Decoding Golgi-Mediated Glycosylation in Hypometabolism-Associated Aging

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This project explores how Golgi-mediated glycosylation influences stress-induced aging in plants. A forward genetic screen identified a rapidly senescing *cog7* mutant, revealing unexpected roles for the Conserved Oligomeric Golgi (COG) complex in glycosylation homeostasis under hypometabolic conditions. Overexpression studies showed that COG6 and COG3 alleviate *cog7*-associated early senescence, while COG8 activates tissue-specific stress signaling. The *cog7/camta3* double mutant uncovered RSRE-independent senescence pathways, pointing to previously unrecognized regulatory networks.

In parallel, multiomics identified novel glycosyltransferases upregulated during senescence. These are being functionally characterized using CRISPR knockouts and LC-MS-based substrate profiling.

A system to isolate glycosylated extracellular vesicles (EVs) via His-tagged TET8 was also established, enabling targeted EV proteomics.

Collectively, this work introduces a novel experimental platform that links Golgi function, glycosylation remodeling, and EV biogenesis with aging. The integration of genetic, structural, and proteomic tools provides a powerful new apparatus for decoding the molecular machinery of hypometabolism-associated aging, offering transformative insights into plant resilience and broader eukaryotic stress adaptation.