

The pea branching *RMS2* gene encodes the PsAFB4/5 auxin receptor and is involved in an auxin-strigolactone regulation loop

Yasmine Ligerot¹, Alexandre De Saint Germain^{1,7}, Sylvie Citerne¹, N. Kadakia³, Romain Novaretti¹, Jean-Paul Pillot¹, Cristelle Troadec⁴, Tanya Waldie², Grégoire Aubert⁶, Frédéric Debellé⁵, Abdel Bendahmane⁴, Mark Estelle³, Ottoline Leyser² and Catherine Rameau¹

¹Institut Jean-Pierre Bourgin, ²Sainsbury Laboratory, University of Cambridge, ³Section of Cell and Developmental Biology and Howard Hughes Medical Institute, University of California San Diego, ⁴Institut des Sciences des Plantes de Paris, ⁵Laboratoire des Interactions Plantes–Microorganismes, INRA/CNRS, ⁶INRA, ⁷Plant Biology Laboratory, The Salk Institute for Biological Studies

Control of shoot branching involves auxin, cytokinin and strigolactones (SLs). In pea, most of the high branching *ramosus* (*rms*) mutants show high expression of the SL biosynthesis *RMS1/CCD8* gene and low xylem-sap cytokinin content. In contrast, the high branching *rms2* mutant displays very low expression of *RMS1* and high xylem-sap cytokinin content. Previous physiological characterization of the pea *rms2* mutant suggested that *rms2* was affected in a shoot-to-root feedback signal controlling both SL biosynthesis and cytokinin level in the xylem sap.

The nature of the feedback signal was investigated by cloning the pea *RMS2* gene. We showed that *RMS2* encodes the pea ortholog of the Arabidopsis auxin receptor belonging to the AUXIN-SIGNALING F-BOX4 (AFB4)/AFB5 clade suggesting that the shoot-to-root feedback signal is very likely auxin. To investigate whether the pea *RMS2* and AFB4/5 from Arabidopsis have the same functions, we analyzed *rms2* phenotypes (e.g. shoot branching, high IAA levels) in the Arabidopsis *afb4*, *afb5* and *afb4 afb5* double mutants and reciprocally, we tested whether the pea *rms2* mutants showed the high selective resistance to the herbicidal auxin picloram of the *afb5* mutant. Complementation studies and biochemical analysis were also performed.

The *rms* branching mutants, rather than being depleted in IAA levels, contained elevated IAA levels. We proposed a model where a non-response to SLs, which occurs in SL-biosynthesis or in SL-response *rms* mutants, stimulates the synthesis of a feedback signal, auxin, which controls via *RMS2*, CK levels in the xylem sap and SL biosynthesis gene expression. We tested this model by quantifying IAA levels after SL treatment using the pea SL *rms* mutants. We demonstrated that SLs repress IAA levels by down-regulating transcript levels of auxin biosynthesis genes in stem (*TAR2* and *YUC1*) independently of polar auxin transport.