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EPIGENETIC CONTROL OF PLANT MALE STERILITY MEDIATED BY DNA METHYLATION

BUNELLO F.*, DE PAOLI E.*, BOUYER D.**

*) University of Udine, Department of Agrifood, Environmental and Animal Sciences, via delle Scienze 206, 33100 Udine, Italy
**) Laboratoire Reproduction et Développement des Plantes (RDP), University of Lyon, Ecole Normale Supérieure de Lyon, Université Claude Bernard Lyon1, CNRS, INRAE, 69342 Lyon, France

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Failure in producing functional pollen and other forms of male sterility are a renowned source of breeding tools with a major application in seed technology for preventing self-pollination and leveraging heterosis in hybrid crops. However, mechanisms of male sterility and fertility restoration have raised scientific interest per se, since the first reported observation of such phenomenon in 1763 and similar findings in hundreds of species thus far. Plant male sterility may be caused by mitochondrial gene mutations combined with mitochondrial-nuclear gene interactions (cytoplasmic male sterility, CMS) or alterations in nuclear genes alone (genic male sterility, GMS). Of particular interest, both for practical purposes and for the understanding of GxE mechanisms, are environment-sensitive GMS systems where pollen fertility is not a fixed property but rather changes in response to environmental cues such as day length and temperature.

In this study, we are investigating a further perspective of male fertility regulation that relies on epigenetic determinants in the model species Arabidopsis. DNA cytosine methylation is an epigenetic mark that does not change massively across the different steps of reproduction, but compelling evidence has emerged that active DNA demethylation occurs in the vegetative cell of the gametophyte and is required for proper pollen tube progression. This process is brought about by DNA glycosylases that act in the vegetative cell to demethylate DNA at specific gene loci, activating their

expression specifically in pollen. Mutant pollen impaired in the DNA demethylation function is capable of resuming fertility upon reestablishment of expression in the affected loci, demonstrating an essential role of active DNA demethylation in regulating genes involved in pollen function. The genome-wide impact of such regulation and the possibility to modulate male fertility by artificial epigenetic dysregulation will be discussed.