# Special Issue

# The Interplay between Autophagy and ROS in Cancer

## Message from the Guest Editors

Cancer cells are commonly characterized by accumulation of reactive oxygen species (ROS). Optimal levels of ROS act as signaling molecules to promote the survival and proliferation of cancer cells, while excessive ROS levels are detrimental to cells. Autophagy is closely involved in ROS-mediated biological events in cancer cells. ROS can directly or indirectly stimulate autophagy. Reciprocally, autophagy serves as a survival mechanism to scavenge excessive ROS and buffer oxidative stress. However, contradictory to the above-mentioned opinions, ROS have also been reported to inhibit, rather than induce, autophagy; and autophagy can promote, rather than suppress, ROS accumulation. The complex reciprocity in ROS and autophagy regulation in cancer seems to be highly context-dependent. In this issue, the roles of ROS, autophagy and their interplay mechanisms in cancer will be highlighted, to enable a deeper understanding of cancer biology and favor the development of future cancer therapeutics.

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