How is Polycomb MEDEA Imprinting Controlled in the Endosperm?

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Genomic imprinting causes genes to be expressed according to their parental origin and is observed exclusively in mammals and flowering plants. Imprinting plays an important role in the reproductive strategies of both groups. In mammals, many of the imprinted genes are expressed in the extraembryonic membranes that serve as a conduit for the flow of nutrients from the mother to the embryo. In plants, the endosperm performs a similar function to the developing embryo and is also known to be a critical site for gene imprinting. Although some imprinted genes are essential for plant reproduction, little is known about how imprinting is initiated and maintained in plants and the mechanisms that control imprinting are unknown.

We have discovered that the Arabidopsis DEMETER (DME) gene mediates endosperm imprinting in Arabidopsis. Inheritance of a dme mutant allele by the female gametophyte resulted in embryo and endosperm abortion even when a wild type paternal DME allele was inherited. Seed viability depends only on the maternal DME allele and the paternal allele is dispensable. The DEMETER (DME) gene encodes a large 1729 amino acid polypeptide with a 200 amino acid Helix-hairpin-helix DNA glycosylases domain and nuclear localization domains. We find that the DME DNA glycosylase functions in vivo to activate maternal MEA allele transcription of imprinted genes. DME transcription is restricted to the central cell of the female gametophyte, the progenitor of the endosperm. DME is required for maternal allele expression of the imprinted MEDEA (MEA) Polycomb gene in the central cell and endosperm.

Helix-hairpin-helix DNA glycosylases are typically small proteins that initiate repair of DNA by excising damaged or mispaired bases. An invariant aspartic acid in the active site is involved in catalyzing the excision reaction. Replacement of this critical reside with an asparagine severely reduces catalytic activity while preserving enzyme stability and structure. We mutated the invariant aspartic acid at position 1304 in DME to asparagine (D1304N) to determine if the catalytic activity of the DNA glycosylase domain is required for DME function in vivo. The results show that the conserved aspartic acid residue is necessary for DME to function in vivo and suggest that an active DNA glycosylase domain, normally associated with DNA repair, promotes gene transcription that is essential for gene imprinting.

Maternal mutant dme or mea alleles result in seed abortion. We identified mutations that suppress dme seed abortion and found that they reside in the MET1 methyltransferase gene, which maintains cytosine methylation. Seeds with both maternal dme and met1 alleles survive, indicating that suppression occurs in the female gametophyte. MET1 suppression requires a maternal wild type MEA allele, suggesting that MET1 functions upstream of, or at, MEA. DME activates whereas MET1 suppresses maternal MEA::GFP allele expression in the central cell fo the female gamotophyte. MET1 is required for DNA methylation of three regions in the MEA promoter in seeds. Our data suggest that imprinting is controlled in the female gametophyte by antagonism between the two DNA modifying enzymes: MET1 methyltransferase and DME DNA glycosylase.