Health Effects of Chronic Exposure to Polychlorinated Dibenzo-P-Dioxins (PCDD), Dibenzofurans (PCDF) and Coplanar PCB (Co-PCB) of Municipal Waste Incinerator Workers

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A national survey of polychlorinated dibenzo-p-dioxins (PCDD) and dibenzofurans (PCDF) in emission gases from the municipal waste incinerators in 1997 revealed that the Nose Bika Center was heavily contaminated by PCDF. Ninety-four workers underwent a physical examination, and blood biochemistry, lymphocyte marker, and NK activity studies were carried out, along with blood dioxin measurements. Information on working history, life-style, and dietary habits was obtained by questionnaire and interview.

The blood dioxin levels were as follows. The median TEQ of dioxins was 39.7 pg I-TEQ/g lipid, and the range was 13.3 to 805.8. The median 2,3,7,8-TCDD concentration was 3.9 pg TEQ/g lipid, and the range was <1 pg TEQ/g lipid (one case) to 13.4 pg TEQ/g lipid. The median TEQ of coplanar PCB was 10.8 pg I-TEQ/g lipid, and the range was 3.1 to 54.2 pg TEQ/g lipid. The congener-specific distribution was quite similar to that in soil around incinerator and waste in the factory. 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.

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. History of hyperlipidemia and allergy had significantly increased odds ratios.

A study on the risk to other workers in the same type of incinerators is under way. Health effects of chronic exposure mainly to PCDF will be clarified by follow-up.

INTRODUCTION

A national survey of polychlorinated dibenzo-p-dioxins (PCDD) and dibenzofurans (PCDF) in emission gases from the municipal waste incinerators in 1997 showed that 50 out of 1500 incinerators emitted more than the national allowable level of 80 ng/Nm³. Bika Center in Osaka Prefecture emitted 180 ngI-TEQ/Nm³ in January 1998, and 150 ng I-TEQ/Nm³ in May 1997. Further investigation of soils at 8 points around the incinerator showed 2,700 pg I-TEQ/g soil at the training farm of a high school located directly adjacent to the incinerator. The Bika Center was placed in a 30,000 m² mountainous area, and had started operations in March 1988. It is equipped with two modern fluidized-type incinerators with and incineration capacity of 26.5 t/16 hr each. Its operations were suspended in June 1997 because of the high dioxin contamination of

Received August 15, 1999; accepted February 28, 2000.
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the surrounding soil.

Bika Center was further investigated at an additional 37 points in farms, rice fields, the bottom of the irrigation water supply system, and well water in December 1997. 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, a pond in a chestnut farm (100 m downwind), a pond for agricultural irrigation, and an irrigating-water stream yielded values of 23,000, 4,100, 3,300, and 26 pg TEQ/g dry soil, respectively. The similar isomer composition of the pollutants in emitted gas and soil suggested that the source of PCDD/PCDF was the incinerator. The level of contamination as the incinerator plant was approached, despite the 40 m high chimney. Such high levels of dioxin contamination were not expected with a modern incinerator. The Ministry of Health and Welfare established an expert committee in the Board of Life and Environment in June 1998, and it investigated the factory and reported the results for dioxin contamination in August 1998. The 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

The number of workers in the Bika Center since March 1988 was 146, and 96 of them (92 males and 4 females), aged 16 to 78 years old, wanted to receive the health examination. A preliminary health check-up was performed in October 1998 by physicians if the subjects would allow collection of 200 ml blood for dioxin analysis, and 88 males and 4 females remained as subjects for this study. The purpose and methods of the study were explained to them, and each gave written informed consent. On October 3 and 4, workers gathered at the local health center, and were interviewed by physicians who were experts in industrial medicine. The subjects described their work history in detail, and underwent a dermatological investigation by an expert dermatologist for Kanemi Yusho. Life habits and dietary habits were collected by questionnaire and checked by trained dieters. 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 AST(GOT), ALT(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. These tests were performed by the Zerox Lab system (Zerox, Tokyo).

As immunological markers, T lymphocytes subsets determined by surface antigens, such as CD3, CD4, CD8, and CD4/CD8 ratio, were measured by Serum Research Laboratory (Tokyo). 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. Because of unsuitable specimens as a result of partial clotting, and decreased lymphocyte viability caused by transportation from Osaka to Tokyo, the number of subjects of these tests was 88.

Blood PCDD/PCDF/Co-PCB was measured by a modification of Patterson's method. Lipids were extracted from 50 g of plasma with a solution of 9 ml saturated ammonium sulfate and 36 ml of ethanol:hexane (1:3) solution after the addition of an internal standard of 13C-labeled mixed dioxin congener solution, which contained 50 pg of 12C2-PCDDs, 13C2-PCDFs, and 13C2-Co-PCBs, except OCDD (100 pg)(Wellington Isotope Laboratories, Massachusetts, USA). All solvents were of dioxin analysis level. Pooled hexane layers were condensed, washed with distilled water, treated with anhydrous sodium sulfate, and evaporated to dryness, and lipid weight was measured.

Clean-up was achieved by a multilayer silica column with 44% sulfuric acid and 33% potassium hydroxide, and after elution with 150 ml of n-hexane, the specimen was evaporated until a small volume of residue was obtained. The columns were washed with n-hexane to remove PCB contamination before use. The residues were then applied to an alumina column. PCB was collected in 80 ml benzene, followed by 200 ml of n-hexane:chloromethane (98:2), and dioxin was collected in the next fraction of 150 ml of hexane:chloromethane (1:1). Both fractions were condensed by evaporation. Coplanar PCBs were separated from other PCBs with another alumina column and non-coplanar PCB eluted with 250 ml of hexane:chloromethane (98:2), and three Co-PCBs were then eluted with 150 ml of hexane chloromethane 1:1.

Analysis of PCDDs/PCDFs/Co-PCBs was carried out by gas chromatography-high resolution mass spectrometry (GC-MS). The analytical conditions were as follows: gas chromatography was performed with a Varian-3400 series unit (Hewlett-Packard, Palo Alto, California) equipped with a Finnigan MAT-90 (Finnigan MAT GmbH, Bremen, Germany). The column used was an DB-dioxin fused silica capillary column, 0.25 mm i.d. × 30 m, with 0.25 mm film thickness (J&W Scientific, Folsom, California). The column temperature was maintained at 100℃ for 3 min, heated to 220℃ at the rate of 20℃/min, heated to 250℃ at the rate of 5℃/min, and maintained at 250℃ for 15 min. The injection temperature was
30°C with a cold injection system, ion source temperature was maintained at 250°C, and the carrier gas (helium) flow pressure was 14 psi. Ionizing current, ionizing energy, accelerating voltage, and ion multiplier were 1mA, 70eV, 5kV, and 2kV, respectively.

PCDDs, PCDFs, and coplanar PCBs were analyzed by the selected ion monitoring method. The analysis of tetrachlorodibenzo-p-dioxins (TeCDDs), pentachlorodibenzo-p-dioxins (PeCDDs), hexachlorodibenzo-p-dioxin (HxCDDs), heptachlorodibenzo-p-dioxin (HpCDDs), and octachlorodibenzo-p-dioxin (OCDDs) used 13C12-1,2,3-TeCDD, 13C12-2,3,7,8-TeCDD, 13C12-1,2,3,7,8-PeCDD, 13C12-1,2,3,4,7,8-HxCDD, 13C12-1,2,3,6,7,8-HxCDD, 13C12-1,2,3,7,8-9-HxCDD, 13C12-1,2,3,4,6,7,8-HpCDD, and 13C12-1,2,3,4,6,7,8,9-OCDD, respectively, as internal standards. The analysis of tetrachlorodibenzo-furans (TeCDFs), pentachlorodibenzofurans (PeCDFs), hexachlorodibenzofurans (HxCDFs), heptachlorodibenzofurans (HpCDFs) and octachlorodibenzofurans (OCDFs) used 13C12-2,3,7,8-TeCDF, 13C12-1,2,3,7,8-PeCDF, 13C12-1,2,3,4,7,8-PeCDF, 13C12-1,2,3,4,7,8-HxCDF, 13C12-1,2,3,6,7,8-HxCDF, 13C12-1,2,3,7,8-9-HxCDF, 13C12-1,2,3,4,6,7,8-HpCDF and 13C12-1,2,3,4,6,7,8,9-HpCDF, respectively, as internal standards. The analysis of 3,3',4,4'-tetrachlorobiphenyl (TeCB), 3,3',4,4',5-pentachlorobiphenyl (PeCB), and 3,3',4,4',5,5-hexachlorobiphenyl (HxCB) used 13C12-3,3',4,4'-TeCB, 13C12-3,3',4,4',5-PeCB and 13C12-3,3',4,4',5,5'-HxCB, respectively, as internal standards. The toxicity of the dioxins was calculated by the WHO TEF method (1997) and is expressed as TEQ/g lipid.

Statistical analysis. SPSS version 8 was used for the statistical analyses. Correlation analysis was performed between PCDD/PCDF/Co-PCB and various variables. Linear regression analysis and logistic analysis were used for evaluating the effects of dioxins, if a significant correlation (p<0.05) was obtained.

RESULTS

Characteristics of the workers and their chief complaints:

The average age of the male subjects was 48.2±15.6 years old, and the average age of the female subjects was 45.5±3.1. The longest residential environment was in a village in 51, a residential area in 30, commercial location in 6, and in industrial area in one. Farming experience was reported by 17. Occupations before Bika Center were clerk in 28, driver in 21, sales person in 33, technician in 23, and laborer in 7 (multiple answers).

The physical characteristics of the subjects were as follows: height; 166.9±7.5 cm in the males and 158.1±4.2 cm in the females; body weight; 65.8±10.8 Kg in the males and 51.9±7.5 Kg in females, body mass index, 23.6±3.4 in the males and 20.8±3.1 in the females; body fat percentage, 20.8±5.1% in the males and 27.8±5.8% in the females; systolic blood pressure, 138.9±24.1 mmHg in the males and 132±23.2 mmHg in the females; and diastolic blood pressure, 84.2±14.5 mmHg in the males and 72.8±11.9 mmHg in the females.

Blood dioxin levels:

The average concentrations of blood PCDD/PCDF/Co-PCB congeners are shown in Table 1. Congener-specific distribution is quite similar to that in soil around the incinerator and waste in the factory. Total amount of PCDD and PCDF was 612.89 pg and 661.97 pg/g lipid, respectively. The median TEQ of dioxins was 39.7 pg TEQ/g lipid, and ranged from 13.3 to 805.8. The median 2,3,7,8-TCDD concentration was 3.9 pg TEQ/g lipid, and ranged from <1 pg TEQ/g lipid (one case) to 13.4 pg TEQ/g lipid. The median TEQ of coplanar PCB was 10.8 pg TEQ/g lipid, and ranged from 3.1 to 54.2 pg TEQ/g lipid.

Working place and dioxin levels:

The concentration of PCDD/PCDF and Co-PCB by age showed a linear increase (Fig. 1). The relationship between dioxin concentrations and work history in the factory showed that incinerator and fly ash treatment contributed to high blood dioxin levels (Fig.2). Maintenance of fluidized bed incinerator and ash treatment significantly contributed to the dioxin level according to the results of the multiple regression analysis. Garbage collection and selection, and crane handling had a negative beta in the regression analysis (Table 2).

Dioxin levels and laboratory data:

Peripheral blood did not show any significant association with dioxin levels. The average and SD of the RBC counts was 4.7±0.5 bil/ml, WBC count 6.4±1.7 mil/ml, platelet count 251±64 mil/ml, and hematocrit 43.8±4.4%. Blood biochemistry data are shown in Table 3. Chief component analysis of these data yielded 5 factors. Correlation analyses between body burden, PCDD/PCDF TEQ, Co-PCB TEQ and various laboratory data showed significantly positive correlations between dioxin levels and GGT, total protein, uric acid, and calcium, and a negative correlation with Fe. However, these correlations disappeared by multivariate analysis adjusted for age, smoking status, and alcohol drinking.

Among the immunological markers, a positive correlation was found between dioxin levels and NK activity and a negative correlation with PHA stimulation (Table 4). The latter trend was also observed when workers who had worked in high risk area were selected. Spontaneous proliferation of lymphocytes without stimulants also showed a negative association. A high CD8 percentage (46.7 %) and a low CD4 percentage (62.8 %) were observed in one subject each.

Life style and dioxin levels:

Former occupation, birthplace, area of residence and air pol-
Table 1. Concentration of Blood PCDDs/PCDFs/Co-PCBs (pg/g fat).

| Compound       | mean  | sd    | median | min  | max  |
|----------------|-------|-------|--------|------|------|
| 2,3,7,8-TCDD   | 4.51  | 2.62  | 3.85   | 0.50 | 13.40|
| 1,2,3,7,8-PeCDD| 14.68 | 15.74 | 10.25  | 3.70 | 104.00|
| 1,2,3,4,7,8-HxCDD| 14.86| 26.37 | 5.85   | 1.30 | 157.00|
| 1,2,3,6,7,8-HxCDD| 74.66| 75.02 | 53.55  | 10.10| 522.20|
| 1,2,3,7,8,9-HxCDD| 19.34| 33.68 | 9.30   | 1.60 | 252.00|
| 1,2,3,4,6,7,8-HpCDD| 65.86| 86.79 | 38.30  | 11.40| 598.00|
| OCDD           | 438.32| 426.39| 281.50 | 94.60| 2100.00|
| 2,3,7,8-TCDF   | 3.37  | 10.77 | 1.90   | 0.50 | 104.00|
| 1,2,3,7,8-PeCDF| 4.82  | 6.61  | 2.20   | 0.50 | 42.10 |
| 2,3,4,7,8-PeCDF| 66.63 | 101.90| 29.60  | 10.80| 644.00|
| 1,2,3,4,7,8-HxCDF| 57.68| 113.13| 15.90  | 4.60 | 651.00|
| 1,2,3,6,7,8-HxCDF| 97.49| 203.42| 24.40  | 4.10 | 1320.00|
| 1,2,3,7,8,9-HxCDF| 5.19 | 11.81 | 0.50   | 0.50 | 68.90 |
| 2,3,4,6,7,8-HxCDF| 96.54| 206.50| 23.55  | 3.60 | 1340.00|
| 1,2,3,4,6,7,8-HpCDF| 214.23| 504.51| 37.15  | 3.00 | 3040.00|
| 1,2,3,4,7,8,9-HpCDF| 103.87| 875.40| 1.90   | 0.50 | 8404.00|
| OCDF           | 12.15 | 21.03 | 5.00   | 2.00 | 117.00|
| D_NEWTEQ       | 30.78 | 27.63 | 22.22  | 8.89 | 157.16|
| F_NEWTEQ       | 62.77 | 112.24| 21.89  | 6.88 | 691.55|
| DF_ITEQ        | 93.54 | 137.74| 47.45  | 15.77| 822.28|
| PCB 77         | 148.59| 92.92 | 149.07 | 32.70| 580.00|
| PCB 126        | 131.81| 98.82 | 98.60  | 25.40| 520.00|
| PCB 169        | 104.55| 60.07 | 90.45  | 1.60 | 272.00|
| PCB_ITEQ       | 14.30 | 10.24 | 10.82  | 3.13 | 54.22 |
| TEQ TOTAL      | 99.69 | 135.47| 51.65  | 7.00 | 831.19|

Figure 1. Congener distribution of blood. Increased level of 2,3,4,7,8-PeCDF, 2,3,4,6,7,8-HxCDF and other frans in contaminated samples reflected the level of blood. Box-plot graph.
Figure 2. Dioxin levels by age. PCDD (round), PCDF (square), and Co-PCB (triangle) slightly increased by age. Ten workers at high risk workplace show exceptionally high PCDF level.

Table 2. PCDD/PCDF Concentration and Work.

| Place          | Conditional Coefficient | Standardized Coefficient | significance |
|----------------|-------------------------|---------------------------|--------------|
|                | B           | se*  | beta  | t   | p        |
| constant       | 147.553     | 29.843 |        | 4.944 | 0        |
| garbage_sel    | -134.09     | 33.556 | -0.37  | -3.996 | 0        |
| crane          | -128.916    | 49.465 | -0.292 | -2.606 | 0.011    |
| burning        | 188.457     | 44.406 | 0.478  | 4.244  | 0        |
| ash treatment  | 139.065     | 45.587 | 0.353  | 3.051  | 0.003    |
| electric ash   | 30.916      | 94.417 | 0.04   | 0.327  | 0.744    |
| chimney wash   | -51.142     | 91.102 | -0.085 | -0.561 | 0.576    |
| cooling        | 16.546      | 81.14  | 0.025  | 0.204  | 0.839    |
| ash transfer   | -8.206      | 32.985 | -0.021 | -0.249 | 0.804    |
| collection     | -107.387    | 32.273 | -0.383 | -3.327 | 0.001    |
| outside work   | 85.607      | 62.08  | 0.154  | 1.379  | 0.172    |
| gardening      | -91.476     | 115.705 | -0.069 | -0.791 | 0.432    |
| clerk          | -64.593     | 37.52  | -0.169 | -1.722 | 0.089    |
| other          | -75.642     | 37.46  | -0.172 | -2.019 | 0.047    |

Se* standard error
Table 3. Result of Blood Analysis.

|    | n  | mean | median | sd  | min | max |
|----|----|------|--------|-----|-----|-----|
| RBC | 94 | 4.7  | 4.8    | 0.5 | 2.7 | 5.7 |
| HB  | 94 | 14.6 | 14.9   | 1.6 | 9.1 | 17.4|
| WBC | 94 | 6.4  | 6.3    | 1.7 | 3.3 | 10.7|
| HT  | 94 | 43.8 | 44.2   | 4.3 | 29.9| 51.7|
| PLT | 94 | 25.1 | 25     | 6.4 | 4.2 | 44.9|
| GOT | 95 | 25.9 | 22     | 13  | 12  | 96  |
| GPT | 95 | 29.2 | 22     | 23  | 8   | 138 |
| LDH | 84 | 226.8| 216.8  | 54  | 133.4| 384.1|
| ALP | 84 | 169.4| 160.4  | 45.1| 89.7| 303.6|
| LAP | 84 | 51.7 | 50.6   | 10.9| 34.5| 94  |
| TBIL| 84 | 0.6  | 0.5    | 0.3 | 0   | 1.6 |
| CPK | 84 | 176.6| 134    | 136 | 65.6| 1087.9|
| AMYL| 84 | 54.2 | 50.6   | 18.8| 25.3| 134.6|
| GLU | 84 | 78.3 | 66.1   | 73.9| 5.8 | 547.4|
| TP  | 84 | 7    | 7      | 0.4 | 6.2 | 8.4 |
| ALB | 84 | 4.7  | 4.7    | 0.4 | 3.8 | 5.9 |
| TCHO| 92 | 171.9| 168.5  | 29.1| 101.2| 279.5|
| HDLC| 92 | 46.2 | 42.6   | 14.6| 19.6| 97.8 |
| TG  | 92 | 139.5| 120.2  | 72.9| 40  | 431.3|
| CRP | 84 | 0.5  | 0.4    | 0.2 | 0.3 | 1.2 |
| CRE | 85 | 1    | 1      | 0.1 | 0.8 | 1.4 |
| UA  | 84 | 6.1  | 6.1    | 1.3 | 3.5 | 9.7 |
| CA  | 84 | 8    | 8.1    | 0.3 | 7.2 | 8.7 |
| NA  | 84 | 167.6| 167.9  | 2.4 | 159.5| 173.7|
| K   | 84 | 3.6  | 3.3    | 0.8 | 2.4 | 5.4 |
| CL  | 84 | 122.4| 122.5  | 2.3 | 115.5| 127.7|
| IP  | 84 | 2.5  | 2.5    | 0.5 | 1.3 | 3.7 |
| NH3 | 84 | 263.8| 238.6  | 88.9| 136.9| 573.9|
| CD3 | 84 | 68.3 | 68.7   | 7.5 | 48.3| 82.4 |
| CD4 | 84 | 44.1 | 46.2   | 9.9 | 0.1 | 62.8 |
| CD8 | 84 | 26.5 | 25.7   | 7.9 | 7.3 | 46.7 |
| CD4/8|84|1.9|1.7|1|0|6.2|
|CD56|84|18.6|17.9|7.4|5.9|36.9|
|NK_ACT|84|27.7|27|12.3|7|70|
|PHA+ | 83 | 42008.3| 42600 | 10866.7| 6368 | 61806 |
|PHA_CONT|83|261.4|221|148.1|83|777|
|CON-A | 83 | 27038.8| 25128 | 8273.5| 3701 | 50642 |
|CONTROL|83|261.4|221|148.1|83|777|

Past and present history of diseases:
Most subjects appeared healthy but reported various diseases during the last 10 years in their past history of diseases: hypertension in 14, diabetes mellitus in 8, hyperlipidemia in 8, allergy and peptic ulcer in 7 each, asthma and gout in 4 each, gastric cancer and atopy in 3 each, liver cirrhosis, chronic hepatitis and renal disease in 2 each, colon cancer and thyroid disease in one each.

Logistic analyses for the above disease history by quarterly categorized PCDD/PCDF levels, age, BMI, and smoking (yes/no) did not show any significant contribution of PCDD/PCDF levels. Only odds ratio by PCDD/PCDF levels (divided at 100 pg I-TEQ/g fat) and the presence of hyperlipidemia became significant (OR=6.08, p=0.023 with 99% confidence interval of 0.019-0.026). The Mann-Whitney test for


**Table 4. Correlation Coefficients between Dioxin Levels and Immunologic Index.**

|          | Dioxins | Furans | Co-PCBs | Total TEQ |
|----------|---------|--------|---------|-----------|
| CD3      | -0.063  | -0.13  | -0.044  | -0.118    |
| CD4      | -0.056  | -0.124 | 0.112   | -0.1      |
| CD8      | -0.079  | -0.105 | -0.174  | -0.11     |
| CD4/8    | 0.011   | -0.019 | 0.168   | 0         |
| CD56     | 0.134   | 0.112  | 0.186   | 0.124     |
| NK_ACT   | 0.216*  | 0.206  | 0.111   | 0.212*    |
| PHA+     | -0.26*  | -0.27**| -0.147  | -0.281**  |
| PHA_CONT | -0.02   | -0.016 | -0.164  | -0.032    |
| CON-A    | -0.152  | -0.17  | -0.033  | -0.167    |
| CONTROL  | -0.02   | -0.016 | -0.164  | -0.032    |

* p<0.05, ** p<0.01 (both side ; p is shown by small letter)

**Table 5. Correlation between Food Frequency and blood PCDD/PCDF/Co-PCB TEQ Concentration.**

| Food Item          | Dioxins | Furans | Co-PCB | Total TEQ |
|--------------------|---------|--------|--------|-----------|
| Ordinary daily food| 0.096   | 0.111  | 0.358**| 0.132     |
| Clam, shrimp, bacon| 0.073   | 0.041  | 0.087  | 0.056     |
| Fatty food         | 0.277** | 0.187  | 0.006  | 0.201     |
| Rice, egg          | 0.023   | -0.028 | -0.007 | -0.019    |
| Mushroom, ham      | 0.253*  | 0.165  | 0.322**| 0.204     |
| Meat               | 0.073   | 0.045  | 0.032  | 0.052     |
| Butter, cheese, lard| 0.232* | 0.297**| -0.165 | 0.267**   |
| Daily product      | -0.042  | -0.082 | 0.078  | -0.07     |
| Crab               | -0.095  | -0.158 | -0.18  | -0.155    |

** p<0.01,  * p<0.05 both side

**Table 6. Correlation Coefficients between Preferent Meals and PCDD/PCDF/Co-PCB Concentration.**

| Meal Type         | Dioxins | Furans | Co-PCB | Total TEQ |
|-------------------|---------|--------|--------|-----------|
| Fatty meals       | -0.153  | -0.081 | -0.395**| -0.121    |
| Fish meals        | -0.019  | 0.002  | 0.318**| 0.02      |
| Noodles           | -0.009  | -0.071 | -0.163 | -0.069    |
| Broiled meat, tempura| 0.273* | 0.195  | 0.119  | 0.215     |
| Grilled eel, fried dumpling| 0.282**| 0.218* | 0.034  | 0.229*    |

** p<0.01,  * p<0.05 both side

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measurement of PCB may be necessary. A Committee of the Environmental Agency estimated the average Japanese takes 0.52-3.53 pg I-TEQ/kg body weight/day in urban areas, 0.5-3.5 pg I-TEQ/kg/day in towns, and 0.29-3.29 pg I-TEQ/kg/day in rural areas. The actual intake of PCDDs from food by any one person will depend on the amount and type of food consumed and the level of contamination. In the US, daily PCDD/PCDF TEQ intake (pg/kg body weight/day) in 1995 was estimated at from 0.52 to 2.57, while PCDD/PCDF/PCBs intake was 1.16 by the low estimate, and 3.57 by the high estimate.

In Japan, 90% of the daily intake of PCDDs, PCDFs, and other dioxin-like compounds is estimated to come from food. Japanese levels of 2,3,7,8-TCDD in the serum of the general population typically range from 3 to 7 pg/g lipid, and rarely exceed 20 pgTEQ/g lipid (Watanabe et al., unpublished data).

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.

A 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. The endocrine disrupter effects of dioxins have recently become an issue. Excess birth of female infant was noted in the report from Seveso. 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.

Prediction of future health effects in the current subjects is difficult. Most previous cohort studies have dealt with blood TCDD measurements. 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, 805.8 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, 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.

**ACKNOWLEDGEMENT**

This study was supported by the Ministry of Labor. The

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