

# Research on wood formation sheds light on plant biology

August 23 2012

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(Phys.org)—Scientists at North Carolina State University have discovered a phenomenon never seen before in plants while studying molecular changes inside tree cells as wood is formed.

In research published online in [Proceedings of the National Academy of Sciences](#) the week of Aug. 20, the team found that one member of a family of proteins called transcription factors took control of a cascade of genes involved in forming wood, which includes a substance called lignin that binds fibers together and gives wood its strength.

The controller protein regulated [gene expression](#) on multiple levels, preventing abnormal or stunted plant growth. And it did so in a novel way.

The controller, a spliced variant of the SND1 family, was found in the [cytoplasm](#) outside the [cell nucleus](#). This is abnormal, because transcription factor proteins are always in the nucleus. But when one of the four other proteins in its family group was present, the spliced variant was carried into the nucleus, where it bound to the family member, creating a new type of molecule that suppressed the expression of a cascade of genes.

"This is nothing that's been observed before in plants," says Dr. Vincent Chiang, co-director of NC State's Forest Biotechnology Group with Dr. Ron Sederoff. Chiang's research team was the first to produce a transgenic tree with reduced lignin. High lignin levels are desirable for

lumber, but [lignin](#) is removed during the process of making paper or manufacturing biofuels.

Chiang, a professor in the College of Natural Resources, described the team's finding as the long-sought path to understanding the hierarchy of [gene regulation](#) for wood formation.

**Abstract:** Secondary Wall-Associated NAC Domain 1s (SND1s) are [transcription factors](#) (TFs) known to activate a cascade of TF and pathway genes affecting secondary cell wall biosynthesis (xylogenesis) in [Arabidopsis](#) and poplars. Elevated SND1 transcriptional activation leads to ectopic xylogenesis and stunted growth. Nothing is known about the upstream regulators of SND1. Here we report the discovery of a stem-differentiating xylem (SDX)-specific alternative SND1 splice variant, PtrSND1-A2<sup>IR</sup>, that acts as a dominant negative of SND1 transcriptional network genes in *Populus trichocarpa*. PtrSND1-A2<sup>IR</sup> derives from PtrSND1-A2, one of the four fully spliced PtrSND1 gene family members (PtrSND1-A1, -A2, -B1, and -B2). Each full-size PtrSND1 activates its own gene, and all four full-size members activate a common MYB gene (PtrMYB021). PtrSND1-A2<sup>IR</sup> represses the expression of its PtrSND1 member genes and PtrMYB021. Repression of the autoregulation of a TF family member by its only splice variant has not previously been reported in plants. PtrSND1-A2<sup>IR</sup> lacks DNA binding and transactivation abilities but retains dimerization capability. PtrSND1-A2<sup>IR</sup> is localized exclusively in cytoplasmic foci. In the presence of any full-size PtrSND1 member, PtrSND1-A2<sup>IR</sup> is translocated into the nucleus exclusively as a heterodimeric partner with full-size PtrSND1s. Our findings are consistent with a model in which the translocated PtrSND1-A2<sup>IR</sup> lacking DNA-binding and transactivating abilities can disrupt the function of full-size PtrSND1s, making them nonproductive through heterodimerization, and thereby modulating the SND1 transcriptional network. PtrSND1-A2<sup>IR</sup> may contribute to transcriptional homeostasis to avoid deleterious effects on xylogenesis

and plant growth.

Provided by North Carolina State University

Citation: Research on wood formation sheds light on plant biology (2012, August 23) retrieved 29 April 2024 from <https://phys.org/news/2012-08-wood-formation-biology.html>

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