) in its lifecycle. During the teleomorphic phase, as previously stated, it forms structures such as pseudothecia with asci and ascospores, while during the anamorphic phase, it produces conidiophores with conidia. The most important characteristics of these stages will be described further below.

Both forms develop almost uniformly on the medium. The colony consists of a low, uniform, medium-grey mycelium covering the surface. At the same time, the reverse of the culture is initially

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white, later forming a dark brown network of fine mycelium (Shoemaker 1962). The fungus grows abundantly on nutrient-rich media and forms upright tufts resembling fan-like and club-like structures. Tufts can be formed throughout the medium in the petri dish, but their highest concentration is usually found in the central area (Smedegård-Petersen 1971).

Pseudothecia are formed on infected plant debris, often on straw. They typically contain asci with ascospores, although conidiophores with conidia can also form. Pseudothecia of 1–2 mm diameter are frequently formed on stubble and are covered with dark setae containing rod-shaped asci, rounded at the top, with short double-walled (bitunicate) stalks at the base measuring 30–61 × 180–274 µm (Webster 1951; Smedegård-Petersen 1972; Poudel 2018). Smedegård-Petersen (1971) reported that, in their experiments, ascus and ascospore sizes were in the 161–260 × 31–44 µm and 42–61 × 16–25 µm range, respectively. Jordan (1981) also observed ascospores of similar size, 50–60 × 18–22 µm. Webster (1951) reported ascus size of 175–235 × 38–40 µm and ascospore size of 36–51 × 14–20 µm, while Shipton et al. (1973) noted ascus size of 140–335 × 30–62 µm, and ascospore size of 34–66.6 × 13–25.9 µm, with 0–5 transverse and 0–4 longitudinal septa. Ascospores are light yellow-brown, ellipsoidal, with rounded ends and have three to four transverse septa and one, occasionally two, longitudinal septa (located in the middle cell and never present in the terminal cell) (Webster 1951; Smedegård-Petersen 1971, 1972).

Very often, in humid conditions, conidiophores with conidia develop on pseudothecia. After primary infections, the fungus produces numerous conidia on conidiophores on infected organs, serving as a source for secondary infections. Conidia are located at the top of cylindrical conidiophores, which have rounded ends and are slightly swollen at the base, usually arising singly or in groups of 2–3, with 2–5 (produced on stems) and 0–8 (produced in culture) transverse septa which are pale yellow to olive-green in colour (Webster 1951; Liu et al. 2011; Tini et al. 2022; Vasileva et al. 2022). The size of conidiophores ranges from 57 µm up to 600 µm, with a width of 6–18 µm (Shipton et al. 1973). Conidiophore dimensions of *P. teres* isolates from Mexico varied considerably (in the 3.64–79.48 × 10.56–16.82 µm range), with an average size of 41.56 × 13.69 µm (Romero-Cortes et al. 2021). Webster (1951) reported conidiophore length ranging from 60 to 230 µm. Conidiophores of the dotted form are usually longer than those of the net form, with a highly variable shape ranging from rounded to straight or zigzag in the upper parts, complicating the measurement of these structures (Louw et al. 1995).

The conidia of both forms are very similar in shape, size, septation and colour, making differentiation based on those characteristics unfeasible. According to Smedegård-Petersen (1971) and Tini et al. (2022), the conidia are cylindrical, smooth, rounded at the ends and hyaline in transmitted light, becoming light brown with dark scars at the sites where they attach to the conidiophores, while Louw et al. (1995, 1996) noted that the conidia of both forms are olive-brown and have a cylindrical shape, typically with a slightly swollen basal cell. Based on their detailed studies, Smedegård-Petersen (1971) reported differences in conidial size between the net (52–138 × 13–46 µm) and dotted (62–138 × 13–18 µm) forms. According to the measurements conducted by Louw et al. (1995), the conidial size of the dotted form isolates ranges considerably (25–160 × 10–18 µm), with 0–7 septa (4 on average), while 18–120 × 10–18 µm, with 0–5 septa (average 3) was reported for the net form isolates. The conidial size of *P. teres* isolates was reported by Scott (1991) as 40–120 × 19–21 µm, occasionally reaching up to 150 µm. More recently, Romero-Cortes et al. (2021) measured 36.37–102.55 × 13.95–25.61 µm, with an average of 69.46 × 19.78 µm. More than seven decades ago, Webster (1951) reported conidial size of 43–80 × 11–18 µm (produced on stems) and 54–180 × 11–19 µm (produced in culture). In Bulgarian isolates, the conidial size of the net form was within the 69.5–181 × 15.28–24.1 µm range (with 3–8 septa), while the dotted form measured 65–143 × 10.2–19.3 µm (with 3–5 septa) (Vasileva et al. 2022).

On infected plant debris as well as in culture, *P. teres* forms pycnidia, which are round to pear-shaped, ranging in size from 64 to 176 µm, with a thin and fragile yellow-brown wall. They have a rounded papilla or a short ostiole at the top, within which spores are formed. These spores are hyaline, spherical to ellipsoidal and non-septate, and measure 1.4–3.2 × 1.0–1.9 µm (Smedegård-Petersen 1972).

Molecular characterization. Due to the existence of morphological similarities among isolates of different *P. teres* pathogen forms, the application of modern molecular diagnostics is recommended,

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Table 4. Primers for PCR detection of *Pyrenophora teres*, formes and hybrids

Target	Primer name	Primer sequence	Amplicon size (bp)	Reference
P. teres f. *teres*	PTT-F PTT-R	CTCTGGCGAACCGTTC ATGATGGAAAAGTAATTTGTA	378	Williams et al. (2001)
P. teres f. *maculata*	PTM-F PTM-R	TGCTGAAGCGTAAGTTTC ATGATGGAAAAGTAATTTGTG	411	
P. teres f. *teres*	DTT471h	F 5'-CCTGAGTAACTTGCCCCACC-3' R 5'-GAAAAGAGATGATGCGGACAC-3'	91	Leisova et al. (2005)
	DTT339i	F 5'-TGATGCGCTGGAGTGAGACAC-3' R 5'-TGTACATACGCCGCATCACG-3'	81	
P. teres f. *maculata*	DTM494d	F 5'-TATTCTGCTAAGAGCTAGCATCCTA-3' R 5'-ACTGCGTACCAATTCTCTACAACTA-3'	161	
	DTM348j	F 5'-CTTGATGCGCTGGAGTGAGA-3' R 5'-TGCATTTCCACCTACTGGTATGTAC-3'	66	
P. teres (GPD)*	PtGPD1-F PtGPD1-R	CGTATCGTCTTCCGCAAC TTGGAGAGCACCTCAATGT	586	Lu et al. (2010)
P. teres f. *teres*	Ptt MAT1-1 F Ptt MAT1-1 R	ATGAGACGCTAGTTCAGAGTCT GATGCCCAGCCAAGGACAA	1 143	
	Ptt MAT1-2 F Ptt MAT1-2 R	TACGTTGATGCAGCTTTCTCAAT AACACCGTCCAAAGCACCT	1 421	
P. teres f. *maculata*	Ptm MAT1-1 F Ptm MAT1-1 R	TGTTAGAGACCCCACCAGCGT CAGCTTTCTTGGCCTTCTGAA	194	
	Ptm MAT1-2 F Ptm MAT1-2 R	ACGCAAGGTACTCTGTACGCA GACGTCGAGGGAGTCCATTT	939	
P. teres f. *teres*	PttQ1	GGATGATGACCTCGCCAGAT-F GCGATGGTATGTTCTGCGAA-R	70	Poudel et al. (2017)
	PttQ2	AACACTCTGAACGTGGTTGC-F TTCAGTTGTAAGCTGCGTGG-R	110	
	PttQ3	CCTCGTCCTAAGTTGACTCGA-F TTACACGGGTTCCCTCCATC-R	130	
	PttQ5	GCATTGTCTAGCACTCGTCG-F CGCGGACTCAGAAGACATTG-R	173	
	PttQ4	CGTCCCGCCGAAATTTTGTA-F CAAGGACTTACGCGCTCAAA-R	150	
	PttQ6	TCAGAATACTCCGCGGACTC-F GTCCGCATTGTCTAGCACTC-R	188	
P. teres f. *maculata*	PtmQ8	ACGCTAAGACCACCTCGTTT-F TCGACCAGAGAGAGCACAAA-R	161	
	PtmQ9	AATGCTCAATTCTGGTGGCG-F TGTTCGAGTGCAAACTTGGG-R	201	
	PtmQ10	TGCTGTGGACTTAGACGAGG-F TGGGGATCCTTGACCAACTC-R	220	
	PtmQ11	GATTAGACCATTACCACACTAGCG-F ACCACCACATCTTTCCTACTAACT-R	260	
	PtmQ7	GTAGAGGCTGTAGGAGATGTGATT-F CATGGCAAATTGTTCGTAATCCTG-R	140	
	PtmQ12	CTAACCAAAGAACTTCACAGACGA-F CCTTATTAGCCAATTCCATGTCGA-R	279	

* GPD – glyceraldehyde-3-phosphate dehydrogenase

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given that the forms of *P. teres* cannot be differentiated based on the morphology of their reproductive structures. Distinguishing between the *maculata* and *teres* forms is possible based on the symptoms they induce in infected plants. In some cases, errors can occur in detection due to symptoms similar to those caused by other pathogens affecting barley. Therefore, the most reliable approach for distinguishing between species within the *Pyrenophora* genus and forms within the *P. teres* species requires the use of molecular markers, employing the amplification of fungal DNA using the Polymerase Chain Reaction (PCR) technique, as well as sequencing portions of the genome of the species and forms within this genus.

The first markers for detecting *P. teres* and its forms using molecular tests based on PCR were developed by Williams et al. (2001) (Table 4). To develop these markers (primers), the authors used RAPD PCR to amplify "decamer" primers. They observed that 28 of 50 primers tested showed at least one RAPD between the pathogen forms. The RAPD marker that was present and specific to *P. teres* f. *maculata* isolates were cloned, and clones were selected based on size similarity with the specific amplicon. The ends were then sequenced, and the designed primers were used to amplify DNA from both forms of *P. teres* (fragments of the same size). Since specificity was not observed, the amplicons were further sequenced to find DNA polymorphism. Sequence differences between the net form isolates were used to design specific primers, and the designed primer sets were tested on isolates of different origins: American, Canadian and German. The primers designed by Williams et al. (2001) amplified bands of different sizes for net form isolates, indicating their specificity for these forms. The authors reported no amplification for *C. sativus*, but DNA amplification occurred for the pathogen *P. graminea*. Considering that *P. graminea* is clearly distinguishable from *P. teres* based on symptoms, it is probably unnecessary to differentiate it using the PCR test. The PTM primer set specifically amplified a band of 411 bp, while the PTT primer set specifically amplified a band of 378 bp.

Newer markers for distinguishing forms within the *P. teres* species, as well as between *P. teres* and *P. raminea* (which was not possible using the previously designed primers), were developed in 2005 by Leisova et al. (2005) (Table 4). To successfully design these primers, the authors used AFLP analysis (sequenced AFLP markers) on a larger number of isolates from *P. teres*, *P. graminea*, *P. tritici-repentis* and *Helminthosporium sativum*, using 33 rimer pairs. The AFLP analysis resulted in high levels of polymorphism, allowing markers specific to the net form isolates to be selected. Additionally, AFLP patterns varied among the different tested species. Specific AFLP markers for the forms were cloned and sequenced to design specific primers. The designed primers were subsequently tested on a larger number of different isolates (30 isolates of *P. teres* f. *teres*, 36 isolates of *P. teres* f. *maculata*, five isolates of *P. graminea*, three isolates of *P. tritici-repentis*, one isolate of *P. flavispora* and four isolates of *Helminthosporium sativum*) to determine the specificity of certain primers for the pathogen forms. PCR analysis confirmed the success of two primer sets for the species and forms. Primers DTT471h and DTT339i are specific for *P. teres* f. *teres*, amplifying bands of 91 and 81 bp, respectively. At the same time, primers DTM494d and DTM348j are specific for *P. teres* f. *maculata*, amplifying bands of 161 and 66 bp, respectively.

To simultaneously determine the pathogen form and mating type (MAT-1 or MAT-2), new primers for the PCR method were designed by Lu et al. (2010) (Table 4). To design the primers, the authors conducted a test to identify mating-type loci using common PCR primers, which were subsequently cloned, sequenced and analyzed. After sequencing analysis, a specific single nucleotide polymorphism (SNP) was identified in idiomorphs MAT 1-1 and MAT 1-2 isolates of the reticulate and spot forms regardless of origin. These new specific SNPs were used for primer design. Two primers specific to the reticulate form and two primers specific to the spot form were designed. They were tested on 37 reticulate isolates and 17 spot form isolates and on isolates of *P. graminea*, *P. tritici-repentis*, *Stagonospora nodorum* and other actinomycetes used as controls. The designed primers successfully differentiated between the forms and mating types of the *P. teres* pathogen by amplifying bands of varying base pair sizes. The authors also designed two control primers to confirm the *P. teres* species. These primers were designed for the glyceraldehyde-3-phosphate dehydrogenase gene of *P. teres*. Verification of the control primers was performed in all tests and proved successful in confirming the species by amplifying a 586-base pair-long band. This set of primers made a significant contribution to *P. teres* research, allow-

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ing for the simultaneous identification of the species, pathogen forms and mating types.

Markers specific to the forms and their hybrids resulting from mating (recombination) between different forms of *P. teres* were developed by Poudel et al. (2017) (Table 4). This was a considerable advancement, given that previous markers were unable to distinguish hybrids within the *P. teres* species. The identification of distinct regions specific to *P. teres* f. *teres* and *P. teres* f. *maculata* was achieved by Poudel et al. (2017) by using whole-genome assemblies and aligning transcripts obtained from RNA sequencing (RNA-seq). Twelve primers were developed, six specific to the net form and six to the spot form. These primers were designed to produce DNA bands ranging in size from 70 to 190 bp for the *teres* form and from 140 to 280 bp for the *maculata* form. The specificity of the primers was confirmed using a large number of isolates, including 86 *P. teres* f. *teres* isolates, 78 *P. teres* f. *maculata* isolates from Australia, 22 *P. teres* f. *teres* isolates, and 24 *P. teres* f. *maculata* isolates from South Africa, seven isolates of *P. teres* obtained from barley grass (*Hordeum leporinum*) in Australia, six isolates of *Bipolaris sorokiniana* and one isolate each of *Exserohilum rostratum* and *P. tritici-repentis*. The primers demonstrated high specificity, amplifying with all six markers present and not amplifying with DNA from other pathogen species. The specificity of the primers was also tested on hybrids resulting from the mating of two pathogen forms, and high success rates were achieved. The number of markers in hybrids ranged from 2 to 11, with at least one marker always specific to a particular form. Interestingly, when isolates were obtained from barley grass, with four out of six primers, amplification occurred only for the *teres* form, while no amplification occurred for the *maculata* form.

Sequencing of ribosomal chains, or sequence analysis, has become the most reliable method for studying phylogenetic relationships among different organisms. Through ribosomal genes and their rate of evolution, the evolution of the entire genome can be observed (White et al. 1990). In one of the first phylogenetic analyses of ITS and gpd regions in the genus *Pyrenophora/Drechslera*, it was demonstrated that the *Pyrenophora* genus is monophyletic and that its asexual stage clusters together with its predicted sexual relatives (Zhang & Berbee 2001). This finding was further confirmed by Ariyawansa et al. (2014), who conducted a phylogenetic analysis of combined ITS, LSU and GPDH datasets, indicating that sequences from isolates of the same species group together. These authors used universal primers ITS4 and ITS5, as well as gpd1 and gpd2, to amplify these two gene regions to achieve reliable identification and characterization of species within the *Pyrenophora* genus. Isolates from various parts of the world were used to obtain sequences, which were then used to construct a phylogenetic tree. Based on the sequences from these gene regions (ITS and gpd), a phylogenetic tree was constructed, and ten groups were identified within the *Pyrenophora* cluster. *P. teres*, *P. graminea* and *P. japonica* were grouped in a subcluster of the sixth group based on the ITS region. However, the gpd region was not sequenced for the species *P. graminea*, as sequences of these species are highly similar based on the ITS and gpd regions (98.6% and 100%, respectively). *P. teres* and *P. japonica* have almost identical ITS sequences at 98.6% similarity (Zhang & Berbee 2001), which confirms the accuracy of earlier studies suggesting that *P. japonica* is synonymous with or a form of the same species as *P. teres*, known as the spot form of net blotch, *P. teres* f. *maculata* (Smedegård-Petersen 1971; Crous et al. 1995; Campbell et al. 1999).

Wallwork et al. (1992) reported on a new barley pathogen species in Australia under the name *P. hordei*, which had smaller ascospores compared to those observed in the *maculata* form. Later, based on ITS sequences that were identical to those of *P. teres* f. *maculata*, it was established that this was the spot form of net blotch and not a new species named *P. hordei* as originally proposed by the author (Stevens et al. 1998; Kinzer 2015).

According to Ariyawansa et al. (2014), for a more detailed characterization of *Pyrenophora* species, the use of a combination of ITS (ITS1/ITS4), LSU (LROR/LR5) and GPDH (gpd1/gpd2) datasets is necessary. The same authors confirmed the similarity of sequences of these three species based on multiple gene regions (ITS, LSU, GPDH). They clustered *P. teres* with *P. japonica* and *P. graminea* in the same clade with bootstrap values greater than 50%. For differentiating and identifying species within the *Pyrenophora* genus, Marin-Felix et al. (2019) suggested that molecular data from various genes—primarily ITS, GAPDH and RPB2—is necessary. The close relationship between *P. teres* and *P. graminea* based on ITS and gpd sequences was recently confirmed

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by Vasighzadeh et al. (2022), who indicated that the two species can be distinguished based on ITS sequences and combined ITS/gpd sequences. As a part of an earlier investigation, Ellwood et al. (2012) conducted a phylogenetic analysis using five different gene regions: actin (ACT), β-tubulin (TUB), cytochrome P450 14α-demethylase (CYP51), translation elongation factor-1a (eEF1A) and glyceraldehyde-3-phosphate dehydrogenase (GAPDH). The obtained findings confirmed the diversity of forms within the *P. teres* species and their close relationship and clear distinctions between the tested species *P. tritici-repentis*, *P. teres* and *Cochliobolus heterostrophus*.

The development of next-generation sequencing (NGS) techniques has made it possible to obtain the complete genomes of both forms of *P. teres* with a size of 41.58 Mbp (*P. teres* f. *maculata*) and 51.76 Mbp (*P. teres* f. *teres*) (Syme et al. 2018). The genomes of both forms contain 12 chromosomes and are highly collinear. Furthermore, they are characterized by insertions and an increase of transposable elements (TEs), which is especially expressed in *P. teres* f. *teres* that has a longer genome, which might explain a longer host relationship and a higher extent of host–pathogen genetic interactions of this form in comparison with *P. teres* f. *maculata* (Syme et al. 2018). Moreover, a higher NRPS expansion in *P. teres* f. *teres* compared to *P. teres* f. *maculata* may be related to its greater host specialization (Moolhuijzen et al. 2020). Using the NGS techniques, 24 *P. teres* genomes originating from the USA, Australia, Canada, Denmark, and Morocco are currently available in the GenBank (Ellwood et al. 2010; Wyatt et al. 2018, 2020; Martin et al. 2020; Duong et al. 2021; Wingfield et al. 2022; Li et al. 2023).

MANAGEMENT OF NET BLOTCH

The highest yields and grain quality of barley are achieved through the implementation of Integrated Pest Management (IPM) measures, which involve a combination of agronomic, biological and chemical strategies, as well as the cultivation of resistant varieties and other available measures that contribute to better disease control and reduced production costs.

Cultural practices. Cultural practices represent one of the essential preventive strategies against numerous plant pathogens. Understanding the development cycle of the *P. teres* pathogen and its maintenance and transmission through infected seeds and crop residues underscores the significant role of preventive agronomic measures in reducing both potential inoculum and subsequent outbreaks of net blotch. Planting healthy seeds, practising crop rotation, and soil management constitute the initial steps in eliminating potential net blotch inoculum and thus contributing to disease control (Van den Berg & Rossnagel 1991; McLean & Hollaway 2019).

Planting healthy seeds ensures a more reliable production process by eliminating the possibility of seedling decay and infection of newly emerged plants, given that infected seeds can serve as a highly suitable source of primary inoculum. Many reproductive structures of the *P. teres* pathogen have been found on the seed surface (Jordan 1981). However, residues of infected seeds, other plant parts, and straw also serve as significant potential sources of inoculum. Straw is an important inoculum source, and infected seeds facilitate the introduction of the pathogen into previously uninfected plots (Youcef-Benkada et al. 1994; Gangwar et al. 2018).

Treatments such as burning the straw left in the fields after harvest or deep ploughing have proven effective in reducing net blotch (Jordan 1981; Gangwar et al. 2018; Abebe 2021). However, due to climate change, high summer temperatures, the threat of causing large fires, reduction of organic matter and destruction of desirable soil microflora, burning crop residues is no longer ecologically acceptable and is prohibited in most parts of the world. For this reason and due to the need for livestock bedding, straw is currently typically collected and removed from the fields through baling.

Based on the evaluation of various methods of straw manipulation and their impact on conidia levels, Jordan and Allen (1984) emphasized that greater quantities of conidia were captured in plots where straw was chopped compared to those where straw was baled, and the least amount was found in plots where straw was burned. Direct seeding into stubble or burnt areas, or plots with chopped straw, led to the occurrence and capture of a higher number of conidia compared to ploughed plots (Jordan & Allen 1984). In their study on controlling *P. tritici-repentis*, Jørgensen and Olsen (2007) found that soil tillage also had an impact on disease occurrence, highlighting that reduced tillage led to a several-fold increase in disease severity.

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McLean et al. (2009) corroborated these findings, noting that reduced tillage practices contribute to increased disease incidence. According to van den Berg and Rossnagel (1991), crop rotation can also significantly reduce the net blotch inoculum levels. Similarly, based on their investigation of the survival and persistence of pathogens on crop residues in barley and wheat, Duczek et al. (1999) determined that a pause of at least two years is mandatory and even longer intervals between barley crops are desirable for better net blotch control. Accordingly, it can be concluded that direct tillage (ploughing) is essential for controlling net blotch in barley but should be accompanied by other agrotechnical measures, with a focus on implementing multi-year crop rotation.

Host plant resistance. Creating resistant varieties represents the most efficient and ecologically sustainable means of disease control with minimal costs for agricultural producers. Only a resistant genotype can partially or even completely eliminate the need for the application of chemical control measures (McLean et al. 2009) while increasing yield and grain quality, which is also a primary objective of numerous breeding strategies. A resistant genotype exhibits a small number of lesions as well as a small lesion size on leaves, reduces or delays sporulation, restricts fungal growth within infected tissue and enhances the secretion of antifungal substances by the plant in its foliage (Graner et al. 1996; Backes et al. 2021).

In various studies, key quantitative trait loci (QTL) genes for resistance to *P. teres* in barley have been identified on seven barley chromosomes. The obtained findings indicate that their resistance can be effective in both young and mature barley plants (Bockelman et al. 1977; Graner et al. 1996; Williams et al. 1999, 2003; Manninen et al. 2000, 2006; Cakir et al. 2003; Friesen et al. 2006; Abu Qamar et al. 2008; Grewal et al. 2008, 2012; McLean et al. 2009; Tamang et al. 2015; Wang et al. 2015; Poudel 2018; Clare et al. 2020). Nearly 70 years ago, the first genes for resistance to *P. teres* were discovered. These genes (denoted as Pt1, Pt2 and Pt3) were found in the barley varieties and lines Tifang (CI 4407-1), Ming (CI 4797), Harbin (CI 4929) and Manchuria (CI 2335), and two accessions CI 4922 and CI 2750 (Graner et al. 1996). These genes were subsequently confirmed on the 3H chromosome of barley. On the same chromosome, another resistance gene, Rpt1b, was later identified in the CI9819 variety (Bockelman et al. 1977), as well as Pt, in the DHL (Doubled Haploid Line) resulting from the cross between the Igri and Franka varieties (Graner et al. 1996).

In various barley lines, QTL genes influencing resistance to the net form of the pathogen have been identified on chromosomes 2H, 3H, 4H, 5H, 6H and 7H (Graner et al. 1996; Steffenson et al. 1996; Cakir et al. 2003, 2011; Raman et al. 2003; Friesen et al. 2006; Gupta et al. 2010; Islamovic et al. 2017; Koladia et al. 2017; Novakazi et al. 2019). However, major QTL resistance genes have been found near the centromere of chromosome 6H, making this genetic region particularly significant for protection against the net form of the pathogen (Manninen et al. 2000; Cakir et al. 2003, 2011; St. Pierre et al. 2010; Adawy et al. 2013; Novakazi et al. 2019). QTL genes have also been detected on chromosomes 3H and 6H in the resistant Pompadour and susceptible Stirling barley varieties and in their DHL lines in the experiments performed by Gupta et al. (2010). On the same chromosomes (6H and 3H), QTL genes for resistance were later mapped in resistant varieties Ciho 5791 and Tifang by Koladia et al. (2017). In the study conducted by Manninen et al. (2000), the major resistance gene in the 6H chromosome (designated as *Rpt5*) was located in DH lines resulting from the cross between the sensitive Rolfi and the resistant CI9819 barley varieties. The authors also identified other minor-effect genes on chromosomes 1H, 2H, 3H, 5H and 7H. The Rpt5 — located in the centromeric region of the 6H chromosome — is considered a complex locus that plays a crucial role in the interaction between *P. teres* f. *teres* and barley (Adawy et al. 2013; Clare et al. 2020). Two major QTL resistance genes are also located on chromosome 6H in bins 2 and 6. St. Pierre et al. (2010) made crosses between the susceptible NB 'Sep2-72' and the resistant NB 'M120' in barley breeding lines. In a population of DH lines resulting from the cross between the varieties Rika and Kombar, two resistance genes named *rpt.r* and *rpt.k* were identified by Adawy et al. (2013) on chromosome 6H against tested isolates of the net form. The most significant QTL genes on chromosome 6H, representing a large genomic region, were found in DH lines resulting from the cross between the resistant AT4 variety (resistant to NB) and the sensitive Femina variety (sensitive to NB) (Adawy et al. 2013). Additionally, Novakazi et al. (2019) discovered 15 QTL resistance genes for seedlings and ma-

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ture plants on chromosomes 3H, 4H, 5H, 6H and 7H among 449 domestic and commercial barley varieties in Russia. These authors also discovered previously unidentified QTLs on chromosomes 3H, 5H, 6H and 7H.

QTL genes for resistance to the spot form of net blotch in both seedlings and mature barley plants have been discovered on chromosomes 1H, 2H, 3H, 4H, 5H, 6H and 7H in numerous studies (Williams et al. 1999, 2003; Friesen et al. 2006; Manninen et al. 2006; Grewal et al. 2008; Cakir et al. 2011; Tamang et al. 2015, 2019; Wang et al. 2015; Burlakoti et al. 2017). In seedling populations of the CI9214/Stirling, Keel/Gairdner and Tilga/Tantangara varieties, a major resistance QTL gene was observed on chromosome 7H. This significant locus for resistance is designated as *Rpt4*. In DH lines of the Chebec/Harrington varieties, a major QTL for resistance was also mapped to chromosome 7H, approximately in the exact location as *Rpt4*. In adult plants from the Galleon/Haruna Nijo population, QTL genes were identified on chromosomes 5H and 7H with significant impacts on resistance and chromosome 4H with minor impacts. On the VB9104 line, Williams et al. (2003) identified the main source of resistance on chromosome 7H. In their earlier study, the authors identified locus *Rpt4*, highly significant for resistance to the spot form, on chromosome 7H in the Galleon variety (Williams et al. 1999). A new source of QTL resistance called *Rpt6* was identified by Manninen et al. (2006) on chromosome 5H in DH lines (Rolfi x CI 9819). Three QTLs — *QRpt6*, *QRpts4* and *QRpt7* — were later identified on chromosomes 6H, 4H and 7H, respectively, in DHL lines of CDC Dolly (susceptible)/TR251 (resistant) varieties, providing good resistance to the spot form (Grewal et al. 2008). Significant QTL loci for resistance have also been identified in the DHL Baudin × AC Metcalfe on chromosome 6H in both seedlings and mature barley plants (Cakir et al. 2011). In the Q21861 line, Friesen et al. (2006) identified the first resistance gene on chromosome 4H, while the *Rtp8* locus was later identified on chromosome 4H and was shown to have a significant effect on the resistance of both seedlings and mature barley plants (Franckowiak & Platz 2013; Clare et al. 2020). In their study, as a part of which association mapping (AM) on 1947 genotypes of spring barley was analyzed, Tamang et al. (2015) confirmed the previously identified loci associated with resistance to SFNB, including *QRpt4*, *QRpt6*, *QRpt7*, *Rpt4*, *Rpt6* and a QTL on 4H without specific nomenclature. The significance of the 7H chromosome region was highlighted by Wang et al. (2015), who identified four highly significant QTLs for resistance in both seedlings and mature plants — *QRptm7-4*, *QRptm7-6*, *QRptm7-7* and *QRptm7-8* — among a total of 29 QTL regions associated with barley resistance to SFNB across all seven barley chromosomes. In addition to the aforementioned findings, the authors identified another highly significant QTL on chromosome 3H, denoted as *QRptm3-4*. Burlakoti et al. (2017) identified a total of 10 significant QTLs in populations of 376 advanced breeding lines, with newly confirmed significant QTLs on chromosomes 2H (*SFNB-2H-8-10*, *SFNB-2H38.03*) and 3H (*SFNB-3H-78.53*, *SFNB-3H-117.1*). In three recombinant biparental inbred lines (Tra_67381, Pin_67381 and Pin_84314) resulting from crosses with malting barley varieties Tradition (six-row) and Pinnacle (two-row), as well as the two world barley core collection lines, PI67381 and PI84314, a total of 12 QTL loci were identified on chromosomes 2H (*QRptm-2H-1-31*, *QRptm-2H-77-83*, *QRptm-2H-126-137* and *QRptm-2H-141-152*), 3H (*QRptm-3H-81-88*, *QRptm-3H-56-65*), 4H (*QRptm-4H-58-64*, *QRptm-4H-120-125*), 6H (*QRptm-6H-55-64*) and 7H (*QRptm-7H-119-137*, *QRptm-7H-138-160*, *QRptm-7H-92-95*). Among these 12 identified QTLs, three new QTLs that were not previously confirmed were discovered on two chromosomes: *QRptm-2H-77-83*, *QRptm-2H-141-152* and *QRptm-7H-92-95*. Common resistance QTL genes were detected on chromosomes 2H and 7H in all three inbred lines (Tamang et al. 2019).

Biological control. The product Cedomon, based on *Pseudomonas chlororaphis* strain MA 342, is the only biological agent currently available for net blotch disease control and is commercially accessible in the United States, Sweden, Norway, Finland and Austria (Abebe 2021). The bacterium competes for nutrients and space, enhances plant defence systems, promotes root and shoot development, and inhibits fungal growth through the production of 2,3-dihydroxy-2,3-didehydrohizoxin, an antifungal compound (Abebe 2021).

O'Brien (2005) highlighted that *Pseudomonas* strains provided excellent protection in controlling net blotch disease in field conditions and significantly reduced disease severity. In laboratory

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conditions, the positive effects of bacteria belonging to the *Trichoderma* genus (*T. viride*, *T. koningii*, *T. pseudokoningii*), actinomycete (*Mycromonospora* sp. α6) and certain unidentified fungi inhibited the formation of sclerotinoid organs of the pathogen *P. teres* on previously infected and incubated barley stems. However, Ali-Haimoud et al. (1993) found that they also induced certain abnormalities in the morphology of sclerotinoid organs. The same authors noted that the net form of the pathogen exhibited greater resistance to the filtrates of these cultures compared to the spot form, which was more sensitive. The antagonistic properties and effectiveness of biotic control of Serbian strains of the *Trichoderma* genus were confirmed in *in vitro* conditions by Tančić-Živanov et al. (2017). The isolates significantly inhibited the radial growth of the pathogen *P. teres*, as well as other fungal pathogens like *Ascochyta pisi*, *A. pinodes*, *A. pinodella*, *Fusarium graminearum*, *F. proliferatum*, *F. verticillioides*, *F. oxysporum*, *Macrophomina phaseolina* and *Sclerotinia sclerotiorum*. In laboratory experiments, numerous species of the *Trichoderma* genus have been found to produce volatile organic compounds (VOCs) as secondary metabolites. Moya et al. (2018) noted that these compounds reduce plant diseases and promote better plant growth. VOCs produced by species such as *T. longibrachiatum* and *T. harzianum* significantly inhibited the growth of *P. teres* mycelium, leading to the appearance of non-pigmented mycelium and the formation of numerous vacuoles within hyphae (vacuolization). According to these authors, the tested isolates significantly reduced the severity of *P. teres* on barley seedlings, stems and leaves in *in vivo* conditions. *Trichoderma* species are present worldwide in various habitats, including agricultural soil, forests, lakes, plant roots, dead plant material, etc., with a high reproductive potential. This makes them highly suitable for isolation and study, with selecting appropriate pathogenic strains being a crucial factor (Tančić-Živanov et al. 2017).

The application of a suspension containing the IK726 isolate of the fungus *Clonostachys rosea* was confirmed by Jensen et al. (2016) to significantly reduce the infection rate of *P. teres* on young barley plants. The fungicidal effect on the pathogen *P. teres* and endophytes isolated from the plant *Elymus repens* (L.) Gould when endophyte suspensions were applied to barley seeds was recently confirmed by Høyer et al. (2022). In their experiments, these authors found three endophytic isolates — *Lasiosphaeriaceae* sp. E10, *Lindgomycetaceae* sp. E13 and *Leptodontidium* sp. E16 — exhibited a positive effect against the pathogen *P. teres*.

Chemical control. Fungi, as the most significant causative agent of plant diseases, are primarily controlled by the application of fungicides. According to Backes et al. (2021), 70% of fungal pathogens in cereals are controlled through fungicide applications. Foliar fungicides can be used effectively to control diseases, especially when the host plant's resistance is weak (McLean et al. 2009; Tini et al. 2022). As the timing of chemical treatments also affects their effectiveness, it is desirable to apply foliar treatments before the disease symptoms appear or before plant infection by the pathogen occurs, necessitating continuous crop monitoring (Martin et al. 2021). Monitoring environmental conditions is also crucial because if the external conditions are dry and unfavourable for disease development, foliar treatments during vegetation may not be required (Tini et al. 2022). The most effective chemical control of net blotch is achieved by applying foliar treatments to the upper leaves later in the growing season during grain filling (Abebe 2021). Turkington et al. (2004) and Tini et al. (2022) recommended later application of foliar treatments to barley, i.e., during the flag leaf emergence or the heading stage. However, under favourable conditions for disease development, it may be necessary to apply treatments several times during the growing season, particularly using a combination of different active substances with different modes of action in subsequent applications.

The main classes of fungicides currently used worldwide for controlling net blotch are succinate dehydrogenase inhibitors (SDHI), demethylation inhibitors (DMI) and quinone outside inhibitors or strobilurins (QoI) (Sierotzki et al. 2007; Sooväli & Koppel 2010; Mair et al. 2016; Rehfus et al. 2016; Stepanović et al. 2016; McLean & Hollaway 2019; Suciu et al. 2021; Tini et al. 2022).

In Canada, during the 1980s, foliar systemic fungicides from the triazole group (DMI), based on etaconazole and propiconazole, were used with high efficacy for controlling important barley diseases (Sutton & Steele 1983). The effectiveness of tebuconazole (DMI) was investigated in field trials for spring barley over three years against two significant barley diseases caused by

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fungi *P. teres* and *C. sativus* by Sooväli and Koppel (2010). Tebuconazole exhibited high efficacy against these diseases and significantly increased barley grain yields under various infection conditions. McLean and Hollaway (2019) also reported that a treatment combining two active substances, prothioconazole and tebuconazole (DMI), applied twice at growth stages Z31 and Z39 (Zadoks scale), showed the highest effectiveness. The efficacy of fungicides from the DMI group, including epoxiconazole, propiconazole and tebuconazole, on Algerian net blotch isolates was confirmed *in vitro* and *in vivo* tests by Lammari et al. (2020b). The net form of the pathogen was more susceptible than the spot form to all tested DMI group active substances in laboratory conditions. The results reported by Jayasena et al. (2002) highlighted the high efficacy of propiconazole applied individually and in combination with iprodione against the spot form of net blotch. On the other hand, Gisi et al. (2000) found that triazoles inhibit the demethylation step of C14 in the fungal ergosterol biosynthesis, which is why they are called demethylation inhibitors (DMI).

In research conducted by Stepanović et al. (2016) in Serbia, the most effective control of net blotch was achieved by applying a combination of two active substances from different chemical groups, namely pyraclostrobin (QoI) and epoxiconazole (DMI), when applied twice during the vegetation period (BBCH 51 and BBCH 61-65). Similar results were reported in Romania by Suciu et al. (2021), indicating that the application of DMI and QoI fungicides was the most effective combination, particularly when the active substances (azoxystrobin and pyraclostrobin) were used alongside cyproconazole and were applied twice at growth stages BBCH 32 and BBCH 49. In Italy, the application of fungicide combinations with different modes of action, such as DMI + SDHI (prothioconazole + bixafen), SDHI + DMI (benzovindiflupyr + prothioconazole) and SDHI + QoI (fluxapyroxad + pyraclostrobin), once during the vegetation period at growth stage BBCH 39 proved highly effective compared to untreated controls (Tini et al. 2022). Lammari et al. (2020b) identified azoxystrobin and pyraclostrobin (QoI) as the most effective in laboratory tests compared to active substances from the DMI and SDHI groups. In the same research, azoxystrobin exhibited satisfactory control of Algerian net blotch isolates at significantly lower doses *in planta* tests conducted in controlled environments. A relatively newer active substance, metyltetraprole, with a broad spectrum of action against significant cereal diseases, demonstrated excellent efficacy against net blotch in laboratory and field studies (Suemoto et al. 2019).

In addition to the use of QoI (strobilurins) and DMI (azoles), succinate dehydrogenase inhibitors (SDHI) are very effective fungicides for controlling net blotch in barley (Rehfus et al. 2016). Among SDHI fungicides, fluxapyroxad was found to be the most effective, followed by azoxystrobin (QoI), both demonstrating high efficacy even at significantly lower doses than recommended, inhibiting tested net blotch isolates *in planta* greenhouse tests (Lammari et al. 2020b).

One of the significant concerns when using fungicides is their potential harm to the environment, the development of resistant pathogen strains and the eventual prevalence of these strains within pathogen populations. The appearance of triadimenol (DMI) fungicide-resistant isolates of *P. teres* in New Zealand was one of the earliest instances, as indicated by Sheridan et al. (1987). Resistance to QoI fungicides was also identified in France, Sweden and Denmark in 2003 (Abebe 2021; Backes et al. 2021).

In addition to foliar treatments, special attention should be given to seed treatment, considering that the pathogen *P. teres* can be transmitted and maintained in infected seeds, leading to infection on new surfaces. The success of barley seed treatments in significantly reducing inoculum levels and the occurrence of net blotch was confirmed by Hampton (1980), Martin (1985) and McLean and Hollaway (2019). The efficacy of seed treatment up to a particular growth stage was also demonstrated by Hampton (1980) and Sutton and Steele (1983), indicating the potential need for later foliar treatments. More recently, the significant efficacy of seed treatment with the active substance fluxapyroxad was confirmed by McLean and Hollaway (2019). These authors noted that seed treatment with fluxapyroxad reduced disease incidence and increased barley yield and grain quality, especially when followed by foliar treatments later in the vegetative growth stage (post-stem elongation) and mainly where conditions are conducive to disease development. However, it is important to note that the effectiveness of seed treatment depends on several factors, including chemical fungitoxicity, fungal susceptibility and seed coverage quality (Reis et al. 2012). For example, Reis et al. (2012) found that seed treatment with the active substance iprodi-

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one was the most effective against fungal pathogens. However, this active substance is no longer approved in the European Union.

CONCLUSION

This review provides a comprehensive profile of a significant pathogen attacking cultivated barley. Based on the study of a large number of literary sources, we can conclude that net blotch, caused by the fungus *P. teres*, is a highly prevalent barley disease worldwide, with significant negative economic impacts on production, yield and the harvested crop quality. Infected plants exhibit symptoms that appear on all above-ground parts, particularly on the leaves, in the form of net-like patterns or spots. The occurrence of symptoms is influenced by the pathogen's form and virulence, host susceptibility (genotype) and external environmental factors. Toxins, protein metabolites and phytotoxic low molecular weight compounds (LMWCs) secreted by the pathogen in infected tissue also significantly influence symptom expression and host sensitivity. The pathogen is transmitted through infected seeds, as well as infected plant parts and plant residues, and its mitigation relies on an integrated approach to control measures, including the essential implementation of long-term crop rotation. Environmental factors such as humidity and temperature play a key role in the occurrence and spread of the disease. Therefore, understanding the epidemiology of net blotch in barley facilitates more effective disease management and control. Molecular markers are useful tools for accurately detecting and identifying this pathogen, as their use can enhance diagnostic efficiency and disease monitoring. Applying integrated control strategies to combat net blotch in barley is important, which presents challenges for future high-quality and profitable barley production. This includes agronomic practices, biological control and chemical treatments, and the indispensable selection of highly resistant barley genotypes (host plant resistance). The integration of these approaches can reduce disease damage, maintain barley crop health and decrease production costs. This study provides a foundation for the further understanding and management of this disease to mitigate its negative consequences on barley crops.

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