

## **A Model Integrating Cytokinin into Regulation of Shoot Branching by Light Signals**

Tesfamichael H Kebrom<sup>1</sup> and John E Mullet<sup>1</sup>

<sup>1</sup>*Department of Biochemistry and Biophysics, Texas A&M University*

Shoot branching is regulated by environmental and hormonal signals. The proportion of red (R) and far red (FR) lights (R:FR ratio) from the sun incident on leaves is one of the environmental signals that regulate shoot branching. Red light is absorbed by leaves for photosynthesis whereas FR is reflected or transmitted. The R:FR ratio is perceived by the photoreceptor phytochrome B (phyB). High R:FR activates phyB that promotes bud outgrowth by modulating the expression of numerous genes including repressing the expression of the bud dormancy inducing *teosinte branched1* (*tb1*) gene. The microenvironment of plants grown in dense canopies is enriched with shade signals of FR light reflected by leaves, and thus the R:FR ratio is lower and phyB signaling is less active. The expression of *tb1* is elevated when phyB is inactivated by low R:FR or in phyB null mutants and buds become dormant soon after they are formed. Among the plant hormones that regulate shoot branching, auxin and strigolactones synthesized in the shoot apex and roots, respectively, induce bud dormancy without entering into buds whereas cytokinins synthesized in roots and stem promote bud outgrowth by acting locally within the bud. The inhibition of bud outgrowth by phyB deficiency in the phyB null mutant (*phyB-1*) sorghum has been linked to the expression of genes that could reduce cytokinin levels in the bud (Kebrom and Mullet, 2016). We propose a model integrating cytokinin in the regulation of shoot branching by light signals perceived by phyB.