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Regulation of Immune Checkpoint Inhibitors by Post-translational Modifications

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Introduction

Immune Checkpoint Inhibitors have become a cornerstone in modern oncology, providing a novel approach to cancer therapy by unleashing the immune system's ability to target and destroy tumor cells. By blocking inhibitory signals that dampen immune responses, ICIs such as those targeting cytotoxic T-lymphocyte-associated protein 4 (CTLA-4) and programmed Death-1 (PD-1)/programmed Death-ligand 1 can enhance T-cell activity against cancers. Despite their success, the efficacy and safety of ICIs are influenced by complex regulatory mechanisms, including post-translational modifications (PTMs). PTMs, which include phosphorylation, ubiquitination, glycosylation, and acetylation, can profoundly affect the function, stability, and localization of immune checkpoint proteins. This review explores the role of PTMs in regulating ICIs, providing insights into potential therapeutic strategies to enhance their effectiveness and overcome resistance [1].

Description

Post-Translational Modifications (PTMs) are essential in regulating the function and efficacy of immune checkpoint inhibitors (ICIs). These modifications include phosphorylation, ubiquitination, glycosylation, and acetylation, each playing distinct roles in modulating immune checkpoint proteins [2]. Phosphorylation, the addition of phosphate groups to specific amino acids, can alter the conformation and activity of proteins like PD-1, influencing its ability to inhibit T-cell activation and thus modulating immune responses. Ubiquitination, which involves attaching ubiquitin molecules to lysine residues, typically tags proteins for degradation by the proteasome [3].

This process regulates the levels of immune checkpoint proteins on the cell surface, affecting the availability and duration of their inhibitory signals. Glycosylation, the addition of carbohydrate moieties, affects the stability and cell surface localization of proteins such as PD-L1, thereby impacting its interaction with PD-1 and the immune evasion of tumour cells [4]. Acetylation, which adds acetyl groups to lysine residues, can modify protein interactions and stability, influencing the function and surface expression of proteins like CTLA-4. These PTMs collectively ensure the dynamic regulation of immune checkpoints, affecting their therapeutic efficacy and the immune system's ability to combat cancer. Understanding and manipulating these modifications offer potential strategies to enhance ICI therapy and overcome resistance in cancer treatment [5,6].

Conclusion

Post-translational Modifications are crucial regulators of immune checkpoint inhibitors, influencing their function, stability, and localization. By modulating these modifications, it is possible to enhance the therapeutic efficacy of ICIs and overcome resistance in cancer treatment. Further research into the specific PTMs affecting ICIs and the development of strategies to manipulate these modifications holds promise for improving outcomes in cancer immunotherapy. Understanding the intricate regulation of immune checkpoint proteins by PTMs not only provides deeper insights into their biology but also opens new avenues for innovative cancer treatment strategies.

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Conflict of Interest

None.

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