Phytopathol. Mediterr. (2011) 50, 203–211

Distribution of races of *Pyrenophora tritici-repentis* in Algeria and identication of a new virulence type

Hamida BENSLIMANE[1], Lakhder LAMARI[2], Abdelkader BENBELKACEM[3], Rachid SAYOUD[4] and Zouaoui BOUZNAD[5]

[1]Université M'hamed Bougra, Boumerdès, Faculté des Science, Département de Biologie, Laboratoire de Biologie Moléculaire, 16 Rue de l'indépendance, Boumerdès, Algérie
[2]University of Manitoba, Department of Plant Sciences, Winnipeg, Manitoba, Canada, RT3 2N2
[3]Institut National de la Recherche Agronomique d'Algérie, Unité de Recherche de Constantine, Station ITGC, Elkhroub, Algérie
[4]SYNGENTA, 5 chemin des citernes Skikda, Algérie
[5]Ecole Nationale Supérieure d'Agronomie, Département de Botanique, Laboratoire de Phytopathologie et Biologie Moléculaire, El-Harrach, Alger, Algérie

Summary. Tan spot, caused by *Pyrenophora tritici-repentis*, is a foliar disease of wheat, responsible for high economic losses in several wheat growing areas in the world. There are eight known races of *P. tritici-repentis* based on ability to induce necrosis and/or chlorosis on a set of differential cultivars. Fifty five isolates of *P. tritici-repentis* originating from diverse wheat growing regions in Algeria were studied to determine which races are present and to identify new races. Races 1, 4, 5, 6, 7 and 8 were found and a new virulence pattern was identified. Isolates with this pattern induced necrosis in durum wheat but failed to induce any disease in the common wheat genotypes in the differential set.

Key words: tan spot, wheat, virulence, isolate, race.

Introduction

Pyrenophora tritici-repentis (Died.) Drechs. (anamorph *Drechslera tritici-repentis* [Died.] Shoem.), the causal agent of tan spot of wheat, is a destructive fungal pathogen which occurs throughout the major wheat growing regions worldwide (Hosford, 1982; Singh *et al*., 2010). The pathogen can attack durum wheat (*Triticum durum* Desf.) and bread wheat (*Triticum aestivum* L.) and other grass species. Yield losses as high as 49% were reported for susceptible wheat, when conditions favoured disease development (Ress *et al.,* 1982).

The tan spot disease syndrome consists of two distinct symptoms: necrosis and chlorosis. Independent genes in host plants control resistance to both the symtoms (Lamari and Bernier, 1991). The wheat-*P. tritici-repentis* system conforms to the toxin model of gene-for-gene hypothesis, in which compatibility results from an interaction between a pathogen-produced toxin and its putative receptor in the host. Two host specific toxins (Ptr ToxA and Ptr ToxB) were well characterized, and their respective encoding genes were cloned (Balance *et al*., 1989; Strelkov *et al*., 1999; Tomas *et al*., 1990; Tuori *et al*., 1995; Zhang *et al*., 1997). The third toxin (Ptr ToxC) has been purified and partially characterized (Effertz *et al*., 2002). Initially, isolates of *P. tritici-repentis* were classified into pathotypes based on their ability to induce necrosis and/or chlorosis. However, since different virulences were observed within the pathotype based classification, Lamari *et al*. (1995) introduced a race classification system to group isolates on the basis of their virulence on individual host differential genotypes. According to this classifica-

Corresponding author: H. Benslimane
Fax: +213 (0) 21391264
E-mail: benslimh@yahoo.fr

tion, eight races of the pathogen have been identified (Lamari *et al*., 2003; Lamari and Strelkov, 2010). The advantage of this classification system is that the number of races that can be accommodated is limited only by the size and effectiveness of the wheat differential set.

According to Lamari and Strelkov (2010), races 5 and 6 are present in Algeria, where tan spot has become one of the fastest growing disease problems (Benslimane *et al*., 2006). However, little is known about proportion and distribution of different races of the pathogen in this country. Breeding resistant wheat cultivars in combination with appropriate crop rotation seems to be the best option for managing this disease. Therefore, knowledge of the structure of pathogen populations is essential for an efficient breeding approach to utilize host resistance.

In this study, 55 isolates of *P. tritici-repentis* were identified and races characterized for the purpose of determining which races are present in different cereal growing areas in Algeria as well as searching for any new races that may be present in this country.

Materials and methods

Fungal isolates

Single conidia derived 55 isolates of *P. tritici-repentis* used in this study. These isolates were identified and isolated from infected wheat leaves placed in a moist chamber. The original isolates were obtained from several infected wheat fields (*T. aestivum* and *T. durum*) in different cereal growing areas in Algeria (Table 1). Three isolates (Asc1, 86-124, Alg-3-24) corresponding, respectively, to three known races (1, 2, and 5) and provided by late Dr. L. Lamari, University of Manitoba, Winipeg, Canada, were used as controls.

Plant material

The virulence of the 55 isolates was tested on a set of differential wheat genotypes to determine their race classification. The seven wheat lines/cultivars used were selected on the basis of their reaction to the eight known races of *P. tritici-repentis* (Table 2) (Lamari and Bernier, 1989; Strelkov *et al*., 2002; Lamari *et al*., 2003). The differential set was made up of four common wheats (6B-365, Glenlea, 6B-662 and Salamouni) and three durum wheats (4B-160, Coulter, and 4B-1149).

Wheat seeds were planted in clay pots (15 cm diam.) each containing soil mix (2:1:1 soil, sand, peat). Seedlings were maintained in a growth room at 22:18°C (day:night) with a 16 h photoperiod. Plants were watered as required. Two genotypes were planted per pot in separated clumps of four seeds each. At the two leaf stage, three plants per differential genotype were retained for inoculation purposes. All treatments were replicated three times.

Inoculum production

Inoculation was produced following the protocols of Lamari and Bernier (1989) with few modifications. Small plugs, 0.5 cm in diameter were transferred singly to 9 cm Petri dishes containing V8-PDA medium (V8 150 mL, agar 10 g, PDA 10 g, $CaCO_3$ 3 g, H_2O 850 mL). Cultures were incubated in the dark for 5 days at 20°C until they grew to 4 cm in diameter. The cultures were then flooded with sterile distilled water, the mycelium flattened with the bottom of a flamed test tube, and excess water was decanted. Plates were placed under intense light for 18 h at room temperature (22°C), followed by 24 h at 15°C in the dark. Conidia were harvested by flooding the Petri dishes with sterile distilled water and dislodging the spores with a wire loop. The inoculum concentration was adjusted to 3,000 conidia mL^{-1} using a cell counter (Hausser Scientific Company). One drop of Tween 20 (polyoxyethylene sorbitan monolaurate) was added per 250 mL of conidia suspension.

Inoculation

The seedlings of each of the seven differential genotypes were sprayed at the two leaf stage until runoff with the conidial suspension. The plants were incubated for 24 h under continuous leaf wetness at 20°C and a 16 h photoperiod (Lamari and Bernier, 1989). Then they were transferred to a growth room and observed daily for symptom development.

Results and discussion

The control isolates (ASC1, 86-124, and Alg3-24) produced symptoms typical for the races they represent on the differential set. The 55 isolates

Table 1. Algerian *Pyrenophora tritici-repentis* isolates tested on seven wheat cultivars and lines.

Isolate	Location	Source
Ptr 1	Oued Otmania-Mila	Bread wheat
Ptr 2	Oued Aba- Ain Defla	Bread wheat
Ptr 4	Berboucha-Tipaza	Durum wheat
Ptr 7	Bouira	Mixture
Ptr 9	Cherchel-Tipaza	Durum wheat
Ptr 10	Oued Smar-Alger	Durum wheat
Ptr 11	Oued ElAlaig-Blida	Durum wheat
Ptr 16	Hamr El Ain-Tipaza	Bread wheat
Ptr 17	Mozaia-Blida	Bread wheat
Ptr 18	El-Harrach-Alger	Bread wheat
Ptr 21	El Kser-Bejaia	Durum wheat
Ptr 22	Guelma	Durum wheat
Ptr 23	Iaazougen-TiziOuezou	Durum wheat
Ptr 24	Laadjel Hela-Tipaza	Durum wheat
Ptr 25	Djendel-Ain Edefla	Durum wheat
Ptr 26	Maskara	Durum wheat
Ptr 36	El Khroub-Constantine	Durum wheat
Ptr 38	Berouaguia-Medea	Bread wheat
Ptr 39	Benihamiden-Constantine	Durum wheat
Ptr 42	BeniSliman-Medea	Bread wheat
Ptr 45	Gramem Gouda-Mila	Durum wheat
Ptr 46	Ain Defla	Durum wheat
Ptr 48	Bouira	Bread wheat
Ptr 53	Ain Sbaa- Bouira	Bread wheat
Ptr 55	Oued Smar-Alger	Bread wheat
Ptr 56	Oued Smar-Alger	Bread wheat
Ptr 61	Oued Elbared-Bouira	Bread wheat
Ptr 62	Ain Aloui-Bouira	Durum wheat
Ptr 63	Benselman-Bouira	Bread wheat
Ptr 64	El Hachimia-Bouira	Durum wheat
Ptr 65	Said Abid-Bouira	Durum wheat
Ptr 67	Tipaza	Durum wheat
Ptr 68	Tipaza	Durum wheat
Ptr 69	Area1-Boumerdès	Durum wheat
Ptr 72	Area4-Boumerdès	Durum wheat
Ptr 75	Hamr El Ain-Boumerdès	Durum wheat
Ptr 76	Hamr El Ain-Boumerdès	Durum wheat
Ptr 77	Oued Smar- Alger	Bread wheat
Ptr 78	Oued Smar- Alger	Bread wheat
Ptr 79	Oued Smar- Alger	Bread wheat
Ptr 80	Ain Bessam-Bouira	Bread wheat
Ptr 81	Ain Bessam-Bouira	Bread wheat
Ptr 82	Ain Bessam-Bouira	Bread wheat
NA-3-1	R. Djamel- East region	Bread wheat
NA-8-2	Anaba	Bread wheat
NA 8-3	Anaba	Bread whet
NA 7-1	R.Djamel-East region	Durum wheat
NA 3-3	R.Djamel	Durum wheat
NA 2-1	East region	Durum wheat
NA-6-1	El-Hassar	Durum wheat
NA 1-3	R.Djamel- East region	Durum wheat
NA 7-4	R.Djamel-East region	Durum wheat
NA 7-2	R.Djamel-East region	Durum wheat
NA 7-3	R.Djamel-East region	Durum wheat
NA 4-2	East region	Durum wheat

Table 2. Reaction of a set of differential wheat genotypes to the races of *Pyrenophora tritici-repentis*.

Genotype	Race[a]							
	R1	R2	R3	R4	R5	R6	R7	R8
Glenlea	S-N	S-N	R	R	R	R	S-N	S-N
6B-662	R	R	R	R	S-C/b	S-C/b	S-C/b	S-C/b
6B-365	S-C/c	R	S-C/c	R	R	S-C/c	R	S-C/c
Salamouni	R	R	R	R	R	R	R	R
4B-160	S-C	S-N	S-N	R	S-N	S-N	S-N	S-N
Coulter	S-N	S-N	S-N	R	S-N	S-N	S-N	S-N
4B-1149	R	R	R	R	R	R	R	R

[a] S-N, sensitive necrosis; S-C_b, sensitive chlorosis induced by Ptr ToxB toxin; S-C_c, sensitive chlorosis induced by Ptr ToxC toxin; R, resistance.

of *P. tritici-repentis* evaluated were grouped into six races (1, 4, 5, 6, 7, and 8) on the basis of their virulence on individual host differential genotypes (Table 3). The distribution of the different races in Algeria is shown in Figure 1.

Isolates belonging to each race were collected both from bread wheat and durum wheat. However most isolates grouped as race 1 originated from durum wheat. The majority of isolates recovered from the samples represented races 1 and 7, while races 4, 5, 6, and 8 were more rare.

Races 1 and 7 comprised 41 and 40%, respectively, of all the isolates tested. Race 1 was obtained from all regions; it was characterized by ability to induce extensive chlorosis on line 6B-365 and 4B-160, as well as necrosis on cv. Glenlea and

Table 3. Reaction of seven wheat differential lines/cultivars to the 55 isolates of *Pyrenophora tritici-repentis* and race determination.

Isolate	Genotype[a]							
	Glenlea	6B-662	6B-365	Salamouni	4B-160	Coulter	4B-1149	Races
Ptr1	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr2	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr4	S-N	S-C_b	R	R	S-N	S-N	R	7
Ptr7	S-N	S-C_b	S-C_c	R	S-N	S-N	R	8
Ptr9	S-N	S-C_b	R	R	S-N	S-N	R	7
Ptr10	S-N	S-C_b	R	R	S-N	S-N	R	7
Ptr11	S-N	S-C_b	R	R	S-N	S-N	R	7
Ptr16	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr17	S-N	S-C_b	R	R	S-N	S-N	R	7
Ptr18	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr21	S-N	S-C_b	R	R	S-N	S-N	R	7
Ptr22	R	S-C_b	R	R	S-N	S-N	R	5
Ptr23	S-N	S-C_b	R	R	S-N	S-N	R	7
Ptr24	R	R	R	R	S-N	S-N	R	*
Ptr25	R	S- C_b	S-C_c	R	S-N	S-N	R	6
Ptr26	S-N	S-C_b	R	R	S-N	S-N	R	7

continues

Table 3. *continued*

Isolate	Genotype[a]							
	Glenlea	6B-662	6B-365	Salamouni	4B-160	Coulter	4B-1149	Races
Ptr36	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr38	S-N	SC/b	R	R	S-N	S-N	R	7
Ptr39	R	R	R	R	R	R	R	4
Ptr42	S-N	R	S-Cc	R	S-C	S-N	R	1
Ptr45	R	S-C_b	R	R	S-N	S-N	R	5
Ptr46	S-N	S-C_b	R	R	S-N	S-N	R	7
Ptr48	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr53	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr55	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr56	S-N	R	S-Cc	R	S-C	S-N	R	1
Ptr61	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr62	S-N	S-C_b	R	R	S-N	S-N	R	7
Ptr63	S-N	R	S-Cc	R	S-C	S-N	R	1
Ptr64	S-N	S-C_b	R	R	S-N	S-N	R	7
Ptr65	R	R	R	R	S-N	S-N	R	*
Ptr67	S-N	S-C_b	R	R	S-N	S-N	R	7
Ptr68	R	R	R	R	S-N	S-N	R	*
Ptr69	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr72	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr75	R	S-C_b	S-Cc	R	S-N	S-N	R	6
Ptr76	R	R	R	R	S-N	S-N	R	*
Ptr77	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr78	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr79	S-N	R	S-C_c	R	S-C	S-N	R	1
Ptr80	S-N	S-C_b	R	R	S-N	S-N	R	7
Ptr81	S-N	S-C_b	R	R	S-N	S-N	R	7
Ptr82	S-N	S-C_b	R	R	S-N	S-N	R	7
NA 3-1	S-N	R	S-C/c	R	S-N	S-N	R	1
NA 8-2	S-N	R	S-C/c	R	S-N	S-N	R	1
NA 8-3	S-N	R	S-C/c	R	S-N	S-N	R	1
NA 7-1	R	S-C/b	R	R	S-N	S-N	R	5
NA 3-3	S-N	R	S-C/c	R	S-N	S-N	R	1
NA 2-1	S-N	R	S-C/c	R	S-N	S-N	R	1
NA 6-1	S-N	S-C/b	R	R	S-N	S-N	R	7
NA 1-3	S-N	R	S-C/c	R	S-N	S-N	R	1
NA 7-4	S-N	S-C/b	R	R	S-N	S-N	R	7
NA 4-2	R	R	R	R	S-N	S-N	R	*
NA 7-2	S-N	S-C/b	R	R	S-N	S-N	R	7
NA 7-3	S-N	S-C/b	R	R	S-N	S-N	R	7

[a] See Table 2.
* New race.

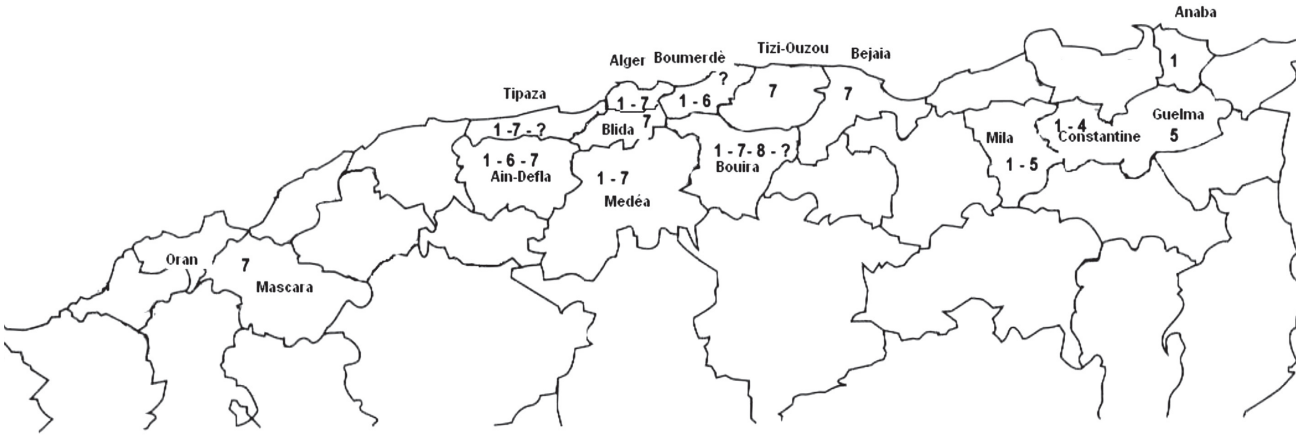

Figure 1. Distribution of races of *Pyrenophor tritici-repentis* in wheat growing regions in Algeria. 1, Race 1; 4, Race 4; 5, Race 5; 6, Race 6; 7, Race 7; 8, Race 8; ? = unknown race (new race).

Coulter. Race 7 was rare inside of the eastern region of Algeria. It was characterized by ability to induce chlorosis on line 6B-662 and necrosis on cv. Glenlea and Coulter, and line 4B-160. Races 4 and 5 were found only in the eastern regions, race 6 in the western and central regions and race 8 only in the central areas. Race 4 was represented by a single isolate; it differed from other races by its inability to produce chlorosis or necrosis on the set of differential wheat genotypes. All cultivars and lines were resistant to race 4. Race 5 comprised 5% of all isolates tested, and it was characterised by ability to induce chlorosis on line 6B-662 and necrosis on Coulter and 4B-160. Race 6 comprised 4% of all isolates tested, and it was characterised by ability to induce chlorosis on both lines 6B-662 and 6B-365 and necrosis on Coulter and 4B-160. Finally race 8 was represented by a single isolate; it was characterised by ability to induce necrosis on Glenlea, Coulter and 4B-160 and chlorosis on both lines 6B-662 and 6B365.

Race 1 induced similar disease reactions in both durum and bread wheat; indeed, Glenlea (bread wheat) and Coulter (durum wheat) developed necrosis. In addition, 6B-365 which is a bread wheat, and 4B-160, which is a durum wheat, developed extensive chlorosis. However, race 5 caused chlorosis in bread wheat but necrosis in durum. This observation coincides with that of Gamba *et al*. (1998). It was also observed that most of the isolates identified as race 1 came from bread wheat. However most of those identified as race 7 came from durum wheat. Aung (2001) suggested that host response could put a selection pressure on the *P.tritici-repentis* population.

The isolates Ptr65, Ptr68, Ptr24, Ptr76 and NA 4-2 all produced a novel virulence pattern. Indeed they were avirulent on the bread wheats; Glenlea, 6B-662, 6B-365, and Salamouni. However, they also infected the durum wheats, 4B-160 and Coulter (Figure 2). The sensitivity of these genotypes was expressed as necrosis, similar to that induced by race 1 on the cultivar Glenlea. Race 1 produced necrosis on Glenlea because its produces the Ptr ToxA toxin. In the case of Ptr65, Ptr68, Ptr24, Ptr76 and NA 4-2, the symptom cannot be the result of the same toxin, since Glenlea, which is sensitive to Ptr ToxA, remains resistant to these isolates. The five isolates obtained from durum wheat, could belong to a new race, able to attack only durum wheat and unable to attack bread wheat. The process of infection could be under the control of a new toxin responsible for the induction of necrosis on the sensitive hosts Coulter and 4B-160. Because this virulence pattern is reported for the first time, we propose that the pathogenicity pattern represented by isolates Ptr65, Ptr68, Ptr24, Ptr76 and NA 4-2 is characteristic of a new race of the pathogen.

Considerable success has been achieved in understanding wheat-*P. tritici-repentis* interactions in the last decades. Our results show there are in Algeria six races of *P. tritici-repentis;* races 1, 4, 5, 6, 7, and 8. Four of these races (1, 4, 7, and 8) are

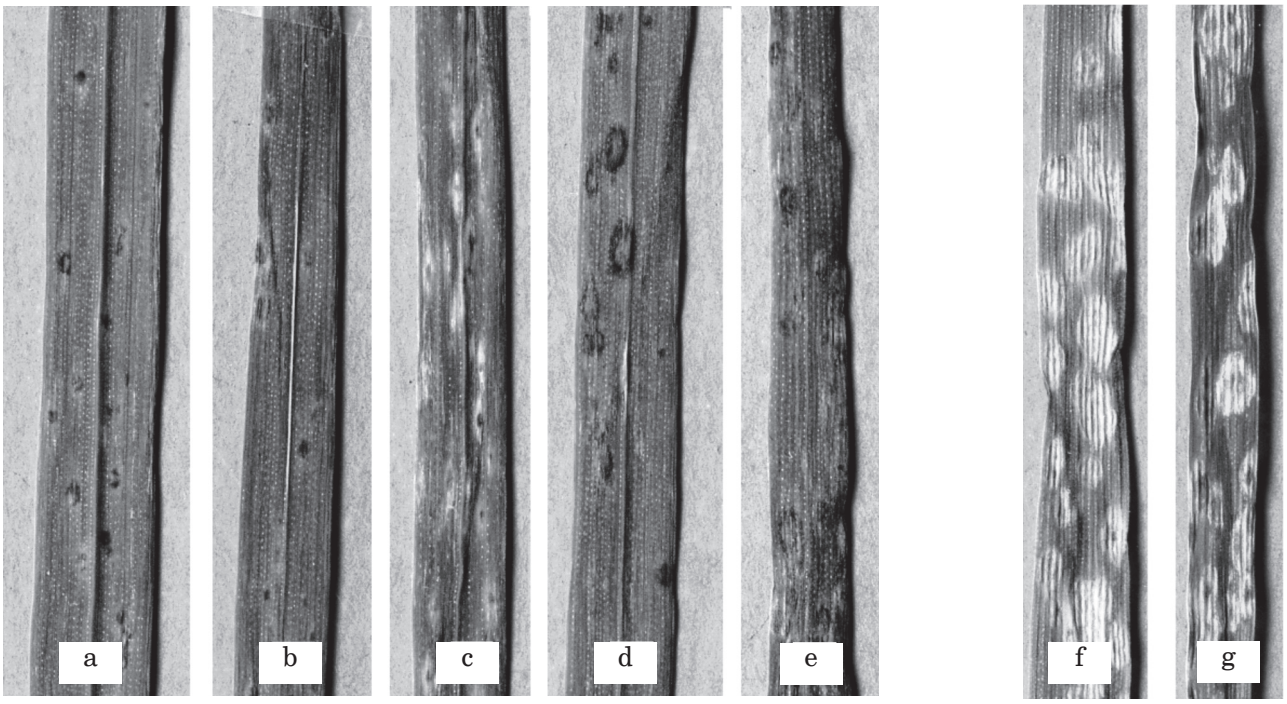

Figure 2. a–e. Resistance of Glenelea (a), 6B-365 (b), 6B-662 (c), Salamouni (d), 4B-1149 (e) to isolate Ptr 65, a new race of *Pyrenophora tritici-repentis*; note the presence of small brown to black spots on leaves. f–g. Sensitivity of Coulter (f) and 4B-140 (g) to Ptr65, new race of *Pyrenophora tritici-repentis*. Note lesions with tan necrosis.

described in Algeria for the first time. Indeed, race 1, which is usually predominant in Australia (Ali *et al*., 2006) and North America and is also present in the Middle-East and the Caucasus (Lamari *et al*., 1995; 1998) has not been reported previously in Algeria. Furthermore, our results indicate that this race is prevalent in Algerian wheat growing areas. This is also true for race 4 which was reported until now only in Canada and in United States (Lamari and Strelkov, 2010); however, this race is rather rare. This rarity was already reported in other regions of the world; for example, in Canada, a study concerning the evolution of races in Manitoba and Saskatchewan between 1990 and 1994, discovered presence of race 4 with a frequency of less than 1% (Lamari *et al*., 1998). In North Dakota, Ali and Francel (2003) reported that 5% of the isolates tested were grouped under race 4. Races 7 and 8 have been found previously only in the Middle-East and the Caucasus (Srelkov *et al*., 2002; Lamari *et al*., 2003; Lamari and Strelkov, 2010).

This study also, confirms the presence of races 5 and 6 already known in the Eastern regions of Algeria (Lamari and Strelkov, 2010). Their virulence patterns were highlighted for the first time from samples collected in the east of this country (Lamari *et al*., 1995; Strelkov *et al*., 2002). Race 5 was found later in the United State (Ali *et al*., 1999), in Canada (Strelkov *et al*., 2002), in Syria and Azerbaijan (Lamari *et al*., 2005). However, to date, race 6 has been found only in Algeria (Lamari and Strelkov, 2010).

In addition, five isolates from durum wheat showed a new virulence pattern. Glenlea, 6B-662 and 6B-365 were resistant to theses isolates, whereas Coulter and 4B-160 were sensitive. Glenlea harbours *Tsn1*, the gene controlling sensitivity to Ptr ToxA (races 1, 2, 7, and 8); wheat line 6B-365 harbours *Tsc1*, the gene controlling sensitivity to Ptr ToxC (races 1, 3, 6, and 8); and wheat line 6B-662 harbours *Tsc2*, the gene controlling sensitivity to Ptr ToxB (races 5, 6, 7, and 8) (Lamari *et al*., 1995; Strelkov *et al*., 2002; Lamari *et al*., 2003; Strelkov and Lamari, 2003; Friesen and Faris, 2004). This suggests that isolates with the new virulence pattern are not able to produce the three HTSs; PtrToxA, PtrToxB and PtrToxC produced by *P. tritici-repentis,* which are virulence deter-

minants that can distinguish races of the fungus (Lamari and Bernier, 1989; Orolaza *et al.*, 1995; Ciuffetti *et al.*, 1997; Effertz *et al.*, 2002). However, these isolates could possess a novel toxin(s) that enable them to induce necrotic symptoms on the durum wheat genotypes, Coulter and 4B-160. Indeed, Lamari *et al*. (2003) suggested that the wheat-*P. tritici-repentis* interaction conforms to the toxin model of the gene-for-gene hypothesis. The compatible interaction between host plants and pathogen leads to susceptibility, which is the result of the pathogen-produced toxin and its toxin receptor in the host at the molecular level.

This study reveals that the tan spot fungus is highly variable in Algeria and a new race has been identified. Lamari *et al*. (2003) hypothesised that more complex races were likely to be found in or near the centre of origin of wheat, which is by definition the region of greatest variability of a plant species (Harlan, 1987; Vavilov, 1951). The presence of these complex races in of *P. tritici-repentis* in Algeria is associated with the variability of the host; indeed, several wheat genotypes are grown in this country. Also, it is likely that the local wild host population and cultivation of plant land races in some areas exerted selection pressure to maintain the various pathogen virulence factors such as Ptr Tox A, Ptr ToxB and Ptr ToxC in high frequency.

Further investigation of the genetic structure of the pathogen and the host in wheat growing regions of Algeria is needed. Host-pathogen interaction studies backed by molecular mapping of the resistance are needed to confirm the existence of new races of *P. tritici-repentis*. These investigations should be of direct benefit to wheat breeding programmes aimed at incorporating resistance to this pathogen. Plant breeders require knowledge of what races are present in the pathogen population and where the cultivar is grown in order to effectively to deploy resistance genes and to determine which gene(s) should be present in resistant cultivars,. Hence, increased efforts should be made to assess and monitor the variability present in the fungus through regular pathongenicity and virulence studies.

Acknowledgments

This manuscript is in memoriam of Dr. Lamari L. and in homage to his outstanding research on tan spot of wheat. We thank Aouali S. and Khalfi A. (Institut Téchnique des Grandes Cultures, Algeirs, Algeria) for supplying several infected wheat samples.

Literature cited

Ali S. and L.J. Francl, 2003. Population race structure of *Pyrenophora tritici-repentis* prevalent on wheat and noncereal grasses in the Great Plains. *Plant Disease* 87, 418–422.

Ali S., L.J. Francl and E.D. De Wolf, 1999. First report of *Pyrenophora tritici-repentis* race 5 from North America. *Plant Disease* 83, 591.

Ali S., T. Adhikari, S. Barbara and H. Cole, 2006. *Pyrenophora tritici-repentis* (Tan spot) races in Australia. In: *2006 APS Annual Meeting, Abstracts of Presentations*. July 29- August 2, 2006, Quebec City, Canada. *Phytopathology* 96(6), S4.

Aung T.S.T., 2001. *Molecular Polymorphism and Virulence of* Pyrenophora tritici-repentis. MSc Thesis, University of Manitoba, Winnipeg, MB, Canada, 82 pp.

Balance G.M., L. Lamari and C.C. Bernier, 1989. Purification and characterization of a host selective necrosis toxin from *Pyrenophora tritici-repentis*. *Physiological and Molecular Plant Pathology* 35, 203–213.

Benslimane H., Z. Bouznad, S. Aouali, A. Khalfi, K. Benbelkacem and R. Sayoud, 2006. Prévalence en Algérie de la tache bronze du blé causée par *Pyrenophora tritici-repentis*. *6éme Journées Scientifiques et Techniques Phytosanitaires*, 20–21 juin 2006, El-Harrach, Alger, Algeria.

Ciuffetti L.M., R.P. Tuori and J.M. Gaventa, 1997. A single gene encodes a selective toxin causal to the development of tan spot of wheat. *Plant Cell* 9, 135–144.

Effertz R.J., S.W. Meinhardt, A.J. Anderson, J.G. Jordahl and L.J. Francl, 2002. Identification of a chlorosis-inducing toxin from *Pyrenophora tritici-repentis* and the chromosomal location of an insensitivity locus in wheat. *Phytopathology* 92, 527–533.

Friesen T.L and J.D. Faris, 2004. Molecular mapping of resistance to *Pyrenophora tritici-repentis* race 5 and sensitivity to Ptr ToxB in wheat. *Theoretical Applied Genetics* 109, 464–471.

Gamba F.M., L. Lamari and A. Brulé-Babel, 1998. Inheritance of race specific necrosis and chlorosis reaction induced by *Pyrenophora tritici-repentis* in hexaploid wheats. *Canadian Journal of Plant Pathology* 20, 401–407.

Harlan J.R., 1987. Center of origin. In: *CRC Handboock of Plant Science in Agriculture* Vol 1. B.R. Christie ed. CRC Press, Boca Rton, FL, USA, 15–21.

Hosford R.M., 1982. Tan spot-developing knowledge 1902–1981, virulence races and wheat differentials, methodology, rating systems, other leaf diseases, literature. In: *Tan Spot of Wheat and Related Diseases Workshop*. July 1981, Fargo, ND, USA. Edited by North Dakota Agricultural Experimental Station North Dakota State

University, Fargo, ND, USA, 1–24.

Lamari L. and C.C. Bernier, 1989. Evaluation of wheat lines and cultivars to tan spot [*Pyrenophora tritici-repentis*] based on lesion type. *Canadian Journal of Plant Pathology* 11, 49–56.

Lamari L. and C.C. Bernier, 1991. Genetics of tan necrosis and extensive chlorosis in tan spot of wheat caused by *Pyrenophora tritici-repentis*. *Phytopathology* 81, 1092–1095.

Lamari L. and S.E. Strelkov, 2010. The wheat-*Pyrenophora tritici-repentis* interaction: progress towards an understanding of tan spot disease. *Canadian Journal of Plant Pathology* 32, 4–10.

Lamari L., R. Sayoud, M. Boulif and C.C. Bernier, 1995. Identification of a new race In: *Pyrenophora tritici-repentis*: implication for the current pathotype classification system. *Canadian Journal of Plant Pathology* 17, 312–318.

Lamari, L., J. Gilbert and A. Tekauz, 1998. Race differentiation in *Pyrenophora tritici-repentis* and survey of physiologic variation in western Canada. *Canadian Journal of Plant Pathology* 20, 396–400.

Lamari L., S.E. Strelkov, A. Yahyaoui, J. Orabi and R.B. Smith, 2003. The identification of two new races of *Pyrenophora tritici-repentis* from the host centre of diversity confirms a one-to-one relationship in tan spot of wheat. *Phytopatholgy* 93, 391–396.

Lamari L., S.E. Strelkov, A. Yahyaoui, M. Amedov, M. Saidov, M. Djunusova and M. Koichbayev, 2005. Virulence of *Pyrenophora tritici-repentis* in the countries of the Silk Road. *Canadian Journal of Plant Pathology* 27, 383–388.

Orolaza, N. P., L., Lamari and G.M. Ballance, 1995. Evidence of a host-specific chlorosis toxin from *Pyrenophora tritici-repentis,* the causal agent of tan spot of wheat. *Phytopathology* 85, 1282–1287.

Rees R.G., G.J. Platz, and R.J. Mayer, 1982. Yield losses in wheat from yellow spot: Comparison of estimates derived from single tillers and plots. *Australian Journal Agricultural Research* 33, 899–908.

Singh P.K, R.P. Singh, E. Duveiller, M. Mergoum, T.B. Adhikari, E.M. Elias, 2010. Genetics of wheat–*Pyrenophora tritici-repentis* interactions. *Euphytica* 171, 1–13.

Strelkov S.E. and L. Lamari, 2003. Host-parasite interactions in tan spot [*Pyrenophora tritici- repentis*] of wheat. *Canadian Journal of Plant Pathology* 25, 339–349.

Strelkov S.E., L. Lamari and G.M. Balance, 1999. Characterization of a host specific protein toxin (PtrToxB) from *Pyrenophora tritici-repentis*. *Molecular Plant-Microbe Interactions* 12, 728–732.

Strelkov S.E., L. Lamari, R. Sayoud and R. Smith, 2002. Comparative virulence of chlorosis-inducing races of *Pyrenophora tritici-repentis*. *Canadian Journal of Plant Pathology* 24, 29–35.

Tomas A., G.H. Feng, G.R. Reeck, W.W. Bockus and J.E. Leach, 1990. Purification of a cultivar-specific toxin from *Pyrenophora tritici-repentis*, causal agent of tan spot of wheat. *Molecular Plant-Microbe Interactions* 3, 221–224.

Tuori R.P., T.J. Woplert and L.M. Ciuffeti, 1995. Purification and immunological characterization of toxin compounds from cultures of *Pyrenophora tritici-repentis*. *Molecular Plant-Microbe Interactions* 8, 41–48.

Vavilov N.I., 1951. *The Origin, Variation, Immunity and Breeding of Cultivated Plants*. Selected writings, Translated from the Russian by K. Starr Chester (Chronica Botanica, Vol. 13, No. 1–6). Waltham, MA, USA.

Zhang H.F., L.J. Francl, J.G. Jordahl and S.W. Meinhardt, 1997. Structural and physical properties of necrosis-inducing toxin from *Pyrenophora tritici-repentis*. *Phytopathology* 87, 154–160.

Accepted for publication: March 2, 2011