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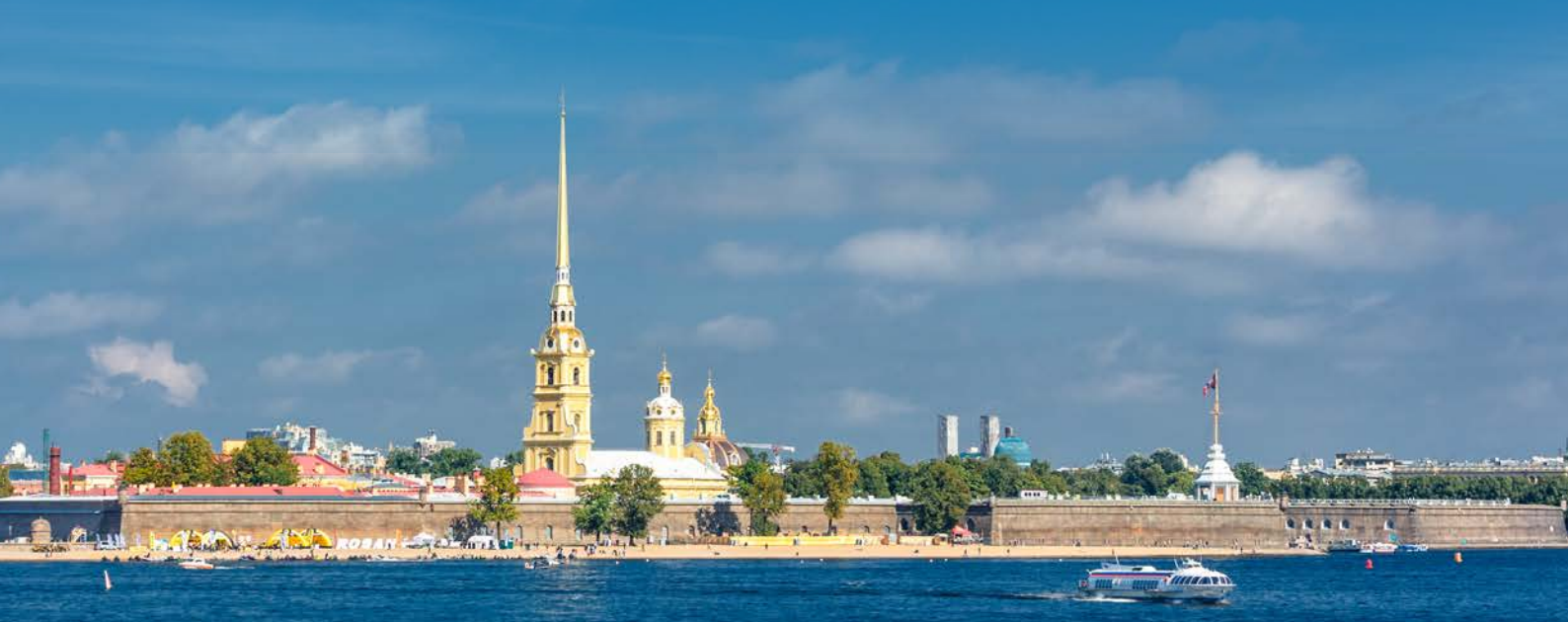

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only 24 h after excision, although the formation of autophagosome was visualized after 2 h. Thus, our data suggest that the up-regulation of *ATG8s* is a hallmark of the response of plants to various abiotic cues. Differential expression of numerous members of *ATG8* family allows the fine-tuning of the autophagic catabolic activity of plant cells.

Autophagic degradation of plant organelles

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Autophagy, an ancient catabolic program with a primarily cytoprotective role, is emerging as a process underlying all sides of plants' life. It is triggered in response to stress, removing cellular components not vital for survival and providing energy released from degraded structures when nutrients are limited. Autophagy also occurs at a low level on a constitutive basis, assuring the turnover of 'worn-out' or damaged cell parts. It seems that autophagy is crucial for the degradation of larger cellular structures, and many organelles in plant cells have been revealed as targets of autophagy. Thus far, these include peroxisomes, ribosomes, mitochondria, endoplasmic reticulum and plastids.
We have investigated the role of macroautophagy in the degradation of peroxisomes and chloroplasts. We used stable transgenic suspension-cultured cell lines of tobacco Bright Yellow 2 that expressed a peroxisome-targeted version of Enhanced Yellow Fluorescent Protein to study the role of autophagy in peroxisome turnover in course of cultivation and ageing of the cultures. We also monitored the size of the cellular peroxisome pool, ROS production and comparative rates of organelle degradation in tobacco BY-2 cells. In leaves of *Hordeum vulgare* and *Arabidopsis thaliana*, wild type as well as *chlorina* mutants lacking chlorophyll *b*, we investigated the relationship between the stability of photosynthetic antenna complexes and the induction of autophagy. While the role of autophagy in the degradation of stromal proteins including Rubisco has long been established (Wada et al 2009 Plant Physiol), the possible role of autophagy in the degradation of components of chloroplast membranes requires further investigation.
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Cytoplastic Glyceraldehyde-3-Phosphate Dehydrogenases Interact with ATG3 to Negatively Regulate Autophagy and Immunity in Plants

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The plant innate immune response includes the hypersensitive response (HR), a form of programmed cell death (PCD). PCD must be restricted to infection sites to prevent the HR from playing a pathologic rather than protective role. We find that the evolutionarily conserved autophagy pathway plays an essential role in plant innate immunity and negatively regulates PCD. Further, we show that autophagy-related protein 3 (ATG3) interacts with the cytosolic glyceraldehyde-3-phosphate dehydrogenases (GAPCs) to regulate autophagy in plants. We found that oxidative stress inhibits the interaction of ATG3 with GAPCs. Silencing of GAPCs significantly activates ATG3-dependent autophagy, while overexpression of GAPCs suppresses autophagy in *N. benthamiana* plants. Moreover, silencing of GAPCs enhances N gene-mediated cell death and plant resistance against incompatible pathogens