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Immunohistochemical localization of PLC in rat brain after chronic ECS

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Chronic electroconvulsive shock(ECS) was shown to increase phosphatidylinositol-4,5-bisphosphate(PIP_2) breakdown and the activity of PLC with the accumulation of inositol-1,4,5-triphosphate(IP3). The purpose of the present study was to determine the effect of ECS on the expression of phospholipase C(PLC) isotypes in rat brain. Two groups of animals were prepared : sham and ECS treated groups. Rats in ECS treated groups received maximal ECS(70mA, 0.5second, 60HZ) by constant current stimulator through ear-clip to induce tonic extension seizures for 12 consecutive days. The expression of PLC isotypes in rat brain was determined by immunohistochemical procedure using sagital section of rat brain. The immunoreactivity of PLCβ1 was observed in corpus striatum, hippocampus, thalamus and that of PLCγ1 in corpus striatum, hippocampus, thalamus, frontal cortex, parietooccipital cortex, limbic forebrain, pons, medulla, superior colliculus, inferior colliculus, rest of midbrain. The amount of PLC was analyzed by Western blot using antibodies against PLCβ1 and PLCγ1. Chronic ECS reduced the immunoreactivity of PLCβ1 in corpus striatum, hippocampus, thalamus but had little effect on PLCγ1. To quantify this change, quantitative Western blot using antibodies against PLCβ1 and PLCγ1 was conducted. The immunoreactivity of PLCβ1 in ECS treated rat whole brain was decreased by 40 % in cytosolic fraction and 26 % in membrane fraction. This different effect of ECS on PLC isotypes may results from the difference of their activation mechanisms and the different effects of ECS on them. The results from the present study suggest that chronic ECS primalily affects neurotransmitter receptors related IP_3 signaling in rat brain.