**GAMMA-BUTYROLACTONE INDUCED ABSENCE EPILEPSY IN WISTAR RATS**

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### Introduction

We studied the resistance to the development of kindling in the chemical model of generalized absence epilepsy induced by gamma-butyrolactone (GBL), a prodrug of gamma-hydroxybutyric acid, excluding the effect of an abnormal genetic background.

### Materials and methods

Three groups of adult wildtype male Wistar rats under anesthesia were implanted with bilateral cortical recording electrodes for the GBL group (GBL) and/or bipolar stimulation electrodes into the right basolateral amygdala for theKindling group (KI) alone and GBL+Kindling group (GBL+KI). Rats in the KI and GBL+KI groups were stimulated twice daily at the afterdischarge threshold until they reached Racine’s stage 5 seizure state. For the GBL+KI group the stimulation was 20 min after intraperitoneal (i.p.) injections of GBL. The GBL rats only received GBL i.p. twice daily over the course of 30 injections.

### Results

The KI animals had stage 5 seizures after 15 stimulations, whereas the GBL+KI rats showed stage 5 seizures after 27 stimulations. The mean numbers of stimulations needed for the development of the first stage 3, 4 or 5 generalized seizures were significantly higher in the GBL+KI group than the KI group.

![Figure 1. Development of kindling in the Gamma-butyrolactone (GBL) + Kindling (KI) and KI groups](image1.png)

By repeated GBL injections, GBL animals displayed spontaneous bilateral synchronous SWDs in the baseline EEG on the Monday morning session after the GBL-free weekend period.

![Figure 2. Mean duration of spontaneous SWDs after 1., 2. and 3. weekend](image2.png)

![Figure 3. Cumulative duration of spontaneous SWDs after 1., 2. ve 3. weekend](image3.png)

### Conclusions

We conclude that the resistance to amydala kindling in the GBL model can be modulated by the absence seizure mechanism alone, without the intervention of an abnormal genetic background.

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