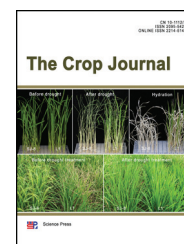
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Reactions of *Triticum urartu* accessions to two races of the wheat yellow rust pathogen

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ABSTRACT

Triticum urartu (AA, $2n = 2x = 14$), a wild grass endemic to the Fertile Crescent (FC), is the progenitor of the A subgenome in common wheat. It belongs to the primary gene pool for wheat improvement. Here, we evaluated the yellow rust (caused by *Puccinia striiformis* f. sp. tritici, Pst) reactions of 147 *T. urartu* accessions collected from different parts of the FC. The reactions varied from susceptibility to strong resistance. In general, there were more accessions with stronger resistance to race CYR33 than to CYR 32. In most cases the main form of defense was a moderate resistance characterized by the presence of necrotic/chlorotic lesions with fewer Pst uredinia on the leaves. Forty two accessions displayed resistance to both races. Histological analysis showed that Pst growth was abundant in the compatible interaction but significantly suppressed by the resistant response. Gene silencing mediated by *Barley stripe mosaic virus* was effective in two *T. urartu* accessions with different resistance responses, indicating that this method can expedite future functional analysis of resistance genes. Our data suggest that *T. urartu* is a valuable source of resistance to yellow rust, and represents a model for studying the genetic, genomic and molecular basis underlying interaction between wheat and Pst.

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1. Introduction

Wheat is the most widely cultivated staple food crop in the world. Although several forms of wheat are cultivated, hexaploid common wheat (*Triticum aestivum*, AABBDD, $2n =$

$6x = 42$) accounts for most of the total wheat production [1]. Wheat is threatened by several major diseases, including yellow or stripe rust caused by *Puccinia striiformis* f. sp. tritici (Pst) [2, 3]. At present, more than 80% of the global wheat production is affected by yellow rust, causing more than five

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million metric tons of yield loss annually [3, 4]. Use of resistant cultivars is the most effective and economic measure for preventing losses to yellow rust; however, resistance when controlled by a single gene is vulnerable to the evolution of new *Pst* races [2–5]. Consequently, identification, characterization and deployment of new sources of resistance are a top priority in the battle against yellow rust [2–6].

Common wheat evolved in the Fertile Crescent (FC) about ten thousand years ago, with its A and D genomes donated by the diploid Triticeae species *T. urartu* (AA, $2n = 2x = 14$) and *Aegilops tauschii* (DD, $2n = 2x = 14$), respectively [7]. The B genome originated from *Aegilops speltoides* or a close relative. The first two grasses belong to the primary gene pool for wheat improvement and genes from *T. urartu* and *Ae. tauschii* can be transferred to common wheat by wide hybridization [8, 9]. To date, more than 70 permanently designated genes (loci/alleles) have been reported to confer resistance to yellow rust [10–12], several of which (*Yr10*, *Yr18*, *Yr36*, and *Yr46*) have been molecularly cloned [13–16]. No formally named yellow rust resistance gene in common wheat is derived from *T. urartu*, although several of them (e.g., *Yr8*, *Yr9*, *Yr28*, *Yr37*, *Yr38*, *Yr40*, and *Yr42*) originated from related species [17]. The yellow rust responses of the *T. urartu* accessions from different parts of the Fertile Crescent (FC) are still not widely reported, with only one report published 20 years ago documenting the reactions of 16 *T. urartu* accessions of unknown geographic origin to a single Mexican *Pst* isolate [18]. Furthermore, genes underlying interaction between *T. urartu* and *Pst* have not been studied at the genetic and molecular levels.

We previously determined the draft genome sequence of *T. urartu* [19], and found that this species is an efficient model for analyzing the molecular basis of resistance to the wheat powdery mildew fungus at the genome-wide level [20]. These findings, together with little knowledge of the interaction between *T. urartu* and *Pst*, prompted us to evaluate if *T. urartu* can be developed as an effective model for studying the genetics of stripe rust resistance in wheat. Towards this end we evaluated the reactions of 147 *T. urartu* accessions from six countries in the FC to major Chinese *Pst* races CYR32 and CYR33 [2]. The infection processes in two *T. urartu* accessions with different reactions were examined. Finally, we tested if the gene silencing approach mediated by *Barley stripe mosaic virus* (BSMV) might be effective in different *T. urartu* accessions in order to employ this strategy in future genetic analyses of interaction between *T. urartu* and *Pst*.

2. Materials and methods

2.1. Plant materials, inoculation conditions and phenotyping

The 147 *T. urartu* accessions and their geographic origins are listed in Table S1. They were grown in a growth chamber at a day/night temperature cycle of 22/20 °C, 16 h light/8 h darkness photoperiod, and 70% relative humidity. *Pst* races CYR32 and CYR33 were maintained in the wheat disease assessment facility of the Institute of Genetics and Developmental Biology, Chinese Academy of Sciences. For each accession and each *Pst* race, 10 uniformly developed seedlings were

inoculated at the two-leaf stage. Briefly, the seedlings were sprayed with 0.02% Tween 20 in distilled water (v/v); the fresh *Pst* spores diluted with talcum powder were then applied to the surface of the leaves using a larynx atomizer. The inoculated seedlings were immediately placed in darkness for 24 h at 16 °C with 90% moisture [21]. Afterwards, the seedlings were returned to the above growth conditions, and reactions were recorded at 14 days post inoculation (dpi).

Phenotype scoring followed a 0–4 scale [21] with some modifications. Specifically, we classified the reactions into five types, viz., IT0 (no visible symptoms; immune), IT1 (necrotic/chlorotic spots without uredinia; high resistance), IT2 (necrotic/chlorotic spots with moderate uredinia; moderate resistance), IT3 (moderate to abundant uredinia with chlorosis; moderate susceptibility), and IT4 (abundant uredinia without necrosis and chlorosis; high susceptibility) (Fig. 1). Tests were repeated to check for reproducibility of reactions.

2.2. Histological staining of *Pst* growth

Histological staining was carried out to examine the development of *Pst* structures in the leaves of inoculated *T. urartu* seedlings as described previously [22, 23]. Accessions G1812 and PI 428322, which were susceptible and resistant to CYR33, respectively (Table S1), were used in this experiment. Leaves

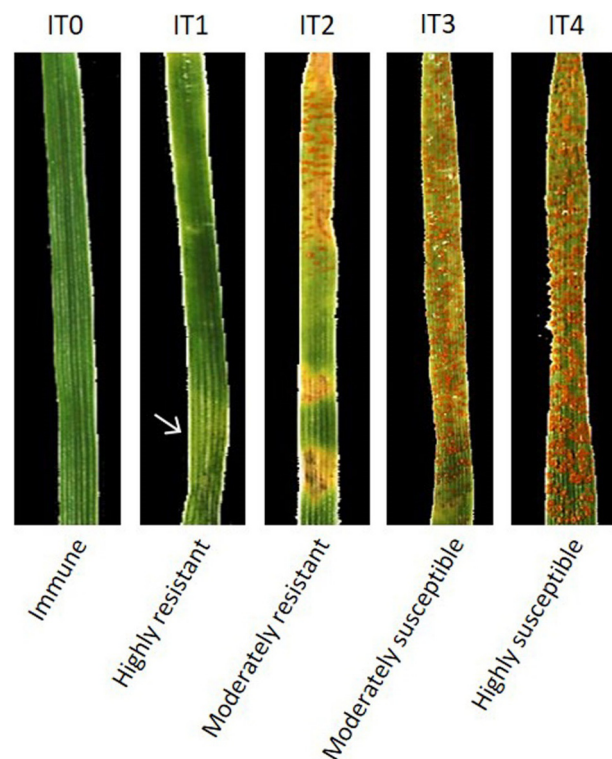


Fig. 1 – Representative yellow rust reactions of *T. urartu* accessions. Accessions exhibiting these infection types (ITs) were regarded as being immune (IT0), highly resistant (IT1), moderately resistant (IT2), moderately susceptible (IT3), and highly susceptible (IT4). The arrow indicates a chlorotic spot. These reactions were obtained with *Pst* race CYR33.

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