**Introduction:** The intracerebral hemorrhage (ICH) represents one of most severe events in the CNS with poor prognosis and outcome. ICH is caused by the extravasation of blood into the brain parenchyma as a result of blood brain barrier disruption. Blood derived products (BDPs) such as hemoglobin, heme and iron, are powerful toxic components which are released and metabolized in cerebral parenchyma after the ICH onset. Here, we investigated the effects of iron on energy and redox metabolism in U-87 glioblastoma cells. **Material and methods:** U-87 cells were cultured for 24 hours in the presence of iron (up to 50 mM) and several cellular and biochemical parameters were evaluated. Cell viability was assessed by the MTT reduction assay. Redox imbalance was determined by measuring the TBARS levels, whereas mitochondrial physiology assessment was carried out by measuring the oxygen fluxes on intact cells in glucose containing medium by high-resolution respirometry (O2k). Citrate synthase activity was determined spectrophotometrically. **Results and discussion:** Iron promoted lipid peroxidation without affecting cell viability. Mitochondrial content, determined by the activity of citrate synthase, was also not affected by iron up to 50 µM. High-resolution respirometry analyses showed that iron caused an overall reduction of the oxygen consumption rates, regardless the mitochondrial metabolic states. **Conclusion:** Iron triggered a signaling cascade that promote functional mitochondrial remodeling in a glioblastoma cell line, which may represent a cellular survival mechanism in response to the BDPs during the ICH events.

Keywords: mitochondria, oxidative stress, stroke

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