Strigolactone signaling and the regulation of shoot branching

Ottoline Leyser[1]
[1]*Sainsbury Laboratory, University of Cambridge*

Mutants with reduced in strigolactone synthesis or response are highly branched and strigolactone addition to buds can inhibit their growth. However, under some circumstances, for example in mutants with compromised auxin transport, strigolactones can promote bud growth. A model for shoot branching control that can explain this apparent paradox involves promotion of endocytosis of specific PIN-type auxin efflux carriers as a primary target for strigolactone action. There is a substantial body of evidence supporting this mode of action. For example, strigolactone triggers PIN1 depletion from the plasma membrane as a rapid cycloheximide independent response. However, the signal transduction pathway for strigolactone is largely nuclear and it has been proposed that strigolactones act by regulating the degradation of transcription repressors, triggering changes in expression of bud regulating genes such as the BRC1 transcription factor. BRC1 is known to inhibit shoot branching, and in many circumstances its BRC1 transcription can be induced by strigolactone addition. Our progress in understanding these different modes of action of strigolcatone signalling in shoot branching control will be reported.