IN BRIEF

A Double Lock on Polyploidy-Associated Epigenetic Gene Silencing

Epigenetic gene silencing is an inheritable loss of gene expression due to modifications to DNA and chromatin that do not involve changes in DNA sequence. Such changes include methylation of DNA cytosine residues and posttranslational modifications to histones, such as acetylation, methylation, phosphorylation, ubiquitylation, and sumoylation. The different epigenetic states of a genetic locus, known as epialleles, can be altered (activated or silenced) in response to various genomic or environmental stresses, including polyploidization (reviewed in Finnegan, 2002). Mittelsten Scheid et al. (2003) described an unusual epiallele of a transgenic locus in Arabidopsis found after tetraploidization. Diploid Arabidopsis carrying a transgenic hygromycin resistance marker gene (HPT) showed stable inheritance of hygromycin resistance over many generations of self-pollination and after outcrossing with diploid wild-type plants. However, tetraploid derivatives generated progeny with a silenced epiallele of HPT (not expressing HPT and hygromycin sensitive). The silenced epialleles were highly stable and exerted a paramutation-like effect in which the silent epiallele led to inactivation of the previously active counterpart in tetraploid crosses.

In new work, Baubec et al. (pages 34–47) investigate the mechanistic basis of this phenomenon, which they termed polyploidy-associated transcriptional gene silencing (paTGS). The authors employ a combination of pharmacological, genetic, and molecular approaches to show that two epigenetic marks, DNA methylation and histone methylation, cooperate to give rise to a double lock on transcriptional silencing, thus generating an extremely stable epigenetic state, as both modifications must be reversed to convert the silent epiallele to an active state. A forward genetic T-DNA screen revealed that the chromatin remodeling factor DDM1 and an S-adenosyl-L-homocysteine hydrolase, HOG1, are required for paTGS. Both DDM1 and HOG1 are known to participate in DNA methylation-dependent gene silencing (Vongs et al., 1993; Furner et al., 1998). The newly identified ddm1 and hog1 alleles were found to have both reduced DNA methylation and heterochromatic histone modifications at the HPT transgene, and both of these effects were required to unlock silencing (see figure). Further experiments using chemical treatment with an inhibitor of the HOG1 gene product corroborated the results and the conclusion that HOG1 plays an important role in chromatin modification in Arabidopsis.

Polyploidy is widespread in plants and is believed to be an important source of genetic diversity and adaptation (Crow and Wagner, 2006). The double lock mechanism might be a general feature of paTGS and would explain why polyploid plants that generate new epialleles represent particularly stable states that do not easily revert.

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REFERENCES


Cooperation of multiple chromatin modifications creates a double lock on epigenetic silencing. paTGS of the HPT transgene can only be released by simultaneous removal of DNA methylation (black lollipops) and the repressive histone modification K9me2 (dimethylation at Lys 9 of histone H3). DDM1 and HOG1 are required to maintain both modifications, and only their lack in ddm1 or hog1 mutants or application of a HOG1 inhibitor (DHPA) can release the double lock. (Figure reproduced from Baubec et al. [2010].)