Emergence of Tan Spot Disease Caused by Toxigenic *Pyrenophora tritici-repentis* in Australia Is Not Associated with Increased Deployment of Toxin-Sensitive Cultivars

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ABSTRACT

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The wheat disease tan (or yellow leaf) spot, caused by *Pyrenophora tritici-repentis*, was first described in the period 1934 to 1941 in Canada, India, and the United States. It was first noted in Australia in 1953 and only became a serious disease in the 1970s. The emergence of this disease has recently been linked to the acquisition by *P. tritici-repentis* of the *ToxA* gene from the wheat leaf and glume blotch pathogen, *Stagonospora nodorum*. *ToxA* encodes a host-specific toxin that interacts with the product of the wheat gene *Tsn1*. Interaction of *ToxA* with the dominant allele of *Tsn1* causes host necrosis. *P. tritici-repentis* races lacking *ToxA* give minor indistinct lesions on wheat lines, whereas wheat lines expressing the recessive *tsn1* are significantly less susceptible to the disease.

Although the emergence and spread of tan spot had been attributed to the adoption of minimum tillage practices, we wished to test the alternative idea that the planting of *Tsn1* wheat lines may have contributed to the establishment of the pathogen in Australia. To do this, wheat cultivars released in Australia from 1911 to 1986 were tested for their sensitivity to *ToxA*. Prior to 1941, 16% of wheat cultivars were *ToxA*-insensitive and hence, all other factors being equal, would be more resistant to the disease. Surprisingly, only one of the cultivars released since 1940 was *ToxA* insensitive, and the area planted to *ToxA*-insensitive cultivars varied from 0 to a maximum of only 14% in New South Wales. Thus, the majority of the cultivars were *ToxA*-sensitive both before and during the period of emergence and spread of the disease. We therefore conclude that the spread of *P. tritici-repentis* in Australia cannot be causally linked to the deployment of *ToxA*-sensitive cultivars.

Additional keywords: horizontal gene transfer, phylogeography.

The spread of plant diseases from one region to another is of immense historical, theoretical, and practical importance in plant pathology (4,20,24,27,36,38). Establishment of a disease in a new location requires that the spores (or other propagules) must both be disseminated and also thrive in the new location. The recent emergence of the wheat disease tan (or yellow) spot caused by *Pyrenophora* (syn. *Helminthosporium* or *Drechslera*) *tritici-repentis* ([Died.] Drechsler) provides a unique opportunity to test hypotheses on the causal factors required for intercontinental disease spread.

Wheat diseases have been carefully studied and catalogued in all wheat growing areas of the world since antiquity. We can therefore be reasonably certain that tan spot, currently a regular and damaging disease of wheat causing major losses in many parts of the world (6,7,23,28,40), did not emerge significantly prior to its description as the causal organism in the period 1934 to 1941 (1,2,5,19,29). The causal pathogen, *P. tritici-repentis* (Ptr) was described by Drechsler in 1923 (12) but was noted only as an occasional pathogen of grasses (18).

Pathogenicity of *P. tritici-repentis* is largely dependent on the production of three toxins of which *ToxA* is the best characterized and most important. *ToxA* is a small, secreted protein and acts as a host-specific toxin causing necrosis in susceptible cultivars (9,37,41). Sensitivity to the toxin is mediated by the *Tsn1* gene

(13) located on wheat chromosome arm 5BL (26). Genotypes with the dominant allele of *Tsn1* are significantly more susceptible to the disease (16).

The disease caused by *P. tritici-repentis* is called either yellow (leaf) spot (in Australia, South Africa, and Russia) or tan spot (in the Americas, Bulgaria, and UK) (10). *ToxA* produces a necrotic reaction in keeping with the name tan spot, whereas the other toxins produce chlorotic reactions, perhaps suggesting the name yellow spot (8,11,21). Isolates carrying each of the toxins are globally distributed (15). We can conclude that the different names given to the disease reflect historical accident rather than consistent phenotypic differences in symptom expression. Therefore, to avoid confusion, the disease will be referred to as tan spot in this paper.

We have recently obtained evidence that the *ToxA* gene was inter-specifically transferred to *P. tritici-repentis* from the related wheat pathogen *Stagonospora nodorum* (Sn) (17). The sequences of *ToxA* genes from *S. nodorum* were shown to be highly variable, consistent with a long history and positive selection for variation (39). In contrast, *ToxA* genes from Ptr isolates from around the world were identical, which is consistent with a more recent acquisition of the gene. In contrast, Sn*ToxA* sequences from *S. nodorum* were shown to be highly variable, consistent with a long history and positive selection for variation (39). Three factors, (i) the very recent emergence of tan spot, (ii) the evidence of interspecific gene transfer, and (iii) the enhanced virulence of strains carrying *ToxA*, led us to hypothesize that the acquisition of the gene was a critical factor in establishing the tan spot disease and promoting its dissemination. It is not clear how Ptr has spread

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throughout the world, but seed has been implicated as a possible means of long distance dissemination (18,43).

The hypothesis. Tan spot was first noticed in Australia in the early 1950s (44). By the 1970s it had spread and become a severe problem in parts of Queensland, New South Wales, and Western Australia (30,32,33). The very recent emergence of the disease in Australia, the excellent records of cultivars released and sown and the availability of those cultivars for testing *ToxA* sensitivity provided a unique opportunity to examine a possible factor behind the establishment of the disease. We have therefore tested the *Tsn1* phenotype and genotype of wheat cultivars released and sown prior to and during the spread of tan spot to determine whether the inadvertent use of the more susceptible *Tsn1* cultivars might have contributed to the spread of the disease in Australia.

Methodology and results. Wheat accessions representing many of the cultivars released between 1911 and 1970 and a

sample of more modern cultivars were obtained from the Australian Winter Cereal Culture Collection and the Department of Agriculture and Food, Western Australia (Table 1). Fifty-three cultivars were tested by infiltrating purified *ToxA* preparations into leaves of 3-week-old plants and examining the reactions 5 days later (25). The leaf reactions fell clearly into only two categories, necrosis or no reaction; no intermediate reactions were detected (data not shown). Only 8 of the 53 accessions were insensitive to the toxin and hence were genotypically *tsn1*. Interestingly the majority of the *tsn1* varieties were released before 1941. Only 1 of the 26 varieties released after 1941 was *tsn1*; Olympic (Table 1). The 53 wheat samples were analyzed using microsatellite markers linked to *Tsn1* (26). The markers have been estimated to flank the *Tsn1* locus at genetic distances of 0.2 and 0.6 cM. All *ToxA*-sensitive wheat lines and six of the eight *ToxA*-insensitive lines displayed the fragment sizes expected for

TABLE 1. The phenotype was assayed using the reaction to *ToxA*; the genotype was assayed using *Tsn1*-linked microsatellite markers

Name ^a	Year released	Areas grown	Phenotype <i>tsn</i> = 0, <i>Tsn</i> = 1	Genotype <i>tsn</i> = 0, <i>Tsn</i> = 1	Years of major use ^b
Merredin	1911	WA	0	0	
Nabawa	1915	NSW, WA, SA, Commonwealth	1	1	1929-48
Ford	1916		1	1	1925-60
Novo	1920	Qld	0	1	
Waratah	1921	NSW	1	1	1925-52
Glucub	1922	WA	1	1	
Flora	1923	Qld	1	1	
Ghurka	1924	Vic, Commonwealth,	0	0	1938-48
Ranee	1924	NSW, SA, Vic	1	1	1929-51
Free Gallipoli	1925	Vic, Commonwealth	1	1	1925-37
Dundee	1927	NSW, SA, Vic	1	1	1932-52
Gular	1927	NSW	0	0	
Bencubbin	1929	WA, Commonwealth, SA, NSW	1	1	1935-74
Sea Foam	1930	Qld	1	1	
Lawrence	1932	Qld	1	1	
Puora	1932	Qld	1	1	
Festival	1936	Qld	1	1	1952-65
Puglu	1938	Qld	1	1	
Bungulla	1939	WA	1	1	1945-51
Magnet	1939	Vic	0	0	1948-57
Puseas	1939	Qld	0	1	
Wongoondy	1940	WA	1	1	
Pindar	1941	Vic	1	1	
Quadrat	1941	Vic, Commonwealth	0	0	1948-57
Warigo	1941	SA	1	1	1948-64
Kondut	1943	WA	1	1	
Charter	1945	Qld,	1	1	1945-59
Gabo	1945	NSW, WA, SA	1	1	1946-70
Insignia	1946	Vic, SA, Commonwealth, WA	1	1	1950-75
Kendee	1946	NSW	1	1	1948-60
Pinnacle	1946	Vic	1	1	1950-74
Glenwari	1948	NSW	1	1	1951-71
Dirk	1949	SA,	1	1	
Sabre	1952	NSW, SA,	1	1	1957-65
Spica	1952	Qld	1	1	1955-76
Olympic	1956	Vic, NSW	0	0	1958-88
Heron	1959	NSW, W A, S A, Vic	1	1	1962-80
Falcon	1960	NSW	1	1	1962-81
Gala	1960	Qld	1	1	
Mengavi	1960	NSW	1	1	1962-71
Eagle	1969	Qld	1	1	
Gatcher	1969	Qld, NSW	1	1	
Condor	1973	NSW	1	1	
Egret	1973	NSW	1	1	
Kite	1973	Qld, NSW	1	1	
Songlen	1975	Qld, NSW	1	1	
Cook	1977	Qld, NSW	1	1	
Tincurrin	1977	WA	1	1	
Miling	1978	WA	1	1	
Banks	1979	Qld, NSW	1	1	
Eradu	1981	WA	1	1	
Gutha	1982	WA	1	1	
Kulin	1986	WA	1	1	

^a Wheat varieties tested in this study.

^b Data available for NSW only.

the *Tsn1* and *tsn1* genotypes, respectively. Interestingly, two *ToxA*-insensitive lines (Novo and Puseas) gave the fragment sizes expected for the *Tsn1* genotype (26) indicating recombination has occurred in these lines (Table 1).

A sample of seven isolates of *P. tritici-repentis* was obtained from P. Williamson, Queensland Department of Primary Industry, to determine whether they carried the *ToxA* gene. These isolates were collected between 1978 and 2001 from sites in Queensland, Victoria, New South Wales, and Western Australia. The *ToxA* primers directed the amplification of the expected 134-bp fragment in all 7 cases (17) (data not shown).

The area planted to each wheat variety is recorded for New South Wales (14). Figure 1 shows the area planted to *tsn1* varieties as a percentage of the total wheat acreage. *ToxA*-insensitive varieties constituted never more than 14% of the area planted, and Olympic was the sole recorded *tsn1* variety cultivated after 1957. The maximum area of *tsn1* utilization was in the period 1960 to 1975 during which tan spot emerged as a wheat disease of major importance.

Significance and conclusions. Amongst the crop diseases to have emerged in recent decades, wheat tan spot is both economically very significant and particularly well documented. Tan spot was first noted in Australia in the 1950s (44). By the 1970s it was a major disease in Australia causing yield losses up to 48% (31,32,34).

ToxA is an important pathogenicity factor for *Ptr* and the *ToxA* gene is commonly found in *Ptr* isolates from diverse geographical regions. Although no large studies of Australian populations of tan spot have been published, all isolates produce necrotic lesions on *ToxA*-sensitive cultivars and all of the seven isolates surveyed carried *ToxA* (35). Thus, the purpose of this study was to test the hypothesis that *P. tritici-repentis* might have become established in Australia as a result of *ToxA*-insensitive cultivars being dropped in favor of sensitive cultivars during the decades before establishment of the disease. To test this, we collected wheat cultivars released in Australia between 1911 and 1986 and tested their reaction to *ToxA*. The pattern we observed did not support our hypothesis. Instead, we found that the large majority of cultivars released in that period were *ToxA*-sensitive, with the areas planted to *ToxA*-insensitive cultivars varying between 0 and 14%. Indeed, the peak period of utilization of *ToxA*-insensitive varieties was in the period 1960 to 1974, by the end of which the pathogen was firmly established. New South Wales contains regions comparable with both the summer (Queensland) and winter rain-fed (Victorian, South Australian, and Western Australian) regions and so can be regarded as indicative of the situation in the rest of Australia. We can conclude therefore that the removal of *ToxA*-insensitive cultivars was not a major factor in the emergence of the disease.

The recessive allele *tsn1* confers toxin insensitivity but only partial disease resistance and only to *ToxA*-producing races of the

fungus. Olympic, the only *tsn1* variety grown between 1958 and the 1980s, was described as susceptible to tan spot with yield losses of 39%, though it performed significantly better than other cultivars described as very susceptible, such as Banks, with losses of 48% (33). The symptoms on Olympic were spreading chlorotic, rather than necrotic, lesions consistent with insensitivity to *ToxA* but sensitivity to *ToxB* or *ToxC* (G. Platz, *personal communication*). Based on overall tan spot score, Rees concluded that the cultivars introduced in the 1960s and 1970s were more susceptible to the disease than the cultivars they replaced (35). More work on the *P. tritici-repentis* populations and tan spot resistance genes in Australia is needed to fully determine whether genetic or, more likely, agronomic factors such as the introduction of minimum tillage, can explain the success of tan spot (3,11).

The possible role of *Tsn1* in the spread of tan spot in western Canada has also been investigated (22). Tan spot remained sporadic until the 1970s but since then has been a regular and damaging disease. *Tsn1* wheats were widely planted from as early as the late 1800s, nearly disappeared by the 1950s but were inadvertently reintroduced in the 1960s. This pattern provides no clear correlation between the incidence of the disease and the relative frequencies of *Tsn1*- and *tsn1*-containing cultivars. Here, as in Australia, other factors appear to have dominated.

The emergence of tan spot invites comparison with southern corn leaf blight (42). This disease, caused by race T isolates of *Cochliobolus heterostrophus*, is also attributed to a host-specific toxin. Maize carrying a specific mutation of the mitochondrial DNA associated with Texas cytoplasmic male sterility (T-cms) was found to be specifically sensitive to the toxin and susceptible to the disease. Race T isolates were first observed in the Philippines in 1961 and in the United States in 1968. The epidemic of 1970 which caused 20 to 30% losses was unprecedented in modern times. Shortly thereafter T-cms corn was abandoned and the disease has retreated (45). T-cms had been introduced into maize cultivars only a decade or so before the emergence of the new epidemic, whereas our results show that *Tsn1* wheat has been present at least 50 years before tan spot became a major disease. The spectacular rise and fall of race T *C. heterostrophus* is explained by the devastating effect of T-toxin on T-cms maize (allied to its hypersusceptibility to other diseases) and the fact that 85% of the United States maize crop carried the susceptibility gene in 1970 to 1971 (42). Such varieties were quickly replaced. In contrast, the effect of *ToxA* on the virulence of *P. tritici-repentis* is more measured and is ameliorated by the presence of at least two other significant toxins. Expression of the sensitivity genes does not lead to catastrophic disease. These factors may explain why the sensitivity allele *Tsn1* seems to have been present at high frequency decades prior to the emergence of the pathogen as a serious disease.

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Fig. 1. A bar graph presenting the percent total area planted in *ToxA*-insensitive wheat varieties from 1935 to 1985 in New South Wales, including the 1970s when the disease emerged as an economic problem (12).

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