Heterochromatin reprogramming and epigenetic inheritance: how to avoid BadKarma

Rob Martienssen, Cold Spring Harbor Laboratory

Epigenetic inheritance is more widespread in plants than in mammals, in part because mammals erase epigenetic information each generation by germline reprogramming. To assess the extent of germline reprogramming in plants, we sequenced the methylome from sperm cells (SC), the vegetative nucleus (VN), and the precursor microspore from developing haploid pollen. We found that asymmetric CHH methylation is lost in microspores and sperm cells, but restored in the VN and in fertilized seed. In the VN symmetric CG methylation is lost from targets of the DNA glycosylases DEMETER (DME) and REPRESSOR OF SILENCING 1 (ROS1) including transposons near imprinted genes, which contributes to imprinting via RNA directed DNA methylation and 24nt siRNA. In contrast, most active transposons give rise to 21nt “epigenetically activated” small RNA in DECREASE IN DNA METHYLATION 1 (DDM1) mutants, in tissue culture and in the VN, which loses heterochromatin. Loss of heterochromatin in the VN is not only due to the loss of DNA methylation but also to histone replacement with variants resistant to modification. Thus genome reprogramming in pollen contributes to epigenetic inheritance, transposon silencing, and imprinting, guided by small RNA. In a real-world example, micropropagation of oil palm clones from somatic cells circumvents germline reprogramming of Karma retrotransposons, and results in heritable epigenetic changes reminiscent of paramutation.