**The role of extracellular NM23**

NM23-H1 (non-metastatic clone 23, isoform H1) was the first identified metastasis inhibitor. NM23-H1 was found to be downregulated in metastases of numerous solid tumors. NM23-H1 was shown to be present in the serum of patients with breast cancer, neuroblastoma and hematological malignancies. We intended to understand the mechanism by which NM23 is secreted into the extracellular environment and determine the role of extracellular NM23. As a model, we established invasive breast carcinoma MDA-MB231T cells transfected by FLAG::NM23-H1, MYC::NM23-H2. Extracellular vesicular fractions were isolated from conditioned media of these cell lines and tested for the presence of the NM23 homologs by flow cytometry and Western blotting. We have shown that exosomes and microvesicles derived from MYC::NM23-H2 overexpressing MDA-MB231T cells, contained the fusion protein. Next, we investigate when tumor cells release NM23 into the extracellular environment during tumor progression. By co-culturing NM23-transfected breast carcinoma cells and fibroblasts, the importance of extracellular NM23 will be examined in communication of tumor cells and microenvironment.