

Epigenetic Change (<https://www.gephebase.org/search-criteria?/and+Aberration+Type=^Epigenetic+Change^#gephebase-summary-title>)

Molecular Details of the Mutation

The taproot white-fleshed mutant is the result of altered DNA methylation in the RsMYB1 promoter. This heritable epigenetic change is due to a hypermethylated CACTA transposon (a 7372-bp TE) which induces the spreading of DNA methylation to the promoter region of RsMYB1. RsMYB1 expression is considerably downregulated and this inhibits anthocyanin biosynthesis in white-fleshed mutants.

Experimental Evidence

Candidate Gene (<https://www.gephebase.org/search-criteria?/and+Experimental+Evidence=^Candidate+Gene^#gephebase-summary-title>)

Main Reference

Transposon-induced methylation of the RsMYB1 promoter disturbs the anthocyanin accumulation in red-fleshed radish (*Raphanus sativus* L.). (2020) (<https://pubmed.ncbi.nlm.nih.gov/31961436/>)

Authors

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Abstract

Red-fleshed radish is a unique cultivar that is rich in anthocyanins beneficial to human health in taproot. However, the frequent occurrence of white-fleshed mutants affects the purity of commercially produced radish and mechanism has puzzled breeders for many years. In this study, we combined QTL-seq and transcriptome analyses to identify a candidate gene (RsMYB1) responsible for the anthocyanin accumulation in red-fleshed radish. However, no sequence variation was found in the coding and regulatory regions of the RsMYB1 genes of the red-fleshed (MTH01) and white-fleshed (JC01) lines, and a 7,372-bp CACTA transposon in the RsMYB1 promoter region occurred in both lines. A subsequent analysis suggested that the taproot white-fleshed mutant was the result of altered DNA methylation in the RsMYB1 promoter. This heritable epigenetic change was due to the hypermethylated CACTA transposon, which induced the spreading of DNA methylation to the promoter region of RsMYB1. Thus, RsMYB1 expression was considerably downregulated, which inhibited anthocyanin biosynthesis in white-fleshed mutants. An examination of transgenic radish calli and the results of a virus-induced gene silencing experiment confirmed the RsMYB1 is responsible for anthocyanins accumulation. Moreover, the mutant phenotype was partially eliminated by a treatment with a demethylating agent. This study explained the molecular regulation mechanism of appearance white-fleshed mutant in red-fleshed radish.

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