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Nanoparticles and immune modulation: A new frontier.

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Introduction

Nanotechnology has redefined the possibilities within biomedical science, introducing novel tools for targeted drug delivery, diagnostics, and vaccine development. Among its most transformative applications lies immune modulation, where nanoparticles (NPs) are engineered to interact directly with immune cells and molecular pathways, either amplifying or suppressing immune responses. As chronic diseases, cancers, and autoimmunity challenge conventional therapeutics, nanoparticle-based immunomodulation emerges as a promising frontier.

Their size, surface charge, shape, and functional groups can be precisely tuned to influence immune cell uptake, trafficking, and activation (Zhang et al., 2018). For instance, PEGylation reduces immune recognition, whereas cationic surfaces enhance dendritic cell activation. Nanoparticles can interact with pattern recognition receptors (PRRs), such as Toll-like receptors (TLRs), NOD-like receptors (NLRs), and scavenger receptors. NPs delivering TLR ligands like CpG or MPLA improve dendritic cell maturation and cytokine release, essential for vaccine adjuvanticity [3, 4].

NPs can steer macrophages toward pro-inflammatory (M1) or anti-inflammatory (M2) phenotypes based on their surface chemistry and payload. Certain NPs activate the NLRP3 inflammasome, leading to IL-1β secretion and heightened inflammation—a dual-edged sword in immunotherapy. These interactions lay the foundation for tailoring immune responses in infection, cancer, and chronic inflammatory disease. One of the most visible successes of nanoparticle-driven immunomodulation is in vaccinology, particularly with COVID-19 mRNA vaccines encapsulated in lipid nanoparticles [5, 6].

NPs protect labile antigens and ensure sustained release and targeted delivery to antigen-presenting cells. NPs themselves may act as adjuvants, enhancing immunogenicity. Polymeric NPs can deliver vaccines intranasally, inducing mucosal IgA responses often lacking in systemic immunization. This approach is now being expanded to other viral and parasitic infections, cancer vaccines, and autoimmune interventions. NPs loaded with anti-PD-1 or anti-CTLA-4 antibodies can enhance tumor penetration and minimize systemic toxicity [7, 8].

NP-based vaccines targeting TAAs elicit robust $CD8^+$ T-cell responses. NPs can deliver siRNA, cytokines, or small molecules to reprogram the tumor microenvironment (TME) and reverse immune. The versatility and tunability of NPs make them ideal vehicles in precision immuno-oncology. Autoimmunity involves dysregulated immune responses against self-antigens. NPs can be designed to restore tolerance or attenuate inflammation: Tolerogenic NPs presenting autoantigens (e.g., insulin peptides in T1D) promote Treg differentiation and dampen autoreactive T cells [9, 10].

Conclusion

NPs delivering IL-10, dexamethasone, or siRNA targeting TNF-α reduce inflammation in diseases like rheumatoid arthritis and IBD. Coating NPs with membrane proteins (e.g., leukocyte membranes) can evade immune detection and deliver payloads precisely to inflamed tissues. NP accumulation in liver or spleen, and off-target effects must be mitigated. Reproducibility in NP synthesis and stability remains a hurdle for regulatory approval.

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