Incidence of malignant tumor is increasing with ageing of population worldwide. Tumors are highly complex tissues, where cancer-associated fibroblasts (CAF), inflammatory cells and blood vessels support the activity of malignant cells. From this point of view, the intercellular interactions seem to be in the center of interest of cancer cell biologists. CAF origin is not fully understood. However, among other sources, they can be formed by induction of transition of normal fibroblasts by TGF-β1 and/or endogenous lectin, galectin-1. These cells usually express smooth muscle actin, but it is not obligatory. CAF differ from normal fibroblasts in expression of almost 600 genes. They are functionally very active because CAF cultured with the normal epithelial cells significantly changed their phenotype to be similar to cancer cells. Interestingly, the fibroblasts under the influence of CAF acquire properties of mesenchymal stem cells. To complete the study of intercellular crosstalk in the tumor we also studied the opposite situation, where normal fibroblasts were cultured with normal/malignant epithelial cells. Normal fibroblasts cultured under the influence of epithelium acquire properties of CAF but for the limited time only. On the other hand, the activation of CAF by cancer cells seems to be more stable. The results demonstrated that IGF-2, BMP-4, CXCL-1, IL-6, IL-8 can play an important role in cancer cell-mesenchymal interaction. Blocking of cancer cell-CAF interaction by targeting of these molecules can have some therapeutic potential in future. Some similarity between the cancer and wound healing has been also observed. The presented data show how the combination of microscopy with genomic approach increases the volume of informations to improve the complexity of cancer microenvironment research.

References:
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