TISSUE-SPECIFIC EXPRESSION OF PVPGIP2 TO IMPROVE WHEAT RESISTANCE AGAINST *FUSARIUM GRAMINEARUM*

TUNDO S.*, JANNI M.**, MOSCETTI I.***, MANDALÀ G.*, SAVATIN D.****, BLECHL A.****, D'OVIDIO R.*

- *) Department of Agricultural and Forestry Sciences (DAFNE), Università della Tuscia, Via San Camillo de Lellis snc, 01100 Viterbo (Italy)
- **) Institute of Biosciences and Bioresources (IBBR) National Research Council of Italy (CNR), Via Celso Ulpiani 5, 70126 Bari (Italy)
- ***) Department of Biological and Ecological Sciences (DEB), Università della Tuscia, Via San Camillo de Lellis snc, 01100 Viterbo (Italy)
- ****) Department of Biology and Biotechnology "Charles Darwin", Università degli Studi di Roma La Sapienza, Piazzale Aldo Moro 5, 00185 Roma (Italy)
- *****) USDA ARS, Western Regional Research Center, 800 Buchanan Street, Albany, CA 94710 (USA)

Fusarium graminearum, PvPGIP2, tissue specific expression, transgenic plants, wheat

Fusarium Head Blight (FHB) is one of the most important wheat diseases caused by some fungi of the genus Fusarium. The pathogen infects the spike at flowering time and causes severe yield losses and deterioration of grain quality due to the secretion of mycotoxins during infection. The understanding of the precise mode of pathogen entering and the subsequent floral tissue colonize is a crucial point to control FHB. Polygalacturonase inhibiting proteins (PGIPs) are cell wall proteins that inhibit the pectin-depolymerizing activity of polygalacturonases (PGs) secreted by microbial pathogens and insects. The constitutive expression of the bean PvPGIP2 limits FHB symptoms and reduces mycotoxin accumulation in wheat. To better understand the spike tissues that play a role in limiting Fusarium infection, we have produced transgenic wheat plants expressing PvPGIP2 or in the endosperm or simultaneously in lemma, palea, anthers and rachis. We showed that this latter approach reduced FHB symptoms caused by F. graminearum compared to control non transgenic plants. The extent of disease symptom reduction was similar to what obtained when PvPGIP2 was expressed constitutively. We showed also that different level of PvPGIP2 accumulation produced similar level of protection. Conversely, the expression of PvPGIP2 only in the endosperm did not affect FHB symptom development, indicating that when the pathogen has reached the endosperm, inhibition of the polygalacturonase (PG) activity of the pathogen is ineffective to prevent fungal spread. Probably the rich source of the endosperm tissue makes the PG activity dispensable for pathogen colonization. Alternatively, the massive growth of the fungus at this stage produces a large amount of PG that is not inhibited by the available PGIP.