Male-sterility induction in transgenic tobacco plants with an unedited atp9 mitochondrial gene from wheat

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Abstract

Cytoplasmic male sterility in plants is associated with mitochondrial dysfunction. We have proposed that a nuclear-encoded chimeric peptide formed by mitochondrial sequences when imported into the mitochondria may impair organelle function and induce male sterility in plants. A model developed to test this hypothesis is reported here. Assuming that the editing process in higher plant mitochondria reflects a requirement for producing active proteins, we have used edited and unedited coding sequences of wheat ATP synthase subunit 9 (atp9) fused to the coding sequence of a yeast coxIV transit peptide. Transgenic plants containing unedited atp9 exhibited either fertile, semifertile, or male-sterile phenotypes; controls containing edited atp9 or only the selectable marker gave fertile plants. Pollen fertility ranged from 31% to 75% in fertile plants, 10% to 20% in semifertile plants, and < 2% in male-sterile plants. Genetic and molecular data showed that the chimeric plasmid containing the transgene is inherited as a Mendelian trait. The transgenic protein is imported into the mitochondria. The production and frequency of semifertile or male-sterile transgenic plants conform to the proposed hypothesis.