

The elicitor surfactin triggers plant immunity by altering membrane lipid properties

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Surfactin (SRF) is a lipopeptide produced by plant beneficial bacilli able to trigger immunity in plants. Previous data strongly suggest that SRF sensing at the plant plasma membrane (PPM) is mainly driven by the interaction between SRF and membrane lipids, contrasting with detection by protein receptors as commonly described for plant immune stimulation by plant elicitors. Nevertheless, the mechanism underneath the lipid-driven sensing of surfactin by plant cells remains largely unknown. To investigate the role of PPM lipids in SRF sensing in plants, we combined functional assays on *Arabidopsis thaliana* root protoplasts with experimental biophysics on biomimetic membrane models.

Isothermal titration calorimetry first revealed a higher binding of SRF for liposomes containing sphingolipids. This SRF-lipid interaction affects the physical properties of the membrane with a thinning of the membrane, a decrease in membrane fluidity, and an increased curvature. Moreover, the *loh1* mutant, impacted in its PM sphingolipid content, showed a strong reduction in SRF-induced ROS burst, one hallmark of plant early immune event, supporting the importance of sphingolipids in SRF sensing.

The involvement of plant lipids in SRF sensing and the impact of SRF on PPM mechanics lead us to investigate the importance in SRF sensing of the PPM-located mechanosensitive (MS) channels, the Mid1-Complementing Activity (*mca*) channels and MscS-like (*msl*) channels. Interestingly, a lower calcium response, another hallmark of early immune event, is observed in root protoplasts from *mca* and *msl* channels-depleted mutants upon SRF treatment compared to Col-0 protoplasts.

Altogether, our results support a SRF eliciting mechanism triggered by an activation of MS channel through a disturbance of PPM lipids organization, which represents a new aspect of plant immune stimulation by beneficial bacterial molecules.