# Biosynthesis of Nicotine as an Anti-insect Defense in Plants

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## Abstract

Nicotine and related pyridine alkaloids are synthesized in the root and then transported to the aerial parts of tobacco plants. Expression of biosynthetic genes for these alkaloids is enhanced 3-4-fold upon insect herbivory to the leaf. Current model suggests that jasmonate acts as a transmissible signal from the damaged leaf to the underground part, where it activates structural genes of nicotine biosynthesis via the conserved COI1-JAZ-MYC2 pathway. In Arabidopsis, the MYC2-family basic-helix-loop-helix transcription factors mediate transcriptional regulation of jasmonate-responsive genes, and their transcriptional activities are suppressed by physical interactions with Jasmonate-ZIM domain (JAZ) suppressors. Regulatory *NIC* loci that positively regulate nicotine biosynthesis have been genetically identified and their mutant alleles have been used to breed low-nicotine tobacco varieties. The *NIC2* locus comprises tandemly arrayed transcription factor genes of an Ethylene Response Factor (ERF) subfamily; in the *nic2* mutant, at least seven *ERF* genes are deleted altogether. Overexpression, suppression, and dominant-repression experiments using transgenic tobacco roots showed functional redundancy and divergence among the *NIC2*-locus *ERF* genes. These transcription factors recognized a GCC-box element in the promoters of nicotine pathway genes, and specifically activated all known structural genes in the pathway. We also demonstrate that tobacco*MYC2* controls nicotine biosynthesis genes in two combinatorial ways, by directly binding G-box in the target promoters, as well as by up-regulating the *NIC2*-locus *ERF* genes.

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