

Induction of isoprenyl diphosphate synthases, plant hormones and defense signalling genes correlates with traumatic resin duct formation in Norway spruce (*Picea abies*)

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Bottom Line: Among defense hormones, jasmonate and jasmonate-isoleucine conjugate accumulated to higher levels in trees with extensive traumatic resin duct formation, whereas salicylate did not. Jasmonate and ethylene are likely to both be involved in formation of traumatic resin ducts based on elevated transcripts of genes encoding lipoxygenase and 1-aminocyclopropane-1-carboxylic acid oxidase associated with resin duct formation. Other genes involved in defense signalling in other systems, mitogen-activated protein kinase3 and nonexpressor of pathogenesis-related gene1, were also associated with traumatic resin duct formation.

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Abstract: Norway spruce (*Picea abies*) defends itself against herbivores and pathogens by formation of traumatic resin ducts filled with terpenoid-based oleoresin. An important group of enzymes in terpenoid biosynthesis are the short-chain isoprenyl diphosphate synthases which produce geranyl diphosphate (C(10)), farnesyl diphosphate (C(15)), and geranylgeranyl diphosphate (C(20)) as precursors of monoterpenes, sesquiterpenes, and diterpene resin acids, respectively. After treatment with methyl jasmonate (MJ) we investigated the expression of all isoprenyl diphosphate synthase genes characterized to date from Norway spruce and correlated this with formation of traumatic resin ducts and terpene accumulation.

Formation of traumatic resin ducts correlated with higher amounts of monoterpenes, sesquiterpenes and diterpene resin acids and an upregulation of isoprenyl diphosphate synthase genes producing geranyl diphosphate or geranylgeranyl diphosphate. Among defense hormones, jasmonate and jasmonate-isoleucine conjugate accumulated to higher levels in trees with extensive traumatic resin duct formation, whereas salicylate did not. Jasmonate and ethylene are likely to both be involved in formation of traumatic resin ducts based on elevated transcripts of genes encoding lipoxygenase and 1-aminocyclopropane-1-carboxylic acid oxidase associated with resin duct formation. Other genes involved in defense signalling in other systems, mitogen-activated protein kinase3 and nonexpressor of pathogenesis-related gene1, were also associated with traumatic resin duct formation. These responses were detected not only at the site of MJ treatment, but also systemically up to 60 cm above the site of treatment on the trunk.

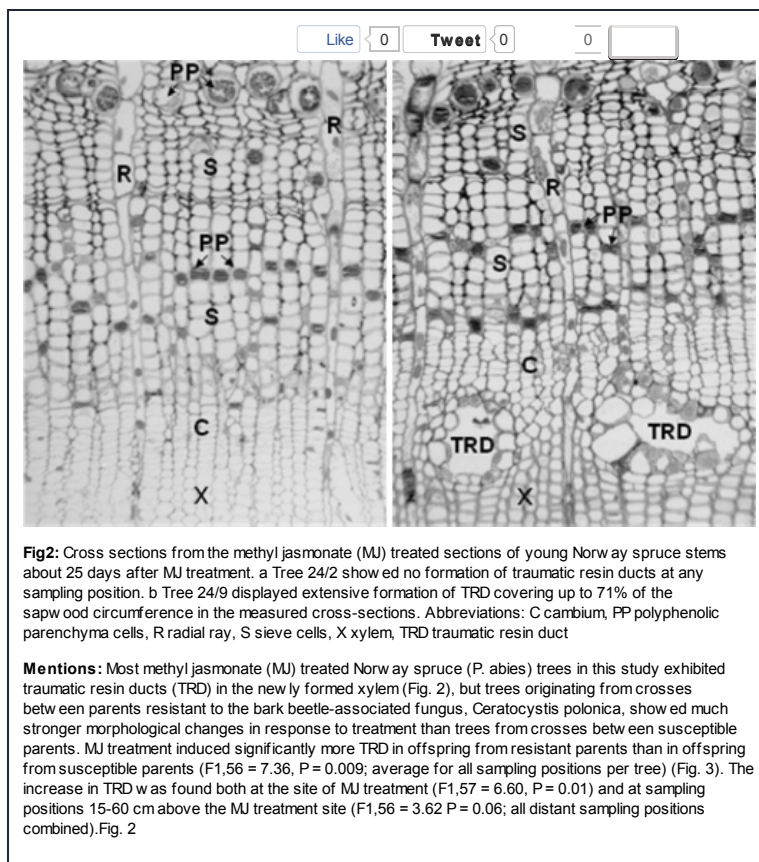


Fig2: Cross sections from the methyl jasmonate (MJ) treated sections of young Norway spruce stems about 25 days after MJ treatment. a Tree 24/2 showed no formation of traumatic resin ducts at any sampling position. b Tree 24/9 displayed extensive formation of TRD covering up to 71% of the sapwood circumference in the measured cross-sections. Abbreviations: C cambium, PP polyphenolic parenchyma cells, R radial ray, S sieve cells, X xylem, TRD traumatic resin duct

Mentions: Most methyl jasmonate (MJ) treated Norway spruce (*P. abies*) trees in this study exhibited traumatic resin ducts (TRD) in the newly formed xylem (Fig. 2), but trees originating from crosses between parents resistant to the bark beetle-associated fungus, *Ceratocystis polonica*, showed much stronger morphological changes in response to treatment than trees from crosses between susceptible parents. MJ treatment induced significantly more TRD in offspring from resistant parents than in offspring from susceptible parents ($F_{1,56} = 7.36$, $P = 0.009$; average for all sampling positions per tree) (Fig. 3). The increase in TRD was found both at the site of MJ treatment ($F_{1,57} = 6.60$, $P = 0.01$) and at sampling positions 15-60 cm above the MJ treatment site ($F_{1,56} = 3.62$, $P = 0.06$; all distant sampling positions combined) (Fig. 2).

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