

Context & Objective

- SEEG recording of prefrontal cortex during thalamic centromedian nucleus stimulation in patient with drug resistant epilepsy
- explain frequency dependent effects of thalamic stimulation on • 10 neocortical dysplasia
- To propose a novel neurophysiologically-plausible computational model

SEEG Data

70

Age/ Gender	Condition
34 y.o./ M	 Drug resistant partial epilepsy Left-sided premotor epilepsy close to the frontal eye field Probable focal cortical dysplasia (FCD) located in an abnormal supernumerary sulcus
Ca	se report \circ 5-second biphasic stimulation at 50 Hz,



CM: Centromedian Nucleus PMC: Premotor Cortex

	Suppression of inter-ictal activity					
Stimulation Frequency	Before stimulation	During stimulation	3-seconds post- stimulation	15-seconds post- stimulation	30-seconds post- stimulation	
50 Hz	\times	\times	×	×	×	
70 Hz	X	\checkmark	\checkmark	X	X	

Simulation results

SEEG of Interictal activity: Recorded & Simulated

- Simulated Signal

Real Signal

- During CM stimulation, inhibitory subpopulations are strongly activated
- Frequency-dependent effects:
 - No change at 50 Hz
 - Minimal suppression at 70 Hz
 - Maximum suppression at 100 Hz



100 Hz Х

A novel model integrating neuroplasticity

Thalamic sub-populations

Thalamic cells (TC, Glu)

alamocortical Networl

the

5

nS

ulatior

do

- Reticular nucleus slow IPSP (RtN1, GABA)
- **Reticular nucleus fast IPSP** (RtN2, GABA)

Cortical sub-populations

Pyramidal (Pyr, Glu)

halamic nuclei: Connections, functions and anatomy

Hz and 100 Hz of

the thalamic

 \times

- 4 types of GABAergic cells :
- Parvalbumin positive (PV+)
- Somatostatin positive (SST+)
- Vasopressin positive (VIP+)
- Neuroglia form cells (NGFCs)



The model shows that the "neuroplastic" behaviour of the cortex is strongly determined by the thalamic input

Extrasynaptic inhibition supplements the connections from RtN1/2 to TC, based on the Ο firing of these sub-populations.



- Extra-synaptic GABA release and reuptake dynamics explains the frequency dependent effect of stimulation
- Recovery of epileptic activity in the PFMC is explained by neuroplasticity in the CM

References

- Köksal-Ersöz, E., et al. Journal of Neural Engineering (2022): 19, 055005.
- 2. Pasnicu, A., et al. Epilepsy research (2013): 104.3, 264-268.
- 3. Mina, F., et al. Frontiers in computational neuroscience (2013): 7, 94.

This project has received funding from the European Research Council (ERC) under the European Union's Horizon 2020 research and innovation program (grant agreement No 855109).

