INVOLVEMENT OF A MYB FACTOR IN THE REGULATION OF PHENYLALANINE PATHWAY IN MARITIME PINE

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Wood is traditionally among the most important commercial products because of the high demand that exits for its derivatives. Trees, including conifers, divert large quantities of carbon into the biosynthesis of phenylpropanoids, particularly to generate lignin. Although lignin and other phenolic compounds do not contain nitrogen, phenylalanine metabolism is required to channel photosynthesis-derived carbon to phenylpropanoid biosynthesis. The phenylpropane skeleton required for lignin biosynthesis is provided by the deamination of phenylalanine in the reaction catalysed by the enzyme phenylalanine ammonia-lyase (PAL). This reaction is quantitatively important in trees because lignin biosynthesis is required for wood formation, and it releases large quantities of ammonium. An efficient and coordinated pathway for the amination of prephenate and the deamination of phenylalanine should be operative in lignifying cells to provide phenylalanine for lignin biosynthesis, and to re-assimilate ammonium.

We hypothesized that one way to ensure efficient photosynthetic carbon channeling for lignin and other phenylpropanoid biosynthesis, together with nitrogen recycling, would be to couple both processes in time and in space by transcriptionally regulating the genes involved in phenylalanine biosynthesis and use.

The experiments described in this communication attempt to test this hypothesis. To this end, we have isolated the promoter region of the three genes involved in the phenylalanine pathway in *Pinus pinaster:* PAL, GS1b and PAT. We have conducted both *in vitro* and *in vivo* studies using three different Myb transcription factors: PtMyb1, PtMyb4 from *P. taeda* and PpMyb8 from *P. pinaster.* We have studied the possible coupling in space and time of gene products for the operative co-regulation of both processes in pine trees, and have proven that Myb8 is a potential candidate to be the transcriptional regulator of phenylalanine metabolism in *P. pinaster* vascular cells.