



# **Autophagy: Chapter 12. Molecular Mechanisms Underlying the Activation of Autophagy Pathways by Reactive Oxygen Species and their Relevance in Cancer Progression and Therapy**

*Noemí Rubio Romero, Patrizia Agostinis*

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Reactive oxygen species (ROS) have emerged as signaling molecules in pathways regulating cell growth and differentiation, inflammation, immune responses, survival, and death. ROS have been shown to promote autophagy, a lysosomal pathway for degradation of dysfunctional unnecessary cellular components. In fact, recent works have revealed a complex cross-talk between these intertwined signals. Whereas ROS can modulate autophagy activation in response to different types of stimuli, autophagy, in turn, may modulate ROS production by degrading, for example, dysfunctional mitochondria that generate aberrant amounts of ROS. Autophagy pathways can act both as tumor-promoter and tumor-suppressor mechanisms, with involvement of ROS in both cases. Paradoxically, whereas ROS and autophagy regulation may contribute to cancer initiation and progression, many antineoplastic treatments are precisely based on the massive production of ROS and activation of autophagy to induce cell death and eradication of diseased tissue. Nevertheless, autophagy activation has also shown a cytoprotective role against the efficiency of the therapy, and the mechanism that controls the switch between these two cellular functions is still unknown. In this chapter we will review the molecular mechanisms by which ROS modulate autophagy, and those modulated by autophagy to control ROS production, in the context both of cancer development and of cancer treatment.

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