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Opinion Article

Nanotherapeutics for Inducing Cell Death and Immunotherapy Cancer

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DESCRIPTION

Immunotherapy appears as a promising treatment for cancer. Despite rapid advances in cancer immunotherapy, the therapeutic efficacy and clinical implementation of immunotherapy have not been as satisfactory as expected, especially for immune cold tumor patients. Immunogenic Cell Death (ICD) is a specific form of tumor cell death that involves the production of tumor-specific antigens that promote the infiltration of effector T cells and enhance the immune response of solid tumors. Therefore, ICD can help stimulate immune cold tumors to become immune hot. Increasing evidence indicates that Photodynamic Therapy (PDT) can effectively induce his ICD. Recently, various photodynamic nanotherapeutics been developed to induce ICD and enhance cancer immunotherapy. Here, in this evaluation, we evaluation recent advances in the field at the interface between PDT, nanotechnology, and ICD, including PDT-induced ICD, synergistic induction of PDT-based ICD, and multimodal immunotherapy based on PDT-induced ICD. Finally, we discuss the prospects and challenges of photodynamic nanotherapeutics in ICD-guided-based cancer immunotherapy.

Malignant tumors remain a serious threat to human health. Immunotherapy displays promising application prospects in clinical practice in recent years. Compared to traditional therapeutic strategies, new immunotherapies not only suppress the growth of primary tumors, but also effectively prevent metastasis and tumor recurrence by harnessing the patient's innate immune system. Typical immunotherapeutic strategies mainly include non-specific immune stimulation, Immune Checkpoint Blockade (ICB) therapy, tumor vaccines, and Adoptive Cellular Immunotherapy (ACI). Among them, programmed death 1 (PD-1)/programmed death ligand 1 (PD-L1) was discovered as an immunotherapy modality capable of blocking immune checkpoints. In particular, PD-1/PD-L1 blockade immunotherapy relies heavily on complete infiltration of activated T lymphocytes into tumor tissue. Nevertheless, a significant proportion of clinical patients with immune cold tumors exhibit low immune responses, leading to poor immunotherapeutic efficacy. Therefore, methods to promote the transformation of immune-cold to immune-hot tumors may be important for efficient immunotherapy.

In recent decades, the immune response has always been thought to be triggered only by substances foreign to the body. However, research in the late 1990s showed that endogenous substances can also induce immune responses under certain conditions. We first showed that tumor cells exposed to Doxorubicin (Dox) can act as a vaccine and activate host immunity during apoptosis, calling this form of cell death Immunogenic Cell Death (ICD). Since the ICD concept was first proposed in 2005, host immunity in cancer therapy has received considerable attention. The advent of ICDs and ICD-based synergistic therapies has closed the gap between conventional and new therapies. In particular, conventional therapy-induced ICD turned out to be a promising strategy for transforming immune-cold tumors into immune-hot tumors in tumor immunotherapy. After ICD induction, dying tumor cells were isolated from Tumor-Associated Antigens (TAAs), proinflammatory cytokines, and risk-related molecules such as Heat Shock Proteins (HSPs), Calreticulin (CRT), and high mobility group and Adenosine Triphosphate (ATP). Released DAMPs stimulate Dendritic Cell (DC) maturation, and mature DC carrying cancer-specific antigens migrate to lymph nodes and activate effector T cells. Ultimately, activated effector T cells migrate to infiltrate tumor tissue and eliminate tumor cells.

ICD is most often induced through apoptosis and is therefore commonly referred to as immunogenic apoptosis. Recently, several non-apoptotic cell death modalities have also been found to be associated with anti-tumor immunity. For example, necroptosis is associated with the release of intracellular contents under DAMPs that induce anti-tumor immunity through activation of Nuclear Factor- κB (NF-κB) or priming and activation of effector T cells, leading to programmed cell death. It's a modality. In addition, ferroptosis is also emerging as a new modality of cell death triggered by iron ion-mediated accumulation of lipid peroxides. We found that tumor cells undergoing ferroptosis can release HMGB1 through autophagy.

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Second, infiltration of CD8+ T cells can promote and increase the development of tumor ferroptosis. Indeed, immunogenic cell death refers to the umbrella term, but several other cell death modalities such as necroptosis and ferroptosis have also been found with immunogenic signatures. However, the exact mechanisms of anti-tumor immunity induced by these non-apoptotic cell death modalities are still not fully understood.

Several conventional treatments such as chemotherapy, radiotherapy and Photodynamic Therapy (PDT) have been found to induce tumor cell death in an immunogenic manner. Among them, PDT has been extensively studied as a non-invasive therapy for local cancer treatment. Under local irradiation with tumor focused Near-Infrared (NIR) light, Photosensitizers (PS) rapidly generate cytotoxic Reactive Oxygen Species (ROS) and kill tumor cells. In particular, ROS generated from PS not only promote tumor cell death, but also induce Endoplasmic Reticulum (ER) pressure, trigger CRT exposure, thereby inducing ICD of tumor cells, and promote proinflammatory cytokines and DAMPs. However, the short half-life of most PSs, the hypoxic tumor microenvironment, and the insufficient accumulation of ROS in the ER complicate the induction of ICD. Moreover, there are still some PDT-specific limitations that are insufficient to elicit a strong immune response. Moreover, PDT-based ICD-based unimodal cancer immunotherapy is inadequate to achieve satisfactory therapeutic outcomes. Therefore, there is a need to improve the *in vivo* delivery efficiency of PS and develop combined therapeutic strategies for efficient PDT-induced immunotherapy.