and -49(G > T) from the transcription start site. We could demonstrate that the variants -186(GGGC)5 ($p < 0.05$), and -49T ($p = 0.05$) were significantly or strongly associated with colorectal cancer as compared to the healthy controls. We found the highest promoter activity to be associated with -186(GGGC)3 and -49T.
Conclusion: These results suggest that the two pKi-67 promoter polymorphisms 186(GGGC)3 > (GGGC)5 and -49G > T located in the basic promoter may play a crucial rule in the development of colorectal cancer.

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P25. MATRIX METALLOPROTEINASE 9 SINGLE NUCLEOTIDE POLYMORPHISM ANALYSIS IN BLOOD OF UROLOGICAL CANCER PATIENTS

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Introduction: Ample evidence indicates that MMPs contribute in multiple ways to all stages of malignant progression, including tumor invasion, blood vessel penetration, metastases and tumor angiogenesis. MMP-9 has been found to be specifically associated with prostate cancer metastasis. A number of DNA polymorphisms in the MMP genes are associated with differences in MMP activity. However, the relationship between the polymorphism and susceptibility of cancer remains ambiguous. A cytosine (C) – thymidine (T) single nucleotide polymorphism (SNP) at position -1562 in the MMP-9 promoter is reported to affect expression of this gene.
Aim: To determine the prevalence of a single nucleotide polymorphism (-1562 C/T) in the MMP-9 gene promoter in cancer patients and evaluate it's correlation with tumor type and stage.
Methods and Materials: DNA from the cancer patients' blood was extracted and amplified with PCR. PCR-RFLP method was used to determine MMP-9 polymorphism in 18 prostate cancer cases, 4 benign prostate hyperplasia cases, 14 invasive bladder cancer cases, 5 non-invasive bladder cancer cases, 4 renal cancer cases and 1 adrenal gland cancer case.
Results: Prevalence of C/C, C/T, T/T genotypes was similar among bladder and renal cancer patients. In prostate cancer patients a significant association ($P = 0.0052$) between clinical stage and MMP-9 polymorphism was found.
Conclusion: Our data demonstrate that MMP-9 (-1562 C/T) polymorphism may modify susceptibility to prostate cancer. We hypothesized that this polymorphism might act as a genetic modifier in the development and progression of prostate cancer. Additional studies with larger population are warranted.

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P26. ANTAGONISTIC FUNCTION OF S100 PROTEINS DURING TUMOR DEVELOPMENT

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Background: Despite compelling data demonstrating a direct link between altered expression of S100 proteins located on human chromosome 1q21 and epithelial malignancies, the knowledge of their function and mode of action in epithelial cells and in tumor promotion or progression is largely unknown.
Methods: Gene expression profiling of the in vivo model of chemically induced skin carcinogenesis revealed differential regulation of genes coding for S100 proteins. Subsequent studies using in vitro models and tissue microarrays are currently extended by to suitable in vivo models.
Results: Here, we have identified a novel signaling pathway in epithelial cells initiated by extracellular S100A8/A9 resulting in the activation of AP-1-dependent gene expression. Importantly, co-expression of S100A3 inhibits S100A8/A9 mediated AP-1 activation, which is in line with repression of this gene during skin carcinogenesis suggesting a negative role for S100A3 in epithelial malignancy. We found elevated levels of MMP2 and MMP9, two well-known AP-1 regulated genes, and identified S100A6 as an additional target gene of S100A8/A9 signaling. Moreover, significant co-expression of S100A8 and S100A9 together with phosphorylation of c-Jun and elevated S100A6 protein levels were evident in eSCC. The in vivo relevance of S100A8/A9 interaction with RAGE is discussed.
Conclusion: Our data suggest that targeting the net activity of S100 induced signaling represents an auspicious strategy for cancer prevention and/or therapy.

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P27. DEFICIENT MITOTIC CHROMATIN CONDENSATION IN RESPONSE TO Chk1 INHIBITION IS MEDIATED BY DEREGULATED Cdc25B

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Background: One of the most common properties of cancer is genomic instability, a leading cause of which are defects of the DNA damage response (DDR). DDR defects in tumors and germline have been linked to clinical outcome and cancer susceptibility, respectively. Among DDR regulators, the nuclear checkpoint kinase Chk1 is an established transducer of ATR- and ATM-dependent signalling in response to DNA damage. Additional functions of Chk1 include regulation of unperturbed cell cycle progression. Recently, we have shown that Chk1 localizes to inter-