The *FAM13A* gene silencing effect on lung cancer cells

According to epidemiological data, lung cancer is the most common cancer, both in man and woman. Lung cancer are caused by the interaction between genetic susceptibility and environmental influences. Decreased availability of oxygen, called hypoxia, has been commonly observed in lung diseases. Low oxygen level during tumor formation progress, causes adaptation of cells to hypoxia which are relevant for proliferation and surviving. Hypoxia-response genes have been identified as negative prognostic factor, promoting more aggressive cancer phenotype. It has been suggested, that lower oxygen tension during hypoxia in lung cancer tumors, may modulate the *FAM13A* activity. In our preliminary study, we have indicated that the *FAM13A* was significantly up-regulated by hypoxia in lung cancer cell lines. However, the biological function of this gene in lung cancer and the precise role in hypoxia response has not been fully explained. The *FAM13A* gene is probably involved in signal transduction and activation of cell adhesion proteins, processes important in carcinogenesis.

The aim of this research is to determine the *FAM13A* gene silencing effect on lung cancer cells and its contribution in hypoxia-response. In the first part of the project the consequences of *FAM13A* gene silencing on lung cancer cells (viability, proliferation, invasive properties and apoptosis), will be done after hypoxia and normal oxygen tensions. In the second part, the *FAM13A* inhibition effect on global gene expression changes in lung cancer cells, after exposition to hypoxia, will be indicated.

The detailed characterization of FAM13A protein function and its regulatory activity, will be described. The global gene expression analysis in lung cancer cells, before and after *FAM13A* silencing, in normoxia and hypoxia, will enable to found genes regulated by *FAM13A* and the new signaling pathways which this gene is involved. If *FAM13A* gene is involved in important signaling pathway activation, its inhibition may cause changes in vitality, proliferation and invasiveness of cancer cells. This offer the new therapeutic targets in lung cancer.