Proceedings of the LXVI SIGA Annual Congress
Bari, 5/8 September, 2023
ISBN: **978-88-944843-4-2**

Poster Communication Abstract – 7.12

THE REDUNDANCY PARADOX: UNCOVERING THE MECHANISMS OF PARALOGOUS COMPENSATION IN THE MAIZE MERISTEM

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dosage, gene duplication, paralog compensation, redundancy, responsive backup circuits

Evolutionary innovations are often achieved by co-opting existing molecular structures to perform new functions, a concept commonly referred to as “molecular tinkering”. Gene duplication is a powerful source of biological innovation, giving rise to duplicates (hereafter, paralogs) that undergo diverse fates and drive evolutionary change. One of the greatest paradoxes in evolutionary genomics is the retention of redundancy among ancient paralogous genes despite the accumulation of mutations. Genetic studies in yeast and plants have suggested that the ability of ancient paralogs to be redundant and to compensate for a loss of function depends on the reprogramming of gene expression, a phenomenon known as active compensation. My research work focuses on the maize trehalose-6-phosphate phosphatases RAMOSA3 and TREHALOSE PHOSPHATE PHOSPHATASE 4, two important meristem development regulators, as a model for studying active compensation. By using promoter editing and chromatin accessibility assays, my work is investigating the hypothesis that non-coding sequences conserved across phylogenetic families over evolutionary time control active compensation by binding to factors that regulate gene expression.

Furthermore, my work is addressing whether the reprogramming of paralogs is linked to the stabilization of their mRNAs following the destabilization of a duplicate, therefore establishing a possible role for post-transcriptional regulation of compensation. Understanding the responsive backup circuits underlying compensation between duplicate genes could allow us to fine-tune traits controlled by redundant paralogs, and improve the

predictability of gene editing outcomes.