ERK1/2 Phosphorylation in the Neocortex of Mice Submitted to PTZ-Kindling

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ERK1/2 are related with neuroplasticity and may be involved with epileptogenesis. Pentylenetetrazole (PTZ)-kindling is a useful model of epileptogenesis. We investigate the ERK1/2 phosphorylation state (P-ERK1/2) in the PTZ-kindling. Adult male Swiss mice were injected with PTZ (35mg/kg i.p.) on alternate days for eight times. Control animals received saline (n=7). Forty eight hours after the last PTZ injection the level of ERK1/2 phosphorylation was determined in the neocortex by western-blotting analysis. After the PTZ-Kindling, 9 animals (28.1%) did not reach the Racine stage 4 or 5. The remained 19 developed at least one stage 4 or 5 seizure. Animals that not developed stage 4 or 5 seizures showed no significant changes in the P-ERK1 (p = 0.11) and P-ERK2 (p = 0.88) in comparison to controls. Animals who developed stage 4 or 5 seizures showed a significant (p < 0.01) decrease of P-ERK1 levels (-25%) and a non-significant trend (-16%; p = 0.06) for P-ERK2 levels in comparison to the control group. There were a significant correlation between the P-ERK1 (Pearson correlation -0.56, p = 0.01) and for P-ERK2 (Pearson correlation -0.55, p < 0.01) and the number of days with crisis in the PTZ treated animals. The total levels of ERK1/2 were not altered by PTZ-kindling. We conclude that PTZ-kindling induced changes in the cortical activation of ERK-1 and 2 that correlates with seizures intensity and frequency. Therefore modulation of ERK1 and 2 may be involved with epileptogenesis in the PTZ-kindling in mice.

Word Keys: ERK1/2, Kindling, Pentylenetetrazole

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