

Tumor immune microenvironment

The [tumor microenvironment](#) (TME) is the cellular [environment](#) in which the tumor exists, including surrounding [blood vessels](#), [immune cells](#), [fibroblasts](#), bone marrow-derived inflammatory cells, [lymphocytes](#), [signaling molecules](#) and the [extracellular matrix](#) (ECM).

The [tumor microenvironment](#) (TME) could be simply characterized into cold (non T cell inflamed) or hot ([T cell-inflamed tumor microenvironment](#)), which is largely attributed to the levels of proinflammatory cytokine production and T cell infiltration

With the advent of [cancer immunotherapy](#), there has been a major improvement in patients' [quality of life](#) and [survival](#). The growth of cancer [immunotherapy](#) has dramatically changed our understanding of the basics of cancer biology and has altered the standards of [care](#) (surgery, radiotherapy, and chemotherapy) for patients. Cancer immunotherapy has generated significant excitement with the success of [chimeric antigen receptor \(CAR\) T cell therapy](#) in particular. Clinical results using CAR-T for hematological malignancies have led to the approval of four [CD19](#)-targeted and one [B-cell](#) maturation antigen (BCMA)-targeted cell therapy products by the US Food and Drug Administration (FDA). Also, [immune checkpoint inhibitors](#) such as antibodies against Programmed Cell Death-1 ([PD-1](#)), Programmed Cell Death Ligand-1 ([PD-L1](#)), and Cytotoxic T-Lymphocyte-Associated Antigen 4 ([CTLA-4](#)) have shown promising therapeutic outcomes and long-lasting clinical effect in several tumor types and patients who are refractory to other treatments. Despite these promising results, the success of cancer immunotherapy in [solid tumors](#) have been limited due to several barriers, which include [immunosuppressive tumor microenvironment](#) (TME), inefficient trafficking, and heterogeneity of [tumor antigens](#). This is further compounded by the high intra-tumoral pressure of solid tumors, which presents an additional challenge to successfully delivering treatments to solid tumors ¹⁾.

The immunosuppressive [tumor microenvironment](#) (TME) of [cancer](#) strongly hinders the anti-tumor [immune responses](#), thereby resulting in disappointing responses to [immunotherapy](#). Chemoattractive and promotive traits of [chemokines](#) exerted on [leukocytes](#) have garnered interest in improving the efficiency of [immunotherapy](#) by increasing the infiltration of [immune cells](#) in the TME. In a study, a [folic acid](#) (FA) -modified gene delivery system based on the self-assembly of DOTAP, MPEG-PCL-MPEG, and FA-PEG-PCL-PEG-FA, namely F-PPPD, was developed to deliver [plasmids](#) encoding the immunostimulating chemokine [CKb11](#). The delivery of plasmid CKb11 (pCKb11) by F-PPPD nanoparticles resulted in the high secretion of CKb11 from tumor cells, which successfully activated T cells, suppressed the M2 polarization of macrophages, promoted the maturation of [dendritic cells](#) (DCs), facilitated the infiltration of [natural killer cells](#) and inhibited the infiltration of immunosuppressive cells in tumor tissues. Administration of F-PPPD/pCKb11 also significantly suppressed the cancer progression. The study demonstrated a [nanotechnology](#)-enabled delivery of pCKb11, that remodeled the immunosuppressive TME, for cancer treatment ²⁾.

Glioma tumor immune microenvironment

see [Glioma tumor immune microenvironment](#).

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Guha P, Heatherton KR, O'Connell KP, Alexander IS, Katz SC. Assessing the [Future](#) of [Solid Tumor Immunotherapy](#). Biomedicines. 2022 Mar 11;10(3):655. doi: 10.3390/biomedicines10030655. PMID: 35327456; PMCID: PMC8945484.

2)

Nie W, Yu T, Liu X, Wang B, Li T, Wu Y, Zhou X, Ma L, Lin Y, Qian Z, Gao X. Non-viral vector mediated CKb11 with folic acid modification regulates macrophage polarization and DC maturation to elicit immune response against cancer. Bioact Mater. 2021 Apr 6;6(11):3678-3691. doi: 10.1016/j.bioactmat.2021.03.031. PMID: 33898872; PMCID: PMC8056185.

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