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Reactive oxygen species induced oxidative stress, neuronal apoptosis and alternative death pathways

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ABSTRACT
Reactive oxygen species (ROS) are produced as a byproduct of cellular metabolic pathways and function as a critical second messenger in a variety of intracellular signaling pathways. The excessive intracellular generation of ROS on the other renders a cell oxidatively stressed. This involvement of ROS in numerous diseases has been documented and at different phases of the apoptotic pathway such as induction of mitochondrial permeability transition and release of mitochondrial death amplification factors, activetion of intracellular caspases and DNA damage has been clearly established. Cell death by apoptosis is a part of normal development and maintenance of tissue homeostasis. Polychlorinated biphenyls, one of the environmental pollutants which are widely used in electrical industries and lipophilic and resistant to biological decomposition accumulate through food chain. They are developmental neurotoxicants which induce neuronal apoptosis. Our studies proved that oxidative stress is induced promoting LPO and a decrease in all the antioxidant enzymes in testis, epididymis, ventral prostate, seminal vesicles, liver, kidney and brain regions. Neuronal damages were observed in all the brain regions after PCB exposure. PCB increased caspase8 mRNA/protein expression in hippocampus of adult rats. This upregulation results in Fas-FasL mediated induction of hippocampal apoptosis. Performin/granzyme induced apoptosis is the main pathway used by cytotoxic lymphocytes to eliminate virus-infected or transformed cells. The production of ROS is greatly increased during reperfusion phase when oxygen becomes available and the mitochondrial respiratory chain is impaired. Furthermore, this is exacerbated by reduced antioxidant defenses.

KEYWORDS
Apoptosis; Caspases; Cell Death; Mitochondria; PCBs; Reactive Oxygen Species

Cite this paper

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