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Cerebellar granule and glial cells prepared from 8-day rat pups were used to investigate the effects of dehydroevodiamine (DHED) on the glutamate release and uptake. The subacute DHED exposure retarded the growth of granule cells and their LC50 was 718.3 μM. However, the viability of glial cells were not affected. The basal release of glutamate from cultured granule cells was decreased (16.1%) by 5 μM of DHED. Also NMDA-induced release of glutamate was inhibited. However, the basal and NMDA-induced release of glutamate from DHED-exposed granule cells (5 μM) for 9 days were not affected by DHED. In addition, DHED (5 μM) significantly inhibited (31%) the glutamate uptake from cultured glial cells. Although DHED did not affect the glutamate uptake from DHED-exposed glial cells (5 μM) for 9 days, DHED potentiated the inhibitory response of L-pyrollidine-2,4-dicarboxylic acid (PDC). In the cAMP-treated glial cells, DHED (5 μM) slightly inhibited (7.8%) and potentiated the inhibitory response of PDC. Although DHED did not affect the glutamate uptake from DHED and cAMP-exposed glial cells, DHED reduced the inhibitory response of PDC. These results indicate that DHED inhibited the glutamate uptake and release. Also the result suggest that DHED might modulate the glutamatergic nervous system.

[PB3-3] [10/20/2000 (Fri) 15:30 - 16:30 / [Hall B]]

Changes in the glutamatergic nervous system of cerebellum after pre - or post-natal nicotine exposure in rats

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To determine changes in the glutamatergic nervous system in cerebellum after the chronic nicotine exposure, nicotine was supplied from the mating through drinking water (25ppm). After delivery, each group was divided into two groups. Groups were continuously exposed to either deionized water or nicotine. Eight week old rats were sacrificed and cerebella were rapidly dissected. The various parameters of glutamatergic nervous activities were measured. The total levels of glutamate in post-natally nicotine exposed rats were only significantly increased (26%), compared with the control. However, those of glutamine (24%) and GABA (14%) in pre-natally nicotine exposed rats were increased. The activitiy of glutaminase was increased (15-19%) in both pre-natally and continuously nicotine exposed rats. And that of glutamine synthetase was also increased (27-87%). While those of glutamate dehydrogenase was decreased (10-37%) in all nicotine-treated rats. In addition, alteration of these enzymatic activities after nicotine exposure was similar with those of previous studies using cultured cerebellar cells prepared from eight day old pups exposed to nicotine with the same dose schedule. These results indicate that the glutamatergic nervous system in cerebellum are changed after the nicotine exposure and suggest that either pre- or post-natal nicotine exposure might affect the excitatory amino acid system during the development. Furthermore, the results suggest that the model of cell culture may be useful for the determination of the alteration by the exogenic agents.

[PB3-4] [10/20/2000 (Fri) 15:30 - 16:30 / [Hall B]]

Impairments of learning and memory following intracerebroventricular administration of AF64A in rats

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