

USING WILD SPECIES OF THINOPYRUM GENUS IN BREEDING WHEAT RESISTANT TO FUSARIUM HEAD BLIGHT

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In recent years, climatic changes have favoured the spreading of previously uncommon fungal diseases, including Fusarium Head Blight (FHB), in several wheat growing areas worldwide. Because of the relevant economic damages to wheat production and quality caused by FHB, and the scarcity of resistant sources among cultivated *Triticum* species, we have looked outside the primary gene pool and targeted wild wheat relatives of the *Thinopyrum* genus as promising sources of effective resistance. Two such species are the decaploid *Thinopyrum ponticum* (carrier of an *el*-type genome) and the diploid *Thinopyrum elongatum* (carrier of an E-type genome). In both species, one or more genes/QTLs for FHB resistance are located on the long arm of a homoeologous chromosome to those of wheat group 7 (named *7el₂L* and *7EL*, respectively). Resorting to chromosome engineering, i.e. aiming at the recombination-based transfer into wheat of small, *7el₂L* or *7EL* chromosomal segments carrying the desired gene(s), we were aware of the different cytogenetic affinity relating the donor and the recipient chromosomes. In fact, bread wheat substitution lines for the *7el₂L* arm or the complete *7E* chromosome for the wheat *7D* counterparts were employed as donor lines, while durum and bread wheat recombinant lines, already carrying *Th. ponticum 7el₁L* portions, have been chosen as recipient lines. These contain the *7el₁L*-derived *Lr19+Yp+Sr25* genes but lack any effective FHB resistance gene. Almost complete homology has been verified to exist between *7el₁L* and *7el₂L* (presumably originating from different *Th. ponticum* accessions), whereas that between the former and *7EL* of *Th. elongatum* is only partial (around 20% pairing as from our records). Thus, homologous recombination is expected to allow pyramiding of all the desired *7el₁L* + *7el₂L* traits with relative ease, while the use of wheat homoeologous pairing mutants (*ph1* and *ph2* mutations) is being included in the transfer schemes to enhance *7el₁L*-*7EL* recombination frequency. The multi-targeted and multi-genomic transfers are being aided by development of suitable polymorphic markers along the *7L* arms and application of GISH (Genomic In Situ Hybridization) in somatic and meiotic cells. As a result, the first recombinants with the desired gene combinations are being isolated.