A2b adenosine receptor enhances melanoma growth and immune suppression mediated by myeloid-derived suppressor cells.

A. Pinto, R. Iannone, S. Morello

Dept. Pharmacy, University of Salerno, Via Giovanni Paolo II 132, Fisciano, Salerno, Italy.

A2b adenosine receptor (A2bR) mediates, at list in part, the adenosine-induced immune suppression in the tumor microenvironment. In this study we examined the role of A2bR in a mouse model of melanoma. Melanoma-bearing mice treated with Bay 60-6583, an A2bR agonist, had increased melanoma growth. This effect was associated with higher levels of IL-10, VEGF and MCP-1, and enhanced number of CD11b+Gr1+ myeloid-derived suppressor cells (MDSCs) in the tumor tissue. In contrast, administration of PSB1115, a selective A2bR antagonist, completely blocked the effects of Bay 60-6583. PSB1115-treated mice exhibited reduced melanoma growth and increased T-mediated response. These data suggest that A2bR promotes melanoma growth and immune suppression mediated by MDSCs and inhibiting A2bR is a promising immune therapeutic approach.