IN BRIEF

Out with the Old: The Fate of Obsolete Peroxisomes

Nutrient reserves in seeds drive early seedling growth. In germinating Arabidopsis thaliana seeds, lipases hydrolyze triacylglycerol into free fatty acids (Kelly et al., 2011), which then undergo β-oxidation in peroxisomes. The resulting acetyl CoA is then catabolized in the glyoxylate cycle into organic acids that are further processed into sugars that can fuel seedling growth. Two key enzymes of the glyoxylate cycle, isocitrate lyase and malate synthase, are present in the peroxisomal matrix of various seedling tissues (Pracharoenwattana and Smith, 2008), but disappear during postgerminative growth (Nishimura et al., 1986). It is unclear how obsolete peroxisomal proteins are removed during the transition from heterotrophic to autotrophic growth (reviewed in Hu et al., 2012), but it has been proposed that these proteins are: (1) degraded in peroxisomes by hitherto unidentified proteases, (2) polyubiquitylated and disassembled by proteasomes, or (3) subject to pexophagy, a type of selective autophagy documented in yeast (Pichia pastoris) and mouse (Mus musculus) that specifically destroys peroxisomes (Iwata et al., 2006).

Kim et al. (pages 4956–4966) investigated the third possibility above. They generated autophagy-defective Arabidopsis lines (autophagy-related 5-1 [atg5-1] and atg7-2) that harbored the peroxisomal marker CFP-SKL. After several days of growth under long-day conditions, the mutant hypocotyls accumulated more peroxisomes than the wild type (see figure), and degradation of peroxisomal enzymes was delayed in the mutants, suggesting that autophagy is involved in peroxisomal degradation during the shift to autotrophic growth. Further analyses suggested that peroxisomal proteins are degraded in the vacuole. Therefore, pexophagy appears to remove obsolete peroxisomal proteins from Arabidopsis hypocotyls during postgerminative growth. The authors propose that this mechanism cleans up old organelles and facilitates cell remodelling as the plant transitions from heterotrophic to autotrophic growth.

A related study conducted by Shibata et al. (pages 4967–4983) suggests that autophagy is a critical quality control mechanism for peroxisomes in Arabidopsis leaves. These authors identified three Arabidopsis mutants that contain aggregated peroxisomes in leaf tissue and found that the disrupted genes were ATG2, ATG18a, and ATG7. They showed that peroxisome aggregation in the mutant leaves is induced by hydrogen peroxide because of catalase inactivation and that autophagy selectively eliminates the damaged peroxisomes. Furthermore, a recent functional analysis of Arabidopsis LON2, a resident peroxisomal protease, also suggested the existence of pexophagy in plants (Farmer et al., 2013). Various atg mutants were found to suppress lon2 defects and peroxisomal degradation via autophagy was shown to be enhanced in the lon2 mutant. Together, these three studies show that pexophagy appears to determine the fate of peroxisomes during both heterotrophic and autotrophic growth.

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Plant Cell 2013;25;4769; originally published online December 24, 2013;
DOI 10.1105/tpc.113.251210

This information is current as of July 9, 2017

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