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Abstracts

Poster Abstracts for the 2nd International Meeting on Molecular Staging of Cancer, 22–26 June 2006, Heidelberg, Germany

P1. OVEREXPRESSION OF THE C4.4A PROTEIN, A uPAR HOMOLOGUE, IN CANCER

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Background: C4.4A is a highly glycosylated GPI-anchor protein with some similarity to uPAR. Unlike uPAR, C4.4A has a very restricted expression pattern. In normal tissues, expression of C4.4A is limited to epithelia of skin, oesophagus and tongue. RNA analysis revealed expression of human C4.4A in different types of cancer including primary melanoma, lymph nodes and skin metastases of melanoma, mammary gland cancer, lung carcinoma and lung tumour derived metastases, transitional cell carcinoma (TCC) of urothelial cell origin and TCC derived metastases.
Results: We produced antibodies against human C4.4A. In normal skin, C4.4A expression is limited only to the keratinocytes of the stratum granulosum, while in wounded skin (chronic ulcers), expression is extended also to the stratum spinosum. C4.4A is expressed by different cancer cell lines including pancreas, prostate and breast cancer cell lines. Glycosylation of the protein changes between different cell lines. Expression analysis of C4.4A on cancer tissues specimens is ongoing. Preliminary analysis shows over-expression of C4.4A in liver metastasis of colon cancer origin.

The C4.4A homologue uPAR is released from cancer cell lines and is present in the sera of cancer patients. Similarly, C4.4A is released as whole molecule and fragments in the supernatant of cancer cell lines.
Conclusion: These data indicate clinical relevance of the C4.4A protein as a new diagnostic and possibly prognostic tumour marker.

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P2. DOWNREGULATION OF UROKINASE-TYPE PLASMINOGEN ACTIVATOR INHIBITOR (PAI-1) IN PANCREATIC CANCER

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Background: Pancreatic cancer has a bad prognosis with an overall 5 year survival rate of less than 3% for all patients. The urokinase-type plasminogen activator system plays a crucial role in tumor invasion and angiogenesis. We analyzed the mRNA expression of urokinase-type plasminogen activator uPA, its receptor uPAR and its inhibitor PAI-1 in pancreatic cancer.
Methods: Twenty-five tumor and corresponding normal tissue samples from patients with pancreatic cancer were analyzed. mRNA expression levels of uPA, uPAR and PAI-1 were detected using quantitative realtime RT-PCR analysis; β-actin was used as internal standard. Gene expression levels in tumor (T) and normal tissue (N) were both expressed as absolute and relative measurement values (T/N).
Results: Gene expressions of uPA and uPAR showed no significant difference in tumor and normal tissue. However, expression of PAI-1 mRNA was significantly ($p < 0.006$) lower in tumor (median 0.07, minimum 0, maximum 13.27) compared to normal tissue (median 0.14, minimum 0.04, maximum 20.32). There was no significant association with pT-, pN-category and grading.
Conclusion: In pancreatic cancer there is a significant downregulation of urokinase-type plasminogen activator inhibitor (PAI-1).

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