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The protein levels of collagen type I, collagen type IV and fibronectin were examined in 6-, 12-, 18-, and 24-month old Fischer 344 rats which were fed ad libitum and diet-restricted. The protein level of type I collagen increased in the kidney and testis by aging and it was modulated by dietary restriction. The m-RNA level of type I collagen in the testis was changed as a similar pattern. Type I collagen in the liver and lung had no change by aging and dietary restriction. The protein level of type IV collagen decreased by aging in the testis and kidney and dietary restriction saved their decrease. However, the m-RNA level of type IV collagen in the testis was not changed by aging and dietary restriction. The protein level of type IV collagen in the liver increased by aging and dietary restriction modulated the increase. However, type IV collagen was not detected in the lung. The protein level of fibronectin increased several times by aging in the testis, kidney and liver and dietary restriction modulated their increase. Even though fibronectin protein level decreased in the lung by aging, dietary restriction had no effects on it. Therefore, the detailed molecular and biochemical studies are further needed to clarify the effects of aging and dietary restriction on the levels of extracellular matrix proteins.

[PB3-1] [10/20/2000 (Fri) 15:30 - 16:30 / [Hall B]]

Nicotine alters the characteristics of cAMP -exposed cerebellar glial cells

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Cerebellar glial cells prepared from 8-day rat pups were used to investigate the effects of subacute nicotine exposure on the glutamate uptake. These cells were exposed to cAMP and nicotine for 2 to 10 days in situ. cAMP and nicotine exposure did not result in any change in cerebellar glial cell viability at concentrations up to 500 μM. Glutamate uptake in the dibutyl cAMP-treated glial cells was significantly increased (30.7%) by 100 μM of nicotine. After subacute exposure with nicotine, the basal glutamate uptake was significantly decreased (11.4%). Furthermore, the IC50 of L-pyrollidine-2,4-dicarboxylic acid, glutamate uptake inhibitor, on the glutamate uptake was 6.7 times decreased compared to the control (184.1 vs 27.4 μM) and the sensitivity of glial cells to PDC was increased. In addition, the activity of glutamine synthetase in subacute nicotine exposed glial cells was 2 times increased compared to the control. After nicotine exposure, the changes in the characteristics of glutamate uptake in cAMP-exposed glial cell were opposite to those in cultured glial cell without cAMP. These results indicate that subacute nicotine exposure modulates the characteristics of the glutamate uptake and the GS activities of glial cells. Also the result suggest that the different states of glial cells during age and in regions might be differently affected by the exposure of subacute nicotine.

[PB3-2] [10/20/2000 (Fri) 15:30 - 16:30 / [Hall B]]

Effects of dehydroevodiamine on the release and uptake of glutamate in cultured cerebellar cells.

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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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