

INCREASED OLEIC ACID CONTENT IN TOBACCO LEAVES DUE TO THE EXPRESSION OF AN OLIVE ACYL CARRIER PROTEIN

BELLUCCI M.*, DE MARCHIS F.*, VALERI M.***, POMPA A.*, BOUVERET E.***, ALAGNA F.*****, GRISAN S.*, STANZIONE V.*****, MARIOTTI R.*, CULTRERA N.*, BALDONI L.*

*) Institute of Biosciences and Bioresources, Research Division of Perugia, National Research Council, 06128 Perugia (Italy)

**) PlantLab, Institute of Life Sciences, Scuola Superiore Sant'Anna, 56127 Pisa (Italy)

***) LISM, CNRS, Aix-Marseille University, 13402 Marseille Cedex 20 (France)

*****) CRA, Agricultural Research Council, research unit for table grapes and wine growing in mediterranean environment, Via Casamassima 148, Turi, 70010 Bari (Italy)

*****) Institute for Agricultural and Forest Systems in the Mediterranean, Research Division of Perugia, National Research Council, Via Madonna Alta 128, 06128 Perugia (Italy)

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The olive (*Olea europaea* L.) is an economically important fruit oil crop that accumulates lipids in fruit mesocarp and, to a minor extent, in the seed. Fatty acid (FA) biosynthesis plays a crucial role in lipid accumulation in olive mesocarp, therefore we investigated the effect of olive acyl carrier protein (ACP) on FA composition by overexpressing an olive ACP cDNA in tobacco plants. Aiming to find the best transformation method to overexpress olive ACP in tobacco, we inserted the OeACP1.1A cDNA both in the nucleus and in the chloroplast DNA of tobacco plants, resulting in extensive transcription of the transgenes. The transplastomic plants accumulated lower olive ACP levels in comparison to nuclear-transformed plants. Moreover, the phenotype of the former plants was characterized by pale green/white cotyledons with abnormal chloroplasts, delayed germination and reduced growth. We suggest that the transplastomic phenotype was likely caused by inefficient olive ACP mRNA translation in chloroplast stroma. Conversely, total lipids from leaves of nuclear transformants expressing high olive ACP levels showed a significant 1.5-fold increase in the oleic acid (18:1) level, together with a small but significant increase in linolenic acid (18:3) and a concomitant significant reduction of hexadecadienoic acid (16:2) and hexadecatrienoic acid (16:3). Interestingly, also the transplastomic plants had increases in the levels of 18:1 and 18:3 FAs, and decreases in 16:2 and 16:3 FAs in comparison to the wild type, but these differences were less marked than those of the nuclear transformed plants and not statistically significant. This implies that in leaves of tobacco transformants, as likely in the mesocarp of olive fruit, olive ACP not only plays a general role in FA synthesis, but also seems to be specifically involved in chain length regulation forwarding the elongation to C18 FAs and the subsequent desaturation to 18:1.