

The *AtSCL26* transcription factor controls cross-talk between GA and Nitrogen control of root architecture in *Arabidopsis thaliana* roots

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SUMMARY

A critical aspect of multicellularity is co-ordination of cell-cell responses. In plants it is known that hormone patterning underlies the regulation of transcriptional networks that regulate cell-cell coordination but much less is known about the cross-talk between cell types that underlie control of whole organ phenotypes. The GRAS/SCARECROW LIKE (SCL) transcription factor family includes genes that respond to the plant growth regulator gibberellin (GA) and some members have already been found to control many aspects of shoot, meristem and root development. We have been able to implicate the previously uncharacterised gene *AtSCL26* (putative homolog of *MtNSP2*) in the cell-specific control of nitrogen-GA response cross-talk to shape root architecture. *AtSCL26* acts to repress lateral root (LR) development specifically under deplete nitrogen (N) levels repressing LR outgrowth. The *AtSCL26* transcription factor functions to balance resource partitioning between growth and secondary processes, and functions in a highly cell type specific manner.

AtSCL26 CONTROLS LATERAL ROOT INITIATION AND OUTGROWTH IN A N-DEPENDENT MANNER

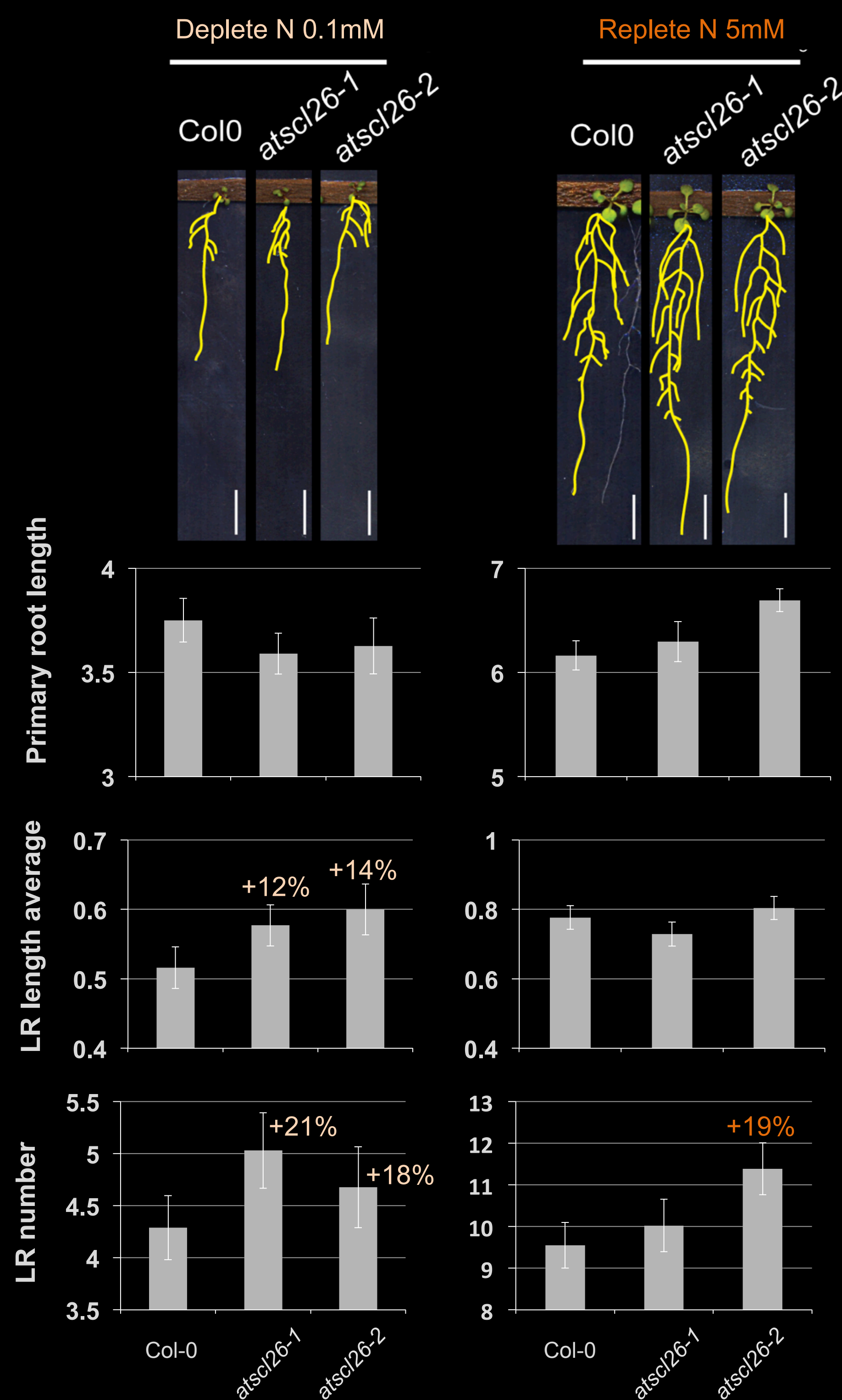


Figure 1. Images of 12 day old seedlings grown on low N or high N for two T-DNA alleles in the *AtSCL26* gene and Col-0; yellow is used to visualise root architecture; scale bars = 1 cm. Graphs show primary root (PR) length, lateral root (LR) length average and LR number. ImageJ was used to perform root measurements.

AtSCL26 is the putative *Arabidopsis* homolog of *MtNSP2*, a transcription factor essential for nodule development (Kalo et al., 2005). The root phenotypic analysis of *atscl26* loss of expression mutants suggests that *AtSCL26* acts to repress LR primordium development under deplete N levels, thus *AtSCL26* affects LR initiation and elongation in a N quantity-dependent manner. These results point to *AtSCL26* as a root plasticity regulator, since it modulates specific root phenes under specific conditions.

CELL TYPE SPECIFIC RESPONSES CONTROLLED BY *AtSCL26*

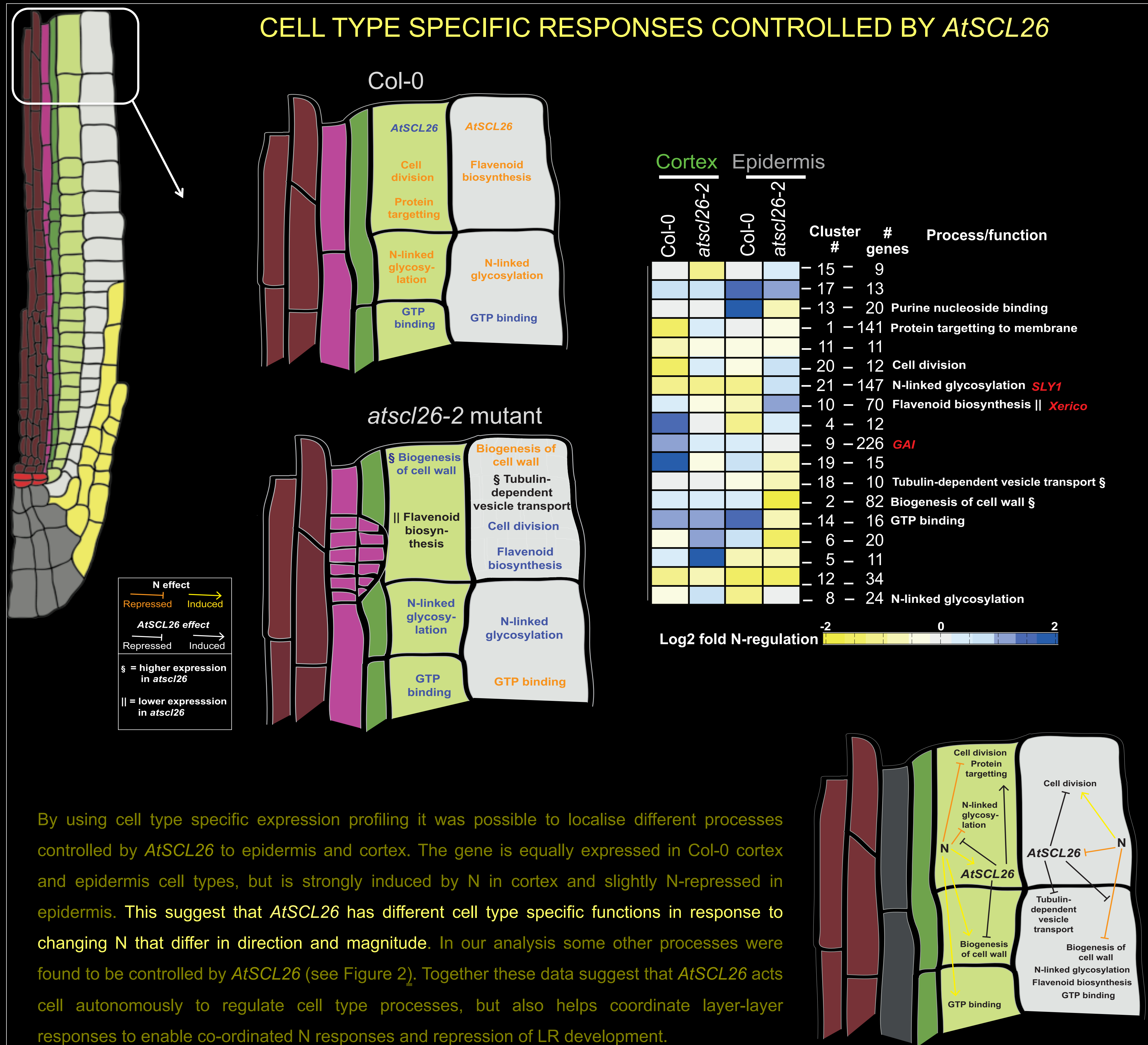


Figure 2. The *atsc26-2* mutant was crossed with *Arabidopsis* root-cell type GFP lines to generate cell type specific reporters in the mutant background. Fluorescent Activated Cell Sorting (FACS) was used to separate epidermal and cortical cell types from 12 day old seedlings treated with high N vs. control for the *atsc26-2* mutant vs. Col-0.

GA₃ / NO₃⁻ / *AtSCL26* CROSSTALK CONTROLLING ROOT ARCHITECTURE

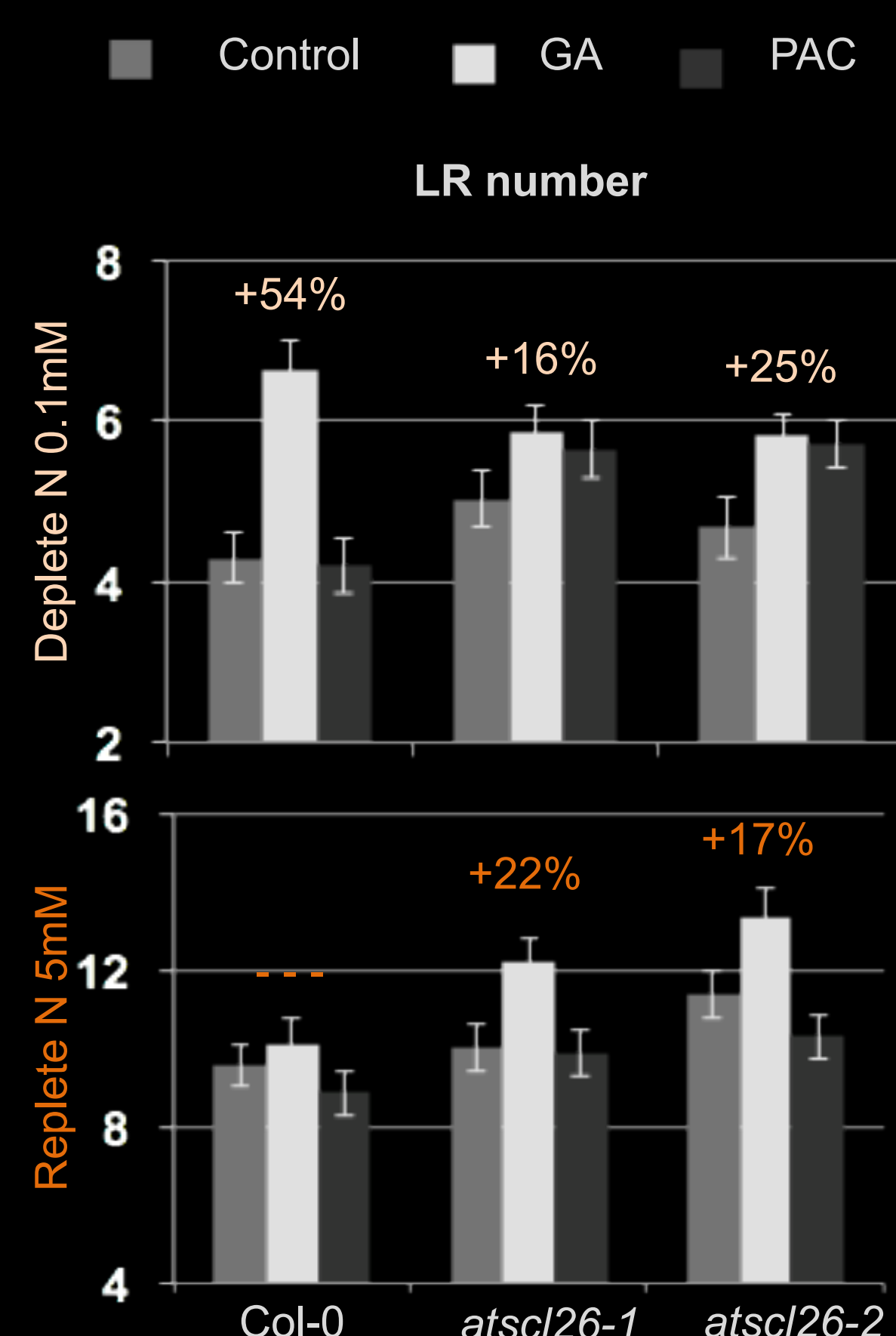


Figure 3. *AtSCL26* controls LR number by modulating crosstalk between N and GA signalling. Graphs representing LR number from 12 day old seedlings grown on low N or high N for two T-DNA alleles in the *AtSCL26* gene and Col-0 after treatment (GA or PAC) on day 9. ImageJ was used to perform root measurements.

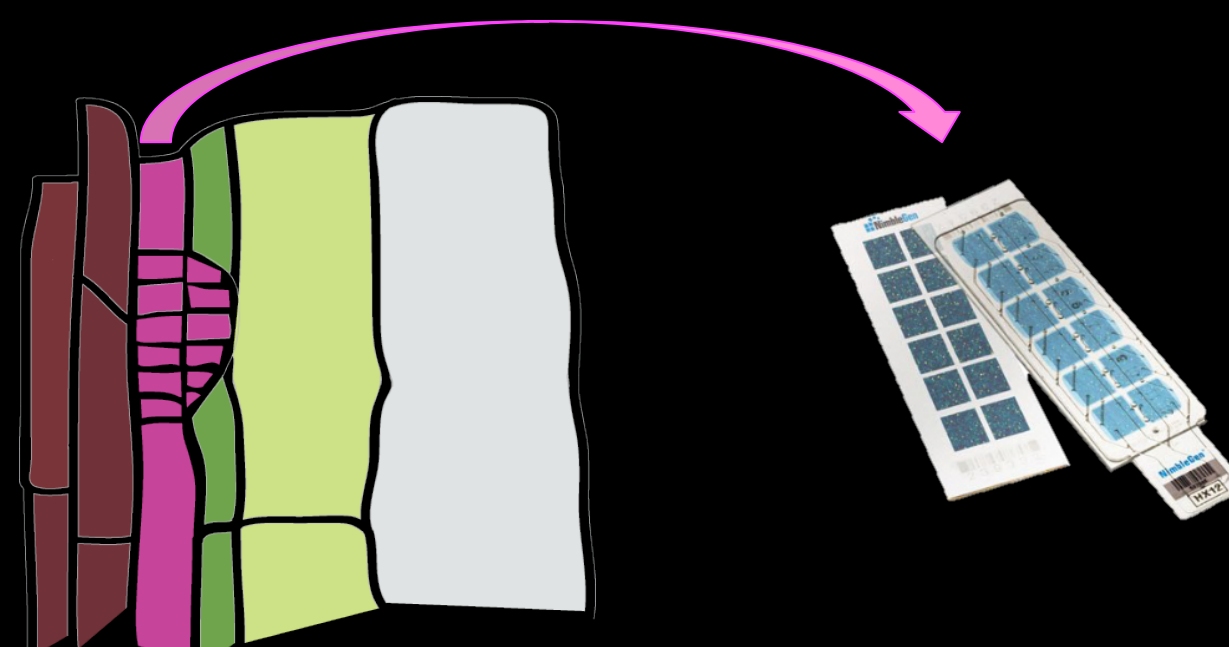
GA₃ and N treatment experiments were carried out to determine if mutant LR phenotype is affected by gibberellic acid (GA), since *NSP2* function in *Medicago* is GA-dependent (Maekawa *et al.*, 2009) and other members of the GRAS family are regulated by this hormone. *AtSCL26* was found to regulate LR number through repressing GA induction of LR in a nitrogen dependent way. A direct function of *AtSCL26* could thus be modulation of the GA signaling pathway since we identify altered N-regulation of GA signalling genes including *GAI/RGA2* and *SLY* and *Xerico* (Fig 2).

CURRENT WORK

Protein localisation: Since these are putative transcription factors we would expect a nuclear localisation



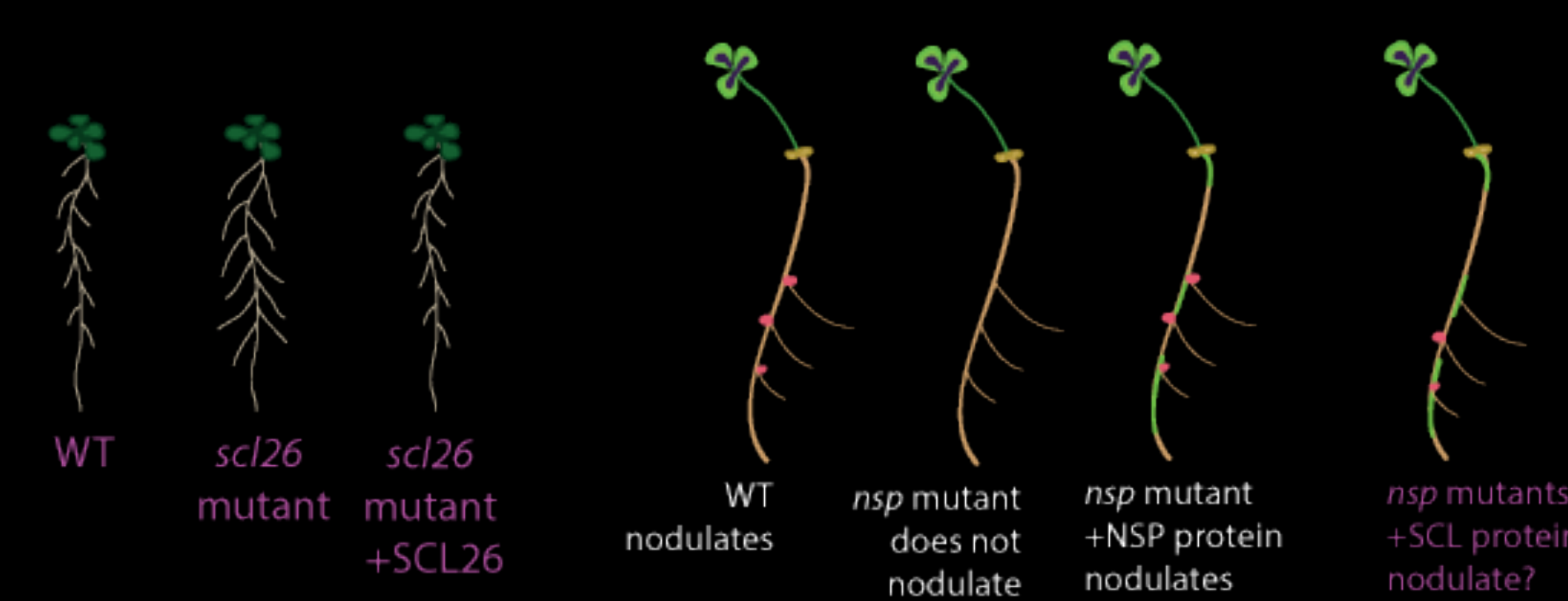
Pericycle gene expression: Complementing gene expression analysis in the root cell type from where lateral roots are formed



Gene and protein regulation: We will perform nitrogen availability experiments followed by microscopy



Protein function: Arabidopsis mutant complementation and test of functional conservation among plant species



REFERENCES

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