【P-27】

Down-Regulation of Palate, Lung, and Nasal Epithelium Clone (Plunc) Gene Expression Induced by Ethanol Exposure in the Developing Mouse Embryo

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Accumulated evidence indicates that maternal alcohol consumption causes fetal alcohol syndrome including fetal enteric damage and growth retardation. Particularly, alcohol is a teratogen that induces a variety of abnormalities including brain and facial defects. In the previous study, cDNA microarray analysis showed that gene expression of the 'palate, lung, and nasal epithelium clone (plunc)', significantly changed due to ethanol exposure. The gene expression of plunc extends to nasal epithelial surfaces in the respiratory tree, digestive tracts, and oral cavity including the palate. We demonstrate that administration of ethanol to mouse embryos results in a dramatic loss of plunc depending on the developmental stage. Expression in the mouse embryo is first noted on gestation day (GD) 14. To investigate the teratogenic effect of acute alcohol exposure, pregnant C57BL/6 mice were exposed to two intraperitoneal infections of 25% ethanol (0.02ml/g), four hours apart, in GD 6 to 8. After exposure, mice were sacrificed by cervical dislocation of GD 10 ~ GD 15. Then the gene expression changes were confirmed by RT-PCR analysis.

Keyword : fetal alcohol syndrome (FAS), palate, lung, and nasal epithelium clone (plunc), RT-PC