Polychlorinated Biphenyls Induce Mitochondrial Dysfunction in SH-SY5Y Neuroblastoma Cells

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Chronic exposure to polychlorinated biphenyls (PCBs), ubiquitous environmental contaminants, can adversely affect the development and function of the nervous system. Here we evaluated the effect of PCB exposure on mitochondrial function using the PCB mixture Aroclor-1254 (A1254) in SH-SY5Y neuroblastoma cells. A 6-hour exposure to A1254 (5 μg/ml) reduced cellular ATP production by 45%±7, and mitochondrial membrane potential, detected by TMRE, by 49%±7. Consistently, A1254 significantly decreased oxidative phosphorylation and aerobic glycolysis measured by extracellular flux analyzer. Furthermore, the activity of mitochondrial protein complexes I, II, and IV, but not V (ATPase), measured by BN-PAGE technique, was significantly reduced after 6-hour exposure to A1254. The addition of pyruvic acid during exposure to A1254 significantly prevent A1254-induced cell injury, restoring resting mitochondrial membrane potential, ATP levels, oxidative phosphorylation and aerobic glycolysis. Furthermore, pyruvic acid significantly preserved the activity of mitochondrial complexes I, II and IV and increased basal activity of complexes V. Collectively, the present results indicate that the neurotoxicity of A1254 depends on the impairment of oxidative phosphorylation, aerobic glycolysis, and mitochondrial complexes I, II, and IV activity and it was counteracted by pyruvic acid.