

OFFICIAL ABSTRACT and CERTIFICATION

Assessing the Crosstalk between CD47 and SIRPα and its Role in Modulating Tumor Cell Growth

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Abstract

CD47 has been found to be over expressed in melanoma, leukemia and lymphoma tumors and to bind with SIRPα to avoid phagocytosis. However, in melanoma cells, blocking CD47 expression does not enhance the phagocytosis process. Surprisingly, we found and confirmed that melanoma cells express SIRPα, and so became curious as to if the CD47 and SIRPα interaction within tumor cells had a biological function to modulate tumor cell growth. To test, cell viability was assessed by MTT assay and flow cytometry. Gemcitabine was used as positive control, and the antibody against murine CD47 (clone MIAP301) was used. Results indicate that gemcitabine was effective in decreasing the viability of cells by around 50% in all tumor cells studied. In RAW264.7 cells and B16 cells, but not MC38 cells, cell viability was decreased upon administration of the CD47 blocking antibody. Flow cytometry data also confirmed the CD47 and SIRPα expression of the corresponding cells. The data suggests that there is a biological function in the crosstalk of CD47 and SIRPα and that it modulates cell viability, as well as a potential biological function of CD47 and SIRPα interaction in RAW 264.7 cells.

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