**Examining the Expression and Function of Septin Proteins in Human Pancreatic Cancer Cells**

Pancreatic cancer is the 4th leading cause of death from cancer and its incidence has risen steadily. The survival rate of pancreatic cancer patients remains the lowest among all cancer types, and is projected to increase further in the future.

Septins family proteins bind to GTP of G protein, which allow to form a stable complex by interacting between a choice of members of the family. Structurally, Septins are related to RAS oncogene, and has been shown as an oncogene by its association with the mixed-lineage leukemia gene. Septin expressions are altered in many cancers, and their mutations were reported in cancers of large intestine, skin, endometrium and stomach. However, the expression and function of Septins in pancreatic cancer has not been examined.

Our preliminary data on PCR demonstrates increased level of transcripts of Septins in pancreatic adenocarcinomas compared to normal pancreatic cells. Our data on Western blot and imunocytochemistry indicates that protein level of various Septins are increased in pancreatic adenocarcinomas compared to normal pancreatic cells. Immunohistochemical analyses also shows higher Septin protein expression in cancer cells or cancer tissues each compared to normal cells or tissues.

We investigated the function of Septins by generating stable clones of Septin knockout using CRISPR approach in pancreatic cancer cells derived from malignant adenocarcinomas. Knockout efficiency was confirmed by GFP expression and Western blot analysis. Compared to control knockout, Septin knocktout induced a significant growth inhibition in cancer cells.

 In summary, our preliminary data indicates that Septins promote the development of pancreatic cancer by increasing their transcripts and proteins. Targeting Septin members may be a potential approach for the development of new therapeutics of pancreatic cancer.