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Assessing the Gene Expression Changes in Oxidative Stress Induced By BDE-99

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Extended Abstract

2,2,4,4,5-Pentabromo diphenyl ether (BDE-99), one of the polybrominated diphenyl ethers (PBDE) species are a commercially available chemical substance used as a flame retardant on a large scale. The use of BDE-99 on such a large scale provides further investigation of its effects on live mechanism due to this pollutant may be exposed to seafood, air, water, household wastes and household dust, as well as the most important exposure route is nutrition. Therefore we aim to investigate the gene expression changes of apoptotic and acute oxidative enzyme induced by BDE-99 in rat liver by qRT-PCR.

Wistar albino rats, 250–300 g, were randomly divided into one control and two experimental groups (n=8 for each). 0,05 mg/kg (dose I) and 0,1 mg/kg (dose II) BDE-99 (in corn oil) were administrated by gavage. 10 µm thick sections of paraffin embedded liver tissue samples from harvested after ten days were used for total RNA isolation. The High Pure FFPET RNA Isolation Kit was used following the manufacturer's recommendations. Complementary DNA (cDNA) generated from extracted RNA samples using High-Capacity cDNA Reverse Transcription kit and random primers according to the kit protocol and 2µL of the reverse transcription reaction was subjected to Quantitative real-time PCR (qRT-PCR) amplification as a template. The cycling program consisted of an initial denaturation at 95°C for 10 min, followed by 50 cycles of 95°C for 15 s, 60°C for 1 min, 60°C for 1 min for all genes. Expression quantities of target genes were normalized using GAPDH as an internal gene. Changes in relative expression levels between control and experimental groups were evaluated for statistical significance according to Student-t test. The results were considered statistically significant in $p<0.05$.

SOD expression levels increased significantly in dose I (2,2 fold) and II (2,91 fold) exposure groups but CAT and GSH levels only increased in dose II (1,9 and 2,08 fold respectively). Changes in SOD, CAT and GSH levels indicate that oxidative stress is induced by BDE-99. mRNA levels of HSP70 (2,08 and 6,04 fold), Cas-9 (12,34 and 20,28), Lamin (1,85 and 5,03 fold) were induced but NuMA levels remained in dose I and II. The increase in HSP70 level, an indicator of cellular stress, also parallels with oxidative stress parameters. Apaf-1 expression level was only increased in dose I (2,54 fold). The changes in expressions of Cas-9 and Apaf-I are also might consider as an evidence of BDE-99-induced cell apoptosis. Besides, it was determined that liver cells responded against nuclear stress by increasing lamin expression. In conclusion, we suggested that BDE-99 cause oxidative stress which leads to cellular apoptosis via increasing the expression levels of apoptotic genes in liver in rat.