

Linking Transcriptomic and Electrophysiological Signatures of MAX2 Function During Ozone Stress in *Arabidopsis thaliana*

Ozone (O₃) stress negatively impacts plant growth and productivity. In *Arabidopsis thaliana*, the F-box protein MAX2 plays a key role in ozone response and stomatal function, with *max2* mutants exhibiting delayed stomatal closure. In addition to MAX2, stomatal opening and closing are regulated by shifts in membrane potential and voltage-gated ion channel activity.

Non-invasive bacterial nanocellulose (BNC) electrode sensors allow quantitative measurement of action potentials and related electrical dynamics reflecting underlying ion fluxes. We hypothesized that the delayed stomatal closure phenotype reflects MAX2-dependent changes in early electrical signaling that occur with altered downstream transcriptional responses. To test this, we exposed WT plants and two independent loss-of-function alleles (*max2-1* and *max2-4*) to ~210 ppb ozone while simultaneously collecting RNA-seq samples and continuous electrophysiological measurements. Transcriptomic analysis implicated disrupted hormonal crosstalk in the *max2* mutant phenotype, with GRN inference identifying key regulatory genes driving these changes. We are currently working to temporally correlate these transcriptional shifts with electrical signaling dynamics in BNC sensor data, toward building a machine learning framework where real-time sensor data serves as an accessible proxy for transcriptional state during plant stress.