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VIRULENCE OF *PYRENOPHORA TRITICI-REPENTIS*: A MINIREVIEW

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Abstract
Pyrenophora tritici-repentis is a major wheat pathogen in all wheat (*Triticum* spp.) growing areas worldwide. Up to date, eight *P. tritici-repentis* races have been described based on chlorosis, necrosis, or both symptoms caused on race differential wheat genotypes: 'Glenlea', 6B662, 6B365, and 'Salamouni'. Symptom development on differential genotypes depends on the interaction of the pathogen's necrotrophic effectors named Ptr ToxA, Ptr ToxB, and Ptr ToxC with host susceptibility genes. Ptr ToxA is encoded by the single copy gene *ToxA* and induces necrosis on sensitive wheat cultivars. Ptr ToxB causes chlorosis and is encoded by the multicopy gene *ToxB*. The Ptr ToxC is the non-proteinaceous, polar, low molecular mass molecule that also induces chlorosis, but up to date, the gene encoding this toxin is unknown. Races producing Ptr ToxA are predominant in the global *Ptr* population. There are several reports about new putative races of *P. tritici-repentis* that do not conform with the current race system, so further research is required. This study aims to collect and systematise available information about the virulence and races of *P. tritici-repentis.*
Key words: races, necrotrophic effectors, population, tan spot, wheat.

Introduction

Tan spot caused by *Pyrenophora tritici-repentis* (*Ptr*), from the phylum *Ascomycota* order *Pleosporales* is a notable wheat foliar disease in all major wheat (*Triticum* spp.) growing regions (Ciuffetti *et al.*, 2014; Strelkov & Lamari, 2003). It is one of the most devastating wheat diseases in Latvia and Lithuania (Ronis *et al.*, 2009; Švarta *et al.*, 2020).

Besides wheat, triticale (× *Triticosecale*), rye (*Secale cereale*), barley (*Hordeum vulgare*), also various grasses from *Poaceae* family are known as host plants of *Ptr* (Ali & Francl, 2003; Hosford, 1971; Krupinsky, 1982; Misra, Pandey, & Misra, 1979).

The first who described and isolated the asexual stage of this pathogen from *Elymus repens* was Diedicke in Germany in 1902 (Diedicke, 1902). Diedicke classified the newly found fungus as *Helminthosporium graminearum* Rab. ex Schlecht. f. sp. *tritici-repentis* Died., with *Pleospora trichostoma* (Fr.) Fuckel. as its teleomorph. In 1903, Diedicke renamed this species as *Helminthosporium tritici-repentis* Died. and *Pleospora tritici-repentis* Died. Later, in 1923, Drechsler classified it as *Pyrenophora tritici-repentis* (Died.) Drechs. based on setae presence on the fruiting body (pseudothecia). In 1928, the fungus asexual stage was isolated from wheat and identified as *Helminthosporium tritici-vulgaris* (Nisikado) by Nisikado. In 1930, Ito, based on conidia germination peculiarities, renamed *H. tritici-vulgaris* as *Drechslera tritici-vulgaris* (Hosford, 1981; Maraite, 1997). All synonyms for both morphs were revised by Shoemaker (1962) as *Pyrenophora tritici-repentis* (Died.) Drechs. for sexual state and *Drecshlera tritici-repentis* (Died.) Shoem. for the asexual state (Hosford, 1981; Maraite, 1997). In 2014, the scientific community supported using the genus name *Pyrenophora* over *Drechslera*, thus adopting *Pyrenophora tritici-repentis* (Died.) Drechs. as a sole name (holomorph) of the pathogen (Wijayawardene *et al.*, 2014).

Up to date 8 different Ptr races have been described, based on necrotrophic effectors (NEs) they produce and symptoms they cause on defined host-plant differential set (Lamari *et al.*, 2003). These NEs are called Ptr ToxA, Ptr ToxB, and Ptr ToxC and can cause two different symptoms on the host plants – necrosis and/or chlorosis (Lamari & Bernier, 1989).

The diversity of the pathogen's population is related to different aspects of disease development and control possibilities, such as overcoming cultivar resistance and sensitivity to fungicides. This study aims to collect and organise available information about *Ptr* populations to understand wheat-pathogen interactions better and gain knowledge for the adaptation of plant protection strategy.

Materials and Methods

In the present study a monographic approach was used. The results of worldwide research on the taxonomy, biology and population diversity focusing on the virulence of *Ptr* were summarised and analysed.

Results and Discussion

Races of P. tritici-repentis

Ptr can cause two different types of symptoms – necrosis and chlorosis. Lamari and Bernier (1989) proposed to group *Ptr* isolates into four pathotypes based on the expressed symptoms on a set of race-differential wheat genotypes. Initially, isolates belonging to pathotype 1 were thought to cause either extensive necrosis or extensive chlorosis (nec^+ chl^+),

until Lamari, Bernier and Smith (1991) reported that wheat genotypes are able to express both necrosis and chlorosis simultaneously. Isolates that induced only necrosis were grouped into pathotype 2 (nec^+ chl^-), while isolates that caused only chlorosis were considered pathotype 3 (nec^- chl^+). Avirulent isolates were classified as pathotype 4 (nec^- chl^-).

Sorting *Ptr* isolates into four pathotypes proved insufficient as Lamari *et al.* (1995) found chlorosis (nec^- chl^+) inducing isolates from Algeria, which had a different virulence pattern from previously described pathotype 3. These isolates were virulent on the wheat cultivar 'Katepwa' and avirulent on the line 6B365, which was directly opposite to pathotype 3, which caused chlorosis on 6B635 and was avirulent on 'Katepwa'. Therefore, Lamari *et al.* (1995) proposed to use race designation to specify *Ptr* virulence (Table 1). Races 1, 2, 3 and 4 represent the formerly described four pathotypes; the newly characterised isolates were assigned as race 5. Race 6 combines the virulence patterns of races 3 and 5, as it causes extensive chlorosis on both 'Katepwa' and 6B635. Likewise, race 5 induces chlorosis on wheat line 6B662 that is resistant to the first four races (Strelkov *et al.*, 2002). Race 7 causes necrosis on 'Glenlea' and chlorosis on 6B662, combining the virulence patterns of races 2 and 5. Race 8 induces necrosis on 'Glenlea', and chlorosis on 6B662 and 6B365, combining the virulence of races 2, 5, and 3 (Lamari *et al.*, 2003; Lamari *et al.*, 2005).

Virulence factors of P. tritici-repentis

Necrotrophic effectors determine virulence factors of the known Ptr races (NEs), historically called host-selective toxins (HSTs) (Ciuffetti *et al.*, 2010; Tomas & Bockus, 1987). The set of race differential wheat genotypes defined by Lamari *et al.* (2003) is based on the symptoms expressed by these genotypes in response to the infection by a specific race of the pathogen (Table 1). Previous research (Ballance, Lamari, & Bernier, 1989; Effertz *et al.*, 2002; Orolaza, Lamari, & Ballance, 1995) revealed that these reactions follow an inverse-gene-for-gene model, as wheat sensitivity to the specific NEs produced by *Ptr* is determined by compatibility between NEs and host susceptibility genes, while insensitivity is observed if the host and NEs are incompatible (Lamari *et al.*, 2003). Ciuffetti *et al.* (1998) published a standardised nomenclature for *Ptr* virulence factors and genes involved to resolve problems arising from the multiple names for the same necrotrophic effector and the regulating gene. NEs are named as Ptr ToxA, Ptr ToxB, and Ptr ToxC in the order of their characterisation. The genes are named according to the NEs and encode *ToxA*, *ToxB*, *ToxC*, respectively (Ciuffetti *et al.*, 2014).

Ptr ToxA produced by races 1, 2, 7 and 8 induces necrosis on susceptible wheat cultivars (Lamari & Bernier, 1989; Lamari *et al.*, 2003). Ptr ToxA is a protein that is synthesized ribosomally and is encoded by a single *ToxA* gene. ToxA encodes a 23 amino acid signal peptide, a 38 amino acid (4.3 kDa) pro-domain

Table 1

The reaction of differential wheat genotypes to the known races of *Pyrenophora tritici-repentis*

Race[a] (ToxA, ToxB, ToxC)	Reported by	Hexaploid genotypes					Tetraploid genotypes		
		Glenlea	6B662	6B365	Katepwa	Salamouni	ND495	Coulter	4B1169
1 (+,–,+)	(Lamari & Bernier, 1989) (Ali & Francl, 2002)	S(N)[b]	R	S(C)	S(N)	R	S(N)	S(N)	R
2 (+,–,–)		S(N)	R	R	S(N)	R	S(N)	S(N)	R
3 (–,–,+)		R	R	S(C)	R	R	R	S(N)	R
4 (–,–,–)		R	R	R	R	R	R	R	R
5 (–,+,–)	(Lamari *et al.*, 1995) (Ali & Francl, 2002)	R	S(C)	R	S(C)	R	R	S(N)	R
6 (–,+,+)	(Strelkov *et al.*, 2002) (Ali & Francl, 2002)	R	S(C)	S(C)	S(C)	R	-	S(N)	R
7 (+,+,–)	(Lamari *et al.*, 2003) (Ali & Francl, 2002)	S(N)	S(C)	R	S(N)	R	S(N/C)	S(N)	R
8 (+,+,+)		S(N)	S(C)	S(C)	S(N)	R	S(N)	S(N)	R

[a] Races are characterised by their production of three NEs - Ptr ToxA, Ptr ToxB, and Ptr ToxC.

[b] S – susceptible; R – resistant; (N) – necrosis; (C) – chlorosis; (N/C) – necrosis and chlorosis; - – symptoms are unknown.

Table 2

Geographic distribution of *P. tritici-repentis* races and their proportion (%) in analysed populations

Country	Race, proportion (%)							
	1	2	3	4	5	6	7	8
USA	40-84	1-4	32	13-32	1	0	0	0
Canada	62-90	36-50	2	0	0	0	0	0
Argentina	2	6	1.5	22	2	2	0	20
Brazil	35	65	0	0	0	0	0	0
Algeria	41	0	0	2	5	4	40	2
Morocco	6	0	0	0	47	44	2	0
Tunisia	0	1	0	1	4	0	49	0
Iran	90	10	0	0	0	0	0	0
Kazakhstan	46-95	5-35	0-23	0	0	0	0	0-34
Azerbaijan	64	3	1	0	13	0	8	11
Kyrgyzstan	100	0	0	0	0	0	0	0
Uzbekistan	100	0	0	0	0	0	0	0
Syria	8	0	58	0	8	0	17	8
Russia	13-23	0-45	0-33	5-61	0	0	0-5	5-13
Finland	19	19	4	10	0	10	0	38
Romania	80	16	2	0	0	0	0	0
Lithuania	48	1	20	5	0	0	0	0
Latvia	90	10	0	0	0	0	0	0

peptide responsible for proper protein folding. Both regions are cut off before synthesizing the final 13.2 kDa (118 amino acid) Ptr ToxA protein (Balance *et al.*, 1996; Ciuffetti *et al.*, 2010; Tomas *et al.*, 1990; Tuori, Wolpert, & Ciuffetti, 1995). Ptr ToxA activity is determined by solvent-exposed loops containing arginyl-glycyl-aspartic acid (RGD) (Sarma *et al.*, 2005). Manning *et al.* (2008) reported that a RGD loop is necessary for Ptr ToxA activity, receptor recognition and internalization. The internalization mechanism of Ptr ToxA is unknown. One of the hypotheses is that Ptr ToxA is uptaken via receptor-mediated endocytosis (Ciuffetti *et al.*, 2014). Chloroplasts are the localization targets of internalized Ptr ToxA, and cell death induced by Ptr ToxA is light-dependent. Ptr ToxA causes the accumulation of reactive oxygen species (ROS), which correlates with the induction of necrosis symptoms. Necrosis is prevented by inhibiting ROS accumulation (Manning *et al.*, 2009; Manning & Ciuffetti, 2005).

Races 5, 6, 7 and 8 produce Ptr ToxB (Lamari *et al.*, 2003; Strelkov *et al.*, 2002). Orolaza, Lamari, and Ballance (1995) were the first who identified Ptr ToxB in culture filtrates of *Ptr*. Ptr ToxB is a small protein of 6.6 kDa mass known to induce chlorosis on sensitive wheat cultivars by a mechanism that affects chlorophyll photooxidation (Strelkov, Lamari, & Ballance, 1998; 1999). Depending on each race and isolate Ptr ToxB coding, *ToxB* is found in multiple copies varying from 2 to 10 copies (Lamari *et al.*, 2003; Martinez, Oesch, & Ciuffetti, 2004). Copy number of *ToxB* correlates with Ptr ToxB expression level and isolate virulence (Amaike *et al.*, 2008; Strelkov *et al.*, 2002; 2006). *Ptr* race 4 has a single copy of the gene *toxb* that is 86% similar to *ToxB* gene and codes a protein that is 81% similar to Ptr ToxB (Martinez, Oesch, & Ciuffetti, 2004) and is avirulent in Ptr ToxB sensitive wheat genotypes (Figueroa Betts *et al.*, 2011). There is evidence that besides chlorosis induction, Ptr ToxB has additional functions connected to basic pathogenic abilities (Aboukhaddour, Kim, & Strelkov, 2012; Amaike *et al.*, 2008).

Ptr ToxC is produced by races 1, 3, 6 and 8 (Gamba, Lamari, & Brülé-Babel, 1998; Lamari *et al.*, 2003; Strelkov *et al.*, 2002). Ptr ToxC is a non-proteinaceous, low molecular mass, non-ionic, polar molecule (Effertz *et al.*, 2002). Like Ptr ToxB, it can cause chlorosis on susceptible hexaploid wheat genotypes while causing necrosis on susceptible tetraploid wheat lines (Lamari *et al.*, 1995). To date, there are no data about which gene encodes Ptr ToxC production.

Geographic distribution of P. tritici-repentis races

The population of *Ptr* is plastic and adapts to various climatic and agroecological conditions. Studies show that the *Ptr* population varies depending on the geographic location and host diversity (Table

Table 3

The reaction of differential wheat genotypes to the potentially new *P. tritici-repentis* races

Strain (ToxA, ToxB, ToxC)	Reported by	Hexaploid genotypes					Tetraploid genotypes		
		Glenlea	6B662	6B365	Katepwa	Salamouni	ND495	Coulter	4B1169
nc1(–,–,–)[a]	(Ali *et al.*, 2002)	S(N)[b]	×	S(N)	S(N)	S(N)	S(N)	×	×
SO3(–,–,–)	(Andrie, Pandelova, & Ciuffetti, 2007)	S(N)	R	R	S(N)	R	×	×	×
PT82(+,–,+)		S(N)	S(C)	S(C)	S(C)	R	×	×	×
AR CrossA5 (–,–,+)	(Ali, Gurung, & Adhikari, 2010)	S(N)	R	S(C)	×	R	×	×	×
AR LonB2 (–,–,+)		R	R	S(C)	S(C)	R	×	×	×
Ptr24(–,–,–)	(Benslimane *et al.*, 2011)	R	R	R	×	R	×	S(N)	R
A029(+,–,–)	(Moreno, Stenglein, & Perelló, 2015)	S(N/C)	R	R	R	R	×	×	×
B028(+,–,–)		R	R	R	R	R	×	×	×
CH007(+,–,–)		S(C)	R	R	S(N)	R	×	×	×
CH009(–,–,–)		R	R	R	R	S(N)	×	×	×
CP021(+,–,–)		S(N/C)	R	R	S(N)	R	×	×	×
CR0819(+,–,+)		R	R	S(C)	R	R	×	×	×
G032(–,–,–)		S(N)	R	R	R	R	×	×	×
G0316(+,–,+)		S(N)	R	S(C)	R	R	×	×	×
G0321(–,–,+)		R	R	S(C)	R	S(C)	×	×	×
G0328(+,–,+)		S(N)	S(C)	S(C)	R	R	×	×	×
G0333(+,–,–)		R	R	R	S(N)	R	×	×	×
H001(+,–,–)		S(N)	S(C)	S(C)	S(N/C)	S(N)	×	×	×
H0014(+,–,–)		S(C)	R	S(C)	R	R	×	×	×
H016(+,–,–)		S(N)	S(C)	S(C)	R	S(C)	×	×	×
25M031(+,–,–)		R	R	R	S(N/C)	R	×	×	×
25M036(+,–,–)		R	R	S(C)	S(N)	R	×	×	×
O0019(+,–,–)		S(N)	S(C)	R	S(N/C)	S(C)	×	×	×
P028(+,–,–)		S(N/C)	S(C)	S(C)	R	R	×	×	×
nc2(–,–,+)	(Abdullah *et al.*, 2017)	S(N)	R	S(C)	×	R	×	×	×
nc3(–,–,–)		S(N)	R	R	×	R	×	×	×
B16 (–,–,–)	(Guo, Shi, & Liu, 2018)	S(N)	R	R	×	R	S(N)	×	×
T128-1(–,+,–)	(Kamel *et al.*, 2019)	S(N)	S(C)	R	×	R	×	×	×

[a] if the name of the strain is not stated in the publication, then the code nc (not conform) is assigned.

[b] S – susceptible; R – resistant; (N) – necrosis; (C) – chlorosis; (N/C) – necrosis and chlorosis; × – symptoms were not investigated;

2). In Canada, races 1 and 2 are predominant in the local *Ptr* population (Aboukhaddour, Turkington, & Strelkov, 2013).

In Argentina, Moreno, Stenglein and Perelló (2015) it was reported that only 54% could be assigned to the eight known races, with races 4 and 8 being the

most widespread. Ptr race 2 is predominant in Brazil (Bertagnolli *et al.*, 2019). These findings contradict the earlier report by Ali and Francl (2002), which indicated that race 1 is the most dominant Ptr race in South America.

Races 1 and 7 formed most of the Algerian *Ptr* population, while races 4, 5 and 6 were rarely observed. (Benslimane *et al.*, 2011). The situation is similar in Tunisia, where, according to Kamel *et al.* (2019), race 7 is predominant, and races 2, 4 and 5 are in the minority. In Morocco, races 5 and 6 are the most typical representatives of the population (Gamba, Bassi, & Finckh, 2017).

The *Ptr* population in Iran is dominated by race 1 (Momeni *et al.*, 2014). Race 1 also is predominant in Kazakhstan, and in some sites, race 8 was found the second most widespread *Ptr* race (Молдажанова, Мауленбай, & Рсалиев, 2020). Lamari *et al.* (2005) analysed *Ptr* race diversity near the centre of wheat origin – the Fertile Crescent and the Caucasus – and found that the most diverse *Ptr* populations are in Syria and Azerbaijan.

Race 4 is predominant in southern Russia, and race 1 is the most widespread in northern Russia. Races 1, 2, 3 and 4 are commonly seen in the western part of Russia, with no race being distinctively predominant. The *Ptr* population of Finland is dominated by race 8 (Мироненко, Коваленко, & Баранова, 2019).

In Europe and the Baltic states, the diversity of the *Ptr* population is insufficiently studied. Abdullah *et al.* (2017) reported that in Lithuania and Latvia, the most common race is race 1. They also observed that in Romania, similarly to the Baltic states, race 1 is predominant. More detailed research is necessary.

Indications of new P. tritici-repentis races and their geographic distribution

Some researchers have reported about possible new *Ptr* races with different virulence patterns (Table 3). Ali *et al.* (2002) reported a putative necrotrophic effector that caused extensive chlorosis on the tetraploid wheat line ND495, which is insensitive to Ptr ToxC. This NE was similar in size to Ptr ToxC, but had distinct chemical properties. Pandelova and Ciuffetti (2005) identified a proteinaceous toxin in the *Ptr* strains lacking *ToxA* gene that induced the same symptoms on the wheat differential set as Ptr ToxA. The current race system can describe only eight races, so discovering the new NEs like Ptr ToxD, Ptr ToxE, etc., and/or new differentials is necessary to incorporate new races in the existing model (Lamari & Strelkov, 2010).

Conclusions

1. Races capable of producing Ptr ToxA are predominant in the global *Ptr* population, and possibly this is the result of the selective pressure from cultivars used in modern agriculture.
2. Various researchers worldwide have reported new *Ptr* races, but further research is needed as the key elements necessary for the description of these races have not yet been found.
3. There are many regions where the diversity of the *Ptr* population is not sufficiently investigated, and these knowledge gaps should be filled to gain a better understanding of the global *Ptr* population and wheat-pathogen interaction mechanisms.

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