30 mg/kg and Group II at a dose of 100 mg/kg. Our results show that 14-day low dose (30 mg/kg) and non-toxic dose (100 mg/kg) caffeine usage decreased protein oxidation in kidney. AOPP levels of kidney tissue decreased significantly compared to control group; however, this decrease is found to be independent from dose. The most decrease is found in Group II. Tissue MDA levels are found to be decreased with caffeine. A statistically significant difference is found between control group and caffeine groups. The most decrease occured in Group II. This antioxidant effect of caffeine is interpreted to be closely related with dose. GST activities in rat kidney showed statistically significant increase with caffeine intake; however, this increase is found to be independent from dose. The most decrease is found in Group II. In SOD activities of kidney, there was no statistically significant difference among the groups. These results support protective effects of caffeine from oxidative stress in short term different doses consumption.

P08-11
Oncogene driven redox cell survival mechanisms

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Reactive oxygen species (ROS) are a group of molecules produced in the cell through metabolism of oxygen. It is now evident that at low physiological levels, hydrogen peroxidefor examplecan act as a classical intracellular signalling molecule regulating phosphatase/kinase driven cell signalling pathways. The oncogenes Bcr-Abl and Flt-3 are central to the development of both chronic and acute leukaemias and when these two genes are expressed on a tet regulated expression system they are associated with increased ROS levels. Using confocal microscopy we have demonstrated that the Flt-3 and Bcr-Abl driven ROS production, in particular hydrogen peroxide, contributes directly to increased genomic instability seen in these tumour cells. We also show by western blotting that there is an increased flux through the Pi3K/Akt survival pathway. Using siRNA and pharmacological inhibitors we demonstrate that the source of the ROS is NADPH Oxidase. Inhibition of this enzyme system lowers ROS levels and also the ability of the tumour cell to survive. Using multi-photon microscopy and hydrogen peroxide specific dyes we demonstrate that the ER is the site of this ROS production. Direct pharmacological inhibition of clinically used Bcr-ABl and Flt-3 inhibitors is associated with a marked reduction in ROS production and we show this is due to ubiquitination and proteasomal destruction of p22phox which is a component of the NADPH Oxidase enzyme system. This in turn facilitates the cell death inducing properties of these chemotherapeutic agents by removing the ROS element of oncogene survival signalling. Thus the tumour survival promoting properties of the Bcr-Abl and Flt-3 oncogenes are driven in part through their ability to stimulate the production of ROS.

P08-12
Effects of some pesticides on catalase and glutation *S*-transferase in Cyprinus carpio carpio

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Free radicals which known reactive oxygen species (ROS) have changing capasity biomolecules like lipids, nucleic acids, considerably reactive and unstabile metabolites. Biological sistems in body developed defence system because of prevent the effect of ROS. This system known 'antioxidant defence systems' or shortly 'antioxidans'. Pesticides may induce oxidative stress, leading to generation of free radicals and cause lipid peroxidation. Due to using of pesticide in agriculture, which fishes are living in polluted freshwaters effect of negative direction. In this study, we investigated some pesticides effects on catalase (CAT) and glutation *S*-transferase (GST) enzymes obtain from Cyprinus carpio carpio. For this study, today using at agriculture of five different pesticides(2,4- dichloroplenoxy acetic acid dimethylamine salt, fenpropathrin, cypermethrin, lambda-cyhalothrin, deltamethrin) was elected. The inhibitory effect of these pesticides on catalase and glutation s-transferase activity, observed under *in vitro* experimental conditions. The pesticides used in this study inhibited the catalase and glutation *S*-transferase activity from Cyprinus carpio carpio to various degrees. These findings observed *in vitro* could be useful in the understanding of the toxic effects that pesticides elicit on aquatic organisms *in vivo*.

P08-13
The effect of sodium tetraborate on antioxidant enzymes

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Antioxidant enzymes have main role in the defense of mammalian blood. Experimental investigations have repeatedly shown that erythrocytes are particularly sensitive to oxidative stres. For this purpose, the *in vitro* biochemical effects of sodium tetraborat on the human blood was investigated in this study. We observed enzyme activities of erythrocyte superoxide dismutase, catalase, glutathione reductase, glutathione s-transferase, glutathione peroxidase, glucose-6-phosphate dehydrogenase and also the effect of sodium tetraborat on antioxidant enzymes by spectrophotometrically. All the heparinized blood samples were taken from 10 healthy people between 25 and 35 who were not exposed to any toxic agents and did not smoke or drink. In this study blood samples were exposed to various doses (2, 4, 8, 17, 33 ppm) of sodium tetraborat *in vitro* conditions. In conclusion, the results suggested sodium tetraborat had shown neither inhibition nor activation effect on antioxidant enzyme activities.

P08r-14
Friedreich Ataxia: rat ventricular myocytes deficient in frataxin have disrupted mitochondria and impaired metabolism

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Friedreich ataxia (FA) is an inherited neurodegenerative disorder characterized by progressive gait and limb ataxia. Other variable features of FA disease include visual defects, scoliosis, and cardiomyopathy. It occurs in about 1 in 50 000 Caucasians. It is caused by mutations in the gene encoding frataxin, a mitochondrial protein which is depleted in FA. Although the exact function of frataxin is still controversial, the current commonly accepted hypothesis confers a role for frataxin in iron metabolism and in oxidative stress protection within the mitochondria. An important number of FA patients suffer from diabetes and most of them have evidence of cardiac dysfunction in live. Even though heart failure is the most frequent cause of death in these patients, the effects of frataxin depletion on cardiomyocytes are poorly understood. Disarrangements in the cardiac function could be caused by alterations in heart myocytes, which are rich