DJ-1 knock-down impairs astrocyte mitochondrial function

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Background
Experimental stress induces DJ-1 translocation from the cytosol to the mitochondria. This may stabilize mitochondrial function.

DJ-1 is overexpressed in reactive glutamatergic Parkinson’s disease (PD) astrocytes, but is unchanged in PD neurons. The DJ-1 expression in astrocytes is thought to be the reason for the cytosolic to mitochondrial translocation. DJ-1’s membrane potential in astrocytes against rotenone (a mitochondrial respiratory chain complex I inhibitor that causes experimental parkinsonism).

DJ-1 knock-down may impair astrocyte mitochondrial functioning as a mechanism through which DJ-1 deficiency impairs astrocyte-mediated neuroprotection.

Methods
DJ-1 knock-down does not alter astrocyte mitochondrial membrane potential

DJ-1 knock-down astrocytes were treated with rotenone then imaged live using a confocal microscope (Olympus IX81) and ImageJ software. Astrocytes were collected. 20 or 40 nM rotenone was added to each rotenone treatment group. The graph represents OCR data normalized to same-well GFAP levels expressed relative to ECAR/GFAP from same-time no rotenone control wells. Mean ± S.E. shown, n=6.

Conclusions
DJ-1 deficiency did not alter mitochondrial respiratory function or glycolytic flux in astrocytes.

DJ-1 deficiency enhanced rotenone-induced depolarization of the mitochondrial membrane potential in astrocytes.

DJ-1 deficiency reduced mitochondrial motility in astrocytes. This was particularly prominent in cellular processes.

DJ-1 deficiency reduced mitochondrial fusion rates in astrocytes. This was particularly prominent with rotenone treatment.

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