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Poster Communication Abstract – 3.05

REINFORCING AND BROADENING RESISTANCE AGAINST *FUSARIUM* DISEASES IN DURUM WHEAT BY AN *UDP-GLUCOSYLTRANSFERASE* TRANSGENE AND ITS PYRAMIDING WITH A *PECTIN METHYL ESTERASE INHIBITOR* TRANSGENE

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Fusarium Head Blight, Fusarium Crown Rot, type II resistance, deoxynivalenol, detoxification

Many species of the genus *Fusarium* are phytopathogenic fungi of a wide range of cereal crop plants, including wheat. *F. graminearum* is the main causal agent of Fusarium Head Blight (FHB), while *F. culmorum* and *F. pseudograminearum* are the main responsible species of Fusarium Crown Rot (FCR). *Fusarium* diseases represent major agricultural problems worldwide, causing reduction of grain yield, grain quality and food safety. The latter is associated with contamination of grains with mycotoxins, particularly deoxynivalenol (DON), which cause health problems in humans and animals. DON is a protein synthesis inhibitor, acting as a virulence factor during pathogenesis and resulting essential for fungal spread along the spike. Conversion of DON to deoxynivalenol-3-β-D-glucoside (D3G) by the activity of specific UDP-glucosyltransferases (UGTs), is one of the mechanisms involved in enhancing plant tolerance to DON. Previous studies demonstrated that the expression of the barley *HvUGT13248* gene confers resistance to DON in *Arabidopsis thaliana* (Shin et al. 2012, J Exp Bot. 63:4731-40) and type II resistance to FHB (i.e. resistance to fungal spread within host tissues) in bread wheat (Li et al. 2015, MPMI 28:1237-46). Improvement of FHB resistance is a major target in both bread and durum wheat. The latter, however, is especially vulnerable, as effective sources of resistance are particularly limited. Therefore, we decided to verify whether the expression of the *HvUGT13248* gene could enhance FHB resistance in durum wheat as well. To this aim, transgenic lines of *Triticum durum* cv. Svevo, constitutively expressing the *HvUGT13248* gene, were produced. Transgenic plants in which presence of transcript and protein was confirmed, were infected with *F. graminearum* and evaluated for FHB severity, DON content and D3G conversion as compared to wild type plants. Our results showed that the *HvUGT13248* gene determines in durum wheat a significant reduction of FHB symptoms (up to 30%) compared to control plants. This effect, however, was mainly evident at early infection stages, progressively decreasing at later stages. This outcome differs from what observed in transgenic bread wheat expressing the same *UGT* gene, in which FHB severity did not exceed 20% up to the last stages of infection (Li et al. 2015). To verify further the effectiveness of the DON-detoxifying approach, durum wheat lines with the same *HvUGT13248* transgene were challenged with *F. culmorum*, also able to produce DON. A significant reduction of FCR symptoms compared to Svevo plants was observed. This represents the first report of improvement of FCR resistance associated with overexpression of an UGT involved in DON-detoxification. Recently, in

order to combine in the same plant genes controlling two different mechanisms of type II resistance to FHB, we have crossed two types of durum wheat transgenic lines, one expressing the *HvUGT13248* gene, the other *AcPMEI*, coding for a kiwi pectin methyl esterase inhibitor, known to increase resistance by strengthening the cell wall pectin fraction. On selected carriers of both transgenes, and in control lines with individual or no transgenes, the efficacy of the novel assembly will be verified against FHB and FCR.