



MASTER'S THESIS

Environmental fingerprints and biological effects of dioxins, with reference to the electronic waste recycling site at Guiyu

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Date of Award: 2008

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Environmental Fingerprints and Biological Effects of Dioxins, with Reference to the Electronic Waste Recycling Site at Guiyu

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A thesis submitted in partial fulfillment of the requirements for the degree of Master of Philosophy

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August 2008

ABSTRACT

In the 21st century, technological advancement accelerates the turnover rate of most electronic equipments (i.e. computers, printers, televisions and mobiles). The disposal, recycling and part salvaging of discarded electronic devices impose adverse health effects to the environment and animal health. The negative impacts are basically attributed by the open burning of the electronic wastes (e-wastes) and the subsequent leaking of persistent toxic pollutants to the ecosystem. China is one of the countries that contribute to the industry of e-wastes recycling. Ewastes contaminated village called Guiyu in ShanTou where was our targeted study area. Soil and dust samples were collected from different sites at Guiyu. The samples were extracted, concentrated and purified for H4IIE-EROD bioassay. All these samples could induce EROD activities in the bioassay with the calculated biological TEQ values ranging of 0.42 pg/ml - 4.32 pg/ml. An anthropogenic compound – 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) is a prototype dioxin, that can cause a large number of apparently unrelated biological effects. It has no industrial or commercial use and was produced as inadvertent trace contaminants in e-wastes recycling process. Epidemiological studies in accidentally exposed populations have established a link between high doses of TCDD and certain types of cancer and cardiovascular disease. At molecular level, most of the effects of TCDD exposure have been known for many years to result from the activation of aryl hydrocarbon receptor (AhR), a ligand-activated transcription factor. AhR dimerization with ARNT is responsible for the up-regulation of Ah gene battery, which comprises several well-characterized genes in the cytochrome P450 CYP1A family and several Phase II detoxification genes. Other possible pathways including the modulation of signaling molecules, like pAkt, mdm2, p53, pStat3 have been reported. Our results indicated that in addition to AhR pathway in human hepatocellular carcinoma (HepG2) and colorectal adenocarcinoma (Caco-2). TCDD could activate other various signaling molecule in the cells. The activation of these pathways might affect some basic cell functions, such as cell differentiation and proliferation. Oral uptake is the major exposure route to TCDD. To determine and simulate the biological effects of TCDD through dietary intake, HepG2 and Caco-2 the human liver and colon cell-lines respectively, were used in

the present study. The biological activities of TCDD can be revealed by the induction of CYP1A1 expression. In this study, four potent AhR antagonists: resveratrol (Rev), 7-ketocholesterol (7-KC), α -naphthoflavone (α -NF), or salicylamide (Sal) was added to TCDD treated HepG2 or Caco-2 cells. Our results indicated that the antagonists may be used as antidotes against TCDD-elicited toxicities.

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