MOLECULAR MECHANISMS OF AUXIN ACTION AND RESPONSE IN LOBLOLLY PINE (*PINUS TAEDA* L.)

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In loblolly pine, very little is known about the genes involved in auxin response, their function or their regulation. We are interested in dissecting the molecular mechanisms underlying auxin response that lead into pathways responsible for different traits in pine, including adventitious root formation.

We previously cloned 5 genes from loblolly pine (LPEAs: Loblolly Pine Early Auxin-induced) that belong to a large family of plant genes known as the Aux/IAA genes. We have been pursuing two main lines of research concerning these genes. One is to determine gene function and the other is to assess factors and cis-elements regulating expression.

We have transformed full length cDNAs of three LPEA genes fused to the CMV 35 S promoter into tobacco, and observed phenotypic variation in the progeny of some of the transgenic lines. Transformants containing LPEA1 and LPEA5, though variable, show similar phenotypes. The most pronounced phenotype is plants with severely impaired growth and developmental characteristics--very slow growth, low flower set and partial sterility. Most lines transformed with LPEA2, however, did not show any abnormal phenotype, except for one line that displays severe morphological abnormalities--altered leaf shape, slow growth, very dense inflorescences, extended pistil and partial sterility.

We are also trying to assess mechanisms and factors involved in regulation of LPEAs. We have found that maturation state significantly affects expression. In juvenile tissues, LPEAs were more strongly induced by auxin and the transcript levels remained high for a longer time period than in mature tissues. Recently, we isolated a genomic clone corresponding to LPEA1 cDNA . We fused different parts from the promoter region to a GUS reporter gene and transformed them into tobacco. Though results of these experiments are preliminary, we observed that most of the GUS activity is localized in the region of the vascular elements, suggesting tissue specificity of the promoter or higher auxin levels in these regions.

In addition to studying the LPEAs, we are using microarray approach to clone other genes that are induced by auxin. We are now in the process of confirming and further characterizing the induction of candidate genes by auxin.

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