**IN BRIEF**

**Lipids in Leaves: Fatty Acid β-Oxidation Affects Lipid Homeostasis**

DNA, RNA, and proteins get a lot of attention, but without lipids, a cell is just a soup. Cellular membranes have a remarkable variety of lipids, and different organelles have different lipid compositions that must be maintained despite changes caused by vesicular trafficking among organelles (reviewed in Holthuis and Menon, 2014). Lipids move via vesicular trafficking, direct membrane contact between organelles, and via so-called pipelines operated by lipid transfer proteins. Changes in lipid composition can alter the surface charge, thickness, and fluidity of a membrane—characteristics that affect, for example, secretion via the Golgi and endoplasmic reticulum. Lipid homeostasis involves a balance between accumulation of membrane lipids and storage lipids such as triacylglycerol. Remobilization of stored triacylglycerol occurs via β-oxidation in the peroxisome, which produces both fatty acid components for membrane lipids and energy for seedling development.

Although lipid metabolism in seeds has received substantial attention, the importance of lipid synthesis and turnover in leaves remains unclear. To examine this, Fan et al. (2014) used trigalactosyldiacylglycerol1-1 (tgd1) mutants, in which a defect in thylakoid lipid synthesis diverts fatty acids, causing accumulation of triacylglycerol in leaves. They found that overexpression of PHOSPHOLIPID:DIACYLGLYCEROL ACYLTRANSFERASE1 (PDAT1) causes increased fatty acid synthesis and turnover, and this requires the lipase SUGAR-DEPENDENT1 (SDP1), as PDAT1-overexpressing sdp1 plants produce high levels of triacylglycerol. Also, tgd1 sdp1 double mutants showed triacylglycerol accumulation in leaves, an effect not seen in double mutants with other lipases (see figure). This turnover appears to occur via β-oxidation in peroxisomes, as double mutants of tgd1 with peroxisomal transporter1 (pxa1) also accumulated triacylglycerol (see figure). However, markers for the β-oxidation pathway did not increase in the tgd1 mutants, indicating that this pathway has sufficient capacity to handle the increased turnover. Also, overexpression of SDP1, but not other lipases, was associated with decreased triacylglycerol accumulation in tgd1 mutants. Mutants lacking the phosphatidic acid phosphohydrolases PAH1 and PAH2 also showed decreased triacylglycerol accumulation in the tgd1 background, indicating that these lipids provide key precursors for triacylglycerol synthesis. Thus, the authors identified a key role for lipid turnover and the β-oxidation pathway in accumulation of triacylglycerol in leaves.

Researchers working on biofuels have taken an interest in lipids in leaves, as lipids make energy-dense, easily extractable biofuels that require no saccharification or fermentation and oil-rich leaves would provide a superb biomass feedstock. However, leaves as source tissues have proven recalcitrant to oil accumulation (Chapman et al., 2013), as their metabolism favors starch accumulation. The increase in triacylglycerol in the double mutants examined here, up to 9% of leaf dry weight in tgd1 pxa1 and tgd1 sdp1 plants, indicates that strategies to increase lipid accumulation should target turnover. However, in addition to increased lipid droplets and changes in membrane lipid composition, these double mutants also show somewhat slower growth and impaired pollen germination (in tgd1 pxa1), indicating that perturbation of lipid homeostasis with the entire plant might also have unintended agronomic effects. Thus, this emerging research informs efforts to produce oil for biofuels by highlighting the importance of peroxisomal β-oxidation in triacylglycerol accumulation in leaves and provides cautionary information on potential problems with such approaches.

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**REFERENCES**


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