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MEETING ABSTRACT

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Different oxidative phosphorylation patterns in healthy mouse brain regions and alteration of oxidative phosphorylation in the epileptic mouse brain

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Background: Mitochondrial dysfunction is common in neurological diseases. Frequently, a regional specificity in vulnerability can be observed. The aim of this study was to understand how mitochondrial failure in particular brain regions contributes to specific pathological conditions.

Methods: We optimized protocols to study oxidative phosphorylation by means of high-resolution respirometry in defined brain regions of heathly mouse brains. With these methods at hand, we investigated alterations in oxidative phosphorylation during the development of epilepsy. For this purpose, we applied the well-established kainic acid model of mesial temporal lobe epilepsy in mice.

Results: In naïve mouse brains, complex I (CI)-linked respiration was highest in motor cortex. Complex II (CII)-linked respiration was especially high in the striatum. In the kainic acid (KA) model of temporal lobe epilepsy in mice, absolute CI- and CII-linked oxygen consumption as well as electron transport system (ETS) capacity were decreased in the injected dorsal hippocampus 2 days after KA. When normalized to ETS capacity, CII-linked respiration was significantly increased compared to controls. Three weeks after KA injection CII-linked oxygen consumption remained elevated when normalized to ETS capacity.

Discussion: The presented high-resolution respirometry protocols allow detailed analyses of oxidative phosphorylation in small amounts of specific tissues (about 2 mg). This allowed the comparison of different brain tissues implicated in neurological diseases of the healthy mouse and in disease models. We observed marked differences in oxidative phosphorylation patterns in healthy mouse brain regions and present alterations in oxidative phosphorylation in a model of epilepsy.

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