Abstract

Hybridization barriers are reproductive barriers that contribute to plant speciation. Understanding the genetic and epigenetic mechanisms of both pre- and post-zygotic hybridization barriers may improve our understanding of evolutionary biology but also may bear applications for breeding purposes.

Among all the hybridization barriers contributing to plant reproductive isolation, pollen rejection, and hybrid seed lethality have received particular attention. Pollen rejection is a prezygotic barrier in which incompatible pollen-pistil interactions prevent a seed between two species from being formed. If a hybrid seed is formed, it might not be viable, leading to a postzygotic barrier. The failure of endosperm development is the major cause of inviability in interspecific hybrid seeds. Epigenetics is well known to play a role in this process. More precisely, the epigenome of the pollen donor seems to have an important impact on hybrid seed viability. However, the role of the maternal epigenome is less well understood. Regarding the implication of epigenetic mechanisms in pollen rejection, very scarce evidence is currently available.

Here, I assessed the role of maternal epigenetics in these pre- and post-zygotic hybridization barriers between two species: *Arabidopsis thaliana* and *Arabidopsis arenosa*. For this purpose, I used *A. thaliana* epigenetically recombinant inbreed lines (epiRILs) that are genetically similar but epigenetically variable and crossed them with *A. arenosa* as a pollen donor. I measured seed set as a proxy for pollen rejection and seed abortion rate as a proxy for hybrid seed viability. There was significant variation between epiRILs in the pre-and post-zygotic barriers, suggesting an effect of maternal epigenetics in both hybridization barriers. To further evaluate this hypothesis, I mapped epigenetic quantitative trait loci (epiQTL) to find the epialleles involved in pollen rejection and hybrid seed viability. We did not find any epiloci associated with these traits, potentially due to technical limitations. Overall, maternal epigenetics likely play a role in the two studied hybridization barriers, with a likely complex epigenetic structure involving several small effect epiloci controlling these barriers.