DEGENRATIVE NEURONAL CHANGES IN THE RAT DORSAL STRIATUM OF 18 DAYS VARIANT INTERVALS INDUCED STATUS EPILEPTICUS.

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INTRODUCTION

Experimental studies indicate that Status epilepticus (SE) causes neurodegenerative changes in the hippocampus, limbic structures, thalamus and neocortical areas. However, basal ganglia (BG) are rarely mentioned. Several clinical studies have reported brain abnormalities (atrophy, metabolic changes) in structures remote from the seizure focus as well as in BG.

AIM OF THE STUDY

To obtain new data on the extent of neuronal degeneration in Dorsal striatum (DS) during development after the SE.

MATERIALS & METHODS

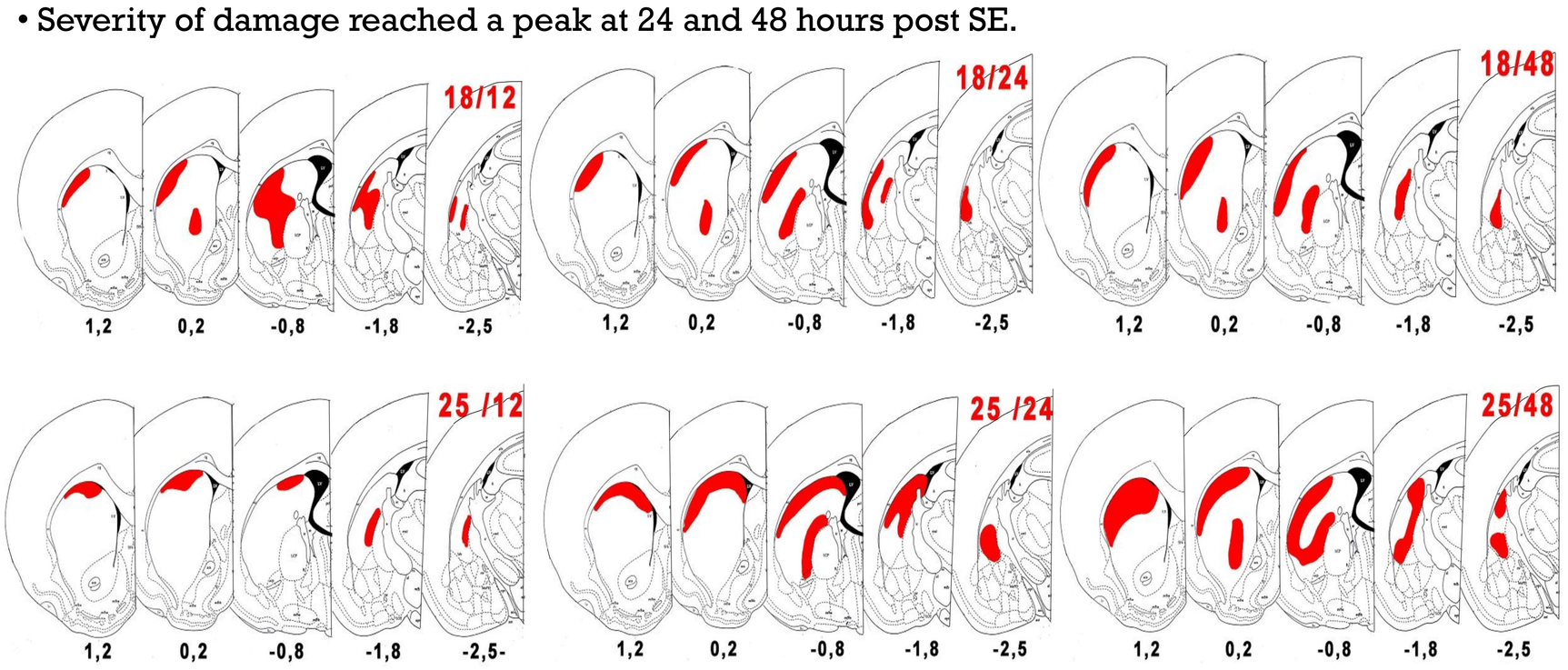
Lithium pilocarpine model of SE: Wistar pups 12, 15, 18, 21 and 25 days old.

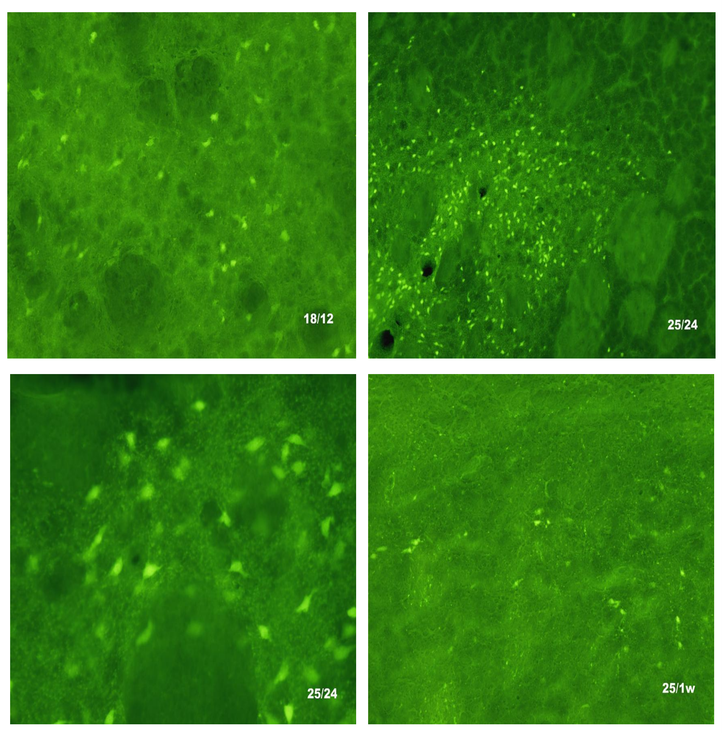
- **Seizure induced:** 3 mmol/kg, i.p. LiCl were injected 24 hours before 40 mg/kg,i.p. pilocarpine. 2 hours after the SE, motor seizures were suppressed with 0.3-0.6 ml/kg i.p. paraldehyde.
- **Survival intervals:** 4, 8, 12, 24, 48 h, 1 week post SE. 3-4 rats per age and interval group.
- **Anesthesia:** 2.5 g/kg, i.p. urethane and perfused with PBS followed by 4% paraformaldehyde in 0.1 M phosphate buffer, pH 7.4.
- **Sections preparation:** Brains were sectioned into 50 µm. **FJB-labeled** degenerated neurons were plotted to standard stereotaxic sections.

*Animal care and experimental procedures were conducted in accordance with the guidelines of **The European Community Council directives 86/609 EEC.**

RESULTS & DISSCUSSION

- In P12 and P15 rats only isolated degenerated neurons were found at interval 24 and 48 hours in the rostral half of the DS.
- In P18, P21 and P24 degeneration of striatal neurons was consistently observed.
- At intervals up to 24 hours post SE, FJB-positive neurons exhibited intense staining of the cell body.
- At longer intervals (48 hours, 1 week) some of positive neurons were shrunken and less intensely stained and surrounded by background of disintegrated fibers.
- Severity of damage reached a peak at 24 and 48 hours post SE.





- Neuronal degeneration within the DS could be explained by the hyperactivity in afferent systems.
- Degenerating neurons in the rostral half of the DS overlap with corticostriatal projections from: parietal cortex, orbital area, MI area, premotor area, and visual association area.
- In the caudal half of the DS overlap with corticostriatal projections from the auditory area with amygdalostriatal projections was evident.

CONCLUSION

SE induced hyperactivity of the corticostriatal and amygdalostriatal glutamatergic projections together with postnatal development of striatal synapses may result in excessive glutamate release and in development of excitotoxic damage of striatal neurons.

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