HEALTH EFFECTS OF CHRONIC EXPOSURE OF MUNICIPAL WASTE INCINERATOR WORKERS TO PCDD, PCDF, AND CO-PCB.

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Introduction

A national survey of polychlorinated dibenzo-p-dioxins (PCDD) and dibenzofurans (PCDF) in emission gases from municipal waste incinerators in 1997 revealed that the Nose Bika Center was heavily contaminated by PCDF. Dioxin contamination of the southern area adjacent to the incinerator was 8,500 pg I-TEQ/g soil, and the bottom sludge of a water adjustment pond yielded values of 23,000 TEQ/g dry soil. Inside the building, PCDD/PCDF contamination of floating sand in the fluidized bed was 1.0-1.1 ng I-TEQ/g sand, 320 ng I-TEQ/g in fly ash in the electrostatic precipitator, 1,500 ng I-TEQ/g in remnants in the ash treating machine to make cement grains, and 120,000 ng I-TEQ/g in piles in the chimney bottom. The tank for water spraying was the most heavily contaminated site, and contained 3,000,000 ng I-TEQ/L in the water and 96,000 ng I-TEQ/g in the bottom sludge.

These unbelievably high levels of contamination in the furnace building prompted us to investigate the health status of the workers, including measurement of their blood dioxin levels, with support from the Ministry of Labor.

Subjects and Methods

Ninety-four workers underwent a physical examination, and blood biochemistry, lymphocyte marker, and NK activity studies were carried out, along with blood dioxin measurements (1). Information on working history, life-style, and dietary habits was obtained by questionnaire and interview. The subjects described their work history in detail, and underwent a dermatological investigation. Height, body weight, and blood pressure were measured. Life habits and dietary habits were collected by questionnaire and checked by trained dieticians. The questionnaire included dietary habits, smoking and drinking habits, residential and work environment, physical activity, past history of diseases and treatments, reproductive history, etc.

Blood was collected into a transfusion bag (200 ml) containing heparin sodium solution (SH-207-Terumo, Japan). About 30 ml of blood was divided to tubes to perform peripheral blood tests, such as RBC, WBC, and platelet counts, and hematocrit, and blood chemistry studies, such as determination of GOT, GPT, gamma-GTP, LDH, ALP, LAP, CPK, amylase, total cholesterol, HDL-cholesterol, triacylglycerol, total protein, albumin, total bilirubin, blood urea nitrogen, creatinine, uric acid, glucose, creatine phosphokinase, sodium, potassium, calcium, iron, and inorganic phosphate.

As immunological markers, T lymphocytes subsets determined by surface antigens, such as CD3, CD4, CD8, and CD4/CD8 ratio, were measured. NK activity was measured by surface antigen (CD56), and natural killer cell activity was determined against K562 cells. Stimulation by PHA and Con A was also applied.

Blood PCDD/PCDF/Co-PCB was measured by a modification of Patterson's method (2). After precise measurement of purified lipids, the samples were sent to Oekometric Bayreuth Institute for Environmental Research (Bayreuth, Germany) to measure dioxins.

Analysis of PCDD/PCDF/Co-PCB was carried out by gas chromatography-high resolution mass spectrometry (GC-MS): Varian-3400 series unit (Hewlett-Packard, Palo Alto, California) equipped with a Finnigan MAT-90 (Finnigan MAT GmbH, Bremen, Germany).

Results and Discussion

The blood dioxin levels were as follows. The median I-TEQ of dioxins was 39.7 pg I-TEQ/g lipid, and the range was 13.3 to 831.19. The median 2,3,7,8-TCDD concentration was 3.9 pg I-TEQ/g lipid, and the range was <1 pg TEQ/g lipid (one case) to 13.4 pg I-TEQ/g lipid. The median I-TEQ of coplanar PCB was 10.8 pg I-TEQ/g lipid, and the range was 3.1 to 54.2 pg I-TEQ/g lipid. The congener-specific distribution was quite similar to that in soil around incinerator and waste in the factory (Fig. 1). The relationship between dioxin concentrations and work history in the factory showed that the fluidized incinerator and fly ash treatment areas were high-risk work areas.

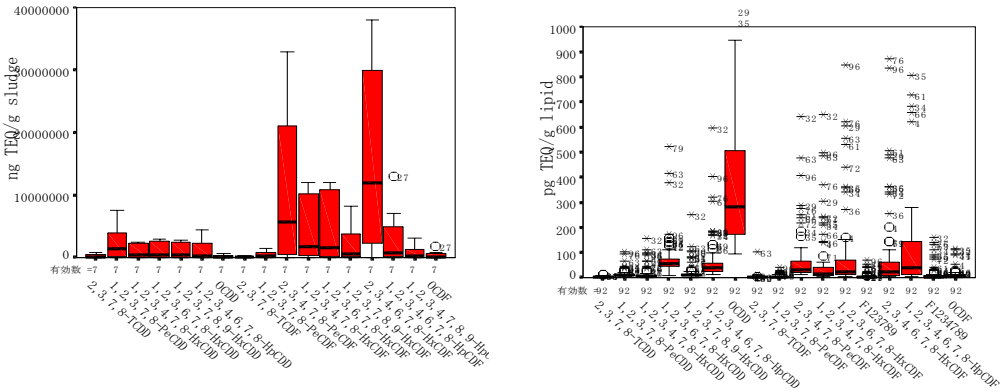

Fig. 1. Congener distribution in sludge inside the incinerator (left) and of blood (right).

Increased level of 2,3,4,7,8-PeCDF, 2,3,4,6,7,8-HxCDF and other furans in contaminated samples reflected the level of blood. Box-plot graph. High individuals are marked by *with id number.

Workers other than at high risk areas did not show remarkably high PCDFs value. Total average amount of PCDD and PCDF in blood was 612.89 pg and 661:97 pg/g lipid, respectively.

Dermatological examinations yielded acne vulgaris in 8 subjects, in which 3 cases may require differential diagnosis from chloracne. One subject had numerous skin pigmentation and eruption-like senile flecks and seborrheic keratosis on his face and upper extremities, and brown, bean-sized

spotty pigmentation on both legs. The dioxin levels of these subjects were 300 to 831 pg I-TEQ/g lipid. The average blood TCDD level of the subjects with acne vulgaris was 13.4 pg/g fat, in contrast to those in Seveso (lowest patient 828 pg/g lipid). In the BASF accident, no chloracne group showed blood TCDD level of 148 pg/g lipid (20-1,279 pg/g lipid), and the level in the severe chloracne group was 1,118 pg/g lipid (493-2,955 pg/g lipid). If skin lesions in this study were chloracne, the level of dioxins was rather low.

Correlation analyses between body burden, PCDD/PCDF TEQ, Co-PCB TEQ and various laboratory data showed significant positive correlations between dioxin levels and GGT, total protein, uric acid and calcium, and a negative correlation with Fe. However, these correlations disappeared as a result of multivariate analysis adjusted for age, smoking status, and alcohol drinking.

Increased NK activity and lower response to PHA stimulation remained significant even after adjusting for age. Reduced immunity has been reported after dioxin poisoning, and thus follow-up study is necessary.

Former occupation, birthplace, area of residence and air pollution there, hours of outside work, driving hours, and frequency of bathing and hair washing were not correlated with dioxin levels. There were 66 smokers, 29 of whom had a Brinkmann index greater than 600. This was positively correlated with PCB-TEQ (ever smoker CC=0.277, p=0.045), but not with dioxins. There were 51 regular drinkers. Alcohol drinking habit was unassociated with dioxin levels.

The effects of dietary habits were analyzed by chief-component analysis. In food frequency, 68.4% of food intake was explained by 9 factors. Correlation analysis between these factors and dioxin levels showed that factor 7 (butter, lard, cheese, bacon) was positively associated with body burden and PCDD/PCDF TEQ, while factor 1 (most ordinary foods, such as fish, clam, egg, squid, vegetables, etc.) was positively associated with PCB levels. This trend obtained by food frequency questionnaire was repeatedly observed by frequency of ordinary Japanese meal intake. Factor 2 (broiled fish, boiled fish, raw fish, and tempura intake) was significantly associated with PCB levels but not with dioxins.

We tried to determine what proportion of dioxin exposure came from food. We assumed that dioxin contamination was present in the fat of fish or animal foods. Our dietary questionnaire covers to about 80% of lipid intake. Correlation analysis suggested that intake of butter, lard, cheese, bacon, etc., was associated with a high body burden and PCDD/PCDF TEQ, while most ordinary foods, such as fish, clams, egg, squid, vegetables, etc. , were positively associated with Co-PCB levels. Japanese consume much more fish than the populations of Western countries, and intake of broiled fish, boiled fish, raw fish, and tempura resulted in high blood Co-PCB levels.

History of hyperlipidemia and allergy had significantly increased odds ratios. The history was self reported, so confirmation of the diagnosis may be necessary, because there was no association between dioxin levels and plasma lipids.

Number of children and their gender: After starting incinerator work the workers had 18 boys and 27 girls (n.s.), and 7 boys and 7 girls were less than 5 years old. When these children were

classified by their parents' dioxin levels, 16 boys and 17 girls were in the low exposure group (less than 47.9 pg I-TEQ/g lipid), and 2 boys and 5 girls were in high exposure group (more than 48 pg I-TEQ/g fat), but the difference was not statistically significant. The endocrine disrupter effects of dioxins have recently become an issue. Excess birth of female infant was noted in the report from Seveso (3). In our study, a small excess of female children was observed in the high-dose exposure group, but this may have been the result of statistical fluctuations.

The relatively small area of contamination around the incinerator and very high level of PCDF in the recirculating cooling water suggested that PCDD/PCDF had spread as mist from the chimney. The average PCDD/PCDF level of the workers who did not come into direct contact with fry ash was 34.2 pg I-TEQ/g lipid, and that of residents within 2 km from the incinerator was 25.3 pg I-TEQ/g lipid (Watanabe et al. in preparation). Three routes of human exposure are thought to exist: eating contaminated food, breathing polluted air, and skin contact with contaminated soil and materials. The difference in body burden between workers and residents outside the incinerator must enter the body through the lungs and skin. We were able to estimate the daily intake dose from body burden. Preliminary estimation of exposure dose suggested a 10-fold difference between persons having a high and low body burden. Obese persons had a higher body burden, and thus the control of obesity seemed to be important in reducing accumulation of PCDD/PCDF in the body.

The incinerator was used for 10 years, and all workers were considered to be chronically exposed. Dioxin toxicity reported in the past has been due to acute and large assessing dose exposure (4). Subclinical exposure as in our subjects is of interest what kind of health effects shall be present in the future. A study on the risk to other workers in the same type of incinerators is under way.

Prediction of future health effects in the current subjects is difficult. Most previous cohort studies have dealt with blood TCDD measurements (5). Summary of previous reports suggested that the maximum TCDD level of the current workers was 13.4 pg/g lipid, i.e. below the level that causes disease. The total TEQ, however, exceeded the minimal effect level in some. If it has similar toxicity equivalence, the person with the highest value, 831 pg I-TEQ/g lipid, may experience some effect in the future. Thus far, the risk of dibenzofurans has only been confirmed by Yusho accident (6), and its carcinogenicity is ranked to 2B by IARC. Further follow-up study of highly exposed incinerator workers may clarify this point in comparison to almost pure 2,3,7,8-TCDD exposure.

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