Unraveling the mechanism of TTL genes in cellulose biosynthesis

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As sessile organisms, plants require mechanisms to sense and respond to the challenging environment, that encompass both biotic and abiotic factors that results in differential development. In these conditions is essential to balance growth and stress responses. As cell walls shape plant growth, this differential growth response cause alterations to the plant cell wall where cellulose is the major component. Therefore, understanding the mechanisms that regulate cellulose biosynthesis is essential to develop strategies to improve plant production. In Arabidopsis, the TETRATRICOPEPTIDE THIOREDOXIN-LIKE(TTL) gene family is composed by four members (TTL1 to TTL4) and mutations in TTL1,TTL3, and TTL4 genes cause reduced growth under salt and osmotic stress due to defects in plant cell wall integrity. We observe association of TTL3 with most core components in traducing BR signalling, such as LRR-RLK BRI1 or GSK3 BIN2 that modulate cellulose biosynthesis through phosphorylating cellulose synthases (CesA). Here, we show that ttlmutants present defects in the plant cell wall, particularly in Isoxaben, salt or sucrose stress. Spinning disk microscopy in etiolated hypocotyls reveals that, TTL proteins are responsible for the cellulose synthase complex (CSC) stability in plasma membrane (PM) upon sucrose stress. Moreover, TTL3 associates with LRR-RLKs that have been shown to be important for cellulose biosynthesis such as FEI1 in the FEI1/FEI2/SOS5 pathway. We aim to investigate the mechanisms by which TTL proteins regulate CesA stability in PM under stress, using a combination of genetics, biochemical, and molecular and cell biology approaches.

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