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THE EFFECTS OF TCDD ON THE TRANSFORMATION AND MICRONUCLEUS FORMATION IN CHANG LIVER, HACAT AND MCF10A CELLS

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2,3,7,8-Tetrachlorodibenzo-*p*-dioxin(TCDD), a prototype of many halogenated aromatic hydrocarbons, is a ubiquitous, persistent environmental contaminant and the most powerful carcinogen categorized by IARC. Despite extensive research, the mechanisms of TCDD-induced carcinogenesis are poorly understood, and its carcinogenic potential in human is not clear. Although TCDD is a powerful carcinogen in several species, limited model system exist to study carcinogenicity of this compound at cellular level.

This study was performed to investigate the neoplastic transformation of human cells in culture with exposure to TCDD. Micronucleus(MN) formation was chosen as index of genetic toxicity. This study used human keratinocyte HaCaT cell, human normal liver Chang liver cell, and human normal breast MCF10A cell. TCDD did not affect on the cell viability of the Chang liver, HaCaT, MCF10A cells treated with 0.1~1000nM. And we observed transformed candidate *foci* in Chang liver cell exposed to 1000nM TCDD for 2 weeks. In HaCaT cell, transformed candidate *foci* were also observed after TCDD(0.1, 1, 10, 100 nM) treatment for 2 weeks. The frequency of micronucleus was increased after treatment of TCDD for 24hr in Chang liver and HaCaT cells. These results indicate that the ability of TCDD to induce numerical chromosomal abnormality may be involved in Chang liver and HaCaT cells transformation. Our TCDD-transformed candidates of Chang liver cells are expected to provide a clue to the elucidation of TCDD-induced transformation pathway as well as acquisition of TCDD resistency through cDNA microarray technique.