Potentiating vascular-targeted photodynamic therapy through CSF-1R modulation of myeloid cells in a preclinical model of prostate cancer

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Résumé en anglais	Vascular-targeted photodynamic therapy (VTP) induces rapid destruction of targeted tissues and is a promising therapy for prostate cancer. However, the resulting immune response, which may play an important role in either potentiating or blunting the effects of VTP, is still incompletely understood. Myeloid cells such as myeloid-derived suppressor cells (MDSCs) and macrophages are often found in tumors and are widely reported to be associated with cancer angiogenesis, tissue remodeling, and immunosuppression. These cells are also known to play a critical role in wound-healing, which is induced by rapid tissue destruction. In this study, we investigated the effects of VTP on the recruitment of tumor-infiltrating myeloid cells, specifically MDSCs and tumor-associated macrophages (TAMs), in the Myc-Cap and TRAMP C2 murine prostate cancer models. We report that VTP increased the infiltration of myeloid cells into the tumors, as well as their expression of CSF1R, a receptor required for myeloid differentiation, proliferation, and tumor migration. As anti-CSF1R treatment has previously been used to deplete these cells types in other murine models of prostate cancer, we hypothesized that combining anti-CSF1R with VTP therapy would lead to decreased tumor regrowth and improved survival. Importantly, we found that targeting myeloid cells using anti-CSF1R in combination with VTP therapy decreased the number of tumor MDSCs and TAMs, especially M2 macrophages, as well as increased CD8 T cell infiltration, decreased tumor growth and improved overall survival. These results suggest that targeting myeloid cells via CSF1R targeting is a promising strategy to potentiate the anti-tumor effects of VTP.
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