

PERICARP COLOR1(P1) GENE MODIFIES PERICARP THICKNESS AND FUMONISINS ACCUMULATION IN MAIZE KERNEL

LANDONI M.***, PUGLISI D.*, BRUNOLDI G.*, COMASCHI C.*, TARENGHI F.*,
BRAMBILLA M.*, BORLINI G.*, CASSANI E.*, CARPINO M.*, REGINELLI D.***,
PILU S.R.*

*) Department of Agricultural and Environmental Sciences - Production, Landscape, Agroenergy,
University of Milano, Via Celoria 2, 20133 Milano (Italy)

**) Department of Biosciences, University of Milano, Via Celoria 26, 20133 Milano (Italy)

***) Experimental farm “A. Menozzi”, Landriano (Italy)

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Mycotoxins are secondary metabolites potentially dangerous for animals and human health. In particular, maize (*Zea mays*) infection caused by *Fusarium* and the consequent fumonisin contamination is widespread in several countries such as Italy. The fumonisins are a group of mycotoxins produced primarily by *Fusarium verticillioides* and *Fusarium proliferatum*, although a few other *Fusarium* species also may produce them. For this problem several official agencies such as the FAO/WHO, U.S. Food and Drug Administration and the European Union, established the threshold fumonisin contents in maize at the level of 2-4 ppm in non-processed maize. So far, no definitive control strategies are available to prevent fumonisin accumulation in kernels. However, it is known that crop growing techniques such as planting date, irrigation, nitrogen fertilization, or damage by insects and inappropriate storage can contribute to fungal growth on kernels. Plant breeding is an environmentally safe method to control fungal infection and reduce mycotoxin levels in maize. Higher resistance levels and reduced fumonisin contamination in maize kernels are possible considering the high genetic variability observed for resistance to fusarium infection and its heritability. In this work we showed that the presence of *pericarp color 1 (p1)* gene modifies pericarp thickness and fumonisins accumulation in maize kernel. *p1* gene maps on short arm of chromosome 1 (Bin 1.03) and encodes a DNA binding protein belonging to the R2R3-MYB transcription factor family: dominant *PI* allele provides pigmentation selectively to plant floral organs, in particular pericarp and cob, due to accumulation of phlobaphene pigments derived by polymerization of flavan-4-ols. We studied and compared different genetic materials carrying *PI* allele and the respective *p1* isogenic lines. Our results indicate that the accumulation of phlobaphene pigments in the seeds lead to an increased pericarp thickness and a reduced level of fumonisin B1. Further work will be necessary to assess if this effect is due to the increased pericarp thickness, or to phlobaphenes accumulation or both.