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Unlocking tumor immunogenicity: A new era in cancer immunology.

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Introduction

Cancer remains one of the most challenging adversaries in modern medicine. Traditional therapeutic approaches such as chemotherapy and radiation have made significant strides, yet they often fail to address the dynamic and evasive nature of tumors. In recent years, the advent of immunotherapy has transformed the oncology landscape, shifting focus toward a fundamental concept: tumor immunogenicity—the capacity of tumor cells to elicit an immune response. Understanding and enhancing tumor immunogenicity has ushered in a new era in cancer immunology, enabling more precise, potent, and durable treatment responses [1, 2].

Tumor immunogenicity refers to a tumor's ability to be recognized and attacked by the host immune system. Highly immunogenic tumors express neoantigens—mutated proteins resulting from genetic aberrations—that are perceived as foreign by immune cells, particularly cytotoxic T lymphocytes (CTLs). However, many cancers are immunologically "cold," lacking recognizable antigens or failing to present them due to mechanisms like MHC class I downregulation or a suppressive tumor microenvironment [3, 4].

Tumors with high mutational burdens (e.g. melanoma, NSCLC) tend to generate more neoantigens. Efficient expression and processing of antigens via MHC molecules are vital for CTL recognition. Dendritic cells and natural killer cells play key roles in initial immune activation. Type I and II interferons enhance antigen presentation and T cell recruitment. Stromal cells, cytokines, and immune checkpoints (e.g. PD-L1) can suppress immune responses and diminish immunogenicity. The concept of converting poorly immunogenic tumors into responsive "hot" tumors has spurred numerous therapeutic strategies: Antibodies targeting PD-1/PD-L1 **and** CTLA-4 pathways unleash suppressed T cells, dramatically improving responses in some tumors. However, efficacy often correlates with preexisting tumor immunogenicity [5, 6].

Vaccines designed to prime the immune system against tumor-specific neoantigens are gaining traction. Personalized mRNA vaccines (like those explored in melanoma) have shown promise in early-phase trials. Genetically engineered viruses selectively infect tumor cells, causing immunogenic cell death and releasing tumor antigens that re-educate the immune system. In addition to direct cytotoxicity, these modalities can induce immunogenic cell death and enhance antigen release. TCR-engineered T cells and CAR T-cell therapy offer tailored immune responses but require tumors to express compatible target antigens [7, 8].

Tumors often adapt, downregulating antigens or upregulating immunosuppressive signals. Enhancing immunogenicity risks triggering immune responses against healthy tissue. Predicting response requires reliable, reproducible biomarkers. Genetic and epigenetic variability within tumors complicates immunogenicity profiling. Agents like histone deacetylase inhibitors may enhance antigen expression. Gut flora can impact systemic immunity and immunotherapy outcomes. Machine learning is helping predict neoantigen quality and therapy response [9, 10].

Conclusion

Unlocking tumor immunogenicity has revolutionized cancer treatment, illuminating paths for more personalized and durable interventions.

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As research deepens, manipulating the immunogenic landscape of tumors may become as standard as chemotherapy once was—ushering in an age where the immune system stands as the most potent weapon against cancer.

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