destructive mechanisms, known as the secondary lesion. The leading mechanisms of damage after SCI are excitoxicity, free radicals' overproduction, inflammation and apoptosis. On the other hand, metallothionein (MT) is a low-molecular-weight cystein-rich protein able to scavenge free radicals, such as hydroxyl radicals. MT participation as a neuroprotective mechanism after SCI is not known. The aim of the present study is to describe the changes of MT protein content in the acute phase after SCI in rats. Female Wistar rats weighing 200–250 g were submitted to spinal cord contusion model, by means of a computer-controlled device (NYU impactor). Rats receiving laminectomy were used as a control group. Animals were killed 2, 4, 8 and 24 h after surgery. MT was quantified by the silver-saturation method, using atomic absorption spectrophotometry. Preliminary results indicate an increased MT content by effect of SCI of 20%, 13%, 83% and 105% at 2, 4, 8, and 24 h, respectively, as compared to sham group values. These results suggest an induction of MT synthesis by SCI and the possible participation of MT as a protective mechanism in response to lesion.

P.315

CAFFEINE CONSUMPTION PREVENTS HIPPOCAMPAL ALTERATIONS IN TYPE 2 DIABETIC MICE

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Diabetes is associated with an increased risk of cognitive dysfunction mainly in older adults, which can be prevented by maintaining good control of glycaemia. Caffeine is the most widely consumed psycho-active substance, is suggested to improve performance in learning and memory tasks, and may also reduce the risk of diabetes possibly by increasing energy expenditure and weight loss. Caffeine mostly acts as an adenosine A2A receptor antagonist when consumed chronically and interestingly the density of this receptor increases upon chronic brain insults such as hyperglycaemia. We now treated a mouse model for type 2 diabetes associated with obesity (NONcNZO10/Ltj from Jax Laboratories) with 1 g/L caffeine in drinking water for 4 months, starting on the 7th month of age. Caffeine reduced body weight and glycaemia of diabetic mice. Histological analysis of brain sections revealed the absence of cellular damage in the diabetic hippocampus evaluated by Cresyl Violet staining of Nissl bodies and FluoroJade-C staining of degenerating cells. Western blot analysis showed that diabetes increased GFAP immunoreactivity in the hippocampus and immunohistochemical analysis indicated an increase in the number of GFAP positive cells in the diabetic hippocampus, which was prevented by caffeine treatment. This astrogliosis may reflect the need of the hippocampus to maintain proper control of glucose homeostasis. The density of the synaptic proteins SNAP25 and synaptophysin was decreased in the diabetic hippocampus, suggesting an impairment of synaptic connectivity. These synaptic modifications were partially prevented by caffeine treatment. The present results suggest that caffeine consumption may help preventing diabetes-induced hippocampal alterations that can lead to cognitive decline on aging.

P.316

INCREASED GLUTAMATE-INDUCED NEURONAL DEATH IN TRANSGENIC MODEL OF HUNTINGTON'S DISEASE. INFLUENCE OF GLYCOLITIC METABOLISM

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Huntington's disease (HD) is a neurodegenerative disease originated by increased glutamine repeats in the huntingtin-protein (mhtt). An excitotoxic mechanism has associated with the degeneration of the medium spiny neurons (MSN) in the caudate-putamen. The R6/2 transgenic mice, which express the N-terminal site of mhtt, show increased NMDA-induced current activation. In addition, altered glutamate uptake system has been described in R6/2 mice. These evidences suggest that mhtt conduces to altered glutamatergic neurotransmission, possibly facilitating excitotoxicity. To test this hypothesis we evaluated the vulnerability of R6/2 mice to the intraestriatal administration of glutamate. Twenty four hours after glutamate administration, larger lesions were observed in the striatum of R6/2 as compared to wild type mice. On the other hand, it is known that glutamate toxicity is exacerbated during metabolic inhibition, and altered glycolysis has been suggested from direct association between mhtt and the glyrceraldehyde-3-phosphate dehydrogenase (GAPDH). Therefore, we have tested glutamate toxicity in mice previously treated with the GAPDH inhibitor iodoacetate (IOA). We observed that GAPDH activity is more sensitive to IOA treatment in transgenic than wild type mice. IOA treatment increased glutamate toxicity both in wild type and transgenic mice.

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P.317

CIS-4-DECENOIC ACID ALTERS RESPIRATORY PARAMETERS IN RAT BRAIN

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Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency is the most frequent disorder of fatty acid oxidation. Biochemically, MCAD-deficient patients present tissue accumulation of the medium-chain fatty acids octanoic, decanoic and cis-4-decenoic (cDA) acids. Clinical presentation of MCAD deficiency is related to fasting and increased metabolic stress, which precipitate acute symptoms such as drowsiness or lethargy that may develop into coma or even death. In the present work, we investigated the *in vitro* effect of cDA on respiratory parameters in mitochondrial preparations from rat brain, namely states III and IV, as well as the respiratory control ratio (RCR), using glutamate/malate and succinate as