Acute Effects of Cocaine on the Respiratory Function of Mitochondria in the Brain

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Mitochondria are the primary source of energy within the cell, producing over 90% of ATP and performing essential functions, ranging from maintenance of Ca2+ homeostasis and the regulation of mitochondrial metabolism. Mitochondria are involved in the regulation of neuronal activity (action potential, internal transport, EPSP, IPSP) and play an important role in cerebral function. Mitochondrial health is maintained by mitochondrial quality control through mitochondrial biogenesis, dynamics (fusion and fission) and mitophagy. Cocaine is a highly addictive psychostimulant that enhances alertness, attention, and energy. This study evaluated the effects of cocaine on brain mitochondria. Tissue that was expose to cocaine was obtained and divided into two groups (control and cocaine-treated). Those tissues were previously treated by another researcher (20 mg/kg, i.p. daily for three consecutive days) before the beginning of the study. Brain tissue was homogenized, and mitochondria were isolated by differential centrifugation. Mitochondrial respiration rates were measured with a Clark electrode connected to an oxygraph. In addition, SDS PAGE followed by western blotting was performed to analyze the expression of mitochondrial fusion and fission proteins. Results showed that tissue treated with cocaine had a lower mitochondrial respiratory function than control tissue. Significant changes in the expression of mitochondrial fusion and fission proteins, such as MFN-2, OPA-1, and Fis-1, were also observed. We concluded that cocaine has a negative effect, decreasing the respiratory function of the mitochondria. Cocaine reduces the expression of mitochondrial fusion and fission proteins, Drp-1, Fis1, Opa-1, and Mfn1, and increases phosphorylation of Drp1.

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