inflammatory breast cancer with 6 cycles of TEC (3-week) prior to the surgical treatment.
Results: Tumour response (pCR, pPR) of more than 70 % can be achieved using neoadjuvant TEC-regimen. The preliminary expression profiling results shown here indicate a subset of 148 genes that classifies all patients with a complete remission (pCR). A comparable separation of the groups could not achieved by established tumor factors, e.g. ER, PgR, HER2, uPA, which are measured simultaneously on the HGSM and also statistically evaluated.
Conclusions: HGSM semi-quantitative expression profiling is promising to have the potential to figure out genes that are related to cancer progression and chemotherapy resistance, especially in primary systemic chemotherapy.

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P63. EXPRESSION OF TISSUE FACTOR IN PANCREATIC ADENOCARCINOMA

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Background: Pancreatic cancer is the tumor with the highest risk for thromboembolic complications. Tissue factor represents the principal initiator of coagulation. The aim of this study is to study expression of tissue factor (TF) in pancreatic cancer.
Methods: TF expression was studied in human pancreatic carcinoma cell lines by Northern blot and immunofluorescence. Expression of alternatively spliced TF (asTF) was assessed by RT-PCR. TF expression was determined by immunofluorescence in tissues of pancreatic adenocarcinoma (PCa), chronic pancreatitis (CP) and normal controls. Plasma samples (30 PCa-patients, 13 CP-patients and 20 controls) were investigated for soluble TF levels and coagulation activation markers (thrombin-antithrombin III complex [TAT], prothrombin fragment 1 + 2 (F1 + 2)).
Results: All pancreatic carcinoma cell lines expressed TF (8/8) and most of them asTF (6/8). All but two pancreatic cancer tissue samples stained positive for TF (17/19). In all samples of cP weak staining was restricted to pancreatic duct cells. TF and TAT levels in PCa patients were significantly elevated whereas elevated F1 + 2 levels did not reach statistical significance compared to controls. In CP patients TAT and F1 + 2 levels proved to be significantly elevated compared to controls, although TAT elevation was less pronounced than in PCa patients.
Conclusion: We conclude that in addition to the upregulated expression of TF on the cell membrane soluble TF might be pivotal for activation of coagulation in pancreatic cancer.

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P64. PROTEIN-BOUND POLYSACCHARIDE PSK INDUCES GROWTH INHIBITION IN PANCREATIC TUMOR CELLS

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Background: Pancreatic carcinoma is particularly aggressive tumor, it has a poor prognosis and at the time of diagnosis, the tumor is generally in an advanced stage no longer suitable for resection. Moreover, this tumor is virtually resistant to conventional radio-/chemotherapy. Thus alternative therapy modalities are urgently needed. Morphologically pancreatic adenocarcinoma is characterized by a dense stromal reaction comprised of activated pancreatic stellate cells. This stroma may protect the tumor cells against the patients immune system and may in part be responsible for the chemoresistance of the tumor cells. Polysaccharide-K (polysaccharide-Kureha; PSK), also known as krestin, is a unique protein-bound polysaccharide derived from the CM-101 strain of the fungus Coriolus versicolor, which has been used successfully as a chemoimmunotherapy agent in the treatment of various cancers in Asia for over 30 years, however there is only one publication concerning the combined treatment of two pancreatic cancer patients with Cisplatin, PSK and UFT. PSK not only boosts the immune system of patients but also has direct antineoplastic effects. So it has been shown, that PSK reduces the invasiveness of a pancreatic tumor cell line by downregulating TGFbeta1 and MMP.
Aim and Methods: In order to analyze, if the reduction of the invasive potential by PSK is a general phenomenon in pancreatic tumor cells, we treated a panel of different pancreatic tumor cell lines with PSK and subsequently analyzed changes in gene expression using RT-PCR. Moreover, the activity of MMPs was analyzed using zymography, proliferation of the cells after PSK treatment was analyzed by WST-1 assay.
Results: Treatment of the pancreatic tumor cells with PSK for up to six days resulted in a short term induction of the cell cycle inhibitor p21/WAF1 at 4 and 24 h, after that expression returned to basal levels. In contrast PCNA and cycD1 expression gradually decreased during the PSK treatment, reaching maximal repression after about three days, which persisted for the rest of the treatment period. In contrast to the published results PSK did not change MMP expression, neither on the RNA level analyzed by RT-PCR nor on the protein level as analyzed by zymography. Moreover, PSK dose dependently decreased the proliferation of all pancreatic tumor cells investigated, reaching a plateau of about 40% decrease of proliferation at a concentration of 250 μg/ml PSK. A further increase of PSK concentration had only marginal additional effects on the proliferation of the cells.
Conclusion: Our results demonstrate a direct antineoplastic effect of PSK on pancreatic tumor cells by decreasing the proliferation of the cells, pointing on the possibility for the use of PSK in the treatment of pancreatic cancer.

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