New Insights Into Ethylene Signalling and Wood Development

Abstract

This thesis describes experimental work concerning the biology of ethylene in the development of xylem in hybrid aspen, Populus tremula L. x tremuloides Michx. Ethylene applied to wood forming tissue has previously been shown to stimulate radial growth in trees. In dicotyledonous angiosperm trees, ethylene has also been implicated in the response to leaning stress when tension wood forms. Tension wood is characterized by a localized stimulation of cambial growth, altered patterning of cell differentiation, and the synthesis of an additional cell wall layer with altered chemistry. Strong functional evidence linking endogenous ethylene to wood formation, however, has remained elusive. Transgenic hybrid aspen trees with reduced sensitivity to ethylene were generated by ectopically expressing the Arabidopsis thaliana dominant mutant allele of the ethylene receptor ETR1 (Atetr1-1). Aspects of wood growth and development suppressed by Atetr1-1 were assayed by applying ethylene or its immediate metabolic precursor, 1-aminocyclopropane-1-carboxylic acid (ACC), to independent transgenic lines and by comparing responses with wild-type. Remarkably, the rate of cell proliferation in tension wood of leaning trees with ectopic expression of Atetr1-1 was reduced. For the first time, this result provides conclusive evidence that ethylene is an endogenous regulator of wood formation. 1-methylcyclopropene (1-MCP), a gaseous structural analogue to ethylene which efficiently inhibits ethylene action was used as a positive control for ethylene insensitivity. Applied ACC and ethylene was observed to influence bulk density, ultrastructure, mechanical properties, and the ratio of S-type to G-type lignin in the secondary cell walls of wood. In addition, a resource describing the transcriptional expression of ETHYLENE RESPONSE FACTORS is also presented as an initial effort to elucidate downstream molecular signalling.

Keywords: wood, ethylene, cambium, lignin, cellulose, growth, ERF, ETR1

Author's address: Jonathan Love, SLU, Department of Forest Genetics and Plant Physiology, 901 83 Umeå, Sweden *E-mail:* Jonathan.Love@genfys.slu.se

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