**HIGHLY METHYL ESTERIFIED SEEDS is a Pectin Methyl Esterase involved in embryo development.**

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[Levesque-Tremblay G](http://www.ncbi.nlm.nih.gov/pubmed/?term=Levesque-Tremblay%20G%5BAuthor%5D&cauthor=true&cauthor_uid=25572606), [Müller K](http://www.ncbi.nlm.nih.gov/pubmed/?term=M%C3%BCller%20K%5BAuthor%5D&cauthor=true&cauthor_uid=25572606), [Mansfield SD](http://www.ncbi.nlm.nih.gov/pubmed/?term=Mansfield%20SD%5BAuthor%5D&cauthor=true&cauthor_uid=25572606), [Haughn GW](http://www.ncbi.nlm.nih.gov/pubmed/?term=Haughn%20GW%5BAuthor%5D&cauthor=true&cauthor_uid=25572606).

**Abstract**

Homogalacturonan pectin domains are synthesized in a highly methyl-esterified form that later can be differentially de-methyl esterified by pectin methyl esterase (PME) to strengthening or loosen plant cell walls that contain pectin, including seed coat mucilage, a specialized secondary cell wall of seed coat epidermal cells. As a means to identify the active PMEs in seed coat mucilage we identified 7 PMEs expressed during seed coat development. One of these, HIGHLY METHYL ESTERIFIED SEEDS (HMS), is abundant during mucilage secretion, peaking at 7 Days Post Anthesis (DPA) both in the seed coat and the embryo. We have determined that this gene is required for normal levels of PME activity and homogalacturonan methyl esterification in the seed. The hms-1 mutant displays altered embryo morphology and mucilage extrusion, both of which are a consequence of defects in embryo development. A significant decrease in the size of cells in the embryo suggests that the changes in embryo morphology are a consequence of lack of cell expansion. Progeny from a cross between hms-1 and the previously characterized PMEI5 over-expression line (OE) suggest that HMS acts independently from other cell wall modifying enzymes in the embryo. We propose that HMS is required for cell wall loosening in the embryo to facilitate cell expansion during the accumulation of storage reserves, and that its role in the seed coat is masked by redundancy.

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