What is claimed is:

1. A method of analyzing a seizure network, the method comprising:

generating an activation map with fMRI data, wherein the fMRI data includes data acquired during administration of an effective amount of light that specifically activates a depolarizing light-reactive protein thereby leading to optogenetic stimulation, wherein the depolarizing light-reactive protein is expressed by a target neuron responsive to delivery of a first polynucleotide.

2. The method of claim 1, wherein the fMRI data further includes data acquired during administration of an effective amount of light that specifically activates a second hyper-polarizing light-reactive protein, wherein the second light-reactive protein is to be administered after the first-light reactive protein thereby inhibiting an afterdischarge generated by the first light-reactive protein, wherein the second light-reactive protein is expressed by the target neuron responsive to delivery of a second polynucleotide.

3. The method of claim 1 or 2 wherein, the fMRI data 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 the fMRI data includes data acquired when optogenetic inhibition is applied during optogenetic stimulation.

8. The method of any of claims 1-7, wherein the fMRI data is combined with LFP recording to generate the activation map.

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 configured to be expressed in the ventral hippocampus.

15. The method of any of claims 1-14, wherein the light-reactive proteins are configured to be 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/mm$^2$ to about 3000mW/mm$^2$.

17. Use of a first polynucleotide that encodes a depolarizing light-reactive protein, in the manufacture of a composition for analyzing a seizure network in a subject, wherein the composition is to be delivered to a target neuron in the subject to express the light-reactive protein in the target neuron, and an effective amount of light that specifically activates the light-reactive protein is to be administered to the subject thereby leading to optogenetic stimulation and generation of an activation map with fMRI.