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In vivo and *in vitro* effects of kinins on preclinical tumor models

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Generation and inactivation of bioactive peptides, like bradykinin (BK) and kallidin (lys-BK), by proteolytic enzymes in the tumor microenvironment may modulate tumor progression. We evaluated the effect of BK and lys-BK on murine melanoma B16F10-Nex2 and murine breast carcinoma 4T1 preclinical models. Both tumor cells express kinin receptors, B1R and B2R. *In vitro* B1R/B2R activation in B16F10-Nex2 cells did not affect cell proliferation or invasion, however, reduced migration. B1R/B2R activation in 4T1 cells did not affect cell proliferation either, but increased cell migration and invasion. B16F10-Nex2 cells were endovenously injected in C57BL/6 mice, and animals were alternate-day treated intraperitoneally with five doses of 20 or 50μM Lys-BK. Fifteen days after tumor challenge, it was observed a significant reduction in the number of lung metastatic nodules in peptide-treated groups. Female Balb/c mice were inoculated into mammary fat pad with 4T1 cells and the same treatment was performed; primary tumors developed similarly in treated and untreated mice. Alternatively, tumor cells were treated *in vitro* with 10 μM of Lys-BK and inoculated into syngeneic mice. It was observed a significant reduction in the number of metastatic melanoma lung nodules, but primary adenocarcinoma breast tumor development was not affected. Our results suggest that kinins interfere with metastasis development in both preclinical models.

Biography

Mariana Ferrazzo Souza graduated in Biomedical Sciences at Federal University of São Paulo, where she is currently attending a Master of Science Program. Her work was presented in 2009, 2010 and 2011, during her undergraduation, at regional meetings at Federal University of São Paulo.

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