

〔目的〕

High fat (HF) diets and platelet-derived growth factor (PDGF) receptor β (PDGFR- β) in the brain, as well as dioxin exposure, affect feeding behavior, which is an important determinant of body growth and obesity. In the present study, the effects of prenatal exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and HF food after weaning on body growth and expression of PDGFR- β in the brain were investigated in rat pups.

〔方法並びに成績〕

TCDD (1.0 $\mu\text{g}/\text{kg}$) was orally administrated to dams on the 15th gestational day. The rat pups from each litter in the control and dioxin exposure groups were assigned to 1 of 3 nutritional groups: standard diet, HF diet as juvenile, or HF diet in adulthood. Body size parameters (length, body weight, body mass index) were measured daily, and expression of PDGFR- β was assessed by western blotting using samples from brain areas including the hippocampus, amygdala, nucleus accumbens (NAc), striatum, parietal cortex and superior colliculus (SC).

〔総括〕

Results indicated that consumption of the HF diet decreased PDGFR- β levels in the amygdala and hippocampus in both sexes compared to the control group, while TCDD decreased PDGFR- β levels in the amygdala and striatum only in females receiving a HF diet. Furthermore, PDGFR- β levels in the hippocampus and striatum were negatively correlated with increases in body length, while those in the amygdala and NAc were related to body weight gain or body mass index. Together with findings from previous studies, the results suggest that the changes in body growth and brain functions due to dioxin and HF diets may be partially mediated by changes in PDGFR- β levels. Furthermore, the present findings also suggest that these dioxin and HF diet-induced changes in brain functions (depression, cognitive deficits, etc.) may be partially mediated by changes in PDGFR- β levels in the brain.