Modelling physiological regulation of bud burst by sucrose and auxin

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Research focus. Branching is an important process for productivity (number of productive branches) and for visual quality of ornamental plants (branches spatial arrangement). Branching is difficult to control in production systems due to the lack of knowledge on the mechanisms regulating branching plasticity and response to environment. Our research aims at modelling the physiological mechanisms responsible for the spatio-temporal patterns of bud burst using rosebush as a plant model.

The burst of an axillary bud is inhibited by auxin, and several decades of research have led to the identification of the physiological pathway by which auxin regulates bud burst (Rameau et al., 2015). Recent studies have enlightened the importance of a novel player, sugars, and that sucrose modulates hormonal mechanisms suggesting a non-trivial interaction between sucrose and auxin (Mason et al., 2014; Barbier et al., 2015). The present study aims at modelling how sucrose interacts with auxin and other key hormones to regulate the burst of a bud. The "bud" model developed will serve as an elementary brick for a FSPM model simulating bud burst patterns on a plant.

Methods. Current knowledge allows defining a model of auxin action on the bud: auxin level in the stem controls the level of two "second messenger" hormones, cytokinins (CKs) and strigolactones (SLs), which would relay the auxin signal by migrating into the bud. We developed a computational model of this system and designed experiments to identify and quantify sucrose action in this model. Experiments consisted in cultivating nodal stem segments of rosebush in vitro with different sucrose and auxin levels, and in quantifying bud elongation, CK level, and the expression of genes involve in SL biosynthesis and signalling.

Results. We observed that increasing sucrose level decreased the inhibition of bud elongation by auxin, so that buds fed with high sucrose level were less inhibited by a given amount of auxin than those fed with low sucrose level. In accordance with literature, auxin repressed CKs and stimulated the expression of SLs biosynthesis genes. We demonstrate that sucrose repressed SL signalling. From these results, we designed a model that reflects the combined action of sucrose and auxin on bud elongation. We validated it for its capacity to predict the effect of external CK supply for different sucrose levels.

Conclusions. We developed the first "bud" model accounting for the combined action of auxin and sucrose on bud burst. Initially observed for rosebush, these results were also validated in pea, demonstrating model genericity. Next step is to integrate the "bud" model at the scale of a plant to understand the role of sugars, together with hormones, in the spatio-temporal regulation of bud burst. In particular, we want to evaluate if sugar availability can explain the variations of bud burst observed in response to light environment.