

What is claimed is:

1. A method of analyzing a seizure network, the method comprising:
 - a) delivering a first polynucleotide that encodes a depolarizing light-reactive protein to a target neuron, to express the light-reactive protein in the target neuron;
 - b) administering to the subject an effective amount of light that specifically activates the light-reactive protein thereby leading to optogenetic stimulation; and
 - c) generating an activation map with fMRI.
2. The method of claim 1 further comprising:
 - i) delivering a second polynucleotide that encodes a hyper-polarizing light-reactive protein to the target neuron; and
 - ii) administering to the subject an effective amount of light that specifically activates the second light-reactive protein following step b) of claim 1 thereby inhibiting the afterdischarge generated by the first light-reactive protein.
3. The method of claim 1 or 2 wherein, the fMRI used is cerebral blood volume (CBV)-weighted fMRI.
4. The method of any of claims 1-3, wherein the first polynucleotide is operably linked to a calmodulin-dependent kinase II (CAMKII) or a human synapsin (hSyn) promoter.
5. The method of any of claims 2-4, wherein the second polynucleotide is operably linked to a calmodulin-dependent kinase II (CAMKII) or a human synapsin (hSyn) promoter.
6. The method of any of claims 2-5, wherein the promoter operably linked to the first polynucleotide is different from the second polynucleotide.
7. The method of any of claims 2-6, wherein optogenetic inhibition is applied during optogenetic stimulation.
8. The method of any of claims 1-7, wherein the fMRI is combined with LFP recording.

9. The method of any of claims 1-8, wherein voxel-wise activation time maps are generated, comparing the spread of seizure activity between ADs originating in the dorsal and ventral hippocampus.

10. The method of any of claims 1-9, wherein the seizure map shows where afterdischarge can be suppressed locally for therapeutic intervention.

11. The method of any of claims 1-10, wherein early spiking is demonstrated to be predictive of successful inhibition of seizures.

12. The method of any of claims 1-11, wherein the first polynucleotide encodes a Chr2 protein.

13. The method of any of claims 2-12, wherein the second polynucleotide is an eNpHR3.0 protein.

14. The method of any of claims 1-13, wherein the light-reactive proteins are expressed in the ventral hippocampus.

15. The method of any of claims 1-14, wherein the light-reactive proteins are expressed in the dorsal hippocampus.

16. The method of any of claims 2-15, wherein the light used for optogenetic inhibition is from about 2000mW/mm² to about 3000mW/mm².