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PYRAMIDING THE GLYCOSIDASE INHIBITORS PVPGIP2 AND ACPMEI OR PvPGIP2 AND TAXI-III TO IMPROVE RESISTANCE AGAINST FUNGAL DISEASES IN WHEAT

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Many pathogens produce a wide range of cell wall degrading enzymes (CWDE) and among them polygalacturonase (PG) is one of the first enzyme produced during the infection process. PG activity is limited by PGIP and by high levels of pectin methyl esterification which is controlled by the activity of the pectin methyl esterase (PME) and its inhibitor (PMEI). In wheat, high accumulation of a bean PGIP (PvPGIP2) or an increased level of pectin methyl esterification, obtained by expressing a kiwi PMEI (AcPMEI), reduced Fusarium Head Blight (FHB) and leaf blotch symptoms caused by the fungal pathogens F. graminearum and Bipolaris sorokiniana, respectively. In the present work we pyramided PGIP and PMEI with the aim to verify whether the combined presence of PG inhibitor and high level of pectin methyl esterification could enhance further wheat resistance against fungal pathogens, We used traditional breeding to cross the transgenic wheat plants expressing PvPGIP2 and AcPMEI. Analyses of F1 and subsequent progenies (up to F6) demonstrated the feasibility to combine the expression of both transgenes and the occurrence of both PvPGIP2 activity and high level of methyl esterification of cell wall pectin. Plants expressing both inhibitors showed a significant reduction of disease symptom against both F. graminearum and B. sorokiniana, although the level of protection was similar to that observed with the parental lines carrying the individual genes. On the contrary, preliminary experiments on durum wheat plants carrying the transgenes encoding PvPGIP2 and the xylanase inhibitor TAXI-III showed a reduced FHB symptoms compared to the parental line PvPGIP2.