MS-B5 + SS-D7

MS-B5-07

NEUROPROTECTIVE EFFECT OF DEPRENYL AND PARA-FLUOR-DEPRENYL

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Oxidative deamination of primary monoamines by monoamine oxidase (MAO) produces neurotoxic agents, which could play a role in the pathomechanism of neurodegenerative diseases. MPTP is the best model to induce parkinsonian symptoms and its effect can be prevented by (-)-deprenyl (D) pretreatment. In addition to the MAO-B inhibitory effect, D or mainly its metabolites inhibit the uptake process which can also play a role in the protection. (-)-Para-fluor-deprenyl (PFD) possesses similar spectrum of action but due to its more prolonged blood and tissue level its uptake inhibitory effect can be more expressed compared to D. D displaces ^{3}H-MPTP from the binding site of neuromelanin and human substantia nigra preparation. The experiments suggest the existence of a low and a high affinity binding site of D and PFD.

D and PFD prevent the neurodegenerative effects of the noradrenergic toxin DSP-4. In these studies CFY-rats were treated with DSP-4 (50 mg/kg i.p.) 1h, 24h or 4 days after the administration of D or PFD (10 mg/kg i.p.) and were killed one week after DSP-4 administration. The hippocampal norepinephrine (NA) content and the MAO-B activity were determined. The two inhibitors showed a similar potency in inhibiting MAO-B activity and both of them prevented the decrease of NA level when they were given 1h before DSP-4 administration. D was not effective when it was administered 24h or 4 days before DSP-4 treatment, while PFD at 24h showed some protective effect.

The preventive effect of the two inhibitors is not related to the MAO-B inhibition. We proved that the metabolites of the two inhibitors (methamphetamine and p-fluor-methamphetamine) were also potent to prevent the decrease of NA level.

MS-B5-08

NERVE GROWTH FACTORS IN NEURODEGENERATIVE DISEASES

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The recent cloning of the nerve growth factor (NGF) as well as new members of the NGF family, namely the brain-derived neurotrophic factor (BDNF) and neurotrophin-3 (NT-3) has greatly expanded our knowledge on the structural properties and neurotrophic activities of these proteins. Elucidation of their developmental and topographical expression and comprehension of the mechanisms regulating their synthesis in nervous system physiology and pathology are proceeding at a brisk pace. This has led to propose a potential pharmacological use of these proteins in some neurodegenerative diseases. For example, the capability of NGF in affecting forebrain cholinergic neurons, has suggested its therapeutic use in patients suffering from Alzheimer's disease, which is associated with profound alterations of basal forebrain cholinergic systems. The possibility of using neurotrophic factors or agents capable of affecting their action and/or synthesis in clinical studies is likely to represent one of the major areas of neurobiological research in the coming years.

MS-B5-09

Potential Use of Lazaroids in Neurodegenerative Disease.

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Superoxide and hydroxyl free radicals, hydrogen peroxide, and lipid hydroperoxides are known to be generated by normal homeostatic processes and a variety of pathological states. If not catabolized by endogenous scavengers such as superoxid dismutase, catalase, glutathione peroxidase, Vitamin E and glutathione, free radicals and peroxides can severely damage and kill cells and tissues through peroxidation of membrane lipids, constitutive protein, and nuclear DNA. Tissue damage by free radicals is implicated in a variety of age-related disorders including neurodegenerative diseases such as Parkinson's disease and Alzheimer's disease. In the aged brain, there are abnormal intraneuronal accumulations of lipofuscin, an end product of lipid peroxidation. The generation of hydrogen peroxide by dopamine catabolism is hypotheized to compromise the viability of mesencephalic dopaminergic neurons resulting in Parkinson's disease. In Alzheimer's disease, the brain appears to be challenged by increased oxidative stress as superoxide dismutase is disproportionately increased in vulnerable neurons, and the activity of enzymes are increased that generate endogenous reducing agents such as NADPH. Examination of cerebral cortex from Alzheimer's patients indicates a higher baseline content of thiobarbituric acid-reactive lipid peroxidation products compared to age-matched controls. Induction of lipid peroxidation by iron *in vitro* is also larger in Alzheimer's samples.

The "Lazaroid" compounds are a group of 21-aminosteroid analogs, the most interesting of which is U-74006F or tirilazad mesylate. These compounds were identified for their ability to inhibit free radical-induced lipid peroxidation. Tirilazad mesylate demonstrates profound efficacy in animal models of closed head injury and cerebral ischemia, and is believed to act by potentiating endogenous radical scavengers and inhibiting radical-induced molecular peroxidation. In samples from Alzheimer's brain, lazaroid will reduce the extent of *in vitro* iron-induced lipid peroxidation. These data suggest that lazaroid therapy may slow the progress of age-related, free radical-induced neurodegeneration. We have begun to examine the efficacy of lazaroid therapy in animal models of Parkinson's and Alzheimer's disease.

SS-D7-01

DISTURBANCES OF HEAD MOTION AFTER WHIPLASH INJURY. A PROSPECTIVE STUDY

Berger M., Gerstenbrand F., Taferner E., Schauer R., Holzmüller G., Baldauf E. Innsbruck / Austria

In the prospective study the three dimensional headmovement of 407 investigations after whiplash injury was documented by cervicomotography at regular intervals (for three times in the first year).

The results of the examinations were compared with the results of 225 healthy volunteers, who had no history of trauma and showed no neurological or neuroorthopaedical abnormities.

The examination included preprogrammed and tracked movements induced by optical and acoustic triggers. The automatic calculation gives 126 different parameters for one examination.

The investigation included slow voluntary movements in the three axes of rotation, preprogrammed movements, slow tracking movements following visual targets and investigation of the passive mobility.

Most of the patients after whiplash- injury showed typical changes of motor patterns during many months. Only a few patients with marked pathological changes had a complete restitution of motor function. The analysis of the motor patterns of the cervical spine seems to be a powerful tool for the diagnosis of acute symptoms and late sequelae after whiplash injury of the cervical spine.

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