

**Possible role of microglia in mental disorder-related hearing impairment**

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Disrupted processing of auditory information including auditory hallucination is a challenging problem that affects the quality of life of patients with mental disorders. Here we report that microglia, the brain resident immune cells, play a key role in the disruption of auditory information processing in a mouse model of adult temporal lobe epilepsy (TLE). We found that the chemoconvulsant kainic acid (KA)-induced status epilepticus significantly activated microglia in the medial geniculate nucleus (MGN), a brain region which sends ascending projection to the auditory cortex. In the MGN, microglia wrapped the soma of MGN neurons and stripped axosomatic inhibitory synapses to these neurons. The synaptic stripping by microglia decreased the inhibitory synapse density, resulting in the elevation of c-fos expression in neurons of the MGN as well as the auditory cortex. Furthermore, after KA-induced prolonged status epilepticus, mice exhibited deficits in an auditory perception test to discriminate whether or not sound stimuli are presented; mice behaved as if the sound stimuli were presented when the stimuli were not actually presented. These results suggest that mice experienced auditory hallucination after KA-induced status epilepticus.