Pancreatic ductal adenocarcinoma (PDAC) remains an important cause of malignancy related death. Despite recent progress in understanding the molecular basis of PDAC further studies are needed to find new molecular markers for diagnostic and therapeutic purposes. We found kallikrein 10 to be distinctly over-expressed in pancreatic ductal adenocarcinoma (PDAC) in a gene expression profiling approach using microdissected PDAC and normal ductal cells. Human kallikrein 10 (hK10) is a secreted serine protease. It has been reported that it is highly expressed in ovarian tissue and represents a novel serological marker for ovarian cancer. The role of hK10 in pancreatic cancer remains unclear.

Therefore we examined the relevance of hK10 in PDAC. We quantified by immunoassay, hK10 in sera from 40 healthy individuals (controls), 24 patients with benign pancreatic diseases, and 76 patients with pancreatic cancer. We then examined the diagnostic value of this measurement in pancreatic cancer and benign pancreatic diseases. We found that normal serum hK10 ranged from 21 to 1100 ng/liter (mean = 563 ng/liter). hK10 concentration in benign pancreatic diseases and pancreatic cancer showed no statistically significant different levels (range for benign diseases: 15-1,080 ng/liter; mean = 470 ng/liter; range for PDAC: 60-1,530 ng/liter; mean = 487 ng/liter).

We conclude that serum hK10 concentration has no prognostic and diagnostic value in pancreatic cancer. The discrepancy between high up-regulation of KLK10 mRNA in cancer tissues and no serum hK10 increase in pancreatic cancer may be due to the following reasons: (a) Transcriptional up-regulation but no translational increase, (b) Mutations in the KLK10 gene that lead to truncated proteins not measurable by ELISA, (c) Digestion of hK10 by pancreatic enzymes before hK10 reaches the circulation, (d) Expression of splice variant forms of mRNA that lead to proteins not detected by the ELISA assay used. These possibilities are under investigation.