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Oxidative Stress-Mediated Apoptosis in Neuronal SK-N-MC cells After Stimulation with 2,2′,5,5′-Tetrachlorobiphenyl

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Polychlorinated biphenyls (PCBs) are widespread environmental contaminants and have become distributed throughout the entire ecosystem. Human populations have been exposed to these chemicals by both chronic and acute exposures. Apoptosis is a programmed cell death mechanism to control cell number in tissue and to eliminate individual cells that may lead to disease states. The present study was designed to investigate whether 2,2′,5,5′-Tetrachlorobiphenyl increase the production of active oxidants, and subsequently promotes apoptosis of neuronal SK-N-MC cells. Upon treatments with 2,2′,5,5′-Tetrachlorobiphenyl, the time- and concentration-dependent inhibition of cell viability were observed. The capability of 2,2′,5,5′-Tetrachlorobiphenyl to induce apoptosis was associated with proteolytic cleavage of specific target proteins such as poly(ADP-ribose) polymerase and beta-catenin proteins suggesting the possible involvement of caspases. This study examined the association of PCB-induced apoptosis with the modulation of biomakers of oxidative damage to lipids (malondialdehyde [MDA]) in SK-N-MC cells treated with 2,2,5,5-Tetrachlorobiphenyl (5, 10, 15, 20μg/ml) for 12 and 24hrs. Increased MDA was observed in cytosol treated with 10, 15, 20μg/ml for 12 hrs and in media treated with 10, 15, 20μg/ml for 24 hrs. The activities of antioxidant enzymes, catalase, CuZn-Superoxide Dismutase, and Glutathione S-Transferase, were also examined.