Autophagy in cancer

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ABSTRACT

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The 2016 Nobel Prize in Physiology and Medicine has been awarded to Yoshinori Ohsumi for the discovery of mechanism for autophagy. His work published in 1993 greatly transformed the understanding of autophagy and now its role in human physiology and disease is well appreciated.

Autophagy is a general term for the degradation of cytoplasmic components within the lysosomes. There are three types of autophagy— macroautophagy, microautophagy, and chaperone-mediated autophagy. The term usually indicates macroautophagy unless otherwise specified. Autophagy is mediated by a unique organelle called the autophagosome.

The most important trigger of autophagy is nutrient starvation. Lack of any type of essential nutrient can induce autophagy. Autophagy plays a housekeeping role in removing misfolded or aggregated proteins, clearing damaged organelles, such as mitochondria, endoplasmic reticulum and peroxisomes, as well as in eliminating intracellular pathogens. Thus, it is actually a survival mechanism. Autophagy can be either non-selective or selective in the removal of specific organelles, ribosomes and protein aggregates. It promotes cellular senescence and cell surface antigen presentation, limits necrosis making its role important in preventing diseases such as cancer, neurodegeneration, cardiomyopathy, diabetes mellitus, autoimmune diseases, liver diseases and certain infections. Thus, it can easily be said that disruption of autophagy leads to diseases. Autophagy can be considered as a cellular ‘recycling factory’ that promotes energy efficiency through ATP generation and mediates damage control by removing non-functional proteins and organelles [1].

Autophagy has a vital role in cancer; both in protecting against cancer and potentially contributing to the cancer growth. Autophagy promotes survival of tumor cells that have been starved, or that degrade apoptotic mediators such as the use of inhibitors of the late stages of autophagy (such as chloroquine). Role of autophagy is both as a tumor suppressor and as a factor in the tumor cell survival. It is more likely to be used as a tumor suppressor. Necrosis and chronic inflammation also have been limited through autophagy which helps protect against the formation of tumor cells. In neoplastic cells, autophagy is used as a way to deal with stress on the cell. Once this autophagy related genes are inhibited, cell death is potentiated. Metabolic stresses include hypoxia, nutrient deprivation, and an increase in proliferation. Autophagy is activated to recycle ATP and maintain survival of the cancerous cells. Autophagy enable continued proliferation of tumor cells by maintaining cellular energy production. By inhibiting autophagy genes in these neoplastic cells, regression of the tumor and extended survival of the organs affected by the tumors has been reported. Inhibition of autophagy has also a role in enhancing the effectiveness of anticancer therapies [2].

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