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Astrocytic gap junctions contribute to potassium redistribution over the neocortex

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Extracellular potassium ion concentration ($[K]_e$) is tightly regulated throughout the brain because it has a major impact on brain functionality. Potassium concentration is disrupted in many brain diseases such as stroke and epilepsy. This project is designed based on a well-developed experimental platform to investigate the effects of extracellular potassium redistribution in physiological states. All experiments were conducted *in vivo* in mouse neocortex. Two double-barreled K-sensitive electrodes coupled with local field potential (LFP) electrodes were placed 4 mm apart. 50 mM KCl solution was injected focally closer to one of the K-LFP electrodes. $[K]_e$ levels and LFP were measured in two different scenarios: pharmacological intervention (gap junction blockage) and optical intervention (optogenetics). Focally increased $[K]_e$ was associated with a transient depolarization which in turn spreads into neighboring tissues so called spreading depolarization. Gap junctional blockade in the peri-injection site simultaneously increased the amplitude and duration of the local $[K]_e$ response, and the local field response was greatly prolonged. While in the remote injection site, $[K]_e$ response was decreased after gap junctional blockade application. Optical stimulation decreased the $[K]_e$ both in the peri-injection and remote site. Our preliminary results are evidence of slow K redistribution (take for minutes) throughout the astrocytic syncytium which is partly mediated via astrocytic gap junctions. Potassium redistribution across a large area of the cortex is not a well-studied area because most studies have limited their focus on focal potassium dynamics. In this project we are addressing this gap using novel tools to elucidate potassium redistribution dynamics.

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