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beta-Ketoacyl-acyl carrier protein (ACP) synthase II (KASII) elongates 16:0-ACP to 18:0-ACP in the plastid, where it competes with three other enzymes at the first major branch point in fatty acid biosynthesis. Despite its key metabolic location, the influence of KASII in determining seed oil composition remains unclear, in part because the biochemical consequences of the *fab1-1* mutation were unresolved. Thus, *fab1-1*, and a newly identified knockout allele, *fab1-2*, were analyzed in the context of the hypothesis that modulating KASII activity is sufficient to convert the composition of a temperate seed oil into that of a palm-like tropical oil. No homozygous *fab1-2* individuals were identified in progeny of self-fertilized heterozygous *fab1-2* plants, 1/4 of which aborted before the torpedo stage, suggesting that *fab1-2* represents a complete loss of function and results in lethality when homozygous. Consistent

with this hypothesis, homozygous

fab1-2

plants were identified when a

fab1-1

transgene was introduced, demonstrating that

fab1-1

encodes an active KASII. Strong seed-specific hairpin-RNAi reductions in

FAB1

expression resulted in abortion of 1/4 of the embryos in an apparent phenocopy of

fab1-2

homozygosity. In less severe

FAB1

hairpin-RNAi individuals, embryos developed normally and exhibited a 1:2:1 segregation ratio for palmitate accumulation. Thus, early embryo development appears sensitive to elevated 16:0, whereas at later stages, up to 53% of 16:0, i.e., a 7-fold increase over wild-type levels, is tolerated. These results resolve the role of KASII in seed metabolism and demonstrate that modulation of

Arabidopsis

KASII levels is sufficient to convert its temperate oilseed composition to that of a palm-like tropical oil.

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