Title: Intron-retained splice variants of the VND6 and SND1 transcription factors are dominant negatives that cross-regulate VND6 and SND1 members in *Populus trichocarpa*.

Ying-Chung Lin[1,*] (ylin14@ncsu.edu), Hao Chen,[1] Quanzi Li,[1,2] Rui Shi,[1] Sermsawat Tunlaya-Anukit,[1] Peng Shuai,[1,3] Wei Li,[1,4] Huiyu Li,[1,4] Ying-Hsuan Sun,[5] **Ronald R. Sederoff**,[1] and **Vincent L. Chiang**[1]

[1] Forest Biotechnology Group, Department of Forestry and Environmental Resources, North Carolina State University, Raleigh, North Carolina 27695

[2] State Key Laboratory of Tree Genetics and Breeding, Chinese Academy of Forestry, Beijing 100091, China

[3] College of Forestry, Fujian Agriculture and Forestry University, Fuzhou 350002, China

[4] State Key Laboratory of Tree Genetics and Breeding, Northeast Forestry University, Harbin, China

[5] Department of Forestry, National Chung Hsing University, Taichung 40227, Taiwan

Vascular-Related NAC-Domain 6 (VND6) is a key transcription factor (TF) involved in xylem and secondary cell wall differentiation. We discovered a splice variant of PtrVND6, called PtrVND6-C1^{IR}, which is a dominant negative regulator of full-size PtrVND6 members. PtrVND6-C1^{IR} lacks a transactivation domain and DNA binding ability, and can be translocated from the cytosol into the nucleus as a heterodimeric partner with any full-size PtrVND6 member. The formation of heterodimers between PtrVND6-C1^{IR} and the full-size PtrVND6 disrupts the function of the full-size PtrVND6, thereby repressing transcription of PtrVND6 direct targets in its network. Secondary Wall-Associated NAC Domain 1 (SND1) also affects secondary cell wall biosynthesis. We previously demonstrated that the splice variant of PtrSND1-A2, PtrSND1-A2^{IR}, can inhibit the PtrSND1 transcription network through the same mechanism. Using laser capture microdissection, we found that PtrVND-6C1^{IR}, PtrSND1-A2^{IR}, and all full-size PtrVND6 and PtrSND1 are expressed in both fiber and vessel cells. We further discovered that either PtrVND6-C1^{IR} or PtrSND1-A2^{IR} can inhibit both PtrVND6 and PtrSND1 transcription by the same mechanism. The cross-regulation between the PtrSND1 and PtrVND6 families through their splice variants suggests a general mechanism for the function of xylem specific NAC TFs controlling wood formation.