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below CMC. Above CMC, however, TDC induces GSH consumption, and oxidative damages of proteins, pointing to decreased mucosal detoxification ability. Ultimately, these findings might partly explain the BS-induced tissue damage of colon mucosa.

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Autonomic nervous system dysfunction in patients with irritable bowel syndrome

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Background: The normal autonomic nervous system (ANS) function is important in regulation of secretion and motility of the gastrointestinal tract and in visceral sensation.

Aim: The aim of this study was to evaluate the ANS activity in irritable bowel syndrome patients (IBS).

Patients and methods: A total of 20 pts (45 ± 8 years) and 30 healthy volunteers matched with age and gender (45 ± 5 years) were included in the study. The ANS activity was assessed by the heart rate variability (HRV) and the blood pressure variability with CNSystem equipment (Task Force Monitor 3040i) were measured in basal conditions in both groups. The plasma levels of epinephrine and norepinephrine were evaluated in resting conditions.

Results: HRV recording: in IBS patients we noted decrease of parameters of the HRV spectral domain analysis in comparison with the control group (LF – 664.1 vs. 811.6 ms²; HF – 422.5 vs. 854.6 ms²; *P* < 0.05 respectively). LF/HF ratio was increased in IBS patients (1.86 vs. 1.21 in the control group), which may indicate sympathetic overactivity. BPV recording: the patients' parameters of the spectral domain analysis of BPV in comparison to the control group were significantly lower (LF – 1.45 vs. 3.45 mmHg²; HF – 0.65 vs. 3.68 mmHg²; *P* < 0.05 respectively). The mean levels of epinephrine (E) and norepinephrine (NE) were higher in patients group than in the control (E: 1.28 ± 0.06 nmol/L vs. 0.65 ± 0.05 nmol/L; NE: 3.54 ± 1.2 nmol/L vs. 2.89 ± 0.8 nmol/L respectively).

Conclusions: Our results show changes of the autonomic system activity in patients with IBS. The increased plasma level of

catecholamine correlated with decreased parasympathetic and increased sympathetic parameters recorded in HRV and BPV measurements. We conclude that symptoms in patients with IBS and ANS dysfunction are caused mostly by high adrenergic drive resulting in gastrointestinal motility disorders.

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Chronic hepatitis C with extrahepatic lesions in the west region of Ukraine

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Background: Hepatitis C virus is estimated to infect 100 million people worldwide. Chronic infection occurs in 85–90% of persons exposed to the virus and may progress to chronic active hepatitis and cirrhosis. Chronic hepatitis C virus (HCV) infection is associated with several extrahepatic disorders. Although the exact pathogenesis of these conditions is not fully understood, several studies have provided insight into the role of HCV in their development.

Materials and methods: We performed study on 54 with chronic hepatitis C. For each patient HCV genotype was determined by polymerase chain reaction (PCR) with genotype specific primer. The presence, concentration, and type of cryoglobulins were tested by immunofixation. Rheumatoid factor (RF) and antinuclear antibody (ANA) were also measured.

Results: Extrahepatic manifestations were found in 26 (48%) patients with chronic hepatitis C (more frequently in hepatic cirrhosis). Renal involvement can occur early in the course of the disease and occasionally is the presenting symptom of HCV infection. Patients with chronic HCV infection reported a wide variety of rheumatic manifestations, impairing their quality of life, with discrete frequency. For the most part in women and in long-lasting disease 21% patients had cryoglobulinemia. Patients with cryoglobulinemia and free of it exhibited varying frequency cutaneous vasculitis (14%), Raynaud's syndrome (7%), affection of the muscles and joints (5%), nodular periarteritis, lesions of lungs, myocarditis, autoimmune thyroiditis, Sjögren's syndrome, monoclonal immunoglobulinopathy of high frequency were activity of rheumatic factor, hypocomplementemia and other immunological disorders.

Conclusion: Chronic hepatitis C associated with a wide variety of extrahepatic manifestations.

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Erythrocyte antioxidant enzymes as early warning bioindicators of exposure to anionic surfactants

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Background: The present study was designed to assess sublethal effects of anionic surfactant in a widely distributed and commercially important fish, gilthead seabream *Sparus aurata* L., in order to identify early warning bioindicators of exposure.

Materials and methods: To get this goal, groups of 30 juvenile specimens were exposed for 24, 48, 72 and 96 h to a sublethal concentration of anionic surfactant sodium dodecyl sulphate (SDS, 1 mg/L). Another group with 10 specimens was used as control (0 mg/L SDS). The activity of erythrocyte antioxidant enzymes [superoxide dismutase (SOD, E.C. 1.15.1.1) and glutathione peroxidase (GPX, E.C. 1.11.1.9)] was assessed from hemolysates. Histopathological changes from exposed gills were assessed by light microscopy (x400). This protocol was approved by a local ethics committee.

Results: Firstly, it should also be mentioned no mortality was observed during the whole experience. The activity of superoxide dismutase (SOD) and glutathione peroxidase (GPX) were

altered significantly from 24 h onward. In this respect, when compared to controls, both SOD (4.46 ± 0.38 vs. 8.92 ± 0.73 U/mgHb; $P < 0.05$) and GPX activities (0.42 ± 0.09 vs. 1.26 ± 0.2 U/mgHb; $P < 0.05$) were significantly increased after 24 h exposure to SDS. On the other side, histopathological changes of the gills were observed as early as at 72 h-exposure.

Conclusions: It is concluded antioxidant enzymes employed (SOD and GPX) changed significantly a long-time before histopathological alterations of gills became evident. Consequently, erythrocyte antioxidant enzymes may be also recommended as early-warning bioindicators of environmental pollution by anionic surfactant sodium dodecyl sulphate.

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The role of apoptosis and cell proliferation regulating genes in mycosis fungoides

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Background: Molecular mechanisms responsible for primary cutaneous T-cell lymphomagenesis are largely unknown. In the present study, K-Ras, p53 and Fas gene mutations and c-myc gene amplification were analyzed in skin samples of patients with MF, as well as p53 and Bcl-2 expression.

Materials and methods: Skin samples were taken from 35 patients, 30 with MF and five with large plaque parapsoriasis. PCR-SSCP analysis was used for detection of mutation in p53 gene, Fas gene, K-Ras and semi-quantitative PCR for detection of c-myc amplification. Immunohistochemistry was used for Bcl-2 and p53 expression analysis.

Results: In analyzed samples K-Ras mutations were not detected, and also, significant amplification of c-myc was not found. In 9/35 patients (25.7%) p53 mutations were found (in exons 4 and 6). p53 protein expression was found in 8/24 (33.3%) analyzed samples, more frequently in the late phase of disease (stage IIB and more). In 4/24 patients (16.6%) Fas gene mutations were found (all in exon 7). In the late phase of disease Bcl-2 expression on malignant T-cells was found to be more frequently low in contrast to the early disease where higher levels of Bcl-2 expression were found.

Conclusion: Alteration in genes that control cell apoptosis were found in 65% of patients, which implies that deregulation of apoptosis plays a central role in molecular pathogenesis of MF.

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Calcium dependent cell death in human platelets: evidence of calpain associated signalling in the intrinsic mitochondrial pathway

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Background: Calcium sensitive mitochondrial membrane potential is an early indicator of cell ageing in platelets (Leaver et al. Platelets, 2006), and metalloprotease inhibitors have been reported to improve haemostatic function in aged platelets in a mouse model (Bergmeir et al. Blood 2003). In this study, the role of calpain in degenerative changes was investigated by using three structurally unrelated inhibitors of calpain in human platelets.

Methods: Twenty four human pooled platelet preparations from 72 donors were incubated in gas permeable platelet storage bags at 22°C with constant agitation from 2–14 days in the presence of Calpeptin 50, 150, 500 µM; ALLN 5, 20, 50 µM; MG-132 5, 15 µM and mitochondrial and cytoplasmic indicators of cell death, including calcium sensitive mitochondrial membrane potential, caspase 3, 8 and 9 were analyzed at 2-day intervals.

Results: Platelet preparations showed decreased calcium sensitive mitochondrial membrane potential from 5 days and increased Caspase 3, 9 (and to a lesser extent) 8, which paralleled changes in platelet count. Calpain inhibitor toxicity was detected using ALLN, and to a lesser extent, MG-132 and Calpeptin in platelet preparations. However, at lower concentrations, platelets incubated with calpain inhibitors showed increased calcium sensitive mitochondrial membrane potential and enhanced survival.

Conclusions: These studies suggest a possible calpain dependent process associated with platelet ageing *in vitro* and indicate that calpain may be associated with previously reported calcium sensitivity and with the intrinsic mitochondrial pathway of cell death in human platelets and support reports of haemostatic function deficits in calpain knockout embryos.

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Changes in lipids profile in patients with ankylosing spondylitis during cryotherapy

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Background: The aim of the study was to examine the influence of whole-body cryotherapy on lipids profile in patients with ankylosing spondylitis (AS).

Materials and methods: This trial was carried out on 16 persons with AS exposed to a cycle of 10 daily, 2 min-lasting whole-body cryotherapy procedures with subsequent 60 min-lasting kinesitherapy. Before and after a cycle of whole-body cryotherapy procedures the following parameters of lipids profile: total cholesterol (TC), LDL-cholesterol, HDL-cholesterol, triglycerides (TG) concentrations in plasma were estimated.

Results: The obtained results are shown in table below.

Conclusions: The results of this study indicate that whole-body cryotherapy evokes a beneficial effect on lipids profile in patients with ankylosing spondylitis.