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## Axillary bud outgrowth regulation by light intensity: modelling hormone and sugar interactions.

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### Introduction

Branching is a key trait of plant adaptation to growth conditions. It involves a regulation of the outgrowth of axillary buds. These are inhibited by the fast-growing apical zone, a phenomenon called apical dominance. The extent of this inhibition depends on plant growth conditions. In this work, we investigate the ability of light intensity to modulate apical dominance and seek to identify the underlying mechanisms.

Apical dominance is mediated by hormonal and sugar competition mechanisms (Rameau et al., 2015; Schneider et al., 2019). The fast-growing apical zone is, on the one hand, a source of auxin, that is transported downwards in the stem and indirectly inhibits bud outgrowth. Auxin rapidly controls the production of two other hormones, the cytokinins (CK) and strigolactones (SL) in bud neighborhood, that are the direct regulators of bud outgrowth. On the other hand, the fast-growing apical zone is a highly important sink organ that deprives axillary buds from sugar (Mason et al. 2014). Such deprivation inhibits bud outgrowth in an unknown manner (Barbier et al., 2015).

Plant incident light intensity dampened apical dominance through increasing CK production in the stem in rose (Corot et al., 2017). The sugar involvement in light effect is unclear, despite light-induced changes in photosynthesis and plant sugar level. Here, we hypothesize that both CK and sugar are involved in incident light-related stimulation of bud outgrowth and antagonize dose-dependently auxin inhibiting effect. Moreover, CK production is supposed to be related to light perceived locally by the bud-bearing stem. Our objective was to test, by a combination of experiments and simulations, whether this hypothesis accounts for bud outgrowth differences observed between three light intensity regimes experienced by rose plants. First, we developed and calibrated a model at the scale of the bud-bearing stem using experiments *in vitro*. It assumes an effect of light on CK production, and an antagonistic and dose-dependent effect between CK-sugar module and auxin on bud outgrowth (see figure, left). Then, we used this model with, as inputs, the values of sugar, light, and auxin measured on intact plants, and tested model ability to simulate the observations of CK and bud outgrowth for the three light regimes.

### Material and Methods

To develop and calibrate the model at the bud-bearing stem scale, single bud-bearing stem segments of rose were grown *in vitro* under different sucrose and hormone concentrations, and light intensities. Bud length was scored each day, so as to estimate the time at which bud outgrowth started (denoted  $T$ ). Moreover, stem CK content was monitored for two auxin and sucrose levels. From all these data, a model was built and calibrated by minimizing the relative errors between measured and simulated values of CK contents and  $T$ . An absence of bud outgrowth was considered as an infinite  $T$ .

To test our light regulation hypothesis, young rose plants were grown under three different light intensity regimes: continuous low light (LL), continuous high light (HH), or temporary light limitation before the expected bud outgrowth period (LH). For each regime, the time at which bud outgrowth started was scored along the primary axis, while physiological variables (sugar, CK, auxin, and light levels in the axis median zone) were recorded before bud outgrowth started. The observed values in auxin, sugar, and absolute light level, were set as inputs of the *in vitro*-established model, and CK contents and the start of bud outgrowth were simulated.

## Results

A first set of *in vitro* experiments was dedicated to model sugar and auxin antagonistic effect on bud outgrowth. They indicated that sucrose availability did not antagonize auxin effect on CK levels but repressed bud response to SL (Bertheloot et al., 2020). Assembling these observations into a computational model was able to reproduce a diversity of observed dose-dependent sucrose-hormones effects on bud outgrowth for bud-bearing stems *in vitro*. A second set of *in vitro* experiments was dedicated to integrate light effect in this model. Adding a light intensity effect on CK production in the stem was able to capture observed bud outgrowth responses to light intensity, in interaction with sucrose and auxin levels. All these experiments led to a quantitative model simulating local light intensity, auxin, and sugar effect on bud outgrowth (see figure, left).

Our experiments in intact plants under different light regimes showed strong differences in bud outgrowth between LL, LH, and HH. This was correlated to differences in starch, CK, and local light levels. Introducing these differences in starch and light as inputs of the above-described model captured the observed differences in CK levels and bud outgrowth between LL, LH, and HH (see figure, right). This supports our initial hypothesis that plant light regimes modulates auxin-mediated apical dominance through changes in both (i) bud light environment that impacts stem CK production, and (ii) sugar availability.

## Conclusion

We have developed a model of bud outgrowth regulation accounting for the effect of local levels of hormone, sugar, and light intensity. The use of this model in intact plants made it possible to explain light intensity impact on the outgrowth of axillary buds by local variations in sugar and hormones in bud vicinity.

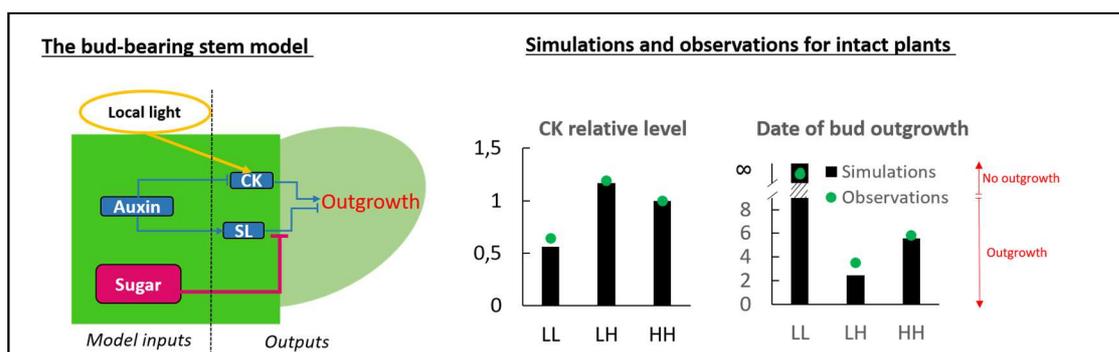


Figure: Bud outgrowth regulation by three different plant light regimes (LL, LH, HH): a model in which sugar and light-driven CK in bud neighborhood antagonizes dose-dependently auxin effect.

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