ABSTRACT - PLANT PHYSIOLOGY

AUTOPHAGY AND REDOX STATES: RECIPROCAL REGULATION

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Autophagy is a self-degradative process, with fundamental roles in cellular and organism homeostasis. Diverse situations of stress have been shown to be inducers of the autophagic process. The major source of ROS in the plant cell is the chloroplast, where different oxygen species can be produced. The aim of this work is to study the effects on the autophagic processes of the different ROS produced in the chloroplast, and how autophagy regulates the redox state of the plant cell. Our results point out that the production of O2– or H2O2 in the chloroplast has an inhibitory effect on autophagy, where treatments with different scavengers rescued this inhibition. This inhibition of autophagy was shown to occur under oxidative conditions, measured by GRX-roGFP2 or roGFP2-ORP1, biosensors of glutathione redox state and H2O2 endogenous respectively. Also, the production of 1O2 shows an increase of the autophagy flux, suggesting a opposite roll of the different ROS in the autophagy process. In order to evaluate the role of autophagy in the regulation of the redox state of the plant cell, atg7 mutant lines expressing the GRX-roGFP2 construct targeting cytoplasm, chloroplast and peroxisome were obtained. The degree of oxidation

between the wildtype and atg7 lines was compared during leaf development, observing a significant increase in the degree of oxidation in both chloroplasts and peroxisomes during leaf senescence. Moreover, it was observed that atg7 mutant plants presented a higher level of oxidation in chloroplasts and peroxisomes after being treated with H2O2. The fact that atg mutants show greater oxidation during senescence and after oxidative stress, would provide more evidence linking the autophagy process in the control and degradation of oxidized organelles, been these processes essential for the regulation of the redox state of the plant cell.