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

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**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-

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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.

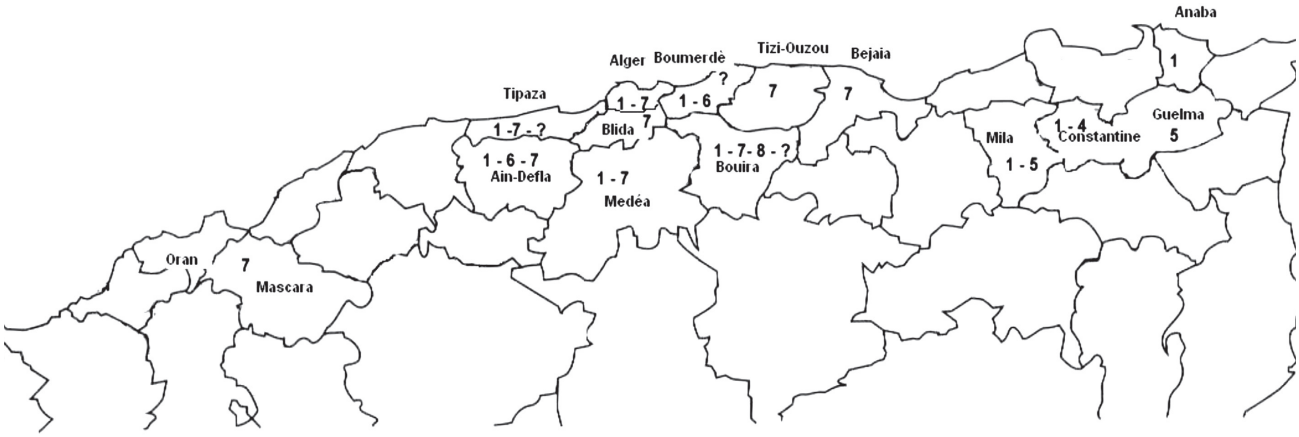


Figure 1. Distribution of races of *Pyrenophor tritici-repentis* in wheat growing regions in Algeria. 1, Race 1; 4, Race 4; 5, Race 5; 6, Race 6; 7, Race 7; 8, Race 8; ? = unknown race (new race).

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

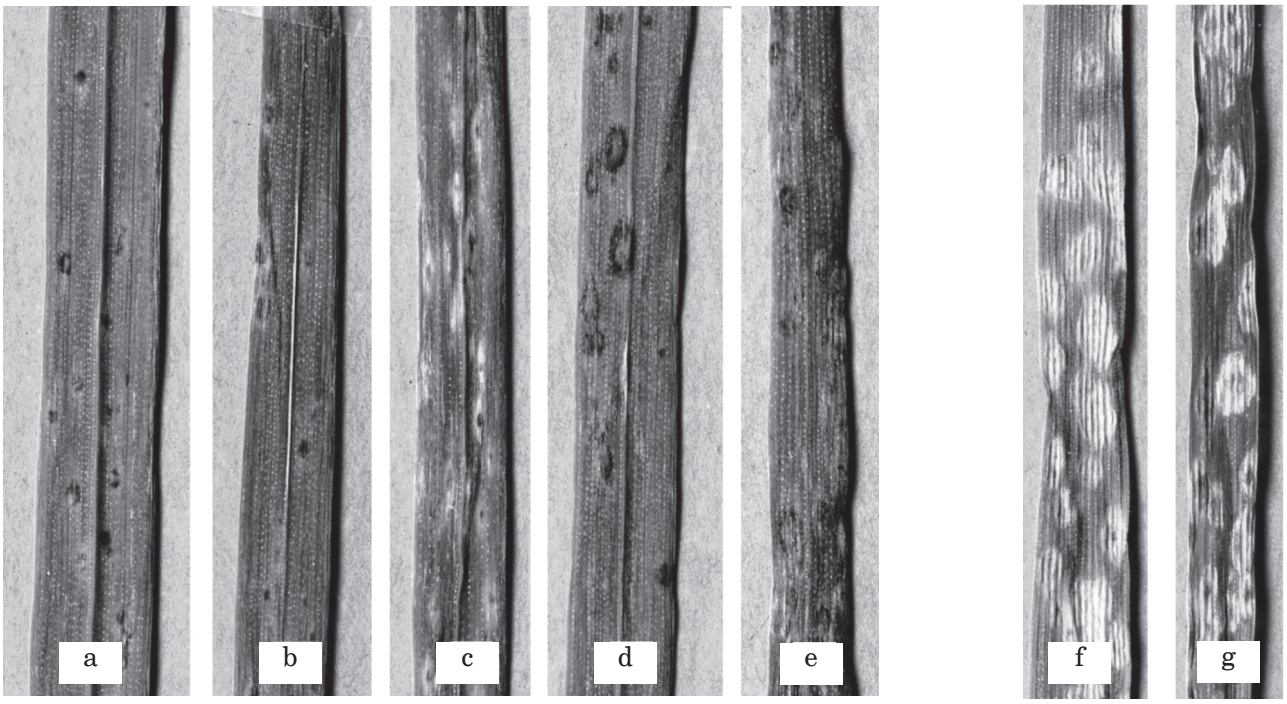


Figure 2. a–e. Resistance of Glenelea (a), 6B-365 (b), 6B-662 (c), Salamouni (d), 4B-1149 (e) to isolate Ptr 65, a new race of *Pyrenophora tritici-repentis*; note the presence of small brown to black spots on leaves. f–g. Sensitivity of Coulter (f) and 4B-140 (g) to Ptr65, new race of *Pyrenophora tritici-repentis*.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

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