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Genomic Immunometabolism and DNA Repair: Drivers of Cancer in Cellular Oncology

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Description

Genomic instability and DNA repair mechanisms are two interconnected concepts that play crucial roles in maintaining the integrity of an organism's DNA and preventing the development of various diseases, including cancer. Genomic instability refers to the tendency of an organism's DNA to undergo various changes, such as mutations, deletions, insertions, and rearrangements. This instability can result from both endogenous (internal) and exogenous (external) factors. Genomic instability can have severe consequences, as it can lead to diseases like cancer and genetic disorders. Understanding these metabolic changes is crucial for optimizing immunotherapies. Cancer cells can exploit their altered metabolism to evade immune detection and destruction. They may produce metabolic by-products that suppress immune responses, or they can create an immunosuppressive TME that inhibits the function of immune cells. Targeting the interactions between cancer cells and the various recruited healthy cells is another avenue for cancer therapy. Researchers are investigating ways to reprogram tumor-associated immune cells to have anti-tumor effects and to inhibit the activity of cancer-associated fibroblasts.

Understanding cancer immunometabolism has led to the development of novel cancer immunotherapies. For example, immune checkpoint inhibitors (e.g., PD-1 and CTLA-4 inhibitors) aim to restore the anti-tumor immune response by blocking inhibitory signals. Additionally, metabolic modulators are being investigated as potential adjuvants to enhance the effectiveness of immunotherapy. Immune cells and cancer cells may compete for essential nutrients within the TME, such as glucose and amino acids. Strategies to selectively target cancer cell metabolism while sparing immune cell metabolism are of interest in developing new therapies. Metabolic markers in tumors and immune cells can serve as biomarkers to predict treatment responses and patient outcomes. Measuring these markers can help tailor treatment strategies for individual patients. Cancer immunometabolism is a complex and rapidly evolving field that examines the metabolic interplay between cancer cells and immune cells within the tumor microenvironment. Understanding these interactions is critical for developing innovative cancer therapies that harness the power of the immune system to target and eliminate cancer cells. Immunotherapy has emerged as a promising approach to overcome immune evasion in cancer. Checkpoint inhibitors, Chimeric Antigen Receptor (CAR) T-cell therapy, and other immunotherapies aim to enhance the immune system's ability to recognize and attack cancer cells. Combining immunotherapy with other treatments like targeted therapy or chemotherapy is an active area of research to improve cancer treatment outcomes.

Cancer cells often undergo significant alterations in their energy metabolism to meet the demands of rapid growth and proliferation. The most well-known metabolic shift in cancer cells is the Warburg effect, where they preferentially use glycolysis for energy production even in the presence of oxygen. This metabolic shift not only provides energy but also generates intermediates needed for the synthesis of macromolecules required for cell division. This altered metabolism can make cancer cells more resistant to certain treatments, as they can adapt to low-oxygen environments within tumors. Targeting cancer cell metabolism is an active area of research. Potential strategies include inhibiting glycolysis or other metabolic pathways specific to cancer cells, as well as exploiting these metabolic differences for diagnostic purposes (e.g., using PET scans that detect increased glucose uptake). The immune system has mechanisms to identify and eliminate abnormal cells, including cancer cells. However, cancer cells can develop strategies to evade immune detection and destruction. This evasion can involve various mechanisms, such as downregulating surface antigens, activating immune checkpoint pathways (e.g., PD-1/PD-L1), and creating an immunosuppressive tumor microenvironment. cancer cells can recruit various healthy cells to support tumor growth and progression. For example, tumor-associated fibroblasts can create a supportive extracellular matrix, while immune cells like macrophages can have both pro-tumor (M2-like) and anti-tumor (M1-like) functions depending on their polarization [1-5].

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Conflict of Interest

The Author declares there is no conflict of interest associated with this manuscript.

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