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Cerebrum Maturity Damage Induced by Fluoride in Suckling Mice

Authors : Hanen Bouaziz, Françoise Croute, Najiba Zeghal

Abstract : In order to investigate the toxic effects of fluoride on cerebrum maturity of suckling mice, we treated adult female mice of Swiss Albinos strain by 500 ppm NaF in their drinking water from the 15th day of pregnancy until the day 14 after delivery. All mice were sacrificed on day 14 after parturition. During treatment, levels of thiobarbituric acid reactive substances, the marker of lipid peroxidation extend, increased, while the activities of the antioxidant enzymes such as glutathione peroxidase, superoxide dismutase and catalase and the level of glutathione decreased significantly in cerebellum compared with those of the control group. These results suggested that fluoride enhanced oxidative stress, thereby disturbing the antioxidant defense of nursing pups. In addition, acetylcholinesterase activity in cerebellum was inhibited after treatment with fluoride. In cerebellum of mice, migration of neurons from the external granular layer to the internal granular layer occurred postnatally. Key guidance signals to these migrating neurons were provided by laminin, an extracellular matrix protein fixed to the surface of astrocytes. In the present study, we examined the expression and distribution of laminin in cerebellum of 14-day-old mice. Immunoreactive laminin was disappeared by postnatal day 14 in cerebellum parenchyma of control pups and was restricted to vasculature despite the continued presence of granular cells in the external granular layer. In contrast, in cerebellum of NaF treated pups, laminin was deposited in organised punctuate clusters in the molecular layer. These data indicated that the disruption of laminin distribution might play a major role in the profound derangement of neuronal migration observed in cerebellum of NaF treated pups.

Keywords : acetylcholinesterase activity, cerebellum, laminin, oxidative stress, suckling mice
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