*Journal of Integrative Agriculture 2026, 25(2): 744–755*

Available online at ScienceDirect

Journal of Integrative Agriculture



RESEARCH ARTICLE

# Revealing inheritance of a Xinjiang isolate BGTB-1 of *Puccinia striiformis* f. sp. *tritici* and the shift of pathogenicity from avirulence to virulence at heterozygous *AvrYr5* locus

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## Highlights

- Revealing complex genetic characteristic and high genetic variation of *Puccinia striiformis* f. sp. *tritici* in relatively isolated stripe rust in Xinjiang by a selfed population of a representative isolate.
- Direct evidence showing the discovery of virulent sexual progenies originated from the avirulent parental isolate at *Yr5* locus *via* sexual reproduction under the background of the current absence of *Yr5*-virulent isolates in Xinjiang.
- Sexual reproduction accounts for the origin of new strains and evolution of *P. striiformis* f. sp. *tritici* in Xinjiang.

## Abstract

In China, Xinjiang is a relatively independent epidemic region of wheat stripe rust, caused by *Puccinia striiformis* f. sp. *tritici*, due to great population genetic divergence of Xinjiang with other inland epidemic regions. In this region, race evolution was usually slower than inland populations. However, many new races have recently been found, and therefore, it is necessary for more understanding of the virulence evolution of the Xinjiang population. So, in this study, a 65 sexual progenies, derived from a Xinjiang single-urediospore isolate BGTB-1 of *P. striiformis* f. sp. *tritici* by selfing on alternate host barberry (*Berberis aggregata*). It was phenotyped on the 25 single *Yr* lines, and genotyped by 19 kompetitive allele-specific PCR-single nucleotide polymorphism (KASP-SNP) markers. As a result, the 65 progenies were identified as 56 various virulence patterns (VPs), and neither of which was identical to the parental isolate, showed 100% virulence variation. Compared with the parental isolate, of all progenies, 39 (60.0%) had increased virulence, and 18 (27.7%) had decreased virulence. All progenies exhibited avirulence to *Yr10*, *Yr15*, *Yr32*, and *YrTr1* loci, and avirulence and virulence segregation at the remaining 21 *Yr* resistance loci. The results showed avirulence to *Yr5*, *Yr7*, and *Yr76* (A:V≈3:1) loci is controlled by a single dominant gene, and that to *Yr6*, *Yr25*, and *Yr44* (A:V≈1:3) loci by a single recessive gene. However, avirulence to the remaining 15 resistant loci including *Yr1*, *Yr2*, *Yr3*, *Yr4*, *Yr8*, *Yr9*, *Yr17*, *Yr26* (=*Yr24*), *Yr28*, *Yr29*, *Yr43*, *YrSP*, *Yr27*, *YrA*, and *YrExp2*, with various avirulence and virulence segregation ratios, is controlled by two genes with different gene effects, indicating complex genetic traits of the parental isolate. Totally, 65 dissimilar genotypes detected among progenies using overall molecular markers, by which a linkage map was constructed, with a genetic distance of 441.0 cM. Interestingly, although the parental isolate was avirulent to *Yr5*, but 17 progenies showed virulence, showing the change of pathogenicity from avirulence to virulence at this resistance locus. It was for the first time to report that progenies with virulence to *Yr5* produced sexually from avirulent parental isolate at this resistance locus. To our knowledge, this study offers an insight into inheritance, sexual reproduction and virulence variation of *P. striiformis* f. sp. *tritici* in Xinjiang, facilitating us to understand evolution of the rust pathogen in this region and accounting for Xinjiang population distinguished form other inland populations. Additionally, it is necessary to further confirm the roles of sexual reproduction in the emergence of new races and affecting population genetic diversity of *P. striiformis* f. sp. *tritici* under natural conditions in Xinjiang.

**Keywords:** wheat yellow rust, *Puccinia striiformis* f. sp. *tritici*, sexual reproduction, virulence variation, new race, SNP, *Yr5*, Xinjiang

Received 6 January 2024; Received in revised form 4 March 2024; Accepted 13 March 2024; Available online 13 April 2024
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doi: 10.1016/j.jia.2024.04.023

## 1. Introduction

Wheat stripe (or yellow) rust, caused by an obligate and heteroecious rust fungus belonging to the phylum Basidiomycota, is a highly destructive disease worldwide, especially in cool and humid regions (Li and Zeng 2002; Chen 2005; Wellings 2011). The disease can cause a yield reduction of 10–30%, or even no grain if early severe infection occurs on highly susceptible wheat cultivars (Doling and Doodson 1968; Roelfs 1978; Chen 2005; Wellings 2011).

Utilizing wheat genetically wheat resistant cultivars is an important approach for controlling wheat stripe rust, because of their effectiveness, low cost, and environment friendly nature. Resistance in wheat cultivars is often overcome by new races within a few years of their release or even within one year (Rapilly 1979; Li 1980). Consequently, the evolution and emergence of new wheat stripe rust races has raised concerns about their origin.

After the discovery of alternate hosts of *Puccinia striiformis* f. sp. *tritici* by Jin *et al.* (2010), worldwide scientists are exploring whether any new races are associated with sexual reproduction. They tested specific isolate through selfing on alternate host barberry and as a result, a new race generation has formed based on genetic and phenotypic variability (Tian *et al.* 2016; Wang *et al.* 2018; Mehmood *et al.* 2020; Rodriguez-Algaba *et al.* 2021; Zhang *et al.* 2023). These studies have demonstrated that the genetic segregation of self-progeny populations at the same avirulence or virulence resistance locus varies from different isolates.

Wheat is grown annually in 0.60–0.75 million hectares since 2000 in Xinjiang, China, in which there are three different wheat growing areas, including spring wheat planting area, winter wheat planting area, and spring and winter wheat mixed planting area (Li *et al.* 2010). In this region, stripe rust is an important disease in wheat, threatening safe crop production. In the 1950s–1960s, moderate to severe epidemics of wheat stripe rust occurred in six years including 1952, 1954, 1956, 1958, 1959, and 1960, resulting in a remarkable yield loss of 30 to 50%, even without any harvest (Zhang 1962). In the 1990s, the large-scale epidemics of the disease were recorded in 1990, 1992, 1993, and 1994, with annually infected wheat field areas of 40–80 thousand hectares. The outbreak of wheat stripe rust took place in 2002, and since then, the affected area has been increasing every year. The situation reached its peak in 2007 when the disease affected 159 thousand hectares of wheat fields (Li *et al.* 2010).

Among different epidemic regions of wheat stripe rust in China, Xinjiang is an important epidemic region and a relatively independent epidemic region of the disease, geographical and ecological characteristics make this region distinct from other inland epidemic regions of country (Li and Zeng 2002; Zhan *et al.* 2016). Epidemically, population evolution of the rust in this region is greatly slower than those of the other inland epidemic regions (Wu *et al.* 1993; Li *et al.* 2010; Zhan *et al.* 2016). But still this region has a high level of genetic diversity and molecular variation (Wan *et al.* 2015; Zhan *et al.* 2016). In recent years, it has been demonstrated that Xinjiang population diverges genetically, distinctly different from those of other epidemic regions of China (Zhan *et al.* 2016; Awais *et al.* 2022). However, the reason contributed to this status has remained unknown. Thus, the objective of this study was to understand inheritance of *P. striiformis* f. sp. *tritici* in Xinjiang, and to provide a theoretical basis for revealing virulence variation and evolution of the wheat stripe rust pathogen in the independent epidemic area of Xinjiang.

## 2. Materials and methods

### 2.1. Plant and fungal materials

The wheat cultivar Mingxian 169, highly susceptible to all known Chinese races of *P. striiformis* f. sp. *tritici*, was used to cultivate pure isolate and increase urediospores. Twenty-five single *Yr* gene lines were used to differentiate pathogenic types of all cultures including the parental isolate and its progeny, and to determine avirulence or virulence at each resistance locus of each culture. Seeds of barberry species (*Berberis aggregata*) as an alternate host for *P. striiformis* f. sp. *tritici* were collected from Tianshui in Gansu in autumn, dried completely in room temperatures, and then sealed in a plastic bag before being stored at 4°C in a refrigerator for future use.

### 2.2. Cultivation of plant seedlings

Seeds of wheat materials were sown in a small pot filled with potting soil mix (Inner Mongolia Mengfei Bio-Tech Co., Ltd., Huhhot, Inner Mongolia, China), transferred in a stainless-steel tray with water, and cultivated till to 10-day-old seedlings with inside a chamber with a dual photoperiod and temperature regime of 20°C for 16 h with light (8,000–10,000 μmol $m^2$ $s^{-1}$ of light intensity) and 16°C for 8 h in the dark in a condition-controlled greenhouse. Seeds of barberry species (*Berberis aggregata*) were put inside of folded and water-soaked three-layer filtering paper in a Petri dish (90 mm in diameter), and incubated at 16°C until germinating. After two cotyledons of a seed grown out completely, and was moved into a pot for cultivation to 6–10 leaf seedlings for inoculation in the growth chamber.

### 2.3. Preparation of single-urediospore isolate and teliospores

Pure culture of the isolate BGTB of *P. striiformis* f. sp. *tritici*, collected in Yili in western Xinjiang was established by picking a single urediospore on a leaf of wheat Mingxian 169 seedlings under a dissecting light microscope (XTL-165-VT, Phoenix Optical Group Co., Ltd., Jiangxi, China). After spraying with deionized water over approximately 40 cm, inoculated wheat plants were transferred into a dew chamber (I-36 D, Percival Scientific, Inc., IA, USA) and kept for 24 h at 10°C without light, and 100% relative humidity for urediospore germination and penetration. After incubation, the inoculated wheat plants were transferred into the growth chamber under control condition (16 h at 16°C with light and for 8 h at 13°C in the dark) until sporulation. Fresh urediospores were collected, and kept in a dessicator with silica gels at 4°C for late use. Re-inoculation was used to increase urediospores.

To obtain teliospores, the single urediospore culture was

inoculated leaves of wheat plants at adult growth stage, and cultivated until sporulation referred the method as above. When primary sporulation on the leaves was observed, the infected wheat plants were continuously cultivated: daytime temperature was altered to 25°C for inducing teliospore formation. As appearance of a great deal of telia, leaves bearing teliospores were collected from the wheat plants, dried at room temperatures avoiding direct sunlight, and put into a paper bag and then kept in a desiccator with silica gels at 4°C until use.

### 2.4. Inoculation of barberry seedlings

Leaves bearing teliospores were cut into short segments, and soaked in deionized water completely for 12–24 h at room temperatures prior to inoculation. The leaf segments were scratched by a clean scalpel and then put onto 2% water agar medium in a Petri dish, and kept at 16°C in an incubator. Periodical observation was conducted to confirm teliospore germination using a light microscope (B301, Chongqing Optec Instrument Co., Ltd., Chongqing, China). Once teliospores initiated to germinate, the plate was overturned and put the top of a plastic transparent cylinder that was covered the barberry seedlings in a pot. The barberry plants were incubated for 3–4 days at 16°C in the dew chamber in the dark, and then moved into the growth chamber under the same conditions as above.

### 2.5. Establishment of single aeciospore progeny population

It was observed that pycnia appeared on the surface of infected leaves and macroscopic nectars (honey dews) accumulated obviously onto yellowish pycnial flecks (ostioles of pycnia). Fertilization was implemented by transferred between nectars using a 1-μL plastic inoculation loop (Changde Bkmam Bio-Tech Co., Ltd., Hunan, China). After fertilization, the barberry plants were continuously cultivated to produce aecia on the abaxial surface of leaves. An individual aecium was detached from leaf blade, placed onto a glass slide disinfected with 70% (v/v) ethanol solution, and gently pressed to expose aeciospores. A single aeciospore was picked by a special-made tool to inoculate on the adaxial surface of a leaf of wheat Mingxian 169 seedlings. It's worth noting that every single aeciospore was come from one single aecium. Conditions with regard to incubation, and cultivation was referenced as mentioned above. One infected leaf of only a wheat plant was kept as a pure progeny isolate, and the remaining wheat plants were discarded. Numerous urediospores produced, and were collected to put in a desiccator with silica gels for a short time storage until use. Increasing urediospores was completed by re-inoculation.

### 2.6. Phenotyping

To determine pathogenic variation, the parent isolate and the progenies were differentiated on the 25 single *Yr* gene lines. The mixture of ~5 mg urediospores of each isolate and 5 mL of methoxy-nonafluorobutane fluid (Novec 7100, 3M, MN, USA) was used to made the spore suspension in a 5-mL atomizer. The spore suspension was sprayed onto leaves of the 10-day old wheat seedlings carrying single *Yr* gene, and wheat Mingxian 169 was used as susceptible control along four directions over a distance of ~40 cm. After spraying with deionized water, inoculated wheat plants were moved into the dew chamber and kept for 24 h at 10°C without light for germination and penetration of the spore, and then moved into the growth chamber with the same conditions as above for the development of the infection. At 18–20 days post inoculation, once infection types (ITs) of each line and the susceptible wheat control were scored according to a 0-to-9 scale (Line and Qayoum 1992), of which ITs 0-to-6 were indicated as avirulent (A), and 7-to-9 as virulent (V), respectively.

### 2.7. Genomic DNA extraction

For each isolate, ~20 mg urediospores were put into a 2-mL plastic centrifuge tube inside which three stainless-steel beads (Ø 2 mm) were added, and ground to fine powder using a tissue lyser (Tissuelyser-II, Qiagen, Dusseldorf, Nordrherin-Westfalen, Germany). Genomic DNA extraction was accomplished with the cetyltrimethylammonium bromide method as described by Chen *et al.* (1993). Evaluation of the quality and concentration of the extracted DNA was carried out with a spectrophotometer (NanoDrop 2000, Thermo Fisher Scientific Inc., MA, USA). The DNA was used for PCR amplification with KASP-SNP primers that were developed by Meng *et al.* (2020), and synthesized by Sangon Biotech (Shanghai) Co., Ltd. (China). FAM and HEX fluorescently labeled adapters were connected at the 5′-end of two forward primers, F1: 5′-GAAGGTGACCAAGTTCATGCT-3′, and F2: 5′-GAAGGTCGGAGTCAACGGATT-3′, respectively. A 2-μL diluted DNA (50 ng $\mu L^{-1}$) was pipetted in a 384 well plate, and dried at 55°C in an incubator (SFG-02.400; Huangshi Hengfeng Medical Devices Co., Ltd., Hubei, China). A 4-μL KASP system, consisting of 2 μL of KASP V4.0 2×Master mix, 1.5 μL of $ddH_2O$, and 0.5 μL primer mix (12 μmol $L^{-1}$ of each of the two forward primers F1 and F2, and 30 μmol $L^{-1}$ of reverse primer). The PCR amplification was run in a PCR thermal cycler (Applied Biosystems, CA, USA) with the following program including predenaturation for 15 min at 94°C; 10 cycles of predenaturation for 20 s at 65°C, annealing for 60 s at decreasing 0.8°C per cycle; and 32 cycles of denaturation for 20 s at 94°C, annealing for 60 s at 57°C. Data of PCR products were read automatically by SNP Analyzer of a FLUOstar Omega (BMG Labtech, Ortenberg, Germany), and genotyped using the Kluster Caller software (ver. 2.24.0.11, LGC Biosearch Technologies).

### 2.8. Data analysis

A binary system was used to transform phenotypic avirulence (A, ITs 0-to 6) or virulence (V, ITs 7-to-9) into "0", and "1", respectively. Using VAT software (Kosman *et al.* 2008), phenotypic data of the parental isolate and the selfed progenies tested on single *Yr* gene lines were utilized to determine virulence patterns of overall isolates, and among which the same virulence pattern was compare. Fit goodness of avirulence and virulence segregation was evaluated by Chi square test ($\chi^2$).

The Chi square test was also used to assess fit goodness of allele of KASP-SNP and model of single locus or double loci at confidence interval of greater than 99% ($P$<0.01). Correlation between phenotype (virulence/avirulence) and genotype (multi-locus genotypes, MLGs) was calculated by NTSYS-pc software (ver. 2.0). Phenotypic data of the selfed progenies was used for determining quantitative trait loci (QTLs) using GACD software (http://www.isbreeding.net/), with a threshold value 2.5 of log of the likelihood odds ratio (LOD). The genotyping data were used to construct a linkage genetic map with Mapchart ver. 2.3 (Voorrips 2002).

## 3. Results

### 3.1. Phenotype and virulence variation

A single urediospore culture BGTB-1 from the isolate BGTB was established (Fig. 1-A), and were tested virulence on the 25 single *Yr* gene lines. The isolate BGTB-1 showed virulence to *Yr1*, *Yr6*, *Yr9*, *Yr17*, *Yr25*, *Yr28*, *Yr29*, *Yr44*, *YrSP*, *Yr76* (previously known as *YrTye*), and *YrA*, and avirulence to *Yr2*, *Yr3*, *Yr4*, *Yr5*, *Yr7*, *Yr8*, *Yr10*, *Yr15*, *Yr26*, *Yr27*, *Yr32*, *Yr43*, *YrTr1*, and *YrExp2*.

Abundant black telia and teliospores were produced on wheat (Mingxian 169) leaves 30 days after inoculation with the BGTB-1 isolate (Fig. 1-B). The basidiospores were produced from germinated teliospores, which were then used to inoculate barberry seedlings. Nectar-to-nectar transfers were made for fertilization as pycnia appeared with nectar accumulation. At 6 day post inoculation, orange-colored aeciospores arose from the opposite side to a fertilized pycnium on the surface of leaves of barberry seedlings (Fig. 1-C and D). After inoculation of wheat Mingxian 169 seedlings with an individual aeciospore from one aecium (aecial cup), as a result, 65 selfed (S1) single aeciospores progeny were obtained, and all of which were determined through avirulence or virulence formula based on tests on each of the 25 single *Yr* gene lines. The VAT analysis indicated that the 65 S1 progenies were classified into 56 various virulence patterns (VPs) (Appendix A). Out of the 65 S1 progenies, 39 progenies (60.0%) had 32 different VPs (57.1%). They showed increased pathogenicity with broader virulence and spectrum compared to the parental isolate BGTB-1. On the other hand, 18 progenies (27.7%) belonging to 18 VPs (32.1%) had decreased pathogenicity with narrower virulence spectrum (Table 1). Among all the VPs, VP1 had more clone, included 4 S1 progeny and accounted for 6.2% of the total isolates.

Among all S1 progenies, none of them exhibited same virulence patterns as the parental isolate BGTB-1. Only VP16 had the closest virulence pattern with the parental isolate. Only difference was at *YrSP* resistance locus. For this locus, which found be virulent in VP16 (Table 1). Compared with the parental isolate, four virulence patterns, VP2, VP18, VP26, and VP51, showed difference of avirulence or virulence at two resistance loci. The most significant difference regarding avirulence and virulence spectrum on overall single *Yr* gene lines was detected by the VP31, which showed difference at ten resistance loci compared with the parental isolate. VP34, involving one S1 progeny, was the broadest of 56 virulence patterns in the virulence spectrum. It attributed virulence to 17 out of 25 *Yr* loci and was found more virulent than the parental isolate. The parental isolate was avirulent to *Yr3*, *Yr5*, *Yr7*, *Yr17*, *Yr27*, *Yr43*, *YrSP*, and *YrExp2*. On the other hand, VP34 was found to be virulent towards them. The VP7, including one S1 progeny, showed avirulence to all of 25 resistance loci, and it was greatly weaker than the parental isolate. Phenotypic observations indicated that all S1 progenies were avirulent to four resistance loci (*Yr10*, *Yr15*, *Yr32*, and *YrTr1*). Neither of the whole S1 progenies was observed with virulence to overall 25 *Yr* resistance loci. Interestingly, the parental isolate showed avirulence to the *Yr5* locus. However, 17 VPs (VP6, VP8, VP9, VP12, VP19, VP22, VP27, VP28, VP31, VP34, VP35, VP36, VP41, VP46, VP48, VP55, and VP56) included 18 S1 progenies and showed virulence to this resistance gene locus, such as S1-13, and S1-65 (Fig. 2). Genetic avirulence and virulence segregation was observed at 21 *Yr* resistance loci, *Yr1*, *Yr2*, *Yr3*, *Yr4*, *Yr5*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, *Yr17*, *Yr27*, *Yr25*, *Yr26*, *Yr28*, *Yr29*, *Yr43*, *Yr44*, *Yr76*, *YrExp2*, *YrA*, and *YrSP*, indicating that the parental isolate was heterozygous at these loci.

### 3.2. Genetic analysis for avirulence/virulence loci

Based on testing of the 65 S1 progenies on the 25 single *Yr* gene lines, it was observed that the overall S1 progenies showed avirulence to four resistance loci (*Yr10*, *Yr15*, *Yr32*, and *YrTr1*), with an observed ratio of 65 avirulence (A):0 virulence (V) (Table 1). It showed that the parental isolate

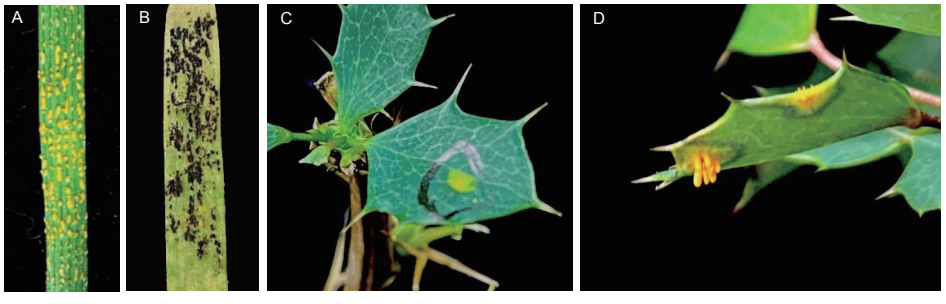



**Fig. 1 Uredia and urediospores (A), telia and teliospores (B), pycnia and pycniospores (C), and aecia and aeciospores (D) of the Xinjiang single-urediospore isolate BGTB-1 of *Puccinia striiformis* f. sp. *tritici* on leaves of wheat (cv. Mingxian 169) and barberry (*Berberis aggregata*) seedlings after inoculation, respectively.**

**Table 1 Avirulence (A) and virulence (V) segregation of 65 selfed progenies at 25 *Yr* gene lines**

| *Yr* gene[1)] | Phenotype of parental isolate | Observed no. of progeny | | Expected ratio (A:V) | $\chi^{2}$ [2)] | *P* | Avirulence genes |
|---|---|---|---|---|---|---|---|
| | | A | V | | | | |
| Yr1 | V | 5 | 60 | 1:15 | 0.05 | 0.82 | Unlocated |
| *Yr2* | A | 55 | 10 | 13:3 | 0.29 | 0.59 | *AvrYr2-1*, *AvrYr2-2* |
| *Yr3* | A | 60 | 5 | 15:1 | 0.05 | 0.82 | Unlocated |
| *Yr4* | A | 64 | 1 | 15:1 | 1.72 | 0.19 | Unlocated |
| *Yr5* | A | 47 | 18 | 3:1 | 0.13 | 0.72 | *AvrYr5* |
| Yr6 | V | 14 | 51 | 1:3 | 0.25 | 0.62 | Unlocated |
| *Yr7* | A | 45 | 20 | 3:1 | 0.87 | 0.35 | Unlocated |
| *Yr8* | A | 63 | 2 | 15:1 | 0.64 | 0.42 | *AvrYr8-1* |
| *Yr9* | V | 9 | 56 | 3:13 | 0.73 | 0.39 | *avrYr9-1*, *avrYr9-2* |
| *Yr10* | A | 65 | 0 | 1:0 | – | – | – |
| *Yr15* | A | 65 | 0 | 1:0 | – | – | – |
| *Yr17* | A | 27 | 38 | 7:9 | 0.05 | 0.81 | Unlocated |
| *Yr25* | V | 19 | 46 | 1:3 | 0.42 | 0.52 | *avrYr25* |
| *Yr26* | A | 64 | 1 | 15:1 | 1.72 | 0.19 | Unlocated |
| *Yr27* | A | 40 | 25 | 9:7 | 0.54 | 0.46 | *AvrYr27-1* |
| *Yr28* | V | 11 | 54 | 3:13 | 0.05 | 0.83 | *avrYr28-1*, *avrYr28-2* |
| *Yr29* | V | 7 | 58 | 1:15 | 1.56 | 0.21 | *avrYr29-1*, *avrYr29-2* |
| *Yr32* | A | 65 | 0 | 1:0 | – | – | – |
| *Yr43* | A | 60 | 5 | 15:1 | 0.05 | 0.82 | *AvrYr43-1*, *AvrYr43-2* |
| *Yr44* | V | 20 | 45 | 1:3 | 0.87 | 0.35 | *avrYr44* |
| *Yr76* | V | 46 | 19 | 3:1 | 0.42 | 0.52 | *AvrYr76* |
| *YrA* | V | 6 | 59 | 1:15 | 0.54 | 0.46 | Unlocated |
| *YrExp2* | A | 54 | 11 | 13:3 | 0.05 | 0.83 | *AvrYrExp2-1* |
| *YrSP* | A | 11 | 54 | 3:13 | 0.05 | 0.83 | *avrYrSP-1*, *avrYrSP-2* |
| *YrTr1* | A | 65 | 0 | 1:0 | – | – | – |

[1)] *Yr26*=*Yr24* (McIntosh *et al.* 2018). *Yr76* previously known as *YrTye* (Xiang *et al.* 2016).
[2)] $\chi^{2}$ indicates Chi square test for the goodness of fit of the observed number of progeny at each of avirulence/virulence loci to the expected ratio.
"–", data not available.

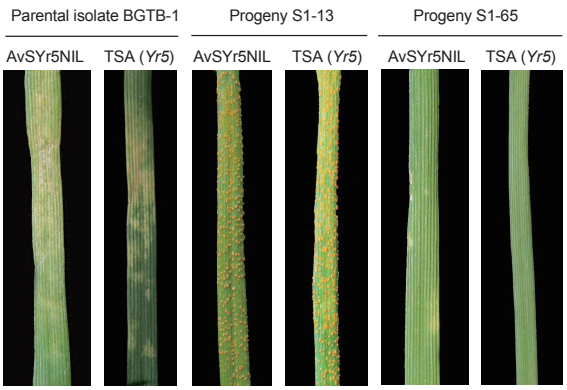



**Fig. 2 Avirulence (low infection type) and virulence (high infection type) of the parental isolate BGTB-1 and progenies S1-13 and S1-65 on AvSYr5NIL, *Yr5* near isogenic line (NIL) with Avocet Susceptible (AvS) genetic background, and *Triticum aestivum* subsp. *spelta* var. *album* (TSA, *Yr5*).**

BGTB-1 was homozygous avirulence at the four *Yr* loci. However, different avirulence and virulence segregations were observed at each of the remaining 21 *Yr* loci (Table 1), showing that the parental isolate was heterozygous at these resistance gene loci. Of the 21 *Yr* resistance loci, *Yr5*, *Yr7*, and *Yr76* displayed an observed avirulence to virulence segregation ratio of 3:1 in the S1 progenies, hinting that a single, dominant gene responsible to avirulence at these 3 *Yr* loci. At *Yr6*, *Yr25*, and *Yr44* loci, a segregation ratio of 1A:3V to the S1 progenies was observed, and indicated that avirulence to the three *Yr* loci was controlled by an individual, recessive gene. However, avirulence to the other 15 *Yr* loci, *Yr1*, *Yr2*, *Yr3*, *Yr4*, *Yr8*, *Yr9*, *Yr17*, *Yr26*, *Yr27*, *Yr28*, *Yr29*, *Yr43*, *YrSP*, *YrExp2*, and *YrA*, was regulated by two genes, with different gene effects. Of these 15 resistance loci, two loci, *Yr2*, and *YrExp2*, segregated with a ratio of 13A:3V, and avirulence to each was controlled by two genes, one dominant gene and one recessive gene. A segregation ratio of 3A:13V at *Yr9*, *Yr28*, and *YrSP* to the S1 progenies was observed, being inferred that avirulence of the parental isolate to each of the three *Yr* loci were theoretically governed by one dominant gene and one complementary suppressor gene, or one dominant gene and one recessive gene. At *Yr27* locus, at which overall S1 progenies showed avirulence and virulence segregation (A:V=9:7), implying that avirulence of the parental isolate at this resistance locus was dominated by two complementary dominant genes at this resistance locus. Avirulence and virulence at *Yr17* locus to the S1 progenies segregated with a ratio of 7A:9V, suggesting that two recessive genes manipulated avirulence of the parental isolate at this resistance locus. A avirulence and virulence segregation (A:V=15:1) of the S1 progenies to five loci, comprising *Yr3*, *Yr4*, *Yr8*, *Yr26*, and *Yr43*, was detected, manifesting that avirulence of the parental isolate at these resistance loci was commanded by two dominant genes. At three *Yr* loci, *Yr1*, *Yr29*, and *YrA*, at which the S1 progenies showed avirulence and virulence segregation with a ratio of 1A:15V, indicating that avirulence of the parental

isolate at each of these resistance loci was governed by two complementary recessive genes.

### 3.3. Genotype polymorphism

A total of 59 pairs of KASP-SNP primers were used to identify genetic variation among the 65 S1 progenies (Appendix B). Out of these, 19 found to be polymorphic. The polymorphism was observed due to heterozygous parental isolate BGTB-1 and segregation in the progenies. Using the 19 KASP-SNP markers (Appendix A), 65 multi locus genotypes (MLGs) were detected among the overall progenies, distinguished from that of the parental isolate BGTB-1. All S1 progeny had distinguished MLGs, showed high multilocus genotype diversity. The 19 KASP-SNP markers displayed varying percentages of heterozygous marker loci among all progeny, showing the minimum of 43.1% at the 159 genotypic locus and the maximum of 89.2% at the 351 genotypic locus, with the mean value of 63.2% (Table 2). Among the 65 MLGs, no one was fully homozygous at the 19 molecular marker loci. The parental isolate displayed heterozygosity at each of the 19 molecular marker loci. The number of homozygous genotypes varied as different loci, ranging from 7 (at marker locus 351) to 37 (at marker locus 159; Table 2). Fourteen of 19 KASP-SNP markers, including KASP55, KASP76, KASP85, KASP102, KASP150, KASP172, KASP177, KASP189, KASP190, KASP201, KASP203, KASP259, KASP271, and KASP359, fit an expected ratio of 1:2:1 for homozygous dominant alleles (AA):heterozygous (Aa):homozygous recessive alleles (aa) at each marker locus, with a probability value range of 0.021–0.982 ($P$>0.01). The correlation efficiency between the phenotype and KASP-SNP genotype was ultralow due to a highly minimal value ($r$ =4E−7). The frequency number of MLGs being homozygous at the 19 molecular marker loci nearly presented a Gaussian distribution, inferring that the S1 progeny population was established randomly regardless of either MLGs or progenies (Fig. 3). So, these KASP-SNP markers were suitable to construct a genetic map.

### 3.4. Linkage map

A genetic linkage map was created using 19 KASP-SNP markers, which showed a genetic distance of 441.0 cM among dominant avirulence (*Avr*) and recessive avirulence (*avr*) genes (Fig. 4; Appendix B). The maximum of genetic distance (44.49 cM) was detected between KASP271 and KASP177 markers, QTLs with virulence to the remaining 13 *Yr* loci, including *Yr2*, *Yr5*, *Yr8*, *Yr9*, *Yr27*, *Yr43*, *Yr44*, *YrSP*, *Yr76*, *YrExp2*, *Yr25*, *Yr28*, *Yr29*, was established on the linkage map. Only miss 8 QTLs with virulence to *Yr1*, *Yr3*, *Yr4*, *Yr6*, *Yr7*, *Yr17*, *Yr26*, and *YrA* loci.

As shown in the genetic map, four *Avr/avr* genes, *AvrYr2-1*, *avrYr9-1*, *avrYr44-1*, and *avrYrSP-1*, were mapped between KSAP271 and KASP177 markers, with an interval of 44.49 cM. *AvrYr5*, *avrYr9-2*, *AvrYr43-1*, and *avrYrSP-2* were located within a 37.13 cM interval between KASP177 and KASP259 markers. *Avr27-1* and *AvrYrExp2-1* were mapped between KASP259 and KASP150, and between KASP201 and KASP359, respectively. *avrYr25* was located between KASP203 and KASP359, and *avrYr29-1* was positioned between KASP359 and KASP76. *AvrYr8-1* was detected in an interval between KASP76 and KASP85. *AvrYr43-2*, *avrYr28-1*, and *avrYr29-2* were located between KASP102 and KASP159 on the linkage map. Both *AvrYr76* and *avrYr28-2* were mapped an interval between KASP159 and KASP351. The genetic map indicated that *AvrYr2-2* was constructed between KASP159 and KASP458, with an interval of 0.01 cM. *AvrYr8-1* showed the maximum of LOD, with a value of 20.313, and *AvrYr2-2*, however, had a LOD of 2.5277, which was minimum among overall *Avr*/*avr* genes on the linkage map (Appendix B).

### 3.5. Linkage and independence between *Avr* and *avr*

Determination of independence and linkage among avirulence/virulence genes was performed using Chi square test ($\chi^2$) (Appendix C). Totally, 231 values regarding probability were generated among 21 heterozygous loci, 61 of which are linked at confidence interval of greater than 95% ($P$<0.05), and 170 are independent at confidence interval of lower than 95% ($P$>0.05). At the level of lower than 0.05 ($P$<0.05), *AvrYr2* is linked to *AvrYr3*, and *avrYr17*. *AvrYr4* is associated with *AvrYr26*. *AvrYr5* is genetically linked to *avrYr25*.

Association of *AvrYr7* with *AvrYr27*, *AvrYr43*, *AvrYrExp2*, *avrYr6*, *avrYr17*, and *avrYr44* was observed. Genetic linkage is noted between *AvrYr8* and *avrYrSP*. Linkage of *AvrYr27* to *AvrYrExp2*, *avrYr6*, *avrYr17*, *avrYr44*, and *avrYrSP*. *AvrYr43* and *AvrYrExp2* was observed. It was noted that *AvrYr76* is linked to each of five *avr* genes including *avrYr17*, *avrYr25*, *avrYr28*, *avrYr44*, and *avrYrSP*. Genetic linkage was also detected between *AvrYrExp2* and *avrYr17*. For genetic linkage among *avr* genes ($P$<0.05), it was observed that *avrYr1* is linked to *avrYr6*, *avrYr25*, *avrYr28*, *avrYr29*, *avrYrSP*, and *avrYrA*, and that *avrYr6* is associated with *avrYr9*, *avrYr17*, *avrYr25*, *avrYr28*, *avrYr44*, *avrYrSP*, and *avrYrA*. The *avrYr9* gene is linked to *avrYr17*, *avrYr25*, *avrYr28*, *avrYr29*, *avrYr44*, *avrYrSP*, and *avrYrA*. Genetic linkage of *avrYr17* to *avrYr25*, *avrYr28*, *avrYr29*, *avrYr44*, *avrYrSP*, and *avrYrA* was observed. *avrYr25* is linked to *avrYr28*, *avrYr29*, and *avrYrSP*. *avrYr28* is associated with *avrYr29*, *avrYr44*, *avrYrSP*, and *avrYrA*. Linkage relationship was also observed between *avrYr29* and *avrYrSP*, between *avrYr29* and *avrYrA*, between *avrYr44* and *avrYrSP*, between *avrYr44* and *avrYrA*, and between *avrYrSP* and *avrYrA*. However, the remaining *Avr* or *avr* genes are independent, especially *AvrYr3* and *AvrYr26* that are completely independent but not linked with any of *Avr* and *avr* genes ($P$>0.05). *AvrYr4*, *AvrYr5*, *AvrYr8*, *AvrYr43*, and *AvrYrExp2* are linked with only one of 21 *Avr* or *avr* loci, and *AvrYr2* is associated with two of 21 *Avr* and *avr* loci ($P$<0.05).

## 4. Discussion

### 4.1. Occurrence of wheat stripe rust and race evolution of *P. striiformis* f. sp. *tritici* in Xinjiang

In Xinjiang, wheat stripe rust has frequently occurred, affecting large area of spring and winter wheat crop (Zhang 1962; Li *et al.* 2010; Zhan *et al.* 2016; Chen *et al.* 2023). The

**Table 2 Homozygous genetic loci and number of multilocus genotypes (MLGs) of 65 selfed progenies (S1 series) and parental isolate BGTB-1 of *Puccinia striiformis* f. sp. *tritici* based on 19 kompetitive allele-specific PCR-single nucleotide polymorphism (KASP-SNP) markers[1)]**

| Isolate | Genotypic locus | | | | | | | | | | | | | | | | | | | No. of MLGs | No. of homozygous loci |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 359 | 205 | 203 | 201 | 190 | 189 | 172 | 159 | 150 | 102 | 85 | 76 | 55 | 351 | 458 | 259 | 177 | 271 | 263 | | |
| BGTB-1 | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | 66 | 0 |
| S1-5 | NN | NN | AC | **CC** | AC | AC | AC | NN | AC | NN | AC | AC | AC | NN | AC | NN | **CC** | AC | AC | 19 | 2 |
| S1-37 | AC | AC | AC | **AA** | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | AC | **AA** | AC | 35 | 2 |
| S1-31 | NN | AC | AC | AC | AC | AC | AC | **AA** | AC | AC | AC | AC | **AA** | AC | AC | AC | NN | **CC** | AC | 57 | 3 |
| S1-26 | AC | NN | AC | **AA** | AC | AC | AC | NN | AC | **CC** | **CC** | AC | AC | AC | AC | AC | **AA** | AC | AC | 33 | 4 |
| S1-21 | AC | AC | **AA** | AC | AC | AC | AC | NN | AC | AC | AC | **AA** | AC | AC | AC | AC | **AA** | AC | **AA** | 8 | 4 |
| S1-47 | AC | NN | **CC** | AC | **CC** | AA | AC | **AA** | AC | AC | AC | AC | AC | NN | AC | AC | NN | AC | AC | 17 | 4 |
| S1-64 | AC | AC | AC | **CC** | AC | AC | **AA** | NN | AC | NN | NN | AC | **AA** | AC | AC | NN | AC | **CC** | AC | 53 | 4 |
| S1-1 | NN | NN | AC | AC | **AA** | AC | AC | NN | **CC** | NN | AC | AC | **AA** | **AA** | AC | AC | NN | AC | CC | 32 | 5 |
| S1-9 | AC | AC | AC | **AA** | AC | AC | **CC** | **AA** | AC | **CC** | AC | NN | **AA** | AC | AC | NN | AC | AC | AC | 59 | 5 |
| S1-4 | **AA** | AC | **AA** | AC | AC | AC | AC | **AA** | AC | NN | AC | AC | AC | AC | AC | AC | NN | **AA** | **AA** | 46 | 5 |
| S1-14 | **CC** | AC | AC | AC | AC | AC | AC | NN | **AA** | AC | **AA** | **AA** | AC | AC | AC | **CC** | NN | AC | AC | 1 | 5 |
| S1-8 | AC | AC | **AA** | AC | AC | AC | AC | **CC** | AC | NN | AC | AC | AC | **CC** | AC | **AA** | CC | AC | AC | 47 | 5 |
| S1-16 | NN | NN | AC | **AA** | AC | NN | AC | NN | AC | **AA** | AC | AC | **AA** | AC | AC | **AA** | NN | **CC** | AC | 12 | 5 |
| S1-41 | AC | AC | **AA** | **AA** | AC | AC | AC | **AA** | AC | AC | AC | **CC** | AC | AC | AC | **AA** | AC | AC | AC | 49 | 5 |
| S1-61 | **CC** | AC | AC | AC | AC | AC | AC | **CC** | AC | NN | **CC** | **AA** | AC | AC | **CC** | NN | NN | AC | AC | 51 | 5 |
| S1-2 | **CC** | AC | AC | **AA** | AC | AC | **AA** | AC | AC | NN | **AA** | AC | AC | AC | AC | AC | **CC** | **CC** | AC | 20 | 6 |
| S1-6 | NN | AC | AC | **AA** | AC | AC | AC | **CC** | AC | **AA** | **AA** | AC | AC | **CC** | AC | AC | NN | **CC** | AC | 31 | 6 |
| S1-18 | **AA** | AC | AC | AC | AC | AC | **CC** | **AA** | **CC** | AC | NN | AA | NN | AC | AC | **AA** | AC | AC | AC | 65 | 6 |
| S1-19 | AC | **AA** | **CC** | AC | AC | AC | AC | NN | **AA** | NN | AC | NN | NN | AC | **CC** | **AA** | NN | **CC** | AC | 23 | 6 |
| S1-29 | **CC** | AC | **AA** | AC | AC | NN | AC | NN | **CC** | AC | **AA** | AC | NN | NN | AC | AC | **AA** | AC | **AA** | 34 | 6 |
| S1-24 | AC | AC | **AA** | AC | **CC** | NN | AC | NN | AC | **AA** | AC | AC | NN | AC | AC | **AA** | **AA** | **CC** | AC | 22 | 6 |
| S1-35 | AC | AC | AC | **CC** | **AA** | **CC** | AC | NN | AC | AC | **AA** | AC | AC | AC | AC | **AA** | AC | **CC** | AC | 7 | 6 |
| S1-45 | NN | AC | **CC** | AC | AC | AC | NN | **AA** | **CC** | NN | **CC** | AC | NN | AC | NN | AC | **CC** | **AA** | AC | 13 | 6 |
| S1-39 | AC | AC | AC | AC | AC | AC | **AA** | **CC** | **AA** | AC | AC | **CC** | AC | AC | AC | **AA** | **AA** | AC | AC | 38 | 6 |
| S1-50 | **AA** | AC | AC | **CC** | AC | AC | AC | AC | NN | AC | **CC** | AC | AC | AC | AC | **CC** | **CC** | **CC** | AC | 27 | 6 |
| S1-58 | NN | **CC** | **CC** | AC | AC | AC | AC | AC | **CC** | NN | **CC** | AC | AC | AC | **AA** | **CC** | NN | AC | AC | 54 | 6 |
| S1-51 | NN | AC | AC | NN | **CC** | **AA** | AC | **AA** | NN | AC | AC | **CC** | AC | AC | AC | **CC** | NN | AC | **AA** | 14 | 6 |
| S1-10 | AC | AC | **AA** | **AA** | AC | AC | **AA** | **AA** | AC | **AA** | NN | AC | AC | AC | AC | AC | **CC** | AC | AA | 60 | 7 |
| S1-7 | AC | AC | **AA** | AC | AC | AC | **CC** | **CC** | **CC** | NN | AC | **AA** | AC | AC | NN | AC | **CC** | **AA** | AC | 63 | 7 |
| S1-15 | AC | AC | AC | AC | **CC** | **AA** | **CC** | **CC** | AC | AA | AC | AC | AC | AC | AC | AC | **CC** | **CC** | AC | 21 | 7 |
| S1-20 | NN | AC | AC | **CC** | AC | AC | AC | **AA** | **CC** | AC | **CC** | AC | AC | AC | **AA** | **AA** | AC | **CC** | AC | 56 | 7 |
| S1-22 | AC | AC | **CC** | AC | **AA** | **CC** | AC | AC | AC | AC | AC | **CC** | **CC** | AC | AC | **CC** | AC | **CC** | AC | 18 | 7 |
| S1-30 | **AA** | **AA** | AC | **AA** | **AA** | NN | AC | **AA** | AC | AC | AC | AC | **CC** | AC | **AA** | AC | AC | AC | AC | 48 | 7 |
| S1-32 | AC | AC | **AA** | **AA** | AC | AC | AC | NN | **CC** | AC | **CC** | AC | **AA** | AC | **CC** | **AA** | NN | AC | AC | 5 | 7 |
| S1-40 | **AA** | **CC** | **AA** | AC | AC | AC | **CC** | **AA** | **CC** | AC | **AA** | AC | AC | AC | AC | AC | AC | AC | AC | 29 | 7 |
| S1-59 | **CC** | AC | **CC** | **AA** | **CC** | NN | NN | **AA** | NN | AC | AC | AC | AC | AC | **AA** | **CC** | NN | AC | AC | 40 | 7 |
| S1-63 | AC | NN | AC | **CC** | **AA** | **CC** | **AA** | AC | AC | AC | **CC** | AC | NN | AC | **CC** | **CC** | NN | AC | AC | 39 | 7 |
| S1-3 | **AA** | NN | **CC** | **AA** | NN | AC | **CC** | **AA** | **AA** | AC | AC | **CC** | AC | AC | AC | AC | AC | **CC** | AC | 30 | 8 |
| S1-13 | **CC** | AC | **AA** | AC | AC | AC | NN | NN | NN | NN | **CC** | **CC** | NN | **AA** | AC | **CC** | NN | **AA** | **CC** | 58 | 8 |
| S1-28 | **AA** | **AA** | AC | AC | **CC** | AC | AC | **AA** | **AA** | AC | **CC** | AC | AC | AC | AC | AC | **AA** | **AA** | AC | 64 | 8 |
| S1-36 | **CC** | AC | **CC** | AC | **CC** | **AA** | **AA** | AC | AC | NN | AC | **CC** | AC | AC | AC | **AA** | AC | **AA** | AC | 24 | 8 |
| S1-46 | **CC** | NN | AC | AC | **AA** | **CC** | **AA** | **CC** | **AA** | NN | **CC** | AC | AC | NN | NN | AC | AC | AC | **CC** | 52 | 8 |
| S1-48 | AC | AC | **AA** | **CC** | AC | AC | **AA** | NN | AC | AC | **CC** | **AA** | **AA** | AC | AC | AC | **AA** | **CC** | AC | 26 | 8 |
| S1-54 | AC | NN | **CC** | **CC** | **AA** | **CC** | **AA** | AC | AC | AC | AC | **CC** | AC | AC | **CC** | NN | **CC** | AC | AC | 41 | 8 |
| S1-55 | **AA** | NN | **AA** | AC | AC | NN | **CC** | AC | **CC** | **AA** | NN | AC | **CC** | AC | AC | NN | NN | **CC** | **AA** | 9 | 8 |
| S1-11 | NN | AC | **AA** | **AA** | **CC** | **AA** | AC | **AA** | **CC** | NN | AC | AC | AC | **CC** | **AA** | AC | AC | **AA** | AC | 2 | 9 |
| S1-17 | AC | AC | **AA** | **CC** | AC | AC | AC | **AA** | **CC** | AC | AC | **CC** | **AA** | AC | **CC** | **AA** | AC | **AA** | AC | 61 | 9 |
| S1-27 | **AA** | **AA** | **AA** | AC | **CC** | **AA** | **CC** | **AA** | NN | AC | AC | AC | AC | AC | AC | AC | **AA** | AC | AA | 45 | 9 |
| S1-23 | **CC** | AC | AC | AC | **CC** | AC | **CC** | **AA** | AC | **CC** | **AA** | AC | **CC** | NN | AC | **AA** | AC | **CC** | AC | 11 | 9 |
| S1-42 | **AA** | NN | AC | **AA** | **AA** | **CC** | **CC** | AC | **CC** | **CC** | AC | **AA** | AC | AC | AC | **AA** | AC | AC | AC | 10 | 9 |
| S1-52 | AC | **CC** | **AA** | AC | AC | AC | **CC** | **AA** | **AA** | AC | **AA** | **CC** | AC | AC | **CC** | AC | AC | **AA** | AC | 37 | 9 |
| S1-12 | AC | AC | **CC** | **AA** | **AA** | **CC** | **CC** | **AA** | AC | AC | **AA** | **CC** | AC | AC | AC | **AA** | AC | **CC** | AC | 62 | 10 |
| S1-44 | **AA** | AC | AC | **AA** | **AA** | **CC** | **AA** | **AA** | **AA** | NN | AC | **CC** | NN | AC | AC | **AA** | **CC** | AC | AC | 44 | 10 |
| S1-60 | **CC** | AC | **CC** | AC | **AA** | **CC** | **AA** | **AA** | **CC** | NN | AC | AC | **CC** | AC | **CC** | **CC** | AC | AC | AC | 50 | 10 |

**(Continued on next page)**

**Table 2 (Continued from preceding page)**

| Isolate | Genotypic locus | | | | | | | | | | | | | | | | | | | No. of MLGs | No. of homozygous loci |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 359 | 205 | 203 | 201 | 190 | 189 | 172 | 159 | 150 | 102 | 85 | 76 | 55 | 351 | 458 | 259 | 177 | 271 | 263 | | |
| S1-53 | NN | **AA** | AC | **CC** | **AA** | **CC** | **AA** | **AA** | AC | AC | AC | AC | **CC** | AC | NN | **CC** | **AA** | **AA** | AC | 4 | 10 |
| S1-56 | **CC** | **CC** | AC | AC | **AA** | **CC** | **AA** | **CC** | **CC** | AC | **CC** | AC | AC | AC | **CC** | AC | NN | **AA** | AC | 16 | 10 |
| S1-25 | **AA** | **CC** | AC | **AA** | **CC** | **AA** | **CC** | **AA** | AC | **CC** | **AA** | AC | NN | AC | AC | **CC** | **AA** | AC | AC | 3 | 11 |
| S1-38 | **AA** | AC | **AA** | AC | **CC** | **AA** | **AA** | **AA** | **CC** | NN | **CC** | **CC** | AC | AC | AC | **AA** | AC | **CC** | AC | 42 | 11 |
| S1-62 | AC | AC | **CC** | **AA** | NN | AA | **CC** | AC | AC | AC | **CC** | **AA** | **AA** | AC | **AA** | **CC** | **CC** | **CC** | AC | 25 | 11 |
| S1-65 | AC | **CC** | **CC** | AC | **AA** | **CC** | **AA** | **AA** | **CC** | AC | **CC** | AC | **AA** | AC | AC | **CC** | AC | **AA** | AC | 28 | 11 |
| S1-33 | **CC** | AC | AC | **CC** | **AA** | **CC** | AC | NN | **CC** | **CC** | **CC** | AC | **AA** | AC | **CC** | **CC** | **CC** | **AA** | AC | 43 | 12 |
| S1-34 | NN | **AA** | AC | **CC** | **AA** | **CC** | AC | **AA** | **CC** | AC | **CC** | AC | AC | **CC** | **CC** | **CC** | NN | **AA** | **AA** | 36 | 12 |
| S1-43 | **AA** | **CC** | **AA** | **CC** | **AA** | NN | **AA** | NN | **CC** | NN | **AA** | AC | AC | **CC** | NN | **AA** | AC | **AA** | **AA** | 55 | 12 |
| S1-57 | **CC** | NN | **AA** | AC | **AA** | **CC** | **AA** | **AA** | **CC** | **AA** | AC | AC | **CC** | AC | **CC** | **AA** | **CC** | **AA** | AC | 15 | 13 |
| No. of heterozygous loci | 38 | 52 | 33 | 34 | 35 | 44 | 34 | 28 | 36 | 53 | 38 | 45 | 47 | 58 | 47 | 31 | 44 | 29 | 54 | | |
| Heterozygosity percentage (%) | 58.5 | 80 | 50.8 | 52.3 | 53.8 | 67.7 | 52.3 | 43.1 | 55.4 | 81.5 | 58.5 | 69.2 | 72.3 | 89.2 | 72.3 | 47.7 | 67.7 | 44.6 | 83.1 | | |

[1] AA and CC in bold indicated different homozygous genotype locus, and NN indicated missing data.

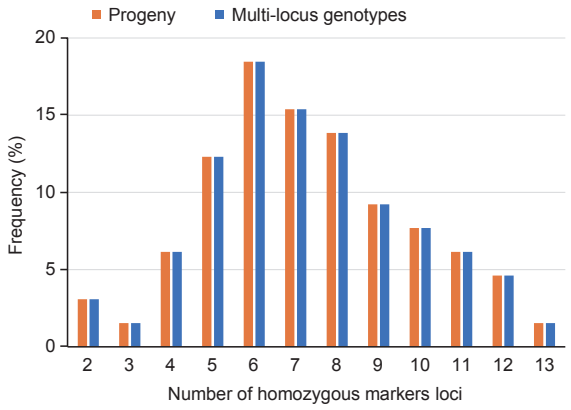



**Fig. 3 A histogram showing frequency of progeny with homozygous marker loci of 65 selfed progenies of the Xinjiang isolate BGTB-1 of *Puccinia striiformis* f. sp. *tritici*.**

past two decades field surveillance data showed increasing the severity of the disease (Li *et al.* 2010). That due to extensively growing of individual wheat variety, as mentioned by Li *et al.* (2010) that wheat cultivar Bima 1 was grown in a large scale since 1950s, resulted in the 1960 outburst of wheat stripe rust. During the 1980s–1990s, the wheat cultivar Tangshan 6898 had been widely cultivated and showed resistance against the stripe rust, but a few years later, it lost resistance and became highly susceptible. Eventually epidemic outbreak in 1993 cooccurred in Xinjiang. Since the 1990s, local wheat cultivars have been mainly grown in this region, which could later be responsible for wheat stripe rust epidemics.

In addition to highly susceptible wheat cultivars, race dynamics is largely contributed to severe occurrence of the disease in Xinjiang. According to annual race surveillance, initial emergence of predominant races of *P. striiformis* f. sp. *tritici* in Xinjiang is usually later than other inland regions in China, with an average of 3 to 5 years (Li and Zeng 2002; Li *et al.* 2010). For example, 1999s, race CYR31 was most dominant countrywide, with a frequency of 13.5%. However, this race was not found in Xinjiang along with CYR17, CYR23, and CYR24 (Li *et al.* 2010). Another example is race CYR32, which possessed an occurrence frequency of 28.8% and accounted for the most prevalence among all identified races across China. It maintained a high occurrence frequency until 2008. However, in Xinjiang, CYR32 was not mainly detected until 2004 but became predominant in 2007 (Li *et al.* 2010). In 2008, CYR33 and CYR32 developed as the predominant races in China, accounting for 25.9 and 25.2% in occurrence frequency, respectively. Meanwhile, in Xinjiang, CYR32 and Suwon 11-13 races were dominant, and CYR33 had not been detected before this year. In recent years, studies by Zhan *et al.* (2016) and Ma *et al.* (2023) reported that the population structure of *P. striiformis* f. sp. *tritici* in Xinjiang differed from those of other inland regions in China. Some old races have not been detected in inland regions for decades but can still maintain their survival in Xinjiang. Therefore, the rust pathogen in Xinjiang evolves uniquely. This mechanism is likely to affect the presence of old races and the emergence of predominant races later than other inland regions.

Altogether with previous studies (Tian *et al.* 2016, 2017; Yuan *et al.* 2018; Wang *et al.* 2018; Zhang *et al.* 2023), this study further confirmed that that an individual race being heterozygous for the avirulence loci can arise numerous new races (recombinants) by sexual reproduction. To some extent, members of these races have capacity of potentially conquering the resistance of genes in wheat varieties against the single race. Therefore, sexual reproduction is important for race evolution. For example, the isolate GS-2013 (Tian *et al.* 2017) and PSTv-11 (Zhang *et al.* 2023) were reported in previous studies, and the isolate BGTB-1 of this study generated virulent progenies to *Yr2* resistance gene after selfing due to heterozygosity for avirulence to *Yr2* locus. In contrast, Pinglan 17-7 (Tian *et al.* 2016) and CYR32 (Wang *et al.* 2018) isolates with homozygous avirulent to this locus did not produced any progeny virulent to *Yr2*. Sexual reproduction is also important for targeting selection of resistance genes, such as *Yr15* that was tested in this

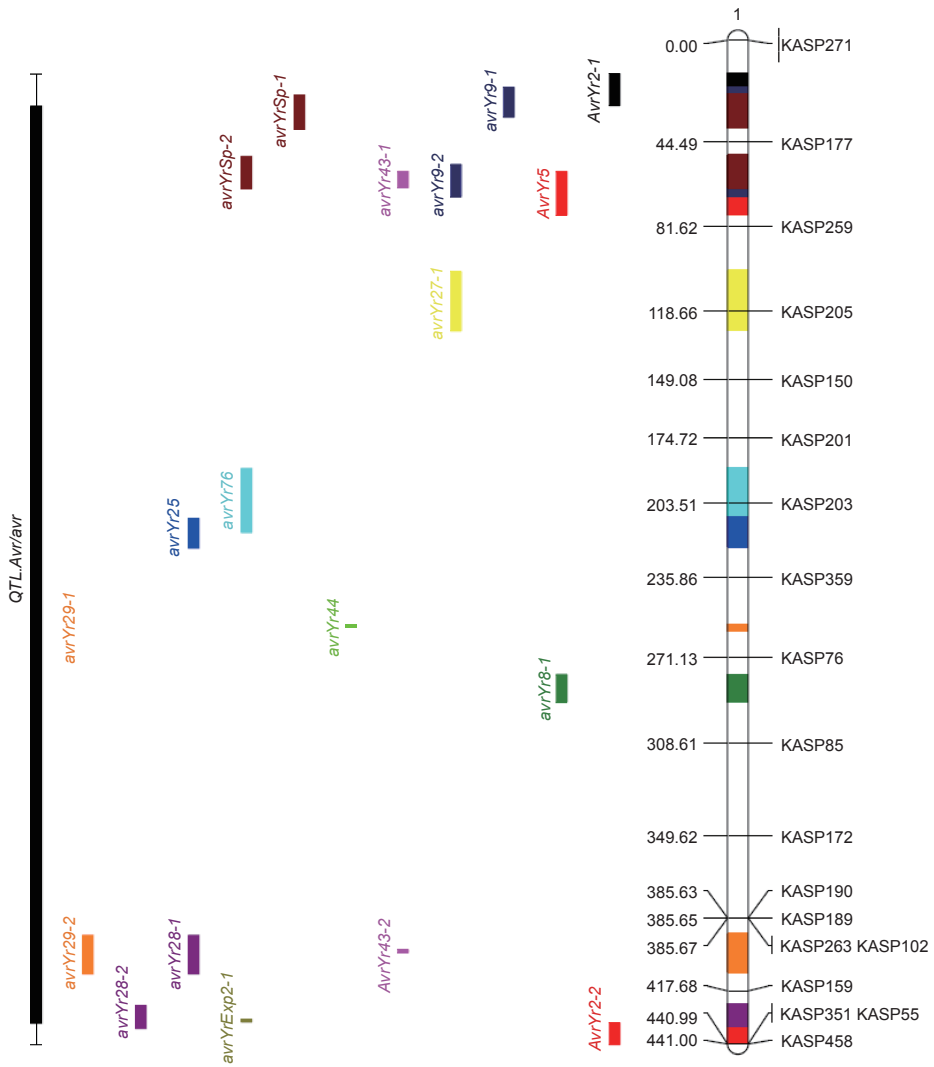



**Fig. 4 A quantitative trait loci (QTL) map of avirulence loci established based on 19 kompetitive allele-specific PCR-single nucleotide polymorphism (KASP-SNP) markers for 65 selfed progenies of the Xinjiang isolate BGTB-1 of *Puccinia striiformis* f. sp. *tritici*.** *Avr* indicates as dominant avirulence gene, and *avr* as recessive avirulence gene.

study as well as some other *Yr* resistance genes in terms of the understanding regarding homo- and hetero-zygosity for avirulence genes in respective progeny populations of the isolates. In China, many new races that emerged historically and subsequently developed to predominant race were characterized by low infections with sporulation on necrotic and chlorotic lesions on wheat cultivars carrying *Yr* gene(s) at initial emergence, and rapidly evolved to highly susceptible in subsequent years. For example, in the 1950s, race CYR1 infected wheat cultivar Bima 1 producing infection type 2 (scored by 0–4 scale) at adult-plant stage as primarily appeared (Liu and Meng 1957). Another sample is herein given that the race CYR34, previously known as pathotype G22-9, was first detected on wheat cultivar Chuanmai 42 (*Yr26*, =*Yr24*) with ITs 0-2, and kept thus low infections until 2008. This race lineage evolved to be highly susceptible to wheat Chuanmai 42, with IT $3^{-}$ in 2019, and IT 3 in 2010 (Kuang *et al.* 2013), and subsequently developed rapidly to be predominant (Liu *et al.* 2017).

### 4.2. Potential role of alternate host barberry in relation to wheat stripe rust

According to the monograph (IPlant 2025), in Xinjiang, six barberry species, embracing *B. heteropoda*, *B. iliensis*, *B. kaschgarica*, *B. sibirica*, *B. thunbergii*, and *B. ulicina*, have been recorded. Based on our field investigations in the past years in this region, three barberry species including *B. heteropoda*, *B. kaschgarica*, and *B. nummularia* (not recorded in the monograph) were visible, and are extensively distributed in geography, even growing as patches of bushes. Under artificial experimental conditions, it is testified that these three *Berberis* species are capable to serve as alternate hosts for *P. striiformis* f. sp. *tritici* (Zhuang *et al.* 2019). In spring, natural infection by the rusts on leaves of barberry bushes is quite common. To date, however, possibility of sexual reproduction of *P. striiformis* f. sp. *tritici* on alternate host barberry has been remained unknown due to the absence of isolate of the rust pathogen obtained from rusted barberry plants in fields. The present study demonstrated

selfing as a way of sexual reproduction, the S1 progenies of the isolate BGTB-1 generated a large proportion of new races that were different from their parental isolate in virulence. Same this rule implemented, under natural conditions, if rust infection of *P. striiformis* f. sp. *tritici* appear on susceptible barberry bushes is present, the rust pathogen can produce new races *via* sexual reproduction. Moreover, Xinjiang population of *P. striiformis* f. sp. *tritici* is genetically diverged from those of other inland regions of China (Zhan *et al.* 2016; Awais *et al.* 2022). As a result, further determination with regard to occurrence of sexual reproduction of the rust on susceptible barberry plants in fields in Xinjiang is necessary for understanding way of virulence variation of the rust pathogen, and for accounting for a high level of genetic diversity of the rust population in this region.

### 4.3. Inheritance complexity at *Yr* loci

A comparison of inheritance of diverse isolates was conducted by Wang *et al.* (2018) based on previous studies (Tian *et al.* 2016, 2017; Yuan *et al.* 2018), which showed difference and complexity of homozygous or heterozygous avirulence/virulence phenotype at *Yr* loci. Even if the same *Yr* locus, an isolate possibly presents same or different avirulence / virulence segregation ratio, hinting that avirulence or virulence for this locus is governed by same number or different number of genes with different gene effects.

Avirulence to *Yr*1 locus was controlled by a recessive gene, with an avirulence/virulence ratio of 1:3, in isolates GS-2013 and PSTv-11 (Tian *et al.* 2017; Yuan *et al.* 2018). However, the Xinjiang isolate BGTB-1 used in this study showed that avirulence to this resistance gene locus was controlled by two recessive genes (A:V=1:15) with gene complementation. This inconsistent inheritance seems to be not in accord with the gene-for-gene rule (Zhang *et al.* 2023). At *Yr1* locus, homozygous virulence (A:V=0:1) was observed selfed progenies of two isolates CYR32 and Pinglan 17-7 (Tian *et al.* 2016; Wang *et al.* 2018).

The avirulence or virulence gene corresponding to *Yr7* is controlled by one gene, but their traits are not the same. The avirulence of BGTB-1, TSA-6, GS-2013, 12–368 to *Yr7* locus was controlled by a single dominant gene (Tian *et al.* 2017; Xia *et al.* 2020; Zhang *et al.* 2023), whereas the avirulence of CYR32, Pinglan17-7, PSTv-11, 574232 to *Yr7* locus was controlled by a single recessive gene (Tian *et al.* 2016; Yuan *et al.* 2018; Wang *et al.* 2018; Mehmood *et al.* 2020).

At *Yr44* locus, Pinglan 17-7 and PSTv-11 isolates showed an avirulence:virulence ratio of 1:15 (Tian *et al.* 2016; Yuan *et al.* 2018), and both isolates TSA-6 and GS-2013 exhibited a segregation ratio of avirulence and virulence (A:V=3:13) (Tian *et al.* 2017; Zhang *et al.* 2023). However, isolates CYR32 (A:V=0:1), 12-368 (A:V=3:1), and BGTB-1 (A:V=1:3, this study) showed various avirulence and virulence ratios at this resistance locus (Wang *et al.* 2018; Xia *et al.* 2020).

For *Yr8* locus, homozygous avirulence was found selfed populations from two isolates CYR32 and Pinglan 17-7 (A:V=1:0) (Tian *et al.* 2016; Wang *et al.* 2018). Both isolates GS-2013 and TSA-6 showed same segregation ratio (A:V=3:1) (Tian *et al.* 2017; Zhang *et al.* 2023). Likewise, an avirulence and virulence segregation ratio of 15A:1V was shown in selfed progenies generated by isolates 12-368 (Xia *et al.* 2020), and BGTB-1 used in this study. Whereas, the isolate PSTv-11 showed a ratio of avirulence and virulence (A:V=1:3) at this resistance gene locus (Yuan *et al.* 2018).

At *Yr43* locus, both isolates Pinglan 17-7 and 12-368 showed same segregation ratio (A:V=3:1) (Tian *et al.* 2016; Xia *et al.* 2020). GS-2013 and TSA-6 isolates had an avirulence and virulence ratio of 13A:3V (Tian *et al.* 2017; Zhang *et al.* 2023). However, different avirulence and virulence ratio was observed at isolates CYR32 (A:V=1:3) (Wang *et al.* 2018), PSTv-11 (A:V=1:15) (Yuan *et al.* 2018), and BGTB-1 (A:V=15:1) in the present study.

Inconstant segregation ratios were also found at different isolates to *YrExp2* locus. The isolates GS-2013 and 12-368 showed an avirulence and virulence of 3A:1V (Tian *et al.* 2017; Yuan *et al.* 2018), and TSA-6 isolates and BGTB-1 used in this study displayed an 13A:3V ratio (Zhang *et al.* 2023). However, the two isolates CYR32 (A:V=1:3), and PSTv-11 (A:V=7:9) showed different segregation ratio at *YrExp2* locus (Yuan *et al.* 2018; Wang *et al.* 2018). The isolate Pinglan 17-7 showed homozygous virulence to this locus (Tian *et al.* 2016).

At *Yr17* locus, except both isolates PSTv-11 (Yuan *et al.* 2018) and BGTB-1 used in the present study that showed an avirulence and virulence of 7A:9V, isolates CYR32 (A:V=3:13), 12-368 (A:V=1:3), and TSA-6 (A:V=1:15) showed different segregation ratios (Wang *et al.* 2018; Xia *et al.* 2020; Zhang *et al.* 2023). Pinglan 17–7 and GS-2013 isolates showed homozygous virulence to this resistance locus (Tian *et al.* 2016, 2017).

At *Yr27* locus, apart from isolates CYR32 (A:V=3:13), TSA-6 (A:V=1:15), and BGTB-1 (A:V=9:7) that showed different avirulence and virulence ratios, four isolates including Pinglan 17-7 (Tian *et al.* 2016), GS-2013 (Tian *et al.* 2017), PSTv-11 (Yuan *et al.* 2018), and 12-368 (Xia *et al.* 2020) had same segregation ratio of avirulence and virulence (A:V=1:3).

In conclusion, according to genetic characteristic of the isolates that have been studied as above, the genetic regularity of avirulence or virulence genes in the wheat stripe rust pathogen is quite complex, and further studies are needed to better understand this mechanism.

### 4.4. Origin of *Yr5*-virulent races and heterozygosity of *Yr5* avirulence locus

*Yr5* was identified by Macer (1966), and further studied by Law (1976) and Chen and Line (1992). Pathogenic races to wheat stripe rust resistance gene *Yr5*, 360 E137 A- and 360 E137 A+, were first detected in Australia in 1984, while it was not detected in subsequent years (Wellings and McIntosh 1990). These two Australian races exhibited narrow virulence spectrums. The former had virulence to *Yr2* and *Yr5* loci, and the latter was virulent to *YrA* in addition to *Yr2* and *Yr5* loci. Subsequently, the race 13 (67S8) was detected in 1934 in India (Gangwar *et al.* 2022), in which it was reported with susceptibility to *Triticum spelta album* (*Yr5*) in 1986 (Nagarajan 1986). It were more virulent than both Australian races, with virulence to *Yr1*, *Yr7*, and Suwon 92×Omar

(resistance genes unknown) besides *Yr5*. This race was sensitive to high temperatures and produced necrosis to shift virulence to avirulence to *Yr5* once temperatures exceed 20°C under laboratory conditions (Nagarajan 1986). Since then, neither of races again found virulent to this resistance gene has been detected in India (Aggarwal *et al.* 2018; Singh *et al.* 2020; Gangwar *et al.* 2022). Most recently, races with virulence to *Yr5* have been detected one after another in 2017 in China (Zhang *et al.* 2020), and in 2020 in Turkey (Tekin *et al.* 2021). In China, the *Yr5*-virulent races were collected from wheat fields adjacent to alternate host barberry (*Berberis shensiana*) bushes in western Shaanxi Province, and were inferred that it could originate from aeciospore inoculum sexually produced on barberry after infection by the rust pathogen. In our recent study by Zhao *et al.* (2023), some isolates of the rust pathogen, isolated from naturally rust-infected barberry bushes in this region in spring in 2016, were found to show low infection types (ITs, 1 or 2 based on 0–4 scale, data not shown) with sporulation and necrosis on leaves of *Triticum aestivum* subsp. *spelta* var. *album* (*Yr5*) genotypes after inoculation with aeciospores of aecial samples. However, no isolate with high virulence to *Yr5* has been directly isolated from rusted barberry to support this inference. In this study, the parental isolate BGTB-1 was avirulent to *Yr5*, to which, whereas, 18 out of 65 S1 progenies were manifested virulent. In addition, some isolates of a natural population collected from Xinjiang showed low infections (ITs, 2 and 3), and high infection (IT 9) on Yr5 line according to the 0–9 scale (unpublished data). Based on the results mentioned above, this study provided the theoretical basis for explaining that *Yr5*-virulent isolates in China likely derived from sexual reproduction.

Inheritance of isolates avirulent to *Yr5*, including Pinglan 17-7 (Tian *et al.* 2016), GS-2013 (Tian *et al.* 2017), CYR32 (Wang *et al.* 2018), and PSTv-11 (Yuan *et al.* 2018), has been studied. These four isolates showed homozygous avirulence to *Yr5* locus due to no change in virulence among their respective selfed progenies. Most recently, however, our study demonstrated the isolate TSA-6, with virulence to *Yr5*, was heterozygous owing to avirulence and virulence segregation at this resistance locus (Zhang *et al.* 2023). In the present study, the Xinjiang isolate BGTB-1, avirulent to *Yr5*-carrying wheat genotype *T. aestivum* subsp. *spelta* var. *album*, and *Yr5* near isogenic line (NIL), segregated at *Yr5* locus, with an avirulence and virulence ratio (A:V=3:1), in consistent with the study of the *Yr5*-virulent isolate TSA-6. Thus, this change in virulence hinted that the Xinjiang isolate BGTB-1 was heterozygous at *Yr5* locus in virulence. These results implied that heterozygosity of an isolate at a resistance locus is benefit for virulence variation, whereas homozygosity is not. Although isolates with virulence to *Yr5* have been detected in a few countries, this gene still maintains effective resistance to striping rust on wheat in most regions of the world. However, the *Yr5*-virulent race and its derivatives were reported to possess high parasitic fitness, alerting the potential risk (Zhang *et al*. 2024), and driving us to strengthen the monitoring of the race dynamics.

## 5. Conclusion

Phenotyping and genotyping of a selfed progeny population derived from a Xinjiang single-urediospore isolate BGTB-1 of *P. striiformis* f. sp. *tritici* (the cause of wheat stripe rust) revealed that the progenies of the isolate can produce a quite high proportion of new races, with outstanding virulence variation. It indicates that selfing, as a way of sexual reproduction, is an important approach for generation of new races in this region. Avirulence and virulence segregation of the Xinjiang isolate at 25 *Yr* resistance loci tested was different. The progenies showed different homozygosity and heterozygosity to the 19 KASP-SNP marker loci, displaying that inheritance of the Xinjiang isolate is complicated. At *Yr5* locus, the Xinjiang isolate, with avirulence to this locus, is heterozygous due to detection of *Yr5*-virulent isolates among the progenies. Such variation in virulence of the isolate *via* selfing can be used to explain our speculation that *Yr5*-virulent races, detected in recent years, resulted from sexual reproduction. The results of this study can be utilized to account for virulence evolution of the rust in Xinjiang, and are benefit for integrated management of wheat stripe rust in this region.

## Acknowledgements

This work was financially supported by Xinjiang Major Science and Technology Projects, China (2023A02009).

## Declaration of competing interest

The authors declare that they have no conflict of interest.

## Declaration of generative AI and AI-assisted technologies in the writing process

The authors declare that they did not use AI in the preparation and writing of this manuscript.

**Appendices** associated with this paper are available at https://doi.org/10.1016/j.jia.2024.04.023

## References

Aggarwal R, Kulshreshtha D, Sharma S, Singh V K, Manjunatha C, Bhardwaj S C, Saharan M S. 2018. Molecular characterization of Indian pathotypes of *Puccinia striiformis* f. sp. *tritici* and multigene phylogenetic analysis to establish inter- and intraspecific relationships. *Genetics and Molecular Biology*, **41**, 834–842.

Awais M, Ali S, Ju M, Liu W, Zhang G S, Zhang Z D, Li Z J, Ma X Y, Wang L, Du Z M, Tian X X, Zeng Q D, Kang Z S, Zhao J. 2022. Countrywide inter-epidemic region migration pattern suggests the role of southwestern population in wheat stripe rust epidemics in China. *Environmental Microbiology*, **24**, 4684–4701.

Chen L, Awais M, Yang H, Shen Y Y, Li G K, Gao H F, Ma J B. 2023. Races *CYR34* and *Suwon11-1* of *Puccinia striiformis* f. sp. *tritici* played an important role in causing the stripe rust epidemic in winter wheat in Yili, Xinjiang, China. *Journal of Fungi*, **9**, 436.

Chen X M. 2005. Epidemiology and control of stripe rust (*Puccinia striiformis* f. sp. *tritici*) on wheat. *Canadian Journal of Plant Pathology*, **27**, 314–337.

Chen X M, Line R F. 1992. Identification of stripe rust resistance genes in wheat genotypes used to differentiate North American races of *Puccinia striiformis*. *Phytopathology*, **82**, 1428–1434.

Chen X M, Line R F, Leung H. 1993. Relationship between virulence variation and DNA polymorphism in *Puccinia striiformis*. *Phytopathology*, **83**, 1489–1497.

Doling D A, Doodson J K. 1968. The effect of yellow rust on the yield

of spring and winter wheat. *Transactions of the British Mycological Society*, **51**, 427–434.
Gangwar O P, Kumar S, Bhardwaj S C, Prasad P, Lata C, Adhikari S, Singh G P. 2022. Elucidating the population structure and genetic diversity of Indian *Puccinia striiformis* f. sp. *tritici* pathotypes based on microsatellite markers. *Phytopathology*, **112**, 1444–1453.
IPlant. 2025. Plant Plus of China (e-version). https://www.iplant.cn/
Jin Y, Szabo L J, Carson M. 2010. Century-old mystery of *Puccinia striiformis* life history solved with the identification of *Berberis* as an alternate host. *Phytopathology*, **100**, 432–435.
Kosman E, Dinoor A, Herrmann A, Schachtel G A. 2008. Virulence analysis tool (VAT). User manual. [2023-9-21]. http://www.tau.ac.il/lifesci/departments/plant_s/members/kosman/VAT.html
Kuang W J, Zhang G S, Zhang Z Y, Ji H L, Shen L, Ni J Y, Wang Z H, Peng Y L. 2013. Studies on virulence of *avrYr10/24/26/ch42* mutants of *Puccinia striiformis* f. sp. *tritici*. *Southwest China Journal of Agricultural Science*, **26**, 2323–2331. (in Chinese)
Law C N. 1976. Genetic control of yellow rust resistance in *T. spelta album*. In: *Plant Breeding Institute Annual Report 1975*. Plant Breeding, Cambridge, UK. pp. 108–109.
Li J, Zeng J, Jiang Y Y, Li H. 2010. Occurrence and epidemics of wheat stripe rust in Xinjiang. *China Plant Protection*, **30**, 16–19. (in Chinese)
Li Z Q, Zeng S M. 2002. *Wheat Rusts in China*. China Agriculture Press, Beijing. p. 379. (in Chinese)
Li Z Q. 1980. Primary studies on the loss of the resistance of wheat cultivars to stripe rust in China. *Northwest A&F University (Natural Science Edition)*, **3**, 83–92. (in Chinese)
Line R F, Qayoum A. 1992. Virulence, aggressiveness, evolution, and distribution of races of *Puccinia striiformis* (the cause of stripe rust of wheat) in North America, 1968-87. *US Department of Agriculture, Agricultural Research Service, Technical Bulletin*, 1788.
Liu B, Liu T G, Zhang Z Y, Jia Q Z, Wang B T, Gao L, Peng Y L, Jin S L, Chen W Q. 2017. Discovery and pathogenicity of CYR34, a new race of *Puccinia striiformis* f. sp. *tritici* in China. *Acta Phytopathologica Sinica*, **47**, 681–687. (in Chinese)
Liu H W, Meng X C. 1957. On the loss of wheat cultivar Bima 1 to stripe rust. *Shaanxi Journal of Agricultural Science*, **6**, 329–334. (in Chinese)
Ma J B, Awais M, Chen L, Yang H, Lai H L, Shen Y Y, Wang H Q, Li G K, Gao H F. 2023. Identification of *Puccinia striiformis* races from the spring wheat crop in Xinjiang, China. *Frontiers in Plant Science*, **14**, 1273306.
Macer R C F. 1966. The formal and monosomic genetic analysis of stripe rust (*Puccinia striiformis*) resistance in wheat. In: Mackey J, ed., *Proceedings of the Second International Wheat Genetics Symposium, Lund*. *Hereditas*, **2(Suppl.)**, 127–142.
McIntosh R, Mu J M, Han D J, Kang Z S. 2018. Wheat stripe rust resistance gene *Yr24*/*Yr26*: A retrospective review. *The Crop Journal*, **6**, 321–329.
Mehmood S, Sajid M, Husnain S K, Zhao J, Huang L L, Kang Z S. 2020. Study of inheritance and linkage of virulence genes in a selfing population of a Pakistani dominant race of *Puccinia striiformis* f. sp. *tritici*. *International Journal of Molecular Science*, **21**, 1685.
Meng Y, Yang C B, Jiang S C, Huang L L, Kang Z S, Zhan G M. 2020. Development and evaluation of SNP molecular markers of wheat stripe rust based on KASP technology. *Journal of Plant Protection*, **47**, 65–73. (in Chinese)
Nagarajan S. 1986. Race 13 (67S8) of *Puccinia striiformis* virulent on *Triticum spelta var. album* in India. *Plant Disease*, **70**, 173.
Rapilly F. 1979. Yellow rust epidemiology. *Annual Review of Phytopathology*, **17**, 59–73.
Rodriguez-Algaba J, HovmØller M S, Villegas D, Cantero-Martínez C, Jin Y, Justesen A F. 2021. Two indigenous *Berberis* species from Spain were confirmed as alternate hosts for the yellow rust fungus *Puccinia striiformis* f. sp. *tritici*. *Plant Disease*, **105**, 2281–2285.
Roelfs A P. 1978. *Estimated Losses Causes by Rust in Small Grain in Cereals in United States 1918–1976*. Miscellaneous Publication (United States Department of Agricultura). No. 1363. p. 85.
Singh H, Kaur J, Bala R, Srivastava P, Bains N S. 2020. Virulence and genetic diversity of *Puccinia striiformis* f. sp. *tritici* isolates in sub-mountainous area of Punjab, India. *Phytoparasitica*, **48**, 383–395.
Tekin M, Cat A, Akan K, Catal M, Akar T. 2021. A new virulent race of wheat stripe rust pathogen (*Puccinia striiformis* f. sp. *tritici*) on the resistance gene *Yr5* in Turkey. *Plant Disease*, **105**, 3292.
Tian Y, Zhan G M, Chen X M, Tungruentragoon A, Lu X, Zhao J, Huang L L, Kang Z S. 2016. Virulence and simple sequence repeat marker segregation in a *Puccinia striiformis* f. sp. *tritici* population produced by selfing a Chinese isolate on *Berberis shensiana*. *Phytopathology*, **106**, 185–191.
Tian Y, Zhan G M, Lu X, Zhao J, Huang L L, Kang Z S. 2017. Determination of heterozygosity for avirulence/virulence loci through sexual hybridization of *Puccinia striiformis* f. sp. *tritici*. *Frontiers of Agricultural Science and Engineering*, **4**, 48–58.
Voorrips R E. 2002. MapChart: Software for the graphical presentation of linkage maps and QTLs. *Journal of Heredity*, **93**, 77–78.
Wan Q, Liang J M, Luo Y, Ma Z H. 2015. Population genetic structure of *Puccinia striiformis* in Northwestern China. *Plant Disease*, **99**, 1764–1774.
Wang L, Zheng D, Zuo S X, Chen X M, Zhuang H, Huang L L, Kang Z S, Zhao J. 2018. Inheritance and linkage of virulence genes in Chinese predominant race CYR32 of the wheat stripe rust pathogen *Puccinia striiformis* f. sp. *tritici*. *Frontiers in Plant Science*, **9**, 120.
Wellings C R, Mcintosh R A. 1990. *Puccinia striiformis* f. sp. *tritici* in Australasia: Pathogenic changes during the first 10 years. *Plant Pathology*, **39**, 316–325.
Wellings C R. 2011. Global status of stripe rust: a review of historical and current threats. *Euphytica*, **179**, 129–141.
Wu L R, Yang H A, Yuan W H, Song W Z, Yang J X, Li Y F, Bi Y Q. 1993. On the physiological specialization of stripe rust on wheat in China during 1985–1990. *Acta Phytopathologica Sinica*, **23**, 269–274. (in Chinese)
Xia C J, Lei Y, Wang M N, Chen W Q, Chen X M. 2020. An avirulence gene cluster in the wheat stripe rust pathogen (*Puccinia striiformis* f. sp. *tritici*) identified through genetic mapping and whole-genome sequencing of a sexual population. *mSphere*, **5**, e00128–20.
Xiang C, Feng J Y, Wang M N, Chen X M, See D R, Wan A M, Wang T. 2016. Molecular mapping of stripe rust resistance gene *Yr76* in winter club wheat cultivar Tyee. *Phytopathology*, **106**, 1186–1193.
Yuan C Y, Wang M N, Skinner D Z, See D R, Xia C J, Guo X H, Chen X M. 2018. Inheritance of virulence, construction of a linkage map, and mapping dominant virulence genes in *Puccinia striiformis* f. sp. *tritici* through characterization of a sexual population with genotyping-by sequencing. *Phytopathology*, **108**, 133–141.
Zhan G M, Wang F P, Wan C P, Han Q M, Huang L L, Kang Z S, Chen X M. 2016. Virulence and molecular diversity of the *Puccinia striiformis* f. sp. *tritici* population in Xinjiang in relation to other regions of western China. *Plant Disease*, **100**, 99–107.
Zhang G S, Liu W, Wang L, Ju M, Tian X X, Du Z M, Kang Z S, Zhao J. 2023. Genetic characteristics and linkage of virulence genes of the *Puccinia striiformis* f. sp. *tritici* TSA-6 isolate to *Yr5* host resistance. *Plant Disease*, **107**, 688–700.
Zhang G S, Sun M D, Ma X Y, Liu W, Du Z M, Kang Z S, Zhao J. 2024. *Yr5*-virulent races of *Puccinia striiformis* f. sp. *tritici* possess relative parasitic fitness higher than current main predominant races and potential risk. *Journal of Integrative Agriculture*, **23**, 2674–2685.
Zhang G S, Zhao Y Y, Kang Z S, Zhao J. 2020. First report of a *Puccinia striiformis* f. sp. *tritici* race virulent to wheat rust resistance gene *Yr5* in China. *Plant Disease*, **104**, 284.
Zhang Z Z. 1962. Occurrence and control strategy of wheat stripe rust in Xinjiang. *Xinjiang Agricultural Sciences*, **3**, 99–100. (in Chinese)
Zhuang H, Zhao J, Huang L L, Kang Z S, Zhao J. 2019. Identification of three *Berberis* species as potential alternate hosts for *Puccinia striiformis* f. sp. *tritici* in wheat-growing regions of Xinjiang, China. *Journal of Integrative Agriculture*, **18**, 2786–2792.
Zhao Y Y, Huang X L, Li Q, Huang L L, Kang Z S, Zhao J. 2023. Virulence phenotyping and molecular genotyping reveal high diversity within and strong gene flow between the *Puccinia striiformis* f. sp. *tritici* populations collected from barberry and wheat in Shaanxi Province of China. *Plant Disease*, **107**, 701–712.

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