

**Abstract**

Nicotinic acetylcholine (nACh) receptors evoke convulsive seizures both in nicotine intoxication and human epileptic disorders (e.g., autosomal dominant nocturnal frontal lobe epilepsy [ADNFLE]). Here, we performed behavioral and immunohistochemical studies to elucidate the mechanisms of nicotine-induced seizures. Nicotine at high doses (4mg/kg, i.p.) evoked convulsive seizures, which was antagonized by non-selective (mecamylamine) and  $\alpha 7$ -selective (methyllycaconitine) nACh receptor antagonists. Nicotine-induced seizures were accompanied by significant and region-specific increments in Fos protein expression, a biological marker of neural excitation, in the piriform cortex (Pir), amygdala (AMG), medial habenular nucleus (MHb), thalamus (Th) and solitary tract, suggesting that these regions are potential causative sites for nicotine-induced seizures. Electrical lesioning (1 mA for 15 sec per side) of AMG significantly suppressed nicotine-induced seizures, whereas neither lesioning of the Pir, MHb nor Th affected the nicotine seizure induction. Furthermore, bilateral microinjection of nicotine (100 or 300  $\mu\text{g}/\mu\text{L}/\text{side}$ ) into the AMG effectively evoked convulsive seizure in a dose-dependent manner. The present results strongly suggest that acute nicotine treatment evokes convulsive seizures by activating amygdala neurons, mainly through  $\alpha 7$ nACh receptors.

**PT668****Development of behavioral dysfunction in mice cohabit with brain disordered cage-mate**

Seungmoon Jun<sup>1</sup>, Daejong Jeon<sup>2</sup>, Yong Jeong<sup>1</sup>, Hyunwoo Yang<sup>1</sup>

<sup>1</sup>Korea Advanced Institute of Science & Technology, Republic of Korea,

<sup>2</sup>Seoul National University, Republic of Korea

**Abstract**

People who live with patients with brain disorders are considered as a potential risk group leading to mental disorder. They suffer from a caregiving burden and distressing situation. Consequently, the longer caregiving, the worse their quality of life. In spite of their devoted effort, caregiver's deteriorated state has been overlooked. Here, we attempted to set a long-term housing model reflecting the particular situation. Mice were housed with a conspecific temporal lobe epilepsy model mouse or inescapable foot-shock stress mouse models. After long-term housing, cage-mate was performed behavior tests and electrophysiological investigation. The conspecific cage-mate showed increased anxiety and depression-like behavior. Furthermore, they showed significantly reduced social interaction with juvenile and anesthetized mouse. Behavioral dysfunction of cage-mate sustained four weeks after removing the mouse model. Interestingly, fluoxetine, serotonin selective reuptake inhibitor, hardly restored their behaviors. Our results suggest that a dweller whose cage-mate having brain disorder could develop abnormal behavior including reduced social and increased depression-like behavior. These findings may help to understand psychosocial or psychiatric symptoms frequently observed in at-risk nursing people or caregivers.

**PT669****Investigation of the antiinflammatory and gastric side effects of pregabalin**

Fatma Sultan Kilic<sup>1</sup>, Bilgin Kaygisiz<sup>1</sup>, Sule Aydin<sup>1</sup>, Hadi Karimkhani<sup>2</sup>, Cafer Yildirim<sup>1</sup>, Setenay Oner<sup>3</sup>

<sup>1</sup> Eskisehir Osmangazi University Medical School, Department of Pharmacology <sup>2</sup> Eskisehir Osmangazi University Medical School,

Department of Biochemistry <sup>3</sup> Eskisehir Osmangazi University Medical School, Department of Biostatistics

**Abstract**

**Objectives:** Nonsteroidal antiinflammatory drugs are used for the relief of inflammation, however gastrointestinal side effects restrict their clinical use. We aimed to investigate the antiinflammatory effects of pregabalin, a drug used in epilepsy, anxiety, neuropathic pain treatment, on carrageenan-induced paw edema and to evaluate its gastric side effects in Wistar rats.

**Methods:** Pregabalin 30,50,100mg/kg; indomethacin 5mg/kg(reference drug), vehicle(saline) were injected intraperitoneally before 100 $\mu\text{l}$  of 1% carrageenan administration into the right hind paws of the rats. Paw thickness was measured by a gauge calipers(Vernier Calipers) before (0<sup>th</sup> hour) and in every hour during 6 hours after induction of inflammation. Paw thickness of treated groups were compared with control group with One-way ANOVA. Also paw thickness in 0<sup>th</sup> and 6<sup>th</sup> hours were compared within each group with two-way ANOVA. Pregabalin was administered orally for 10 days to evaluate gastric side effect. At the end of 10 day treatment, rats were sacrificed, gastric tissues were removed out, mucus secretion was determined spectrophotometrically, ulcer index was scored from score 0:(no petechia) to score 3:(petechia>5mm).

**Results:** There was no significant difference between 0<sup>th</sup> and 6<sup>th</sup> hours in paw thickness of all groups, except carrageenan group. Carrageenan significantly increased paw thickness in 6<sup>th</sup> hour compared to 0<sup>th</sup> hour. All doses of pregabalin and indomethacin significantly reduced paw thickness in 6<sup>th</sup> hour compared to carrageenan group. Pregabalin 50 and 100mg/kg similar to indomethacin significantly reduced mucus secretion and increased ulcer index compared to control while pregabalin 30mg/kg did not.

**Conclusion:** All doses of pregabalin exerted antiinflammatory effects comparable to indomethacin, 50 and 100mg/kg pregabalin showed gastric side effects as reduced mucus secretion and ulcer formation similar to indomethacin and 30mg/kg pregabalin may be reasonable dose for antiinflammatory effect without showing gastric side effects.

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**Key words:** pregabalin, carrageenan-induced paw edema, gastric mucus, ulcer index

**PT670****Antiinflammatory and gastric side effect of gabapentin**

Sule Aydin<sup>1</sup>, Basak Donertas<sup>1</sup>, Fatma Sultan Kilic<sup>1</sup>, Setenay Oner<sup>2</sup>, Bilgin Kaygisiz<sup>1</sup>

<sup>1</sup> Eskisehir Osmangazi University Medical School, Department of Pharmacology <sup>2</sup> Eskisehir Osmangazi University Medical School, Department of Biostatistics

**Abstract**

**Objectives:** Nonsteroidal anti-inflammatory drugs are effective in the treatment of inflammation. However, they have been associated with gastrointestinal complications such as gastric ulcer formation. Gabapentin is a drug that is used in the treatment of epilepsy, anxiety, depression and neuropathic pain. We aimed to study the antiinflammatory effects of gabapentin on carrageenan-induced paw edema and to determine its gastric side effects on gastric mucus secretion in Wistar rats.