Raloxifene attenuates oxidative stress and preserves mitochondrial function in astrocytic cells upon glucose deprivation

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Oxidative stress and mitochondrial dysfunction induced by metabolic insults are both hallmarks of various neurological disorders, whereby neuronal cells are severely affected by decreased glucose supply to the brain. Likely injured, astrocytes are important for neuronal homeostasis and therapeutic strategies should be directed towards improving astrocytic functions to improve brain's outcome. In the present study, we aimed to assess the actions of raloxifene, a selective estrogen receptor modulator in astrocytic cells under glucose deprivation. Our findings indicated that pretreatment with 1 μM raloxifene results in an increase in cell viability and attenuated nuclei fragmentation. Raloxifene's actions also rely on the reduction of oxidative stress and preservation of mitochondrial function in glucose-deprived astrocytic cells, suggesting the possible direct effects of this compound on mitochondria. In conclusion, our results demonstrate that raloxifene's protective actions might be mediated in part by astrocytes in the setting of a metabolic insult.

astrocytes

glucose deprivation

mitochondria

neuroprotection

raloxifene

cardiolipin

glucose

hydrogen peroxide

raloxifene

reactive oxygen metabolite

superoxide

cardiolipin

glucose

neuroprotective agent

protective agent

raloxifene

reactive oxygen metabolite

selective estrogen receptor modulator

Article

astrocyte

astrocyte cell line

brain mitochondrion

cell nucleus

cell survival

cell viability

concentration response

mitochondrial membrane potential

MTT assay

oxidative stress

priority journal

astrocyte

cell line

cytology

drug effect

human

metabolism

mitochondrion

oxidative stress

Astrocytes

Cardiolipins

Cell Line

Cell Survival

Glucose

Humans

Membrane Potential, Mitochondrial

Mitochondria

Neuroprotective Agents

Oxidative Stress

Protective Agents

Raloxifene Hydrochloride

Reactive Oxygen Species

Selective Estrogen Receptor Modulators