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Data Article

Data on effects of rotenone on calcium retention capacity, respiration and activities of respiratory chain complexes I and II in isolated rat brain mitochondria

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ABSTRACT

The data presented in this article are related to the research article entitled "Rotenone decreases ischemia-induced injury by inhibiting mitochondrial permeability transition in mature brains" (Rekuviene et al., 2017) [1]. Data in this article present the direct effects of rotenone on calcium retention capacity (CRC) in isolated normal cortex and cerebellum mitochondria, effects of rotenone intravenous infusion on leak and phosphorylating respiration rates of isolated cortex and cerebellum mitochondria, on activities of respiratory chain complexes I and II in freezed-thawed/sonicated cortex and cerebellum mitochondria after brain ischemia. In addition, detailed experimental procedures of isolation of brain mitochondria, measurements of CRC, respiration, activities of respiratory chain complexes and H_2O_2 generation in cortex and cerebellum mitochondria are described.

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Specifications Table

Subject area	*Biology*
More specific subject area	*Biology of brain disorders*
Type of data	*Figures, Table*
How data was acquired	*CRC was determined fluorimetrically (Fluorescence Spectrometer Perkin Elmer LS55).* *Mitochondrial respiration was measured with high-resolution respirometry OROBOROS Oxygraph-2 k (Oroboros Instruments, Innsbruck, Austria).* *Respiratory chain complex I and complex II activities were determined spectrophotometrically (Nanophotometer).*
Data format	*Analyzed*
Experimental factors	*CRC of isolated normal brain mitochondria was measured with directly added rotenone (50–1000 nM), brain mitochondrial respiration and activities of respiratory chain complexes I and II were measured after infusion of single rotenone dose (0.01 mg/kg) into the tail vein of rats and the exposure of 120 min brain ischemia.*
Experimental features	*Fluorimetric determination of mitochondrial CRC with Calcium Green-5N, spectrophotometric determination of activities of respiratory chain complexes I (by NADH oxidation) and II (by 2,6-dichlorophenolindophenol reduction) in freezed-thawed/sonicated mitochondria, respirometric measurements (oxygen consumption rate) of isolated brain mitochondria.*
Data source location	*Kaunas, Lithuania*
Data accessibility	*The data are available with this article*

Value of the data

- Data from this research highlights the effects of rotenone on rat brain mitochondrial functions.
- The direct effects of rotenone (50–1000 nM) on CRC of isolated normal brain cortex and cerebellar mitochondria were measured.
- The effects of the intravenous infusion of rotenone (0.01 mg/kg) on leak and phosphorylating respiration of isolated cortex and cerebellum mitochondria as well as on activities of mitochondrial respiratory chain complexes I and II after 120 min brain ischemia were measured.
- These data may be relevant for (i) other researchers using various doses of rotenone in their experiments with mitochondria; (ii) research that focuses on the mitochondrial respiratory chain complex I inhibition during brain ischemia.

1. Data

The data reported include direct effects of rotenone (50–1000 nM) on calcium retention capacity (CRC) in isolated control cortex and cerebellum mitochondria (Fig. 1). We also measured the effects of intravenous rotenone infusion (0.01 mg/kg) on respiration rates (leak and phosphorylating) of isolated normal and 120 min ischemia damaged cortex and cerebellum mitochondria respiring with substrates pyruvate/malate and succinate (Table 1) as well as original recording of respirometric curve of mitochondria is presented (Fig. 2). Effects of rotenone intravenous infusion (0.01 mg/kg) on complex I (Fig. 3) and complex II (Fig. 4) activities of mitochondria isolated from control rat cortex and cerebellum and after 120 min brain ischemia are shown. The detailed experimental procedures of