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# Camphorquinone induced cytotoxicity, cell cycle alterations, apoptosis-related gene and protein expression to human dental pulp cells

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**Introduction:** Camphorquinone (CQ) is popularly-used as photosensitizer in resin composites and dentin bonding agents (DBA). The purpose of this study is to investigate the influences of CQ on cytotoxicity to human dental pulp cells. Then, its effects on the expression of cell cycle and apoptosis related genes and proteins are evaluated. Besides, the relationship between ROS formation and its toxicity is also observed.

**Materials and methods:** Human dental pulp cells were treated with different concentrations of CQ (0.1 to 2 mM). Cell morphology was observed under a phase contrast microscope. Cell proliferation was evaluated by MTT assay. Cell cycle analysis, cell death pattern and ROS formation were investigated by flow cytometry. Changes in mRNA expression were determined by reverse-transcription polymerase chain reaction (RT-PCR). Changes in protein production were evaluated by western blot. In some experiments, cells were pre-treated for 30 min with NAC (1 mM), catalase (2000 U/ml) or hemeoxygenase-1 (HO-1) inhibitor ZnPP (2.5 and 5 μM) before co-incubation with 2 mM CQ. Then, MTT assay was used to investigate the changes of cell viability. One-way ANOVA and post hoc Tukey test was used to analyze differences between experimental and control groups.

**Results:** CQ induced morphological changes and a significant decrease of cell viability, to about 70% and 50% respectively, at the concentrations of 1 mM and 2 mM. At these concentrations CQ led to $G_2$/M cell cycle arrest. The expression of cdc2, cyclin B, p-cdc2 and cdc25C was inhibited by CQ. 1 mM CQ caused an increase of apoptotic cells, and at the concentration of 2 mM, obvious increases of apoptotic, necrotic as well as apoptotic/necrotic cells were observed. Besides, exposure to CQ higher than 0.5 mM for 3 hours caused a dose-dependent increase of ROS, and an increase of HO-1 expression was noted after 24 hours. The reduction of cell viability caused by CQ can be inhibited by NAC or catalase pre-treatment, and can be promoted by 5 μM ZnPP pre-treatment.

**Conclusions:** CQ may cause morphological changes, reduction of cell viability, $G_2$/M phase cell cycle arrest and cell death, especially apoptosis of dental pulp cells at relevant concentrations. These changes may be related to ROS formation, which then cause expressional variations of many genes, such as cdc2, cyclin B, cdc25C, p21 and HO-1. These results of CQ toxicity may partly explain the pulpal response to dentin bonding agents and composite resin filling when remaining dentin thickness is scarce. (This study is supported by National Science Council, Taipei, Taiwan, ROC and National Taiwan University Hospital)

## Biography

Dr. Jiiang-Huei Jeng is a specialist in both Endodontics and Periodontics. He got IADR Hatton Award and his Doctor of Dental Surgery degree in 1994. Dr Jiiang-Huei Jeng practiced endodontic, periodontic, and implant dentistry in the National Taiwan University Hospital since 1991 and the chair in Department of Endodontics. He is also a professor, researcher and teaching staff in the National Taiwan University Dental School. He has published many papers in scientific journals including Biomaterials, Acta Biomaterialia, J Endod, Int Endod J, Carcinogenesis, J Periodontol etc. He received International Association of Dental Research (IADR) distinguish scientist award of PTT group in Brazil (2012). He also served as a reviewer and editorial board member of many journals.

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