Genes regulating flowering and dormancy in *Populus*

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The prolonged juvenile, non-flowering phase of forest trees is a major barrier to breeding. On the other hand, delay or prevention of flowering may be desirable in plantations to maximize vegetative growth and provide genetic containment. We are studying poplar homologs of genes that control flowering in the model annual plant *Arabidopsis*. Specifically, we studied homologs of the meristem identity genes *TERMINAL FLOWER1/ CENTRORADIALIS (TFL1/CEN), LEAFY (LFY)* and *APETALA1 (AP1)* using both overexpression and RNAi-mediated suppression transgenic poplars as well as suppression transgenics for the flowering time gene *SOC1*. Though gain- or loss-of function of these genes in Arabidopsis induces early flowering, most of the poplar transgenics do not show earlier flowering. However, downregulation of poplar *CEN1* modestly accelerates age of flowering onset and increases flowering intensity. To begin to decipher the regulatory pathways controlling flowering in poplar, we conducted transcriptome analysis of overexpression/suppression transgenic plants and wild type controls using a whole genome oligo-microarray and will present these results.

In addition to a conserved role in flowering, studies have begun to reveal that genes controlling flowering in trees have additional functions related to the woody perennial growth habit. Under growth chamber conditions that mimicked a winter dormancy cycle, we found that poplar *CEN1* overexpression transgenics cease growth earlier than control and require longer time for bud-flush, meanwhile, *CEN1* RNAi suppression transgenics show earlier bud flush. This result concurs with previous field observations that the level of *CEN1* overexpression correlated positively with date of bud flush (Mohamed et al. 2006). The poplar *AP1* homolog may also have a role in regulating vegetative shoot growth. Under nutrient deficient conditions that induced terminal bud set in wild-type trees, all transgenics overexpressing *AP1* maintained active shoot growth. Ongoing studies are investigating whether *AP1* also has a role in regulating winter dormancy.

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