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Distinctive serum protein signatures associated with chronic pancreatitis, autoimmune pancreatitis and pancreatic cancer

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Abstract

Pancreatic ductal adenocarcinoma (PDAC) is a devastating disease and the most effective approach to reduce the mortality from this cancer is successful early detection. The clinically available imaging modalities lack the sensitivity and specificity to diagnose asymptomatic PDAC and precursor lesions. This uncertainty leads to a postponement of surgical intervention for months, during which time often incurable tumors arise from precursor lesions or small premalignant foci. The detection of PDAC-associated biomarkers that could be routinely and reliably measured in the serum of PDAC-patients would provide an elegant non-invasive approach for the early diagnosis. Currently available tumor-marker assays are based on biomarkers which usually only appear at late stages of the disease with variable high false-negative and/or false-positive detection rates. Mass spectrometry (MS) and 2D-DIGE has been applied to analyze serum protein alterations associated with chronic pancreatitis (CP), autoimmune pancreatitis (AIP) and PDAC, to identify signatures indicative for the diseases. The sera were either fractionated by chromatography or immuno-depleeted from the 20 most prominent serum proteins prior further MS-analyses. The identity of biomarkers detected was determined by a combination of protein-fractionation, chromatographic purification steps, PAGE, and MS. The identified proteins were validated by ELISAs, and showed that they can easily be assessed by simple tests of patient sera. The lower levels of some of these proteins in PDAC sera could be the result of disturbed activity or balance of proteases in the tumor microenvironment and protease inhibitors, contributing to accumulation of truncated forms. Our data and collaborative studies support this hypothesis.

Biography

Felix, trained biochemist obtained his Ph.D. at the University of Tübingen, Germany in 1990. After a post-doctorate at the Radiation Biology Inst, LM-University Munich, he joined the Molecular Biology and Tumor Genetics Institute at the GSF-Munich in 1993 studying genomic instability, and subsequently moved to the NCI, NIH in Bethesda, MD, US working on oxidative-stress, genomic-instability and developing mouse models for mutagenesis and tumorgenesis. In 2003 he returned to Heidelberg, Germany, leading a research laboratory at the European Pancreas Center, Dept. of Surgery Univ. Heidelberg.