## Features of the interaction of the effector genes *ToxA* and *ToxB* with the susceptibility genes *Tsn1* and *Tsc2* in different species of wheat

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The Triticum aestivum-Pyrenophora tritici-repentis pathosystem is well studied in durum and common wheat. The purpose of our study is to assess the distribution of the sensitivity genes Tsn1 (5BL) and Tsc2 (2BL) in species of the genus Triticum L. from the VIR collection and the response of wheat accessions containing these genes to infection by isolates with complementary genes effectors ToxA and ToxB. All 72 accessions of 16 wheat species were evaluated for resistance to two isolates ToxA<sup>+</sup> originating from Kazakhstan and Russia, and one ToxB<sup>+</sup> from Greece. Using gene-specific primers, Tsn1 and Tsc2 were not detected in the diploid species T. urartu, T. boeoticum, and T. monococcum. In the wild tetraploid wheats T. dicoccoides and T. araraticum and six cultivated tetraploid species, the Tsc2 gene and the polymorphism of the Tsn1 were detected. In the *T. timopheevii*, only the *Tsc2* gene was identified. Polymorphism for both genes was observed in all hexaploid species (genome BBAADD). The manifestation of necrosis and/or chlorosis on wheat leaves is observed when the plant and the pathogen have both of the dominant genes Tsn/ToxA and/or Tsc2/ToxB, respectively. All diploid wheat species had no susceptibility reactions. The gene-on-gene Tsc2/ToxB gene interaction was observed in accessions of T. aethiopicum and T. turgidum, as well as for most accessions of hexaploid species. All accessions of *T. dicoccoides* and *T. dicoccum*, despite the presence of the Tsc2 gene, were resistant to the ToxB<sup>+</sup> isolate. The reasons for this resistance are being studied. When evaluating the interaction of the Tsn1/ToxA, 11 accessions of different tetra- and hexaploid species of wheat aroused particular interest. The *Tsn1* was not detected in these accessions, but a strong necrosis was observed when infected with ToxA<sup>+</sup> isolate, which is possibly due to the presence of other unknown susceptibility and effector genes.

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