

# Abstract

Auxin is phytohormone that regulates several developmental processes and environmental responses. One of the most well-described outcome of the auxin signalling pathway is regulation of gene transcription. Aux/IAA proteins play an important role in this process, acting as transcriptional repressors. Recent studies revealed that several root growth responses are too rapid to be explained by changes in the level of transcription. The correlation between the amount of Aux/IAs and the root growth rate suggests that these proteins might be involved in root growth regulation, especially during rapid growth responses that are not associated with transcriptional reprogramming. This work is focused on one of the 29 *Arabidopsis* Aux/IAA proteins - the IAA17/AXR3 protein.

First, we produced stable transgenic lines of *Arabidopsis thaliana* expressing different combinations of fluorescently labelled AXR3-1 proteins and/or fused to subcellular localization tags under the control of different tissue-specific promoters, in order to characterize the subcellular localization of the studied protein. Subsequent visualization by confocal microscopy methods confirmed information about the role of IAA17/AXR3 protein in root growth responses, its involvement in auxin signalling, and gravitropism.

Next, we showed that the induction of AXR3-1 mutant protein stimulates root growth rate in the initial stages. Moreover, in these mutant lines, an unusual loss of DII-Venus signal in roots indicates either high concentration of auxin or a disruption of its signalling. The dynamics of both IAA17/AXR3 and AXR3-1 protein in *Arabidopsis* root were studied with unexpected results. Although the AXR3-1 protein was driven by cell specific promoter, the signal was also observed outside the expression domain - in the vascular cylinder, indicating its possible intercellular transport.

This work provides in a basis and a toolset for further research that could help understand the molecular principles of the rapid root growth response and their relationship to auxin signalling.