Dioxin and human health

DIOXIN AND HUMAN HEALTH Introduction The major pollutants of the environment are as a result of unwanted by-products of thermal processes, ly polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/PCDF). The paper industry, chlorine industry, herbicide industry, car-trafficking, melting processes yield PCDD/PCDF. Tetrachlorodi bezodioxin (TCCD) is a by-product of herbicide 2, 4-dichloroacetophenol (2, 4D) and 2, 4, 5-trichloroacetophenol (2, 4, 5,- T). The toxicity of TCCD is known since 1950. It was first identified in Japan in 1979. Burning of trash in an incinerator from chlorinated materials and aromatic carbohydrates results in the production of dioxins. The producibility is relatively high in presence of a catalyst like iron. Chemistry of Dioxins Para-dibenzodioxins are formed when two oxygens combines two benzenes (or chlorinated benzenes). The number of isomers is several as both could have upto 8 chlorines – 75 for PCCDs and 135 for PCDFs. These compounds are called as congeners. Toxicity is expressed as Toxic Equivalent Factor (TEF) and toxicity is based on 2, 3, 7, 8 TCCD which is expressed as TEF. The toxicity is caused because of the four chlorine atoms at positions 2, 3, 7, 8 on the dioxin molecules. The toxicity is expressed as pg TEQ/g lipid (ppt) and is summed up based upon their toxicity equivalent concentrations. OCCD is the most abundant isomer related to human contamination followed by the 2, 3, 7, 8 substituted hepta and hexa-chloro congeners. 2, 3, 7, 8 TCCD is normally less abundant than PCCD. The activation of Ah receptor determines the TEF. The oxygen bond between the two benzenes could be substituted to sulphur or azide. Chlorine could be replaced by bromine. Effect of Dioxins on Human Health Eating, breathing, and polluted air and skin contact with contaminated soil and materials are the major routes for human exposure. The PCCDs reach the

blood stream after passing from the gastrointestinal tract and eating food containing the PCCDs. Breathing of polluted air could lead to the concentration of PCCDs in the lungs. Half-life of 2, 3, 7, 8 TCCDs in the body is variable and may take 7-21 years. Dioxin binds to the Ah receptor in the cytoplasm and the complex moves into the nucleus, where it leads to gene activation (Watnabe 1). The critical effects are of TCCD is observed on the reproductive, developmental and endocrine systems. Exposure to humans is through milk, milk-products, diary products fish and meat. The effects include enzyme induction, immunotoxicity and developmental effects. No specific cancer appears to predominates, but there is a risk for specific cancers, which include lymphoma, multiple myeloma, soft tissue carcinoma, lung and liver cancer, breast cancer, testicular cancer, endometrium (Kogevinas, 33). Endocrine System There is a an alteration in a hormonal metabolism through the induction of the cytochrome CYP dependent enzymes. Thyroid dysfunction has been associated with dioxin exposure. Decreased testosterone and increased gonadotrophin concentrations were found with high TCCD concentrations. Dioxin exposure is more associated with women. There is a high mean glucose concentration with TCCD exposed subjects leading to diabetes. Oral medication is used to control diabetes, with increasing exposure to TCCD, whereas, the time to diabetes onset decreased with dioxin exposure. . Neurological Effects Neurological effects were observed with dioxin exposure. Subjective signs such as lassitude, weaknesss of lower limbs, muscular pains, sleepiness, loss of appetite, increased perspiration, mental and sexual disorders have been reported. A study also reported depression, hypochondria, hysteria, and schizophrenia. Adverse effects have also been observed in prenatal children, particularly on

neurodevelopment and behavior of children. Immunological Effects In utero and lactational exposure are a sensitive period for immunotoxic effects. Thymic atrophy is a consistent finding. In vitro studies using peripheral blood lymphocytes showed a decrease in B-cells, CD4+ (helper T-cells) and relative increase in CD8+ (suppressor T cells). These changes in T-lymphocyte populations could persists in later child or adulthood and could result in allergy, autoimmunity, and immunosuppression. Other Effects Upper respiratory tract infections and appendicitis are also reported. PCCDs are able to pass the placental barrier during pregnancy. This results in the exposure of embryo and fetus during a critical period of organ growth and development. The breast-fed infants are exposed to high level of these contaminants. Subacute exposure of TCCD led to hepatic toxicity. Hypercholestelonemia, hyperlipemia and hyperphospholipidemia were observed. Gastrointesinal effects, such as peptic ulcers were also reported. Porphyric cutanea tarda, hypertrichosis, hirsutism and hyperpigmentation were also observed in workers exposed to TCCD. Exposure to high concentrations of dioxin has been associated with dental defects. Normal breast-fed children have shown hypomineralized enamel defects which is also associated with background concentrations of dioxins Conclusion Exposed workers are subjected to high risk to cancer and ischaemic heart disease. The other sensitive organs are the endocrine and reproductive systems. Few of these effects have been evaluated by epidemiological studies. Though, data suggests that dioxin levels in human tissues have decreased. Survey of municipal incinerators revealed 1% incinerators emit dioxin above allowable emission levcls. Holistic environmental control and regular check-up of residents around the incinerators could lead to

decreased exposure of dioxin. Bibliography: 1. Watnabe, Shaw., Kimi Yoshi Kitamura, and Matsahito Nagahashi. “ Effects of Dioxins on Human Health: A Review”. J. Epidemiol. 9 (1999): 1-13. 2. Kogevinas , M. “ Human Health Effects of Dioxin : Cancer, Reproductive, and Endocrine System Effects”. Human Reproduction Update. 7 (2001) : 331-339.