Review of: "TOR kinase controls shoot development by translational regulation of cytokinin catabolic enzymes"

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The article “TOR kinase controls shoot development by translational regulation of cytokinin catabolic enzymes” is an interesting work where authors found a connection between TOR kinase and hormone signaling in plants´ stem cells. After RNA-seq data analysis of samples treated with different TOR inhibitors, authors found enrichment for genes related to hormone signaling pathways. Citokinins (CK) related genes were notably reduced after TOR inhibition, in particular the six type A Arabidopsis response regulators (ARR). Further observations support these data including that the synthetic CK, 6-BA, did not induced type A ARR after TOR inhibition. To narrow down the TOR interaction with CK signaling they used CK response assay. In this assay, mRNA levels of *ARR5 were* reduced in the WT in the presence of trans-zeatin (tZ) after TOR inhibition. Also, CK homeostasis was compromised where tZ derivates were downregulated either in active and conjugated forms. On the top of this, the external addition of tZ derivates rescue *WUS*expression in the absence of TOR activity further indicating the interaction of this signaling pathway and hormonal control of shoot development. Authors further investigated on the differential tZ derivates accumulation and found that citokinin oxidases (CKX) are involved in this process by interacting with TOR at the translational level.

Overall, this is an important study to understand the implications of TOR signaling in plant development and also reinforces the interaction of this kinase with hormonal control. In my opinion this work deserves publication in a peer review journal, although I have some suggestions for the authors that may be included in this manuscript or even as a follow up work.

1. Regarding CKs related targets, I would suggest to validate the results obtained in RNA seq by monitoring protein levels using immunoblotting (when possible). This would strengthen the results obtained in the histochemical GUS assay (Figure 1).
2. I wonder what happens with TOR activity after external addition of CK. Authors should include these data as a control reference.
3. Authors claimed that “TOR is the central gatekeeper for light-, sugar- or CK dependent activation of GUS, respectively, once sugar and CK act together they can bypass TOR control”. Is it TOR as active in darkness as it is in the light?
4. Regarding TOR affecting CKX enzymes, authors should mention that TOR can be involved directly or

indirectly in PTMs in these enzymes.

5. Minor comments:

-Figure 6 C is not mentioned in the text

-Results section includes references and comments that should be included in the discussion.