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TMP1 significantly suppresses mammary tumor metastasis via inhibition of granulocytic myeloid-derived suppressor cell differentiation

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Myeloid-derived suppressor cells (MDSCs) are a heterogeneous population of immature myeloid cells including immature granulocytes, macrophages, and dendritic cells that expand extensively during cancer, inflammation and infection. MDSCs are increasingly recognized as having a crucial role in protecting tumor cells from immune surveillance by suppressing anti-tumor immunity. This study aimed to investigate the immune-regulatory and antitumor activities of TMP1 on MDSC expansion and tumor metastasis. The results showed that TMP1 effectively suppressed mammary 4T1 tumor metastasis and increased mouse survival in a mammary tumor resection mouse model. In addition, TMP1 significantly decreased tumor-induced splenomegaly and the number of granulocytic MDSCs in test mice. In ex vivo cell culture assays, TMP1 did not decrease the level of granulocyte colony stimulating factor (G-CSF, a key cytokine for granulocytic MDSC differentiation) produced by tumor tissue, tumor associated stromal cells and 4T1 cells. However, TMP1 significantly inhibited granulocyte-macrophage colony stimulating factor (GM-CSF) or G-CSF-induced MDSC differentiation from bone marrow cells. TMP1 also strongly inhibited GM-CSF-induced expression level of G-CSF receptor in bone marrow cells. Taken together, our results suggest that TMP1 significantly suppresses mammary tumor metastasis via inhibition of granulocytic MDSC differentiation.

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Soluble CD83 inhibits human peripheral blood mononuclear cell differentiation into dendritic cells *in vitro*

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Human CD83 is type I transmembrane glycoprotein, mainly expressed on mature dendritic cells (DCs). The CD83 is not only a molecular marker for mature DCs, but a regulatory molecule, especially its extracellular domain released from DCs, called soluble CD83 (sCD83), possessing many functions in immune regulations. However, whether the sCD83 has the regulation on human peripheral blood mononuclear cell (PBMC) differentiation into DCs is unknown. To investigate its regulatory function on human PBMC differentiation into DCs in vitro, we prepared the glycosylated and deglycosylated sCD83 in HEK293T cells and used the sCD83 to treat human PBMCs being differentiated into DCs. The results showed that both glycosylated and deglycosylated sCD83 could bind the PBMCs and significantly up-regulated CD14 expressions ($P<0.01$) and down-regulated CD1a and CD80 expressions ($P<0.01$), indicating that the sCD83 can inhibit PBMC differentiation into DCs, suggesting that sCD83 possesses the negative feedback regulation on human PBMC differentiation into DCs in vitro.

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