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Advances in the Study of the Dual Role of Cellular Autophagy in Tumours and Its Targeted Therapeutic Strategies

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Abstract. Cell autophagy shows a two-way effect in the pathogenesis and development of cancer. In the early stage of cancer, autophagy (the process of cell degradation of cell components) has the function of tumor suppression by degrading the wrongly coded cell components. In the later stage of cancer, autophagy has a protective effect on tumor cells, allowing cells to survive, invade and be resistant to treatment after exposure to initial drug treatment. The signaling pathway that regulates autophagy is strictly regulated. Inhibition measures include Akt (protein kinase B) and mTOR (mammalian target of rapamycin) pathways involving inhibiting autophagy, and the AMPK/mTOR pathway makes autophagy possible. The aberration of Beclin1 (loss of expression or mutation) is an important regulator of autophagy, which is associated with malignant tumors, including breast cancer and ovarian cancer. Autophagy not only regulates cell degradation but also regulates the progression of tumors through the synergy of immune regulation, cell metabolism, tumor microenvironment remodeling or lysosomal activity. The priority or targeting of regulatory networks involving autophagy - especially the AMPK/mTOR pathway or Beclin1 signal is an emerging strategy for low-toxic biomarker therapy. However, they are worth considering whether the context comes from the tumor stage and/or histology. In summary, this paper evaluates the two-way effect of autophagy on tumor progression and treatment, including its role in the tumor microenvironment, and provides new insights for targeted therapy and biomarker treatment regulation.

Keywords:The Tumor microenvironment, bidirectional regulation of autophagy, targeted therapies

1. Introduction

Autophagy is an important metabolic reaction of cells, which plays an important role in many biological processes. It helps the degradation of cell components and uses the degradation products in different cell processes.

There are many types of autophagy: micro autophagy, macro autophagy, companion-mediated autophagy and selective autophagy [1].

Cancer is one of the most serious problems today, which has a significant impact on the lives of patients. Autophagy is an important part of cancer development, which plays an important role in the maintenance of adult stem cells and may contribute to the formation of cancer stem cells. Autophagy

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also helps cancer cells to be resistant to chemotherapy. It is reported that autophagy inhibition increases immunotherapy, which is a gap in the study of anti-cancer immune response. It is necessary to study autophagy in depth and develop cancer targeted therapy by properly regulating autophagy-related pathways. Comparative research is also needed to understand the effects of autophagy inhibition and autophagy promotion on cancer in different tumor stages.

Despite the growing interest in studying autophagy, the implementation of autophagy regulation in cancer treatment remains to be seen. The role of autophagy in the tumor microenvironment increases the complexity of its use. Autophagy not only affects tumor cells, but also indirectly affects the progression of tumors by regulating the functions of tumor-related immune cells, angiogenesis and stromal cells [2]. The clinical application of autophagy in cancer is further limited by technical and methodological restrictions. For example, in combination therapy, the synergistic mechanism between autophagy-regulating drugs and other therapies is unknown, which may lead to unpredictable treatment results [3].

2. Autophagy-related signaling pathways and regulatory mechanisms

2.1. Core signaling pathways: inhibition of the PI3K/Akt/mTOR pathway and activation of the AMPK/ mTOR pathway

Mutations that lead to the activation of serine/threonine kinase Akt are common in cancer. Akt has several downstream targets involving tumor occurrence, including mTOR (mammalian targets for rapamycin) and Ark. Akt also inhibits autophagy, which may be used as a tumor inhibitor in some cases. Akt inhibits autophagy by activating mTOR, which in turn inhibits the ULK1 kinase complex that drives autophagy. Research shows that active Akt can inhibit autophagy through a mechanism unrelated to mTOR.

The AMPK pathway is also associated with treatment; as an autophagy regulator, AMPK activates the increase in stimulating autophagy, which is supported by clinical research. In addition, the "toxic autophagy" mediated by AMPK/mTOR decomposes important cell components, thus providing therapeutic benefits against cancer. Recently, studies have shown that ginseng glycoside Rk1 can significantly alleviate liver damage, fibrosis and cirrhosis during the progression of liver cancer (HCC), as depicted in Figure 1. Transcriptome studies on mouse liver tissue showed that ginseng glycoside Rk1 significantly regulates the AMPK/mTOR signaling pathway, as well as autophagy and apoptosis-related pathways. Supportive studies show that ginseng glycoside Rk1 activates AMPK and increases the autophagy marker LC3-II, thus promoting autophagy. In addition, ginseng glycoside Rk1 down-regulation of Bcl-2 leads to caseinase cascading reaction, leading to AMPK/mTOR-mediated "toxic autophagy" to stimulate autophagy-dependent cell death. In addition, if ginseng glycoside Rk1 is combined with autophagy inhibitors, the apoptosis of HCC cells is not developed, indicating further evidence of autophagy-dependent cell death induced by Rk1. In short, these findings show that ginseng glycoside Rk1 stimulates toxic autophagy by the AMPK/mTOR pathway to drive apoptosis, thus reducing the progression of liver cancer [4].

2.2. Key regulatory factors: autophagy proteins (Beclin1) and their loss or mutation in tumors

Beclin1 is an important autophagy protein with anti-tumor activity and direct interaction with Akt. The expression of the autophagy defect mutant Beclin1 phosphorylated by Akt induces autophagy, reducing growth unrelated to anchoring, and limiting Akt-mediated tumor progression [5].

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Therefore, we can think that Beclin1 is a key regulatory switch that induces malignant cell autophagy modified by Akt-induced phosphorylation.

The Beclin1 gene required for autophagy is often lost in human breast cancer, ovarian cancer and prostate cancer. Studies show that the loss of Beclin1 increases the susceptibility to breast cancer, for example, in animal models in which the absence of Beclin1 (Beclin1+/-) hybrid leads to more tumors [6], which is illustrated in Figure 2. These studies show that autophagy-related regulatory proteins such as Beclin1 are important in carcinogenesis. Although much is known about the potential differentiation of autophagy in malignant tumors, the duality of autophagy in cancer has not been proposed.

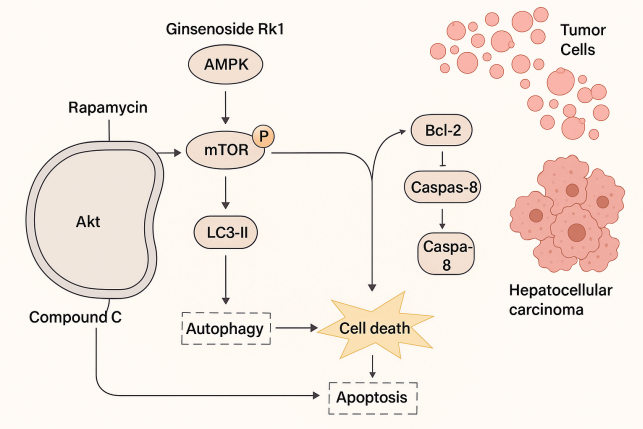

Figure 1. AMPK/mTOR pathway

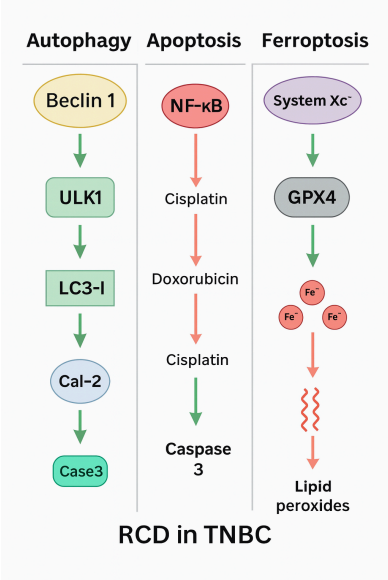

Figure 2. Beclin1 as a key regulatory switch that induces malignant cell autophagy

3. The dual role of autophagy in tumorigenesis and progression

As shown in Figure 3, autophagy plays a complex, context-dependent role in cancer initiation, progression, and treatment. Depending on the tumor type, stage, and microenvironmental conditions, autophagy can either inhibit or promote tumor development [7].

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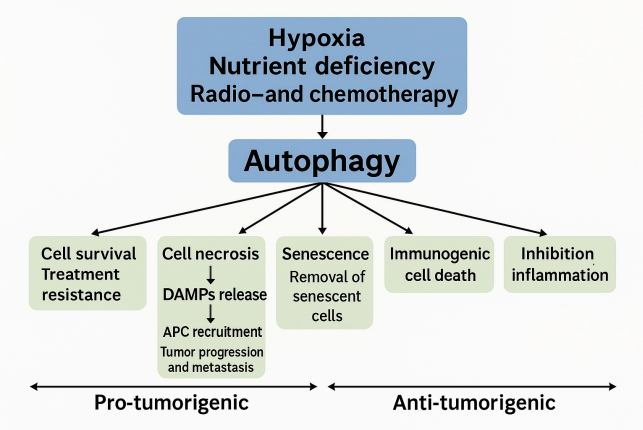

Figure 3. Modulation of inflammation by autophagy

3.1. Tumor suppression phase: protective mechanisms of autophagy

In tumor development, early autophagy helps ensure genome stability by removing aggregates of damaged mitochondria and damaged proteins. Autophagy can ensure genomic stability by reducing the accumulation of reactive oxygen (ROS) after removing damaged mitochondria and protein aggregates, thus reducing malignant transformation [8]. Mitochondrial autophagy or mitochondrial phagocysis is a specific type of autophagy, which targets the damage of mitochondria to remove damaged organelles to maintain cell homeosis.

3.2. Tumor promotion phase: autophagy as a survival support mechanism

3.2.1. Autophagy-mediated therapeutic resistance in tumor cells (KRAS G12D mutant pancreatic cancer as an example)

The KRAS G12D mutation is of significant clinical relevance in pancreatic ductal adenocarcinoma (PDAC), providing potential targets for precision therapy. In KRAS G12D-mutant PDAC cells, autophagy activation reduces chemotherapy-induced apoptosis (for example, in response to gemcitabine), thereby promoting tumor cell survival and proliferation. Interactions between tumor cells regulating autophagy are shown in Figure 4.

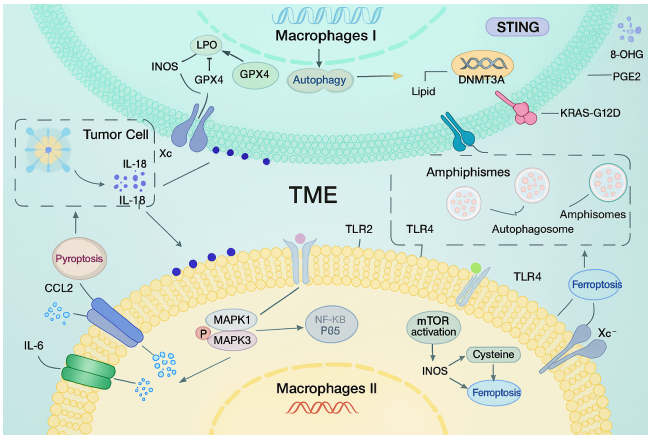

Figure 4. Autophagy and autophagy-related pathways in cancer

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KRAS G12D-mutant pancreatic cancer cells also evade immune surveillance through autophagy-mediated mechanisms. Autophagy activation reduces the expression of MHC class I molecules on tumor cell surfaces, diminishing recognition and killing by CD8+ T cells. Furthermore, autophagy weakens anti- tumor immune responses by inhibiting the release of immunostimulatory cytokines (such as CXCL10 and CXCL11) from tumor cells.

3.2.2. Role of autophagy in tumor metabolic reprogramming

The KRAS G12D mutation is of significant clinical relevance in pancreatic ductal adenocarcinoma (PDAC), providing potential targets for precision therapy. In KRAS G12D-mutant PDAC cells, autophagy activation reduces chemotherapy-induced apoptosis (for example, in response to gemcitabine), thereby supporting tumor cell survival and proliferation.

For instance, by degrading proteins and organelles, autophagy can supply amino acids for new protein synthesis and energy metabolism [9]. Similarly, autophagy breaks down lipid droplets to release fatty acids [10], which can be used for β-oxidation to produce ATP or for the synthesis of new membrane lipids [11].

The provision of these metabolic substrates supports tumor growth and also promotes immune evasion by modulating immune cell function in the tumor microenvironment [12].

The dual function of autophagy in the metabolic adaptation of cancer cells gives it great therapeutic potential. Autophagy attenuation specifies one aspect of inhibition, that is, the consumption of metabolic substrates will limit the growth and/or survival of tumors. On the contrary, autophagy activation can enhance the anti-tumor effect by inducing ionization in cancer cells.

Most specifically, there are several autophagy inhibitors, including chloroquine (CQ) and hydroxychloroquine (HCQ), which can block the formation of autophagias and the degradation of lysosomes, and cut off the nutrient supply of tumor cells, thus reducing growth. In contrast, autophagy activators, such as rapamycin (RAPA), can induce ionization in tumor cells and further enhance the anti-tumor effect.

In addition to regulating cell metabolism, thus affecting the progression of tumors, autophagy can also regulate the immune microenvironment of tumors.

4. Autophagy and the tumor microenvironment and immunity

4.1. Regulation of immune cell function in the tumor microenvironment by autophagy

Autophagy may also regulate the immune system through immune cell function and anti-tumor immunity in the tumor microenvironment, thus helping to regulate tumor growth. A preliminary understanding of the effects of autophagy on antigen presentation, cytokine secretion and immune cell metabolism has been established, but a clear understanding of the mechanism and potential of clinical care continues.

4.2. Autophagy's impact on tumor immune evasion and therapeutic response

Autophagy is not strictly protective. In some cases, it may also support the adaptive metabolism and survival of tumor cells as a supporting mechanism for tumor formation. Data shows that the lack of autophagy can lead to tumor-related macrophages (TAMs) dysfunction, promoting tumor immune escape and promoting progress. For example, excessive ROS species caused by autophagy deficiency 36, the loss of Tim-4+ TAMs in the somatic cell microenvironment of ovarian cancer 36,

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and the lack of FIP200 of P-53 leads to the loss of Tim-4+ TAMs, which induces a downward regulation of the immune response [13]. In KRAS G12D mutant PDAC, autophagy enhances the production of glutamic acid, cysteine and glycine to maintain the production of glutathione to further reduce ROS and induce the loss of apoptosis.

5. Therapeutic strategies targeting autophagy in tumors

5.1. Pharmacological approaches targeting AMPK/mTOR pathways to modulate autophagy

Autophagy participates in the initiation and progression of cancer by using several different pathways, and has conducted extensive research on important signaling pathways. AMPK and mTOR pathways are important components of autophagy signals.

For example, ginseng glycoside Rk1 has been identified as a new activator and autophagy stimulant for AMPK signals, but Rk1 also activates AMPK/mTOR-mediated toxic autophagy to detoxify cells by degrading the substrates required by HCC cells, thus producing therapeutic effects.

The signal network that regulates autophagy also regulates the activation of lysosomes, and lysosomal activity can in turn regulate the growth and progression of tumors. The relationship between lysosome function and autophagy is important, but it is also complicated, because inhibiting mTOR signals, especially mTOR complex 1 (mTORC1), will lead to an increase in lysosome function. After inhibiting mTORC1, TFEB activation is necessary for the production of new lysosomes and activated lysosomes, because the process is complex and strictly regulated. In addition, the activation of lysosomes and the degradation of autophages required for subsequent autophages must be integrated with lysosomes. Lysosomes are not only compartments responsible for degrading autophagy goods, but they also play an indispensable role in regulating autophagy and cell homeosidism signaling pathways. In the group of patients with diseases caused by lysosomal dysfunction, the next direction of therapy that causes damage to lysosomal function is to further study the way to specifically regulate lysosomal function during autophagy.

Autophagy can enhance the invasion and migration potential of tumor cells by regulating the signaling pathway responsible for the transition from epithelium to mesenchymal (EMT). Research shows the relationship between M2 polarization of tumor-asorenated macrophages (TAMs) and the progression of EMT and the rapid changes in tumor cell migration and invasion in the later stage of the disease [14].

5.2. Autophagy intervention approaches targeting Beclin1 complex function

In practice, we have observed the efficiency of regulating stimulation and inhibiting autophagy through targeted pathways, and broad pathway regulation is a hybrid strategy. The Beclin1 complex provides an important regulation node in our targeted pathway regulation strategy. Beclin1 is considered to act as an intermediate for regulating the signal of growth factor receptor by regulating the maturity time of the early endoss. In the absence of Beclin1, the growth factor receptor takes longer to activate the early endossor, activates the Akt pathway and the center of the ERK signaling pathway [15], and promotes greater invasiveness to breast cancer cells. This study shows an example of Beclin1's ability to inhibit cancer progression by regulating membrane transport and receptor signals. In addition to or in parallel with these findings, there are many new studies showing the lack of directional regulation control of mTOR for autophagy: this occurs through the phosphorylation of Beclin1 in this site by Akt dependence, and can transform Beclin1 from promoting autophagy to apoptosis-inducing function [16].

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6. Recent advances and frontier directions in autophagy research

6.1. Application of CRISPR gene editing technology in autophagy research

Through its specificity and effectiveness, CRISPR/Cas9 has become a powerful new tool in the field of autophagy research. Autophagy is a cell degradation process, which is one of the most conservative cell degradation processes in evolution, in which cells degrade and remove damaged organelles and misfolded proteins, and promote homeostasis. The molecular mechanism behind the external factors that regulate autophagy is very complex, because considerable effort is required to dissise the many genes and signals that occur in the autophagy pathway. Traditional genetic function research usually takes a lot of time and energy to discover the influence/degree/function of specific genes in autophagy. In contrast, CRISPR/Cas9 technology provides a simple and effective method to screen the functional autophagy genes of the entire genome through one reading (i.e. whole genome screening). Therefore, this utility provides a new method for examining molecular mechanisms in the context of autophagy signals.

One of the main applications of CRISPR/Cas9 technology is to generate a whole genome knockout library, which can be used for high-throughphout screening of autophagic genes. Researchers can generate a library containing tens of thousands of single-guided RNAs (sgRNAs), each of which aims to knock out specific genes/regions of the human genome. When the library is established, it is transferred to the cell line, and then the cells can induce autophagy or inhibit autophagy (through next-generation sequencing), and high-through volume screening can be used to identify the genes that guide the autophagy process very quickly [17].

6.2. Novel techniques for tracking autophagic flux (real-time reporting systems and imaging methods)

Autophagy fluce includes the whole autophagy process, from its start to its terminal degradation process, including the formation of autophages, fusion with lysosomes and the degradation of the final goods. Traditional methods for detecting autophagy (such as transmission electron microscopy, TEM or LC3 Western imprint) can be used to quantify the morphology of autophagy or the overall protein level, but cannot track the dynamics of dynamic biology in real time.

A new autophagy fluetic flue tracking technology has been developed using fluorescence reporters and in vivo imaging methods to evaluate the autophagy treatment of complete organisms with high space-time resolution in real time, and can also be evaluated at the single-cell level of biological form.

Fluorescent protein-based reporters are vital tools for tracking autophagic flux. The mRFP-GFP-LC3 dual- fluorescent labelling system, for instance, distinguishes autophagosomes from autolysosomes by exploiting the pH-dependent fluorescence of GFP and mRFP. GFP fluorescence is quenched in acidic environments (like the inside of lysosomes), whereas mRFP remains stable. Consequently, autophagosomes appear as yellow puncta (showing both GFP and mRFP signals), while autolysosomes appear red (mRFP signal only). This dual-marker system has been extensively applied in cell culture and animal models, providing a reliable approach for quantitative analysis of autophagic flux [18].

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6.3. Interplay between autophagy and immune checkpoints (autophagy's influence on PD-1/PD-L1 and other checkpoint pathways)

There are complex interactions between autophagy and immune checkpoint pathways such as PD-1/PD- L1. Autophagy can affect the immune evasion capacity of tumor cells by regulating PD-L1 expression. Studies show that autophagy may promote the degradation of PD-L1 protein, thus reducing PD-L1 on the surface of tumor cells and enhancing the anti-tumor properties mediated by T cells. On the contrary, autophagy can also stabilize PD-L1 under certain conditions and enhance the immune escape of tumor cells. For example, autophagy supports the stability of PD-L1 under the condition of nutritional depletion in the tumor microenvironment, enabling tumor cells to escape immune monitoring [19].

Autophagy interacts with PD-1/PD-L1 through a variety of signaling pathways, including the mTOR signal, which is the main regulator of autophagy, and it also affects the expression/function of PD-1/PD-L1. In addition, autophagy can indirectly change the immunosuppressive effect of PD-1/PD-L1 by changing the level of metabolites in the tumor microenvironment (i.e. lactic acid and adenosine), as depicted in Figure 5. Overall, this complex regulatory network further shows that the autophagy-PD-1/PD-L1 interaction is layered and multidimensional [20].

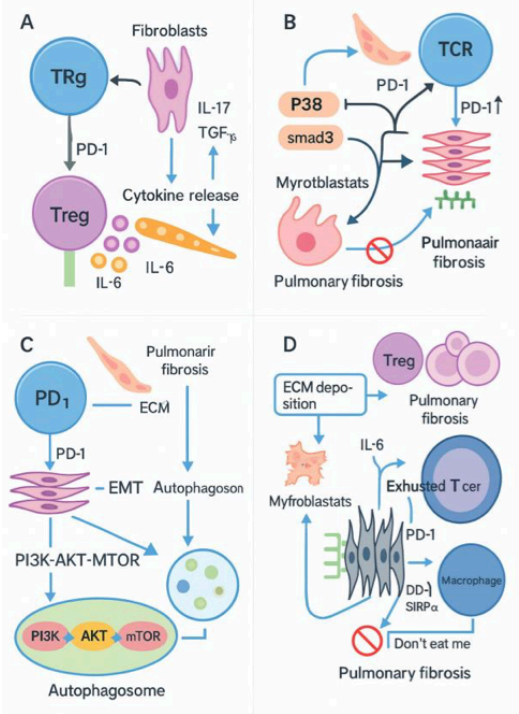

Figure 5. The role of PD-1/PD-L1 axis in idiopathic pulmonary fibrosis

7. Conclusion

This article reviews the potential mechanism of tumor autophagy, current problems and the potential future direction of autophagy for cancer treatment.

According to our current understanding, autophagy has become an important topic in cancer research, because it plays a crucial role in the pathogenesis and clinical management of destructive diseases, especially malignant tumors. Through the regulation of multipath mechanisms, autophagy has a considerable impact on tumor growth and metastasis and the biology of cancer. In recent years,

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people have been increasingly interested in autophagy because of its slowing and inhibiting effect on cancer biology: autophagy usually inhibits the initiation of tumors in the early stages and promotes progression and treatment resistance, especially in the late and late stages of cancer.

This variability increases the complexity of autophagy and means that controlling it may be a potential new cancer treatment strategy. Future research should explore the molecular autophagy mechanisms that occur in different cancer subtypes and environmental contexts, and explore whether there is a synergistic relationship with other programmed cell death pathways, such as apoptosis and iron apoptosis. Future research using high-throughput methods will help describe other potentially important autophagy-related genes (such as MAPK8, ATG10, BCL2), and help verify prognostic research related to autophagy with patient samples. It is recommended to add autophagy targeting methods in cancer.

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