able to distinguish each other at high resolution using all the data obtained from women. According to these results, monitoring the plasma amino acid balance is a promising biomarker to diagnosis of various cancers.

GI-cancer prevention

P21
Sensitivity of colorectal cancer cell lines to cytostatics is not modulated by the calcium-binding protein calretinin

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One of the major obstacles for successful chemotherapy is that tumors frequently are resistant to certain forms of chemotherapy. Calretinin is an EF-hand calcium-binding protein mostly expressed in specific neurons and additionally in a variety of normal tissues, but is also observed in certain tumors derived from mesothelial and colon cells. Recent studies have postulated that treatment of colon cancer cells with either 5-fluorouracil (5-FU) or oxaliplatin leads to a resistant subpopulation of cells characterized by elevated calretinin expression levels. It was hypothetized that over-expression of calretinin may be one of the reasons leading to increased resistance of cells to treatment with either 5-FU or oxaliplatin. To test this, we investigated the sensitivity of calretinin-expressing HT-29 and calretinin-negative CaCo-2 colon cancer cells to 5-FU and cisplatin using the MTT assay. To directly assess the role of calretinin, the sensitivity of CaCo-2 cells stably transfected with either calretinin or the alternative splice variant, calretinin-22k, was determined. Our results have shown that HT-29 cells are more sensitive to 5-FU than CaCo-2 cells, while CaCo-2 cells were more sensitive to cisplatin. However, we did not observe any significant differences in sensitivity to either 5-FU or cisplatin of calretinin and calretinin-22k transfected CaCo2 cells when compared to mock-transfected CaCo-2 control cells. Although both calretinin variants affect adenocarcinoma cell differentiation, expression of either protein does not modulate sensitivity of CaCo-2 cells to the investigated cytostatics. Supported by grant aAV/1106/2004 from Ministry of Education of Slovak Republic.

P22
Bone mass density, subsequent risk of colon cancer and survival in postmenopausal women

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Objectives: To test the hypothesis that high bone mass density (BMD), a potential marker for cumulative exposure to endogenous estrogen, calcium and vitamin D intake, is associated with a lower risk of colon cancer, and that women with a lower BMD are likely to develop a more aggressive form of colon cancer, as defined by mortality.
Study design and Setting: BMD was measured in three different sites (Ward's triangle, trochanter, femoral neck) in 1,471 women 60 years of age. All incident cases of colon cancers were identified through record-linkage of cancer registry. The women were followed for a mean of 9.5 years.
Results: Overall 31 cases of colon cancer were observed among 28.6 expected (standardized incidence ratio (SIR) = 1.09, 95 percent confidence interval: 0.79–1.25). The SIR decreased with increasing bone mass density showing a significantly decreasing risk of 20% for women who were at the higher bone mass density comparatively to women who were at the lower bone mass density in all the skeletal sites. The 10-year survival rates showed that survival was increasing with increased BMD, but not significantly.
Conclusion: The findings suggest that postmenopausal women with lower BMD have an increased risk of colon cancer, but not more aggressive cancer.

P23
No association between VDR gene polymophisms (FoqI, TaqI and Apa1) and colorectal cancer in Romanian patients?

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Introduction and Purpose of the paper: Apart from the regulation of calcium metabolism, vitamin D3 plays an essential role in cell proliferation and differentiation in several tissues, including colonic epithelium. Polymorphisms in the vitamin D receptor (VDR) gene have been reported in several studies to be associated with colorectal cancer. o The goal of this study was to analyse the association between VDR gene polymorphisms (Foq, TaqI and Apa1) and colorectal cancer in Romanian patients.
Means and Methods: Blood samples were obtained, after informed consent from individuals, the test group including 70 colorectal cancer patients and 65 healthy persons. Genomic DNA was extracted from peripheral blood leukocytes using Promega Wizard kits. Three restriction fragment length polymorphisms (RFLPs) were genotyped: FoqI in exon 2, ApaI in intron 8 and TaqI in exon 9 of VDR gene. These polymorphic regions were amplified by standard unlabeled oligonucleotides followed by restriction enzyme digestion corresponding to each RFLP.
Results:

- These polymorphisms for the case-control association studies were conform to Hardy-Weinberg equilibrium expectation ($p > 0.05$) in both case and control groups.
- For FoqI polymorphism, distribution of genotype frequencies and allele frequencies do not differ significantly between all cancer patients and control (p 0.3983, respectively p 0.6113).
- For ApaI polymorphism, distribution of genotype frequencies and allele frequencies do not differ significantly between all cancer patients and control (p 0.2770, respectively p 0.4811).
- For TaqI polymorphism, distribution of genotype frequencies and allele frequencies do not differ significantly between all cancer patients and control (p 0.1095, respectively p 0.4118).

Conclusions:

- In our study, we do not find any association between analyzed polymorphisms and colorectal cancer patients.
- The role of the VDR gene polymorphisms should be further studied, by increasing the power of the study and the number of polymorphisms.