

# Function of Autophagy in Chemotherapy-Induced Cancer and Novel Treatment Approaches

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## Commentary

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## DESCRIPTION

The main issue with modern cancer treatments is patient resistance to chemotherapy and radiation. The therapy outcome is still far from adequate despite much research. The role of autophagy in the complex network of variables that lead to drug resistance in cancer cells is clearer. In order to provide the cells with nutrition and building blocks, the process of autophagy outlines how the cytoplasm can be selectively ingested and then destroyed in autophagolysosomes. Stresses including hypoxia and the removal of growth factors can cause autophagy, which regulates cellular homeostasis.

Although maintaining cellular metabolic balance is autophagy's primary physiological function, dysregulated autophagy has been linked to a number of illnesses, including cancer. It's interesting to note that autophagy in cancer can either promote or inhibit tumour growth. For instance, by regulating cell proliferation and the creation of reactive oxygen species, autophagy can prevent the growth of tumours. On the other hand, under conditions where nutrition is limited, autophagy can give nutrients to the tumour cells to enable tumour growth, which encourages the formation of tumours.

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Anticancer therapy also exhibits this ambiguous behaviour: Autophagy has been discovered to increase the cytotoxicity of chemotherapeutic medications by causing autophagic cell death, but autophagy has also been connected to drug resistance since suppressing autophagy has been found to make tumour cells more susceptible to cell death caused by anticancer drugs. This chapter will concentrate on the dual functions of autophagy in carcinogenesis and chemotherapy, classify the autophagy inducers and inhibitors used to treat cancer, and explore recent issues with the creation of new drugs.

The information now available suggests that autophagy has a potentially significant impact on how neuroblastoma cells behave when being treated. Hence, the degree of autophagy activation may be a major determinant of the effectiveness of the provided (chemo) treatment, radiation, or other more targeted anticancer therapies. According to this approach, a greater knowledge of the circumstances in which autophagy may impact the nature of the neuroblastoma cells' response to medications supplied may pave the way for more efficient therapy plans. As a result, treating cancerous cells through autophagic regulation may yield more encouraging outcomes in the treatment of people with neuroblastoma, especially among patients who do not respond well to the present therapy combinations.

We cannot ignore autophagy in the era of precision medicine that will soon be here. Undoubtedly, this biological activity is a component of the molecular landscape that we are currently striving to identify in each patient in order to develop a more successful treatment. Of However, since autophagy is a very dynamic process, it may be important to monitor it from the time of the initial diagnosis until the end of the treatment.

We might be able to determine if autophagy is a troublesome route that needs to be managed in order to ensure a patient's recovery in this way. Furthermore, considering that this biological process is inextricably linked to the condition of cell energy supply, cell metabolism would also be an important activity to be researched in conjunction with autophagy in neuroblastoma.

Treatment of severe neuroblastoma phenotypes may benefit from the development of novel techniques and investment in ground-breaking medicines that precisely target compromised regulatory mechanisms and cellular processes, such as autophagy. Additionally, as a component of precision medicine, this line of thinking could guarantee that we have taken into account all viable therapy alternatives and used every practical tool to increase the recovery rate of paediatric neuroblastoma patients. It is unknown if some of the planned clinical trials suggested for HR patients with neuroblastoma will include pharmacological intermediates that affect autophagy.