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How Do Chlorpyrifos Affect Programmed Cell Death in Rat Kidney?

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Extended Abstract

Pesticides are widely use in modern agriculture. The use of organophosphate pesticides including chlorpyrifos (CRP) has increased significantly over time leading to an inevitable increase in environmental contamination. Common usages of CRP conduce to public concern due to pollution and toxicity problems, mainly due to its easy transmission to humans through the food chain. In this study we aimed to explain the toxicity of CRP in rat kidney.

Wistar albino rats, 200–250 g, were randomly divided into two groups (n=8 for each). 4.05 mg/kg (LD 50/10) CPF was administrated to animals by gavage. Rats were sacrificed after ten days. Total RNA was isolated from paraffin embedded kidney tissue samples (10 µm thick sections) using The High Pure FFPET RNA Isolation Kit as recommended by manufacturer. 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 amplification as a template. Amplifications of PCR product were monitored via SYBR Green I dye which is an intercalator-based method. 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 experimental groups and control group were assessed for statistical significance according to Student-t test. The results were considered statistically significant in $p<0.05$.

qRT-PCR analysis were demonstrated that mRNA levels of caspase-3, caspase-9, Apaf-1, topoisomerase, vimentin, lamin, NuMA were significantly increased but no significant change was determined in mRNA levels of PCNA and p53 ($p<0.05$). Induced gene expressions of caspase-3, caspase-9 and Apaf-1 and also no change of p53 gene expression demonstrate that CRP could induce apoptosis in kidney via mitochondrial pathways. Over expression of caspase-3 and caspase-9 caused an increase in mRNA levels of NuMA and lamin which can be considered as the adaptive response of nucleus against CRP toxicity. In addition, significant increases in topoisomerase and vimentin expression might be considered as an evidence of a degenerative stress on DNA. Therefore, the remarkable toxicity issues were based on CRP usage was concluded that it is necessary to investigate, monitor and minimize the effects.