



Cancer Immunotherapy: Chapter 26. JAK/STAT Signaling in Myeloid Cells: Targets for Cancer Immunotherapy

Saul J. Priceman, Jiehui Deng, Richard Jove, Hua Yu

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Tumor-associated immune cells, in particular myeloid cells, have opposing roles during cancer development by facilitating antitumor immune responses and driving cancer-promoting inflammation. Defective antitumor immunity is prevalent in cancers, and it is now clear that overcoming the myeloid cell-mediated immunosuppressive microenvironment poses tremendous interest for future cancer therapies. JAK/STAT signaling has come to the forefront as a crucial pathway to induce immunosuppression and procancer inflammation. Specifically, STAT3 activation is critical for the phenotype of myeloid cells by regulating immunosuppressive and prometastatic factors, thereby providing myeloid cells with a multitude of tumor-promoting functions. Genetic ablation of STAT3 in the myeloid compartment induces potent innate and adaptive antitumor immunity along with an inhibition of tumor growth and metastasis. Recently, therapeutic targeting of JAK/STAT3 has shown great promise in blocking immunosuppression in preclinical models. One such example is the use of novel siRNA to selectively target STAT3 in myeloid cells, through conjugation to CpG oligonucleotides that agonize Toll receptor TLR9 on myeloid cells. Along with other novel therapeutic strategies to inhibit JAK/STAT signaling, it seems likely that future efforts to target this pathway will be made in single and combination approaches for effective anticancer immunotherapy.



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