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Emissions of Polychlorinated Dibenzo-p-dioxins and Dibenzofurans from Various Stationary Sources

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Abstract

This work investigated the characteristics of polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/F) in stack-flue gases from six stationary emission sources in five types of incinerators: industrial waste incinerator (IWI), small-scale municipal solid waste incinerator (MSWI), medical waste incinerator (MWI), cement kilns (CK), and crematories (CR). These characteristics were further investigated using factor analysis and cluster analysis. Experimental results reveal that PCDDs dominate MSWI and CR, and PCDFs dominate IWIa, IWIb, CK and MWIs. The factor analysis results showed that CR and MSWI have similar fingerprints, as do IWIb and MWI3. The cluster analysis showed that if a vertical line is cut at a rescaled distance of four, then the PCDD/F congener profiles fall into four groups. The indicators of PCDD/Fs are OCDD, 1,2,3,4,6,7,8-HpCDF, 2,3,4,7,8-PeCDF, and 1,2,3,4,6,7,8-HpCDD. The emission factors of PCDD/Fs herein were from 0.0433 (CK) to 18.7 (MSWI) μg I-TEQ/ton-feedstock.

Keywords: Dioxin emission, incinerators, congener profile, factor analysis, cluster analysis

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INTRODUCTION

Dioxins, especially 2,3,7,8-TCDD, are of great concern, because they are highly carcinogenic. PCDD/Fs are formed during combustion from: (i) precursors, (ii) de novo, and (iii) through pyrosynthesis (Tuppurainen et al., 2003). Major sources of atmospheric PCDD/Fs include stationary emissions, especially from various incinerators, including secondary aluminum smelters (ALS), sinter plants, small-scale municipal solid-waste incinerators (MSWI), medical-waste incinerators (MWI), electric-arc furnaces (EAF), industrial-waste incinerators (IWI), cement kilns and crematoria. Hence, this work studies PCDD/F emissions from MSWI, MSI, IWI, crematory cement kilns (CK), and cement crematoria (CR).

Cement kilns use coal as burner fuel; the other sources use diesel. The feedstock of CK is mostly cement as a raw material and a few waste tires, IWIa is fed with general waste from nuclear power plants, and IWIb is fed mostly with waste-oil sludge. For pollution control, MSWI uses a semi-dry washing tower and a baghouse, MWI uses a Venturi scrubber and a quench tower, CK uses an electrostatic precipitation (ESP), IWIa uses a baghouse, and IWIb uses a cyclone and an ESP.

This paper reports on measurements of PCDD/Fs from the stacks of six incinerators. Emission characteristics of PCDD/Fs from these incinerators are presented, including concentrations, fingerprints and emission factors. Also, similar and dissimilar features between them are further studied using factor analysis and cluster analysis. The data derived from this study can provide guidance to improve operating conditions of the incinerators and to assess the potential health risk to the neighboring community.

EXPERIMENTAL

PCDD/FS Sampling

PCDD/Fs in stack-flue gases were collected using the Taiwan EPA method NIEA A807.73C, which is based on the US EPA Method 23A. Prior to sampling, XAD-2 resin was spiked with isotopically labelled PCDD/F surrogate standards. Each stack gas sampling took 3 h. One trip blank and one field blank were also obtained during field sampling to ensure that the collected samples were not contaminated.

PCDD/FS Analysis

PCDD/Fs were analyzed for stack-flue gases, according to U.S. EPA modified Method 23, using high-resolution gas chromatographs/high-resolution mass spectrometers (HRGC/HRMS). The analysis was conducted at the Super Micro Mass Research and Technology Center at Cheng Shiu University in Taiwan. The HRGC (Hewlett-Packard 6970) was comprised of a DB-5 MS

fused silica capillary column (0.25 mm × 60 m, 0.25 μm) (J&W Scientific) with splitless injection. Helium was used as the carrier gas. The HRMS (Micormass Autospec Ultima, Manchester, UK) had a positive electron impact (EI+) source. The selected ion-monitoring mode (Park et al., 2004) had a resolving power of 100,000. The specified electron energy and source temperature were 35 eV and 250 °C, respectively.

RESULTS AND DISCUSSION

PCDD/F Concentrations in Stacks

Table 1 shows that the total PCDD/F concentrations in the stack gases of IWIa, IWIb, MSWI, MWI, CK, and CR were 0.604, 1.397, 30.1, 1.14, 0.350, and 29.8 ng/Nm^3, respectively, and the order was MSWI > CR > IWIb > MWI > IWIa > CK, indicating that PCDD/F concentrations in the stacks of MSWI and CR greatly exceeded those of other incinerators. The efficiency of a baghouse in removing PCDD/Fs was around 37.6% (Lee et al., 2004); so, highly concentrated PCDD/Fs could be formed from MSWI during combustion. CR should include pollution control equipment to reduce the concentration of PCDD/Fs in the stack-flue gases. The PCDD to PCDF ratios were 0.464, 0.415, 0.903, 0.423, 0.292, and 0.869, respectively. The PCDD/Fs ratios in the stacks followed the order CR > MSWI > IWIa > MWI > IWIb > CK, indicating that PCDDs dominated CR and MSWI and PCDFs dominated the other burners. The total PCDD/Fs I-TEQs were 0.030, 0.137, 3.35, 0.168, 0.062, and 3.00 ng I-TEQ/Nm^3, respectively. The order of total PCDD/F I-TEQ was MSWI > CR > MWI > IWIb > CK> IWIa, similar to that of the total PCDD/F concentration in stacks.

In summary, when considering PCDD/F concentrations, PCDD/F ratio and PCDD/F I-TEQ, MSWI and CR are two leading PCDD/F contributors among the six incinerators.

PCDD/F Congener Profiles of Stationary Emission Sources

Fig. 1 presents the congener profiles of the 17 2,3,7,8-chlorinated substituted PCDD/Fs detected in six stack-flue gases. Each selected congener was normalized to the total weight of all 2,3,7,8-congeners (mean ± SD). The PCDD/F congener profile of IWIa was dominated by 1,2,3,4,6,7,8-HpCDF, OCDD, and OCDF, while that of IWIb was dominated by 1,2,3,4,6,7,8-HpCDF, OCDD, and 2,3,4,7,8-PeCDF. The profile of MSWI was dominated by 1,2,3,4,6,7,8-HpCDD, OCDD, and 1,2,3,4,6,7,8-HpCDF. The profile of MWI was dominated by 2,3,4,7,8-PeCDF, 1,2,3,4,6,7,8-HpCDF, and 1,2,3,7,8-PeCDF. The profile of CK was dominated by 2,3,7,8-TeCDF, 1,2,3,7,8-PeCDF, and 2,3,4,7,8-PeCDF. However, the profile of CR was dominated by 1,2,3,4,6,7,8-HpCDF, 1,2,3,4,6,7,8-HpCDD, and OCDD.

Fig.1 also indicates that MSWI and CR were dominated by PCDD, and IWIa, IWIb, CK and MWI were dominated by PCDF. Moreover, the major and minor dominating congener profiles of IWIb were similar to those of IWIa; and MSWI and CR had similar congener profiles.

Table 1. The PCDD/F concentrations in the stack-flue gases of six emission sources.

PCDD/PCDFs (ng/Nm^3)	IWI a (n=3)		IWI b (n=3)		MSWI (n=3)		MWI (n=3)		CK (n=3)		CR (n=3)	
	mean	RSD(%)	mean	RSD(%)	mean	RSD(%)	mean	RSD(%)	mean	RSD(%)	mean	RSD(%)
2,3,7,8-TeCDD	0.001	12.7	0.006	13.8	0.29	36.0	0.016	88.6	0.018	139	0.319	27.0
1,2,3,7,8-PeCDD	0.003	17.9	0.022	6.67	1.18	37.7	0.042	92.8	0.01	105	1.15	23.7
1,2,3,4,7,8-HxCDD	0.005	32.6	0.013	12.5	0.823	43.9	0.02	93.3	0.003	57.1	0.834	23.3
1,2,3,6,7,8-HxCDD	0.009	26.8	0.022	11.5	1.41	43.8	0.044	92.3	0.004	50.6	1.39	16.7
1,2,3,7,8,9-HxCDD	0.006	27.3	0.018	4.84	0.987	43.9	0.042	91.5	0.003	52.8	1.40	17.9
1,2,3,4,6,7,8-HpCDD	0.056	38.4	0.117	12.0	5.19	51.1	0.089	74.8	0.014	39.6	4.55	28.1
OCDD	0.105	42.2	0.213	36.0	4.38	55.8	0.076	43.0	0.022	24.3	4.05	47.1
2,3,7,8-TeCDF	0.009	14.7	0.064	9.53	1.54	33.5	0.095	81.3	0.114	119	1.66	33.7
1,2,3,7,8-PeCDF	0.013	18.0	0.096	8.06	1.64	43.6	0.11	86.5	0.04	95.3	1.25	32.5
2,3,4,7,8-PeCDF	0.023	23.9	0.127	18.4	2.49	44.7	0.152	88.5	0.039	78.9	1.82	23.3
1,2,3,4,7,8-HxCDF	0.03	41.5	0.116	14.1	1.69	46.4	0.096	88.0	0.013	40.6	1.65	37.5
1,2,3,6,7,8-HxCDF	0.032	39.2	0.122	12.4	1.82	48.6	0.105	90.5	0.018	42.9	1.76	34.9
2,3,4,6,7,8-HxCDF	0.004	116.4	0.009	11.7	0.086	50.2	0.004	76.5	0.001	173	0.061	30.0
1,2,3,7,8,9-HxCDF	0.036	38.5	0.107	15.4	2.17	50.5	0.067	86.1	0.016	42.9	1.60	32.9
1,2,3,4,6,7,8-HpCDF	0.149	58.1	0.232	16.0	3.43	55.0	0.125	71.9	0.022	32.0	5.02	46.3
1,2,3,4,7,8,9-HpCDF	0.02	56.8	0.035	18.3	0.397	57.7	0.015	64.0	0.004	40.8	0.357	40.6
OCDF	0.102	63.0	0.077	37.2	0.567	58.3	0.044	30.6	0.009	15.2	0.964	51.3
PCDDs	0.185	34.4	0.412	23.5	14.3	49	0.329	75.4	0.074	69.8	13.7	29.2
PCDFs	0.419	49.3	0.985	15.7.	15.8	47.7	0.813	80.8	0.276	85.3	16.1	36.1
PCDD/Fs ratio	0.464	20.3	0.415	7.92	0.903	9.43	0.423	12.4	0.292	14.3	0.869	11.6
Total PCDD/Fs	0.604	43.8	1.397	17.9	30.1	47.9	1.14	79.2	0.35	82	29.8	32.8
PCDDs ng I-TEQ/Nm^3	0.005	21.6	0.024	8.5	1.26	39.3	0.049	90.7	0.024	128	1.30	22.9
PCDFs ng I-TEQ/Nm^3	0.025	30.7	0.113	15.8	2.10	44.8	0.12	87.6	0.038	87.1	1.70	28.6
PCDD/Fs TEQ ratio	0.21	10.3	0.217	10.3	0.616	9.47	0.395	7.42	0.495	53.3	0.78	9.18
Total ng I-TEQ/Nm^3	0.030	29.1	0.137	14.3	3.35	42.7	0.168	88.5	0.062	103	3.00	25.8

Factor Analysis

To understand the underlying factors affecting the formation of PCDD/Fs in incinerators, factor analysis (FA) was employed. In this work, factors were extracted using principal component analysis (PCA), which involves varimax orthogonal rotation to determine the eigen values of variance matrix of variables (Wilkinson et al., 1996; Johnson and Wichern, 2002). Usually, those factors with eigen values exceeding unity were chosen. In Table 2, two factors, Factor1 and Factor2, are shown corresponding to the two leading eigenvalues of 6.251 and 5.506,

respectively. Factor1 explains 39.9% of all variance, and Factor2 explains 29.0% of all variance; altogether representing 68.9% of the total variance. Factor1 was strongly related (> 0.7) to: 1,2,3,7,8-PeCDD, OCDD; 2,3,4,7,8-PeCDF; 1,2,3,4,6,7,8-HpCDF; 1,2,3,4,7,8,9-HpCDF; and OCDF. Factor2 was strongly related (> 0.7) to: 1,2,3,4,7,8-HxCDD; 1,2,3,6,7,8-HxCDD; 1,2,3,7,8,9-HxCDD; and 1,2,3,4,6,7,8-HpCDD.

Also, Fig. 1 shows that Factor1 explains the major congeners of IWIa, IWIb, and MWI; while both Factor1 and Factor2 explain the major congeners of MSWI, CK, and CR. Chlorine-containing wastes and precursors are known to be responsible for the formation of PCDD/Fs (Dickson et al., 1989; Milligan and Altwicker, 1993); therefore, Factor1 and Factor2 may likely represent these two factors—chlorine-containing wastes and precursors; the latter is associated with the products of incomplete combustion. Notably, formation of PCDD/Fs in relatively low temperatures (250–350 °C) in dust-control device is also one important mechanism (Dickson et al., 1992; Luijk et al., 1994).

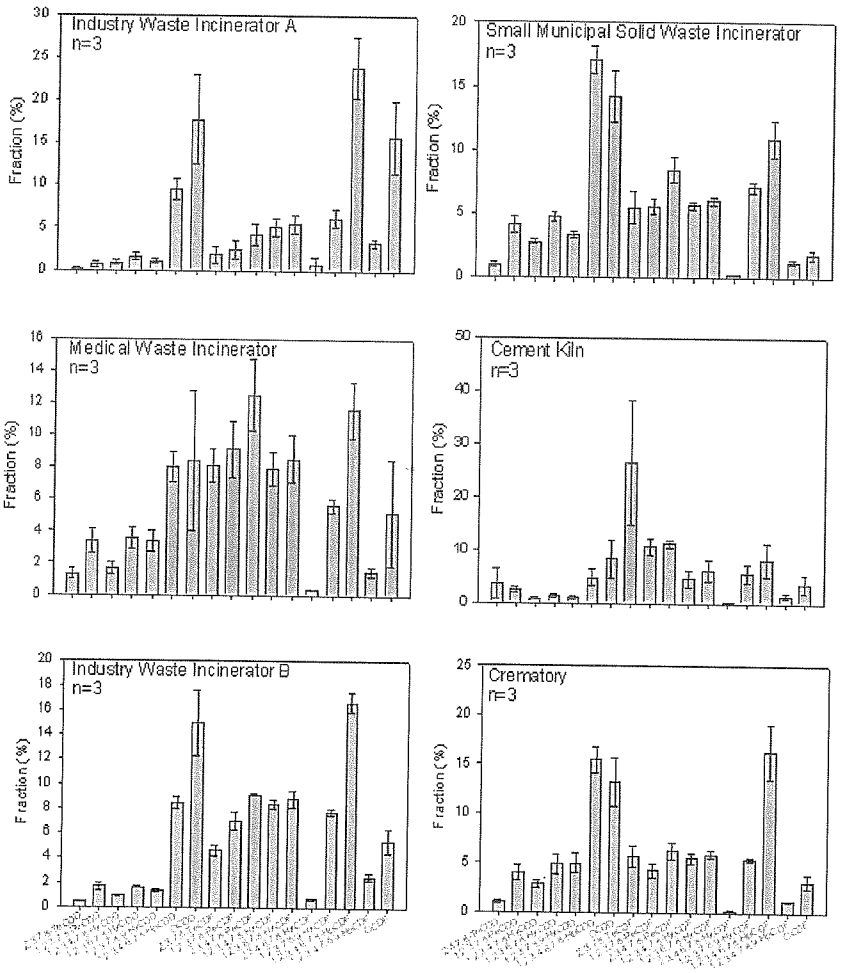

Fig. 1. Congener profiles of seventeen 2,3,7,8 -PCDD/Fs of various emission sources.

Fig. 2 shows the component plot, with Factor1 as the horizontal axis and Factor2 as the vertical axis. In the plot, the closeness of the emission sources to each other implies the similarity in their congener profiles. The plot shows that CR and MSWI have similar fingerprints; similarly for IWIb and MWI3.

Table 2. Factor analysis of PCDD/Fs in six stack-flue gases.

PCDD/Fs	Factor1	Factor2
2,3,7,8-TeCDD	.608	-.584
1,2,3,7,8-PeCDD	**.872**	.446
1,2,3,4,7,8-HxCDD	.519	**.845**
1,2,3,6,7,8-HxCDD	.539	**.825**
1,2,3,7,8,9-HxCDD	.560	**.748**
1,2,3,4,6,7,8-HpCDD	.086	**.952**
OCDD	**-.727**	.353
2,3,7,8-TeCDF	.556	**-.708**
1,2,3,7,8-PeCDF	.698	-.660
2,3,4,7,8-PeCDF	**.700**	-.440
1,2,3,4,7,8-HxCDF	.028	.041
1,2,3,6,7,8-HxCDF	.114	-.101
2,3,4,6,7,8-HxCDF	-.555	-.057
1,2,3,7,8,9-HxCDF	-.224	.181
1,2,3,4,6,7,8-HpCDF	**-.863**	.276
1,2,3,4,7,8,9-HpCDF	**-.959**	-.149
OCDF	**-.879**	-.131
Eigen value	6.251	5.506
Percentage of total variance	39.9	29.0

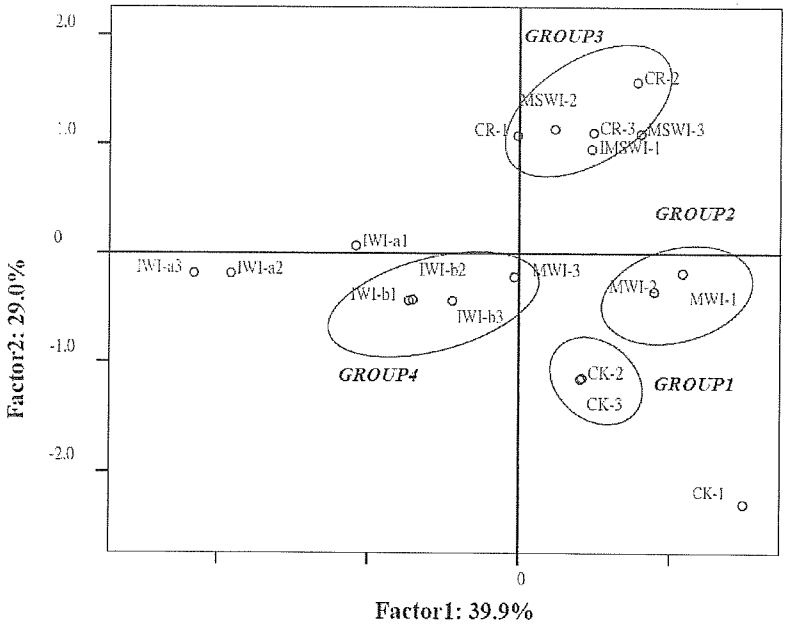

Fig. 2. Component plot from factor analysis.

Cluster Analysis

Cluster analysis, using nearest-neighbour method, was employed to divide the congener profiles from different stack-flue gases into several groups (Johnson and Wichern, 2002). The dendrogram in Fig. 3 from cluster analysis shows that if a vertical line is cut at a rescaled distance of four, then the PCDD/F congener profiles fall into four groups, namely: GROUP1, GROUP2, GROUP3, and GROUP4. GROUP1 comprises CK2 and CK3, and GROUP2 comprises MWI1 and MWI2. GROUP 3 comprises MSWI (MSWI1–MSWI3) and CR (CR1–CR3) which is consistent with the results of similar fingerprints in these two processes discussed earlier. GROUP4 is comprised of IWIb (IWIb1–IWIb3) and MWI3. IWIa (IWIa1–IWIa3) and CK1 do not belong to any group. Generally, cluster analysis results (Fig. 3) agree fairly well with factor analysis results shown in Fig. 2.

Indicators of PCDD/FS

Table 3 shows, based on the results of indicatory PCDD/F analysis, that IWIa, IWIb, MSWI, MWI, and CR have similar indicatory PCDD/Fs, which are OCDD, 1,2,3,4,6,7,8-HpCDF, 2,3,4,7,8-PeCDF, and 1,2,3,4,6,7,8-HpCDD. However, CK has quite different indicatory PCDD/Fs from the other five incinerator types. This may be due to the fact that the feedstock components of CK contain fewer organic compounds than the other incinerators do.

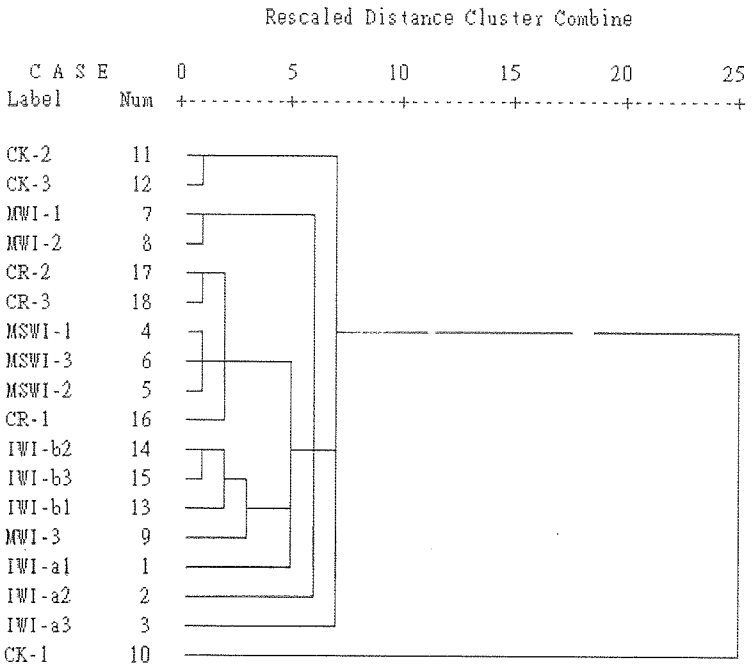

Fig. 3. Dendrogram from cluster analysis.

Table 3. The highest three indicatory PCDD/Fs of various PCDD/F emission sources.

PCDD/F emission sources	Indicatory PCDD/Fs (ratio)
IWIa – Industry Waste Incinerator	1,2,3,4,6,7,8-HpCDF (0.149)
	OCDD (0.105)
	OCDF (0.102)
IWIb – Industry Waste Incinerator	1,2,3,4,6,7,8-HpCDF (0.232)
	OCDD (0.213)
	2,3,4,7,8-PeCDF (0.127)
MSWI – Small Municipal Solid Waste Incinerator	1,2,3,4,6,7,8-HpCDD (5.193)
	OCDD (4.38)
	1,2,3,4,6,7,8-HpCDF (3.433)
MWI – Medical Waste Incinerator	2,3,4,7,8-PeCDF (0.152)
	1,2,3,4,6,7,8-HpCDF (0.125)
	1,2,3,7,8-PeCDF (0.110)
CK – Cement Kiln	2,3,7,8-TeCDF (0.114)
	1,2,3,7,8-PeCDF (0.04)
	2,3,4,7,8-PeCDF (0.0385)
CR – Crematory	1,2,3,4,6,7,8-HpCDF (5.020)
	1,2,3,4,6,7,8-HpCDD (4.547)
	OCDD (4.047)

Table 4. PCDD/F emission factors of various emission sources.

Stationary Source	Emission factor (μg I-TEQ/ton-feedstock)	Reference
IWIs with baghouse	0.059 ± 31.3	This study
IWIs with cyclone and ESP	0.368 ± 14.3	This study
MSWIs with semi dry washing tower and baghouse	18.7 ± 43.2	This study
MWIs with Venturi scrubber and quench tower	3.70 ± 88.7	This study
CK with ESP	0.0433 ± 103	This study
CR without pollution control equipment	41.1 ± 27.6*	This study
Secondary ALS	50.1 ± 62.5	(Chen et al., 2004)
Secondary ALS	0.63 – 200	(Lee et al., 2005)
Sinter plants with SCR	0.970	(Wang et al., 2003b)
Sinter plants without SCR	3.13	(Wang et al., 2003b)
EAFs	0.52 – 3.2	(Lee et al., 2005)
CR with bag filter	6.11*	(Wang et al., 2003a)
CR without bag filter	13.6*	(Wang et al., 2003a)

* Unit: μg I-TEQ/body

Emission Factors of PCDD/FS

Table 4 shows that the emission factors of PCDD/Fs herein were from 0.0433 (CK) to 18.7 μg I-TEQ/ton-feedstock (MSWI), whereas previous studies yielded 0.52 to 200 μg I-TEQ/ ton-feedstock (Wang et al., 2003a and 2003b). The PCDD/F emission factors of CR were 41.1 μg I-TEQ/ body herein, a value which is around three times greater than the value reported by Wang et al. (2003a). The PCDD/F emission factors of MSWI and MWI ranked second and third herein.

It is known that precursors are responsible for the formation of PCDD/Fs. Therefore, the control of feedstock components and the complete combustion of wastes are important in reducing the emission of PCDD/Fs from the stack-flue gas.

CONCLUSIONS

(1) The total PCDD/F I-TEQ concentrations in the stack gases of IWIa, IWIb, MSWI, MWI, cement kiln (CK) and crematory (CR) were 0.030, 0.137, 3.352, 0.168, 0.062, and 3.003 ng I-TEQ/Nm3, respectively. The total PCDD/F I-TEQ followed the order MSWI > CR > MWI > IWIb > CK > IWIa.

(2) The factor analysis results showed that CR and MSWI have similar fingerprints; similarly between IWIb and MWI3. The cluster analysis showed that if a vertical line is cut at a rescaled distance of four, then the PCDD/F congener profiles fall into four groups. These two analyses generally yielded consistent results.

(3) The indicators of PCDD/Fs of IWIa, IWIb, MSWI, MWI, and CR were very similar. They were OCDD, 1,2,3,4,6,7,8-HpCDF, 2,3,4,7,8-PeCDF, and 1,2,3,4,6,7,8-HpCDD, which were quite different from those of CK.

(4) The emission factors of PCDD/Fs herein were between 0.0433 (CK) and 18.7 (MSWI) μg I-TEQ/ton-feedstock.

Since precursors are responsible for the formation of PCDD/Fs, additional studies should be conducted to provide further understanding on their formation mechanisms during combustion-related processes.

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REFERENCES

Chen, S.-J., Lee, W.-S., Guo-Ping, C.-C., Wang, L.-C., Lee, W.-J., Kao, J.-H. and Hu, M.T.M.-T., (2004). Characterizing polychlorinated dibenzo-p-dioxins and dibenzofurans in the surrounding environment and workplace of a secondary aluminum smelter. *Atmospheric Environment.* 38(22): 3729-3732.

Dickson, L.-C., Lenoir, D. and Hutzinger, O. (1989). Surface-catalyzed formation of chlorinated dibenzodioxins and dibenzofurans during incineration. *Chemosphere.* 19: 277-282.

Dickson, L.-C., Lenoir, D. and Hutzinger, O. (1992). Quantitative comparison of de Nova and formation of polychlorinated dibenzo-p-dioxins under simulated municipal solid waste incinerator post-combustion conditions. *Environmental Science and Technology.* 26: 1822-1828.

Johnson, R.-A. and Wichern, D.-W. (2002). *Applied Multivariate Statistical Analysis.* 5th ed., Prentice-Hall, Inc., New Jersey, USA.

Lee, W.-S., Chang-Chien, G.-P., Chen, S.-J., Wang, L.-C., Lee, W.-J. and Wang, Y.-H., (2004). Removal of Polychlorinated Dibenzo-p-dioxins and Dibenzofurans in Flue Gases by Venturi Scrubber and Bag Filter. *Aerosol and Air Quality.* 4(1): 27-37.

Lee, W.-S., Guo-Ping, C.-C. and Wang, L.-C., (2005). Emissions of Polychlorinated Dibenzo-p-Dioxins and Dibenzofurans from Stack Gases of Electric Arc Furnaces and Secondary Aluminum Smelters. *Journal of the Air & Waste Management Association.* 55: 219-226.

Luijk, R., Akkerman, D.-M., Slot, P., Olie, K. and Kapteijn, F. (1994). Mechanism of formation of polychlorinated dibenzo-p-dioxins and dibenzofurans in the catalyzed combustion of carbon. *Environmental Science and Technology.* 28: 312-321.

Milligan, M.-S., Altwicker, E. (1993). The relationship between de Nova synthesis of polychlorinated dibenzo-p-dioxins and dibenzofurans and low-temperature carbon gasification in fly ash. *Environmental Science and Technology.* 27: 1595-1601.

Park, S., Kim, S.-J., Kim, K.S., Lee, D.S. and Kim, J.G., (2004). Influence of an Industrial Waste Incinerator as Assessed by the Levels and Congener Patterns of Polychlorinated Dibenzo-p-dioxins and Polychlorinated Dibenzofurans. *Environ. Sci. Technol.* 38: 3820-3826.

Tuppurainen, K., Asikainen, A., Ruokojärvi, P. and Ruuskanen , J., (2003). Perspectives on the Formation of Polychlorinated Dibenzo-p-dioxins and Dibenzofurans during Municipal Solid Waste (MSW) Incineration and Other Combustion Processes. *Accounts of Chemical Research.* 36 (9).

Wang, L.-C., Lee, W.-J., Lee, W.-S., Chang-Chien, G.-P. and Tsai, P.-J., (2003a). Characterizing the Emissions of Polychlorinated Dibenzo-p-dioxins and Dibenzofurans from Crematories and Their Impacts to the Surrounding Environment. *Environ. Sci. Technol.* 37(1): 62-67.

Wang, L.-C., Lee, W.-J., Tsai, P.-J., Lee, W.-S. and Chang-Chien, G.-P., (2003b). Emissions of polychlorinated dibenzo-p-dioxins and dibenzofurans from stack-flue gases of sinter plants. *Chemosphere.* 50(9): 1123-1129.

Wilkinson, L., Blank, G. and Gruber, C. (1996). *Desktop Data Analysis with SYSTAT.* Prentice-Hall, Inc., Upper Saddle River, NJ, USA.

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Influence of start-up on PCDD/F emission of incinerators

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Abstract

This study aims to evaluate the influence of start-up on polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans (PCDD/Fs) concentration in the stack flue gas of incinerators and its contributing PCDD/F emission. The PCDD/F emission of the first sample among three consecutive stack flue gas samples of five intermittent incinerators, which sampled at a stable combustion condition after start-up, is 2–3 times higher than the mean of the others. For verifying the PCDD/F characteristics of incinerators during start-up, one continuous MSWI was investigated for two years. The elevated PCDD/F emissions of the MSWI during start-up could reach 96.9 ng I-TEQ N m^{-3} and still maintained a high PCDD/F emission (40 times higher than the Taiwan emission limit) even 18 h after the injection of activated carbon, indicating the memory effect. Taking the MSWI for example, which consists of four incinerators, the estimated annual PCDD/F emission from normal operational conditions was 0.112 g I-TEQ. However, one start-up procedure can generate ~60% of the PCDD/F emissions for one whole year of normal operations. And the PCDD/F emission, which is the result of the start-ups of four incinerators, was at least two times larger than that of a whole year's normal operations, without consideration for the PCDD/F emission contributed by the long lasting memory effect.

Keywords: PCDD/Fs; Start-up; Incinerator; Memory effect

1. Introduction

After polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) were discovered in the flue gases and fly ash of municipal solid waste incinerators (MSWIs) in 1977 (Olie et al., 1977), PCDD/Fs have become a serious issue in many countries because of their toxicological effects and associated adverse health implications.

Most research investigated the PCDD/F characteristics and the induced hazardous effect of incinerators during normal operations. Till recently, several studies (Gass et al., 2002; Löthgren and van Bavel, 2005; Neuer-Etscheidt et al., 2006) have focused on the high PCDD/F emission during the start-up of incinerators. The start-up of continuous MSWIs is usually a cold start-up, which occurs after a plant revision and consists of the following procedures: (1) Oil burning with a maximum fuel feed rate until the suitable temperature for incineration (850 °C) is reached. (2) Start of waste feeding and increasing feed rate until design load is reached (Gass et al., 2002). However, for intermittent incinerators, the start-up procedure is a warm start-up, which can be characterized by the furnace's remaining temperature when it is started up again the next day.

Löthgren and van Bavel (2005) measured the PCDD/F levels after a polishing wet scrubber continuously for

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18 months using long-time sampling equipment at a hazardous waste incineration facility in Sweden. Each sampling period lasted two weeks. Two dramatic risings of TEQ levels were observed, both in the period just after a maintenance stop of the plant, from 0.02 to 0.25 ng TEQ m^{-3} and from 0.03 to 0.15 ng TEQ m^{-3}. Gass et al. (2002) reported raw flue gas PCDD/F concentrations during the start-up of a MSWI of up to 250 ng I-TEQ m^{-3} in the heat-up phase. In contrast, Neuer-Etscheidt et al. (2006) reported that PCDD/F I-TEQ crude gas concentrations during the heat-up period were a little lower than those during normal operations with waste as the fuel. One reason for this difference may be the state of cleaning of the boiler section. Immediately after waste was fed, PCDD/F concentrations (46 ng I-TEQ m^{-3}) in the crude gas increased by one order of magnitude compared to normal operating conditions (3–4 ng I-TEQ m^{-3}). Even for a ship's main engine, the highest PCDD/F emissions have been measured for the start-up samples (0.1–0.4 ng WHO-TEQ kW h^{-1} vs 0.03–0.1 ng WHO-TEQ kW h^{-1} during normal operations), which are characterized by relatively poor combustion conditions (also high CO emissions) (Cooper, 2005). However, still little research estimated the generated PCDD/Fs during start-up to what extent. Consequently, it is not yet possible to evaluate the exposures and potential health risks during these conditions (Mckone and Hammond, 2000).

In this study, five intermittently operating incinerators, including one industrial waste incinerator (IWI), one waste liquid incinerator (WLI) and three medical waste incinerators (MWIs) were measured for PCDD/Fs in the stack flue gases when combustion condition reached stabilization after start-up to evaluate the influence of memory effect caused by start-up on PCDD/F emission. For verifying the PCDD/F characteristics of incinerators during start-up, one large scale continuously operating MSWI was chosen to be investigated for two years. In the first year, a total of five start-up stack flue gas samples were collected. For more detailed characterization, the next year, a total of 10 stack flue gas samples were sampled and the sampling time was changed so it's more accordant with the start of waste feeding. Only the 2,3,7,8-PCDD/F congeners in the stack flue gases of the incinerator were measured because of their toxicities. The obtained results were not only compared to the typical concentrations during normal operations of the MSWI but also evaluated for the PCDD/F emission during start-up.

2. Experimental section

2.1. Basic information concerning the incinerators

The basic operation information concerning the intermittent incinerators, including one IWI, one WLI and three MWIs is described in Table 1. The feeding waste and air pollution control devices (APCDs) between these three categories of incinerators are not similar, but each kind of incinerator possesses its representation.

The continuously operating MSWI investigated for two years in this study consists of four 450 ton day^{-1}, two-stage, starved-air modular incinerators, each of which includes its own heat recovery system, dry scrubber, activated carbon injection, bag filter and stack. During start-up, the incinerators were preheated by burners operated with diesel.

2.2. PCDD/F sampling

On a usual day, the intermittent incinerators were started up in the morning and operated during the day. At night, the combustion chamber cooled down. However, in this study, the feeding waste was accumulated to enough for three consecutive 3 h stack flue gas samples during normal operations after start-up, which is characterized by the stable reading of combustion temperature and traditional pollutant, like CO concentration.

The sampling time of each stack flue gas sample of the MSWI during start-up was about 2–3 h and a total of five samples were collected in the first year. For more detailed characterization, the next year, the sampling time was changed to once an hour and a total of 10 stack flue gas samples were sampled.

The PCDD/F samples were collected isokinetically from the stack flue gas of the selected incinerators according to US EPA modified Method 23. The sampling train adopted in this study is comparable with that specified by US EPA

Table 1
Basic information concerning these five intermittent incinerators

Emission sources	IWI	WLI	MWI	MWI	MWI
Denotation	A	B	C	D	E
Feeding waste (kg h^{-1})	Industrial waste (420)	Waste liquid (200)	Infectious and pathological waste (400)	Infectious and pathological waste (300)	Infectious and pathological waste (300)
Auxiliary fuel (l h^{-1})	–	Diesel (0.1)	Diesel (22)	Diesel (21)	Diesel (19)
APCDs in sequence (operation temperature) (°C)	Cyclone (200) BF (160)	VS (90)	DS (250) ACI BF (150)	QC VS (90)	DS (250) ACI BF (160)
Mean stack flue gas flow (N m^3 h^{-1})	8500	2900	4500	4000	6000

Activated carbon injection: ACI; Bag filter: BF; Dry scrubber: DS; Quench chamber: QC; Venturi scrubber: VS.

Modified Method 5. Prior to sampling, XAD-2 resin was spiked with PCDD/F surrogate standards pre-labeled with isotopes, including $^{37}Cl_4$-2,3,7,8-TCDD, $^{13}C_{12}$-1,2,3,4,7,8-HxCDD, $^{13}C_{12}$-2,3,4,7,8-PeCDF, $^{13}C_{12}$-1,2,3,4,7,8-HxCDF and $^{13}C_{12}$-1,2,3,4,7,8,9-HpCDF. The recoveries of PCDD/F surrogate standards were 101–123%, and met the criteria within 70–130%. To ensure the free contamination of the collected samples, one trip blank and one field blank were also taken during the field sampling was conducted. Details are similar to that given in our previous work (Wang et al., 2003).

2.3. Analyses of PCDD/Fs

Analyses of stack flue gas followed the US EPA modified method 23. All chemical analyses were carried out by the Super Micro Mass Research and Technology Center at Cheng Shiu University – the accredited laboratory in Taiwan for PCDD/F analyses. Prior to analysis, each collected sample was spiked with a known amount of the $^{13}C_{12}$-labeled internal standard to the extraction thimble. Add toluene to fill the reservoir approximately 2/3 full. Adjust the heat source to cause the extractor to cycle three times per hour. After being extracted for 24 h, the extract was concentrated, treated with concentrated sulfuric acid, and then followed by a series of sample cleanup and fractionation procedures, including multilayer silica gel column, alumina column and activated carbon chromatography. The eluate was concentrated to approximately 1 ml and transferred to a vial. The concentrate was further concentrated to near dryness, using a stream of nitrogen. Immediately prior to analysis, the standard solution for recovery checking was added to the sample. The recoveries of PCDD/F internal standards for the tetra- through hexachlorinated homologues were between 65% and 98%, and met the criteria within 40–130%, while that for the hepta- and octachlorinated homologues were between 52% and 101%, and met the criteria within 25–130%.

A high-resolution gas chromatograph/high-resolution mass spectrometer (HRGC/HRMS) was used for PCDD/Fs analyses. The HRGC (Hewlett Packard 6970 Series gas, CA, USA) was equipped with a DB-5MS fused silica capillary column ($L = 60$ m, ID = 0.25 mm, film thickness = 0.25 μm) (J&W Scientific, CA, USA), and with a splitless injection. Helium was used as the carrier gas. However, if a valley between peaks is more than 25% of the lower of the two peaks for 2,3,7,8-TCDD and 2,3,7,8-TCDF, the column had to be changed to DB-225 to recheck isomer. The oven temperature program was set according to follows: begin at 150 °C (held for 1 min), then increased at 30 °C min^{-1} to 220 °C (held for 12 min), then increased at 1.5 °C min^{-1} to 240 °C (held for 5 min), and finally increased at 1.5 °C min^{-1} to 310 °C (held for 20 min). The HRMS (Micromass Autospec Ultima, Manchester, UK) was equipped with a positive electron impact (EI+) source. The analyzer mode of the selected ion monitoring (SIM) was used with resolving power at 10000. The electron energy and source temperature were specified at 35 eV and 250 °C, respectively. Details of analytical procedures are given in our previous work (Wang et al., 2003).

3. Results and discussion

3.1. Influence of memory effect resulted from start-up on PCDD/F emissions of the intermittent incinerators

The PCDD/F profiles of three consecutive stack flue gas samples of these five intermittent incinerators were illustrated in Fig. 1 and revealed that the PCDD/F emission of the first stack flue gas sample, which was sampled at a stable combustion condition after start-up, was considerably higher than that of the follow-up samples. For toxicity basis, the first stack flue gas sample was 2–3 times higher than the mean PCDD/F emission of the second and third one. Fig. 1 also revealed that the PCDD/F profiles of the three consecutive stack flue gas samples for each incinerator declined with time. The reason for this is that the memory effect that resulted from the high PCDD/F emission of the start-up contributed and changed the subsequent concentration and profiles significantly. But as time went by, the influence of the memory effect on the PCDD/F emission faded away.

The memory effect caused by the wet scrubber resulted from that PCDD/Fs are adsorbed on scrubber fillings (typically polypropylene (PP) plastics) and thus leading to unexpected PCDD/F releases when the plant is running under more stable conditions (Hunsinger et al., 1998; Adams et al., 2000; Takaoka et al., 2003). Compared to that caused by start-up procedure, the memory effect caused by the start-up can occur in all kinds of incinerators with different APCDs, not only for those with wet scrubbers, for examples, the incinerators in this study are no exception to this.

3.2. PCDD/F emission in the stack flue gases of the MSWI under normal conditions

For more understanding of the memory effect that resulted from the start-up, one continuous MSWI was chosen to characterize the PCDD/F emission during start-up. In the first instance, we need to establish the PCDD/F characteristics of the MSWI during normal operation.

The stack flue gases of the MSWI were sampled five times in different months under normal operation conditions and a total of 25 samples were collected for PCDD/F measurement. The mean PCDD/F concentration (normalized to the dry flue gas conditions of 273 K and 11% O_2) in the stack flue gas was 0.0358 ng I-TEQ N m^{-3} (range: 0.00273–0.0946 ng I-TEQ N m^{-3}, relative standard deviations (RSD): 66.4%), while their corresponding mean PCDD/F emission factor was 0.249 μg I-TEQ ton $waste^{-1}$ (RSD: 69.0%), which is close to that (mean: 0.251 μg I-TEQ ton $waste^{-1}$, range: 0.0512–0.561 μg I-TEQ ton $waste^{-1}$) of the other 13 MSWIs in Taiwan (Lee et al.,

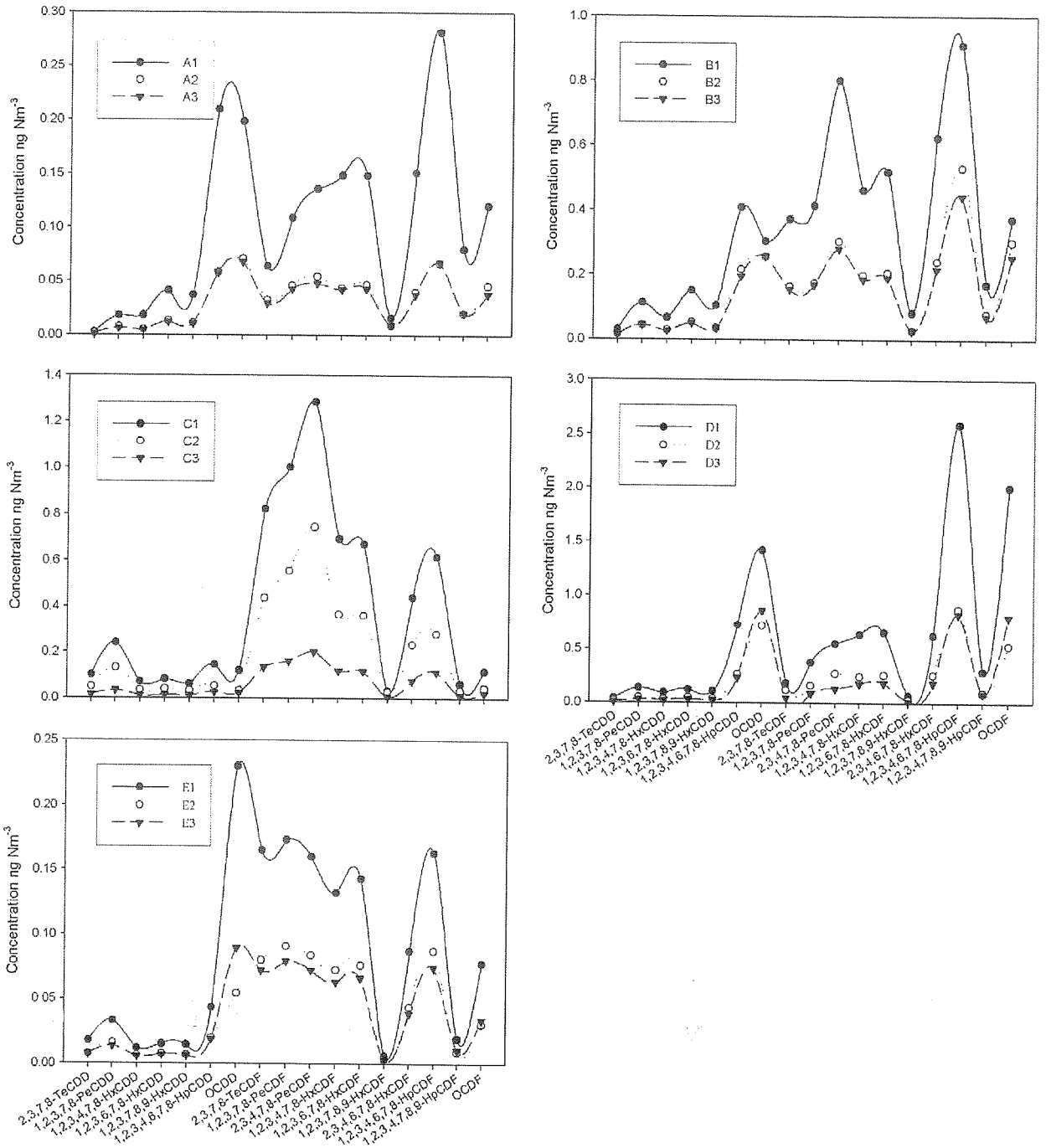

Fig. 1. The PCDD/F profiles of three consecutive stack flue gas samples of the five intermittent incinerators.

2005) and is comparable to that (0.17 μg I-TEQ ton waste^{-1}) of the MSWI (400 ton day^{-1}) in accordance with the best control technologies (Giugliano et al., 2002). Furthermore, five raw flue gas samples sampled in front of the activated carbon injection and bag filter were also measured for PCDD/Fs and the mean PCDD/F concentration was 0.610 ng I-TEQ N m^{-3} (range: 0.231–1.52 ng I-TEQ N m^{-3}, RSD: 86.1%).

According to statistical data, there were ~448 000 tonnes of waste incinerated by the MSWI investigated in this study in 2005. By directly adopting the mean emission factors that were obtained from this study (0.249 μg I-TEQ ton waste^{-1}), the total emission amount of PCDD/Fs from the MSWI is ~0.112 g I-TEQ yr^{-1}.

3.3. PCDD/F emission in the stack flue gases of the MSWI during start-up

Table 2 listed the operation conditions and their corresponding PCDD/F emission in the stack flue gases during

Table 2
PCDD/F emission in the stack flue gases of the MSWI during start-up in the first year's investigation

Sample denotations	Sampling day				
	Day one		Day two		
	a	b	c	d	e
Sampling time	15:30–17:40	19:25–20:55	23:40–3:15	10:10–12:35	13:42–16:10
Passed time after burner started (h)	5.5–7.7	9.4–11	13.7–17.3	24.2–26.6	28–30.2
Passed time after waste fed (h)	–	–	–	5.2–7.6	9–11.2
Auxiliary fuel ($l\,h^{-1}$)	660	830	650	4	7
Temperature of combustion chamber (°C)	310	490	850	900	920
Primary air flow rate ($N\,m^3\,h^{-1}$)	37500	34300	30900	50400	51000
Temperature in the inlet of superheater (°C)	190	240	360	490	500
Temperature in the inlet of economizer (°C)	150	210	300	330	340
O_2 Concentration after economizer (%)	22	22	22	11	7.3
Temperature of dry scrubber (°C)	130	160	210	240	230
Lime feeding rate ($m^3\,h^{-1}$)	0	0	0.6	2.2	4.0
Temperature of bag filter (°C)	120	150	180	160	150
Activated carbon feeding rate ($kg\,h^{-1}$)	0	0	6	10	10
CO concentration in the stack flue gas (ppm)	–	–	–	42	32
O_2 concentration in the stack flue gas (%)	18	17	14	8.3	8.7
PCDFs/PCDDs ratio	0.66	0.62	0.72	0.92	0.75
Total PCDD/Fs ($ng\,N\,m^{-3}$)	83.8	328	68.2	42.9	10.7
PCDFs/PCDDs TEQ ratio	3.0	2.1	2.4	2.6	2.4
Total I-TEQ ($ng\ I\text{-}TEQ\,N\,m^{-3}$)	2.09	12.2	3.34	2.28	0.507

–: No data.

the cold start-up of the MSWI in the first year's investigation. The PCDD/F concentrations (normalized to the dry flue gas conditions of 273 K) in the stack flue gases during the heat-up phase (samples a–c) were ranged at 2.09–12.2 ng I-TEQ $N\,m^{-3}$ while those that were ranged at 0.507–2.28 ng I-TEQ $N\,m^{-3}$ were observed 5 h after the waste feeding process had started.

For more detailed characterization, during the following year, the sampling time was changed to once an hour so it's more accordant with the start of waste feeding (see Table 3). The PCDD/F concentration in the stack flue gases during the heat-up phase (samples a–d) was between 5.16 and 41.5 ng I-TEQ $N\,m^{-3}$, which was higher than the first year's results (2.09–12.2 ng I-TEQ $N\,m^{-3}$). However, the drastic PCDD/F emissions in the stack flue gases during the heat-up phase of the two years' investigation both occurred at the combustion chamber's temperature reaching 490 °C. When the combustion chamber's temperature reaching 490 °C, the temperatures in the inlet of the superheater and economizer were between 210 °C and 290 °C, which were a little below the temperature region (250–450 °C) of the PCDD/F optimal formation. Otherwise, Clarke (2000), Gass et al. (2003) and Neuer-Etscheidt et al. (2006) reported that PCDD/F precursors (e.g., benzenes, phenols and chlorinated forms) are created in the furnace at the highest rate in these temperature regions. In fact, Benestad et al. (1990) reported that PAHs concentration during the start-up period was 2–16 times higher than that during normal operation for three small MSWIs. Yasuda and Takahashi (1998) also determined the PAHs emission of four MSWIs during start-up, burning, and burn-out and reported that during start-up conditions, PAH concentrations (7.7–248 $\mu g\,N\,m^{-3}$) have been shown to be 12–77 times higher during burning (0.1–63.2 $\mu g\,N\,m^{-3}$). Consequently, the drastic PCDD/F emissions occurred during the heat-up phase is more related to the operational temperature of the combustion chamber than to that of superheater and economizer.

But when the temperature of the combustion chamber raised from 490 °C to 850 °C, the PCDD/F emissions decreased from 12.2 ng I-TEQ $N\,m^{-3}$ (no activated carbon injection) to 3.34 ng I-TEQ $N\,m^{-3}$ (activated carbon feeding rate: $6\,kg\,h^{-1}$) during the heat-up phase in the first year's investigation. The injection of activated carbon and the elevated temperature of the combustion chamber, i.e. the more completely combustion condition, may be the reasons of drastic reduction of the PCDD/F emissions, however, due to the intervals of up to several hours where no samples were taken, which one is the most influence factor is needed to be clarified further.

In the first year, the temperature of the combustion chamber reached 490 °C after burning for 10 h with the average auxiliary fuel feeding rate of $710\,l\,h^{-1}$ and the average primary air of $35900\,N\,m^3\,h^{-1}$ while in the second year's investigation, reaching the same combustion condition with a higher average auxiliary fuel feeding rate of $1150\,L\,h^{-1}$ and a less primary air of $27700\,N\,m^3\,h^{-1}$. The results revealed that the worse combustion efficiency occurred in the second year and might result in the PCDD/F concentration in the stack flue gases during the heat-up phase (5.16–41.5 ng I-TEQ $N\,m^{-3}$) which has a recording that is higher in the second year than that of the first year (2.09–12.2 ng I-TEQ $N\,m^{-3}$). Otherwise, Neuer-Etscheidt et al. (2006) proposed that the state of

Table 3
PCDD/F emission in the stack flue gases of the incinerator during start-up in the second year's investigation

Sample denotations	Sampling day									
	Day one			Day two						
	a	b	c	d	e	f	g	h	i	j
Sampling time	18:00–19:00	20:30–21:25	23:10–00:07	01:50–02:57	05:00–06:08	09:50–11:09	12:45–13:58	15:20–16:30	18:04–19:09	20:30–21:41
Passed time after burner started (h)	2–3	4.5–5.4	7.2–8.1	9.8–11	13–14.1	17.8–19.2	20.8–22	23.3–24.5	26.1–27.2	28.5–29.7
Passed time after waste fed (h)	–	–	–	–	1–2.1	5.8–7.2	8.8–10	11.3–12.5	14.1–15.2	16.5–17.7
Auxiliary fuel ($l\ h^{-1}$)	1100	1150	1300	1060	525	2	0	0	0	0
Temperature of combustion chamber (°C)	155	230	400	490	900	900	910	880	900	900
Primary air flow rate ($N\ m^3\ h^{-1}$)	32000	28700	26700	23200	53800	39100	39800	44100	48600	43900
Temperature in the inlet of superheater (°C)	140	190	220	290	360	460	490	490	500	500
Temperature in the Inlet of economizer (°C)	110	160	190	250	310	330	340	340	350	350
O_2 concentration after economizer (%)	19	19	17	17	7.0	9.2	9.1	10	8.6	8.8
Temperature of dry scrubber (°C)	90	140	180	210	210	220	230	230	230	230
Lime feeding rate ($m^3\ h^{-1}$)	0	0	0	0	0	0	0	0	0	0
Temperature of bag filter (°C)	80	130	170	170	160	160	150	150	150	150
Activated carbon feeding rate ($kg\ h^{-1}$)	0	0	0	0	6	6	6	6	6	6
CO concentration in the stack flue gas (ppm)	–	–	–	–	–	49	43	53	37	21
O_2 concentration in the stack flue gas (%)	19	19	13	9.2	8.9	9.1	8.3	8.9	10	8.8
PCDFs/PCDDs ratio	0.64	0.59	0.95	1.0	1.0	0.69	0.74	0.67	0.69	0.68
Total PCDD/Fs ($ng\ N\ m^{-3}$)	180	259	111	384	909	339	86.3	99.7	66.5	64.3
PCDFs/PCDDs TEQ ratio	4.1	3.4	4.0	1.7	2.0	1.9	2.0	2.0	2.1	2.0
Total I-TEQ ($ng\ I\text{-}TEQ\ N\ m^{-3}$)	6.19	8.64	5.16	41.5	96.9	23.1	6.18	6.41	4.30	4.19

–: No data.

cleaning of the boiler section could also resulted in the PCDD/F crude gas concentrations during the heat-up period ranging from a little lower than those during normal operations to 250 ng I-TEQ N m^{-3}, which was reported by Gass et al. (2002).

Another drastic elevated PCDD/F concentrations (from 41.5 to 96.9 ng I-TEQ N m^{-3}) in the stack flue gas occurred one hour after waste feeding (Table 3) and it declined to 23.1 ng I-TEQ N m^{-3} five hours after waste feeding, but still maintained high PCDD/F concentration, 4.19 ng I-TEQ N m^{-3}, 40 times higher than the Taiwan emission limit of 0.1 ng I-TEQ N m^{-3} even 18 h after the injection of activated carbon, indicating the long lasting memory effect. Neuer-Etscheidt et al. (2006) also observed that immediately after waste was fed, PCDD/F concentrations (46 ng I-TEQ m^{-3}) in the crude gas increased by one order of magnitude compared to normal operating conditions (3–4 ng I-TEQ m^{-3}). In this study, the raw gas in front of the APCD had not been sampled so that it is unclear that if the raw gas PCDD/F levels were still elevated at 18 h after the injection of activated carbon. However, the CO concentration (21 ppm, see Table 3) at that time was already in the range of normal operation condition, representing the well combustion condition and little PCDD/F precursors, like benzenes, phenols and PAHs formed, so we extrapolated that the high PCDD/F concentration, 4.19 ng I-TEQ N m^{-3} at 18 h after the injection of activated carbon, resulting from rather the memory effect than the incomplete combustion at that time.

During the heat-up phase, no activated carbon is injected prior to the bag filter in order to avoid ignitions of the activated carbon due to the high oxygen concentration in the flue gas (see Tables 2 and 3). Although it may be one of the reason that the PCDD/F concentration in the stack flue gases was between 5.16 and 41.5 ng I-TEQ N m^{-3}, however, by comparing to the PCDD/F concentration in the raw flue gases in front of the activated carbon injection and bag filter of the MSWI during normal operation (mean: 0.610 ng I-TEQ N m^{-3}, range: 0.231–1.52 ng I-TEQ N m^{-3}), it revealed that a high PCDD/F emission during the heat-up phase occurred due to the low furnace temperature and the poor combustion condition. The surfaces of the boiler and pipe were thus contaminated by the soot particles as well as hydrocarbons, which improved de novo synthesis of PCDD/F formation during the subsequent increase of the temperatures at these locations. Otherwise, the high PCDD/F emission occurred during the start-up procedure also increased the PCDD/F adsorption on the APCDs, like wet scrubber and bag filter and then released over a longer period of time.

3.4. PCDD/F emission quantity of the MSWI during start-up

The profile of the PCDD/F emission rate of stack flue gas with time during start-up of a two year investigation is illustrated in Fig. 2 and the shadowed areas represent

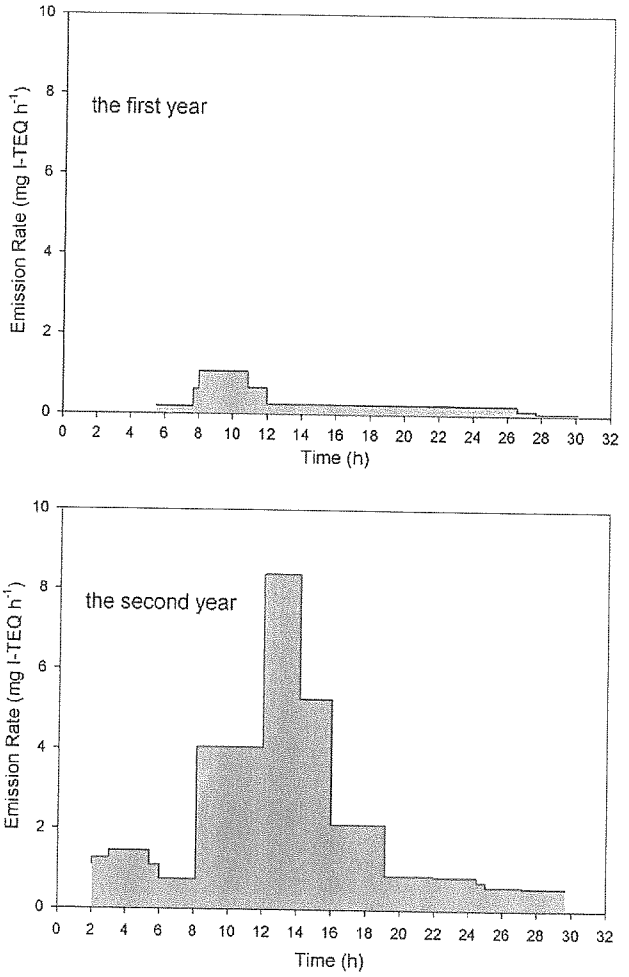

Fig. 2. PCDD/F emission rate from the stack flue gases of the MSWI during startup.

the PCDD/F emission quantity of MSWI during start-up. It revealed that one PCDD/F emission peak exists when the combustion chamber's temperature reaches 490 °C in both the first and second year. However, owing to the inexperience regarding the sampling strategy, another high PCDD/F emission peak resulted from the poor combustion when waste being fed was not observed in the first year's investigation. After changing the sampling time to be more accordant with the start of waste feeding, the PCDD/F profile clearly showed the phenomenon of PCDD/F generation and two high PCDD/F emission peaks with subsequently slow declination on emission in the second year's investigation. After calculating the shadowed areas, the estimated PCDD/F emission quantity during start-up was 0.0658 g I-TEQ in the second year's investigation.

Incinerators are usually at least shut-down and started-up once a year for maintenance. Taking the MSWI investigated in this study for example, which consists of four incinerators, the estimated annual PCDD/F emission from normal operational conditions was 0.112 g I-TEQ. However, one start-up procedure can generate ~60% of that and the PCDD/F emission quantity resulted from

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Emissions of Polychlorinated Dibenzo-p-dioxins and Dibenzofurans from Various Stationary Sources

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Abstract

This work investigated the characteristics of polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/F) in stack-flue gases from six stationary emission sources in five types of incinerators: industrial waste incinerator (IWI), small-scale municipal solid waste incinerator (MSWI), medical waste incinerator (MWI), cement kilns (CK), and crematories (CR). These characteristics were further investigated using factor analysis and cluster analysis. Experimental results reveal that PCDDs dominate MSWI and CR, and PCDFs dominate IWIa, IWIb, CK and MWIs. The factor analysis results showed that CR and MSWI have similar fingerprints, as do IWIb and MWI3. The cluster analysis showed that if a vertical line is cut at a rescaled distance of four, then the PCDD/F congener profiles fall into four groups. The indicators of PCDD/Fs are OCDD, 1,2,3,4,6,7,8-HpCDF, 2,3,4,7,8-PeCDF, and 1,2,3,4,6,7,8-HpCDD. The emission factors of PCDD/Fs herein were from 0.0433 (CK) to 18.7 (MSWI) μg I-TEQ/ton-feedstock.

Keywords: Dioxin emission, incinerators, congener profile, factor analysis, cluster analysis

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INTRODUCTION

Dioxins, especially 2,3,7,8-TCDD, are of great concern, because they are highly carcinogenic. PCDD/Fs are formed during combustion from: (i) precursors, (ii) de novo, and (iii) through pyrosynthesis (Tuppurainen et al., 2003). Major sources of atmospheric PCDD/Fs include stationary emissions, especially from various incinerators, including secondary aluminum smelters (ALS), sinter plants, small-scale municipal solid-waste incinerators (MSWI), medical-waste incinerators (MWI), electric-arc furnaces (EAF), industrial-waste incinerators (IWI), cement kilns and crematoria. Hence, this work studies PCDD/F emissions from MSWI, MSI, IWI, crematory cement kilns (CK), and cement crematoria (CR).

Cement kilns use coal as burner fuel; the other sources use diesel. The feedstock of CK is mostly cement as a raw material and a few waste tires, IWIa is fed with general waste from nuclear power plants, and IWIb is fed mostly with waste-oil sludge. For pollution control, MSWI uses a semi-dry washing tower and a baghouse, MWI uses a Venturi scrubber and a quench tower, CK uses an electrostatic precipitation (ESP), IWIa uses a baghouse, and IWIb uses a cyclone and an ESP.

This paper reports on measurements of PCDD/Fs from the stacks of six incinerators. Emission characteristics of PCDD/Fs from these incinerators are presented, including concentrations, fingerprints and emission factors. Also, similar and dissimilar features between them are further studied using factor analysis and cluster analysis. The data derived from this study can provide guidance to improve operating conditions of the incinerators and to assess the potential health risk to the neighboring community.

EXPERIMENTAL

PCDD/FS Sampling

PCDD/Fs in stack-flue gases were collected using the Taiwan EPA method NIEA A807.73C, which is based on the US EPA Method 23A. Prior to sampling, XAD-2 resin was spiked with isotopically labelled PCDD/F surrogate standards. Each stack gas sampling took 3 h. One trip blank and one field blank were also obtained during field sampling to ensure that the collected samples were not contaminated.

PCDD/FS Analysis

PCDD/Fs were analyzed for stack-flue gases, according to U.S. EPA modified Method 23, using high-resolution gas chromatographs/high-resolution mass spectrometers (HRGC/HRMS). The analysis was conducted at the Super Micro Mass Research and Technology Center at Cheng Shiu University in Taiwan. The HRGC (Hewlett-Packard 6970) was comprised of a DB-5 MS

fused silica capillary column (0.25 mm × 60 m, 0.25 μm) (J&W Scientific) with splitless injection. Helium was used as the carrier gas. The HRMS (Micormass Autospec Ultima, Manchester, UK) had a positive electron impact (EI+) source. The selected ion-monitoring mode (Park et al., 2004) had a resolving power of 100,000. The specified electron energy and source temperature were 35 eV and 250 °C, respectively.

RESULTS AND DISCUSSION

PCDD/F Concentrations in Stacks

Table 1 shows that the total PCDD/F concentrations in the stack gases of IWIa, IWIb, MSWI, MWI, CK, and CR were 0.604, 1.397, 30.1, 1.14, 0.350, and 29.8 ng/Nm^3, respectively, and the order was MSWI > CR > IWIb > MWI > IWIa > CK, indicating that PCDD/F concentrations in the stacks of MSWI and CR greatly exceeded those of other incinerators. The efficiency of a baghouse in removing PCDD/Fs was around 37.6% (Lee et al., 2004); so, highly concentrated PCDD/Fs could be formed from MSWI during combustion. CR should include pollution control equipment to reduce the concentration of PCDD/Fs in the stack-flue gases. The PCDD to PCDF ratios were 0.464, 0.415, 0.903, 0.423, 0.292, and 0.869, respectively. The PCDD/Fs ratios in the stacks followed the order CR > MSWI > IWIa > MWI > IWIb > CK, indicating that PCDDs dominated CR and MSWI and PCDFs dominated the other burners. The total PCDD/Fs I-TEQs were 0.030, 0.137, 3.35, 0.168, 0.062, and 3.00 ng I-TEQ/Nm^3, respectively. The order of total PCDD/F I-TEQ was MSWI > CR > MWI > IWIb > CK> IWIa, similar to that of the total PCDD/F concentration in stacks.

In summary, when considering PCDD/F concentrations, PCDD/F ratio and PCDD/F I-TEQ, MSWI and CR are two leading PCDD/F contributors among the six incinerators.

PCDD/F Congener Profiles of Stationary Emission Sources

Fig. 1 presents the congener profiles of the 17 2,3,7,8-chlorinated substituted PCDD/Fs detected in six stack-flue gases. Each selected congener was normalized to the total weight of all 2,3,7,8-congeners (mean ± SD). The PCDD/F congener profile of IWIa was dominated by 1,2,3,4,6,7,8-HpCDF, OCDD, and OCDF, while that of IWIb was dominated by 1,2,3,4,6,7,8-HpCDF, OCDD, and 2,3,4,7,8-PeCDF. The profile of MSWI was dominated by 1,2,3,4,6,7,8-HpCDD, OCDD, and 1,2,3,4,6,7,8-HpCDF. The profile of MWI was dominated by 2,3,4,7,8-PeCDF, 1,2,3,4,6,7,8-HpCDF, and 1,2,3,7,8-PeCDF. The profile of CK was dominated by 2,3,7,8-TeCDF, 1,2,3,7,8-PeCDF, and 2,3,4,7,8-PeCDF. However, the profile of CR was dominated by 1,2,3,4,6,7,8-HpCDF, 1,2,3,4,6,7,8-HpCDD, and OCDD.

Fig.1 also indicates that MSWI and CR were dominated by PCDD, and IWIa, IWIb, CK and MWI were dominated by PCDF. Moreover, the major and minor dominating congener profiles of IWIb were similar to those of IWIa; and MSWI and CR had similar congener profiles.

Table 1. The PCDD/F concentrations in the stack-flue gases of six emission sources.

PCDD/PCDFs (ng/Nm^3)	IWI a (n=3)		IWI b (n=3)		MSWI (n=3)		MWI (n=3)		CK (n=3)		CR (n=3)	
	mean	RSD(%)	mean	RSD(%)	mean	RSD(%)	mean	RSD(%)	mean	RSD(%)	mean	RSD(%)
2,3,7,8-TeCDD	0.001	12.7	0.006	13.8	0.29	36.0	0.016	88.6	0.018	139	0.319	27.0
1,2,3,7,8-PeCDD	0.003	17.9	0.022	6.67	1.18	37.7	0.042	92.8	0.01	105	1.15	23.7
1,2,3,4,7,8-HxCDD	0.005	32.6	0.013	12.5	0.823	43.9	0.02	93.3	0.003	57.1	0.834	23.3
1,2,3,6,7,8-HxCDD	0.009	26.8	0.022	11.5	1.41	43.8	0.044	92.3	0.004	50.6	1.39	16.7
1,2,3,7,8,9-HxCDD	0.006	27.3	0.018	4.84	0.987	43.9	0.042	91.5	0.003	52.8	1.40	17.9
1,2,3,4,6,7,8-HpCDD	0.056	38.4	0.117	12.0	5.19	51.1	0.089	74.8	0.014	39.6	4.55	28.1
OCDD	0.105	42.2	0.213	36.0	4.38	55.8	0.076	43.0	0.022	24.3	4.05	47.1
2,3,7,8-TeCDF	0.009	14.7	0.064	9.53	1.54	33.5	0.095	81.3	0.114	119	1.66	33.7
1,2,3,7,8-PeCDF	0.013	18.0	0.096	8.06	1.64	43.6	0.11	86.5	0.04	95.3	1.25	32.5
2,3,4,7,8-PeCDF	0.023	23.9	0.127	18.4	2.49	44.7	0.152	88.5	0.039	78.9	1.82	23.3
1,2,3,4,7,8-HxCDF	0.03	41.5	0.116	14.1	1.69	46.4	0.096	88.0	0.013	40.6	1.65	37.5
1,2,3,6,7,8-HxCDF	0.032	39.2	0.122	12.4	1.82	48.6	0.105	90.5	0.018	42.9	1.76	34.9
2,3,4,6,7,8-HxCDF	0.004	116.4	0.009	11.7	0.086	50.2	0.004	76.5	0.001	173	0.061	30.0
1,2,3,7,8,9-HxCDF	0.036	38.5	0.107	15.4	2.17	50.5	0.067	86.1	0.016	42.9	1.60	32.9
1,2,3,4,6,7,8-HpCDF	0.149	58.1	0.232	16.0	3.43	55.0	0.125	71.9	0.022	32.0	5.02	46.3
1,2,3,4,7,8,9-HpCDF	0.02	56.8	0.035	18.3	0.397	57.7	0.015	64.0	0.004	40.8	0.357	40.6
OCDF	0.102	63.0	0.077	37.2	0.567	58.3	0.044	30.6	0.009	15.2	0.964	51.3
PCDDs	0.185	34.4	0.412	23.5	14.3	49	0.329	75.4	0.074	69.8	13.7	29.2
PCDFs	0.419	49.3	0.985	15.7.	15.8	47.7	0.813	80.8	0.276	85.3	16.1	36.1
PCDD/Fs ratio	0.464	20.3	0.415	7.92	0.903	9.43	0.423	12.4	0.292	14.3	0.869	11.6
Total PCDD/Fs	0.604	43.8	1.397	17.9	30.1	47.9	1.14	79.2	0.35	82	29.8	32.8
PCDDs ng I-TEQ/Nm^3	0.005	21.6	0.024	8.5	1.26	39.3	0.049	90.7	0.024	128	1.30	22.9
PCDFs ng I-TEQ/Nm^3	0.025	30.7	0.113	15.8	2.10	44.8	0.12	87.6	0.038	87.1	1.70	28.6
PCDD/Fs TEQ ratio	0.21	10.3	0.217	10.3	0.616	9.47	0.395	7.42	0.495	53.3	0.78	9.18
Total ng I-TEQ/Nm^3	0.030	29.1	0.137	14.3	3.35	42.7	0.168	88.5	0.062	103	3.00	25.8

Factor Analysis

To understand the underlying factors affecting the formation of PCDD/Fs in incinerators, factor analysis (FA) was employed. In this work, factors were extracted using principal component analysis (PCA), which involves varimax orthogonal rotation to determine the eigen values of variance matrix of variables (Wilkinson et al., 1996; Johnson and Wichern, 2002). Usually, those factors with eigen values exceeding unity were chosen. In Table 2, two factors, Factor1 and Factor2, are shown corresponding to the two leading eigenvalues of 6.251 and 5.506,

respectively. Factor1 explains 39.9% of all variance, and Factor2 explains 29.0% of all variance; altogether representing 68.9% of the total variance. Factor1 was strongly related (> 0.7) to: 1,2,3,7,8-PeCDD, OCDD; 2,3,4,7,8-PeCDF; 1,2,3,4,6,7,8-HpCDF; 1,2,3,4,7,8,9-HpCDF; and OCDF. Factor2 was strongly related (> 0.7) to: 1,2,3,4,7,8-HxCDD; 1,2,3,6,7,8-HxCDD; 1,2,3,7,8,9-HxCDD; and 1,2,3,4,6,7,8-HpCDD.

Also, Fig. 1 shows that Factor1 explains the major congeners of IWIa, IWIb, and MWI; while both Factor1 and Factor2 explain the major congeners of MSWI, CK, and CR. Chlorine-containing wastes and precursors are known to be responsible for the formation of PCDD/Fs (Dickson et al., 1989; Milligan and Altwicker, 1993); therefore, Factor1 and Factor2 may likely represent these two factors—chlorine-containing wastes and precursors; the latter is associated with the products of incomplete combustion. Notably, formation of PCDD/Fs in relatively low temperatures (250–350 °C) in dust-control device is also one important mechanism (Dickson et al., 1992; Luijk et al., 1994).

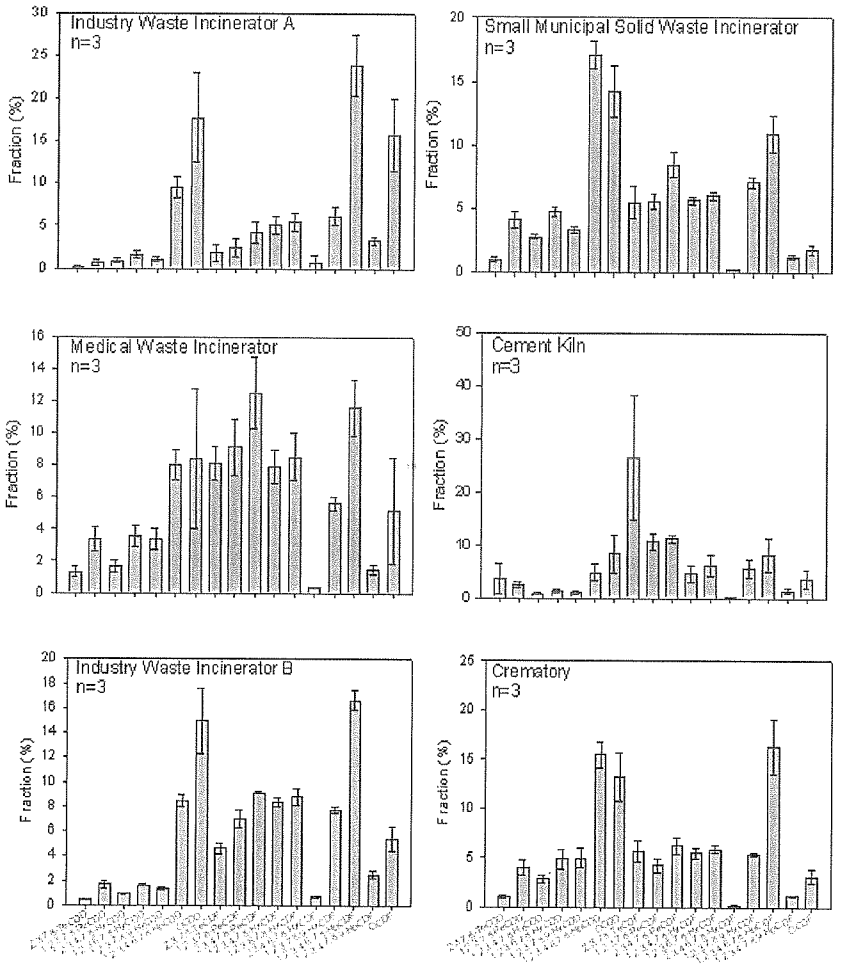

Fig. 1. Congener profiles of seventeen 2,3,7,8 -PCDD/Fs of various emission sources.

Fig. 2 shows the component plot, with Factor1 as the horizontal axis and Factor2 as the vertical axis. In the plot, the closeness of the emission sources to each other implies the similarity in their congener profiles. The plot shows that CR and MSWI have similar fingerprints; similarly for IWIb and MWI3.

Table 2. Factor analysis of PCDD/Fs in six stack-flue gases.

PCDD/Fs	Factor1	Factor2
2,3,7,8-TeCDD	.608	-.584
1,2,3,7,8-PeCDD	**.872**	.446
1,2,3,4,7,8-HxCDD	.519	**.845**
1,2,3,6,7,8-HxCDD	.539	**.825**
1,2,3,7,8,9-HxCDD	.560	**.748**
1,2,3,4,6,7,8-HpCDD	.086	**.952**
OCDD	**-.727**	.353
2,3,7,8-TeCDF	.556	**-.708**
1,2,3,7,8-PeCDF	.698	-.660
2,3,4,7,8-PeCDF	**.700**	-.440
1,2,3,4,7,8-HxCDF	.028	.041
1,2,3,6,7,8-HxCDF	.114	-.101
2,3,4,6,7,8-HxCDF	-.555	-.057
1,2,3,7,8,9-HxCDF	-.224	.181
1,2,3,4,6,7,8-HpCDF	**-.863**	.276
1,2,3,4,7,8,9-HpCDF	**-.959**	-.149
OCDF	**-.879**	-.131
Eigen value	6.251	5.506
Percentage of total variance	39.9	29.0

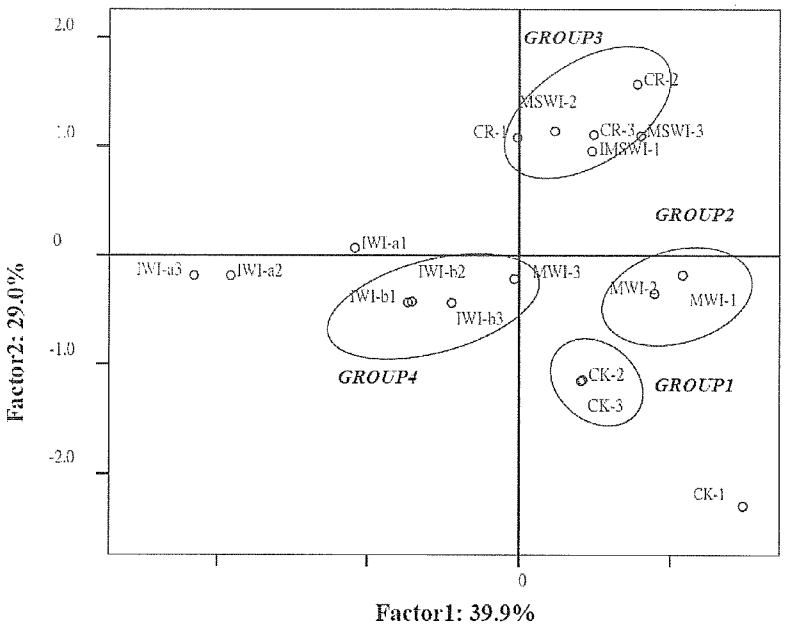

Fig. 2. Component plot from factor analysis.

Cluster Analysis

Cluster analysis, using nearest-neighbour method, was employed to divide the congener profiles from different stack-flue gases into several groups (Johnson and Wichern, 2002). The dendrogram in Fig. 3 from cluster analysis shows that if a vertical line is cut at a rescaled distance of four, then the PCDD/F congener profiles fall into four groups, namely: GROUP1, GROUP2, GROUP3, and GROUP4. GROUP1 comprises CK2 and CK3, and GROUP2 comprises MWI1 and MWI2. GROUP 3 comprises MSWI (MSWI1–MSWI3) and CR (CR1–CR3) which is consistent with the results of similar fingerprints in these two processes discussed earlier. GROUP4 is comprised of IWIb (IWIb1–IWIb3) and MWI3. IWIa (IWIa1–IWIa3) and CK1 do not belong to any group. Generally, cluster analysis results (Fig. 3) agree fairly well with factor analysis results shown in Fig. 2.

Indicators of PCDD/FS

Table 3 shows, based on the results of indicatory PCDD/F analysis, that IWIa, IWIb, MSWI, MWI, and CR have similar indicatory PCDD/Fs, which are OCDD, 1,2,3,4,6,7,8-HpCDF, 2,3,4,7,8-PeCDF, and 1,2,3,4,6,7,8-HpCDD. However, CK has quite different indicatory PCDD/Fs from the other five incinerator types. This may be due to the fact that the feedstock components of CK contain fewer organic compounds than the other incinerators do.

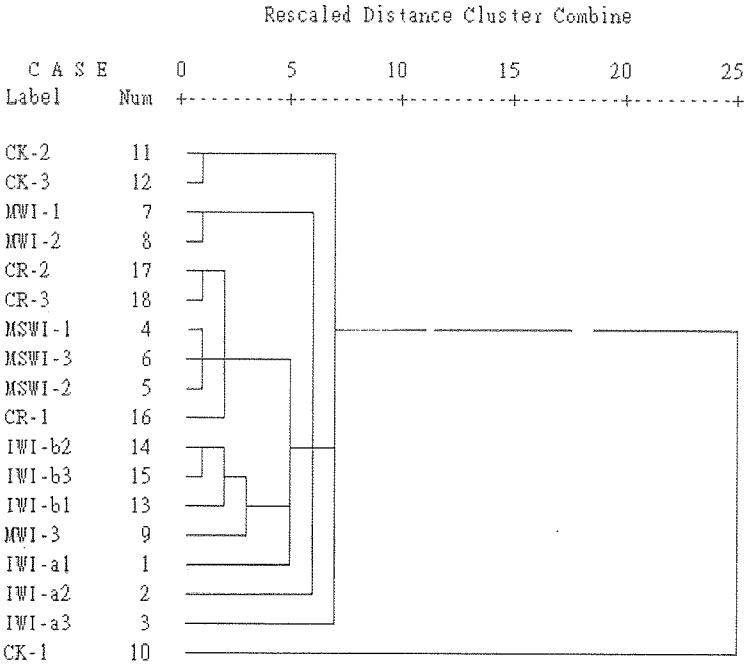

Fig. 3. Dendrogram from cluster analysis.

Table 3. The highest three indicatory PCDD/Fs of various PCDD/F emission sources.

PCDD/F emission sources	Indicatory PCDD/Fs (ratio)
IWIa – Industry Waste Incinerator	1,2,3,4,6,7,8-HpCDF (0.149)
	OCDD (0.105)
	OCDF (0.102)
IWIb – Industry Waste Incinerator	1,2,3,4,6,7,8-HpCDF (0.232)
	OCDD (0.213)
	2,3,4,7,8-PeCDF (0.127)
MSWI – Small Municipal Solid Waste Incinerator	1,2,3,4,6,7,8-HpCDD (5.193)
	OCDD (4.38)
	1,2,3,4,6,7,8-HpCDF (3.433)
MWI – Medical Waste Incinerator	2,3,4,7,8-PeCDF (0.152)
	1,2,3,4,6,7,8-HpCDF (0.125)
	1,2,3,7,8-PeCDF (0.110)
CK – Cement Kiln	2,3,7,8-TeCDF (0.114)
	1,2,3,7,8-PeCDF (0.04)
	2,3,4,7,8-PeCDF (0.0385)
CR – Crematory	1,2,3,4,6,7,8-HpCDF (5.020)
	1,2,3,4,6,7,8-HpCDD (4.547)
	OCDD (4.047)

Table 4. PCDD/F emission factors of various emission sources.

Stationary Source	Emission factor (μg I-TEQ/ton-feedstock)	Reference
IWIs with baghouse	0.059 ± 31.3	This study
IWIs with cyclone and ESP	0.368 ± 14.3	This study
MSWIs with semi dry washing tower and baghouse	18.7 ± 43.2	This study
MWIs with Venturi scrubber and quench tower	3.70 ± 88.7	This study
CK with ESP	0.0433 ± 103	This study
CR without pollution control equipment	41.1 ± 27.6*	This study
Secondary ALS	50.1 ± 62.5	(Chen et al., 2004)
Secondary ALS	0.63 - 200	(Lee et al., 2005)
Sinter plants with SCR	0.970	(Wang et al., 2003b)
Sinter plants without SCR	3.13	(Wang et al., 2003b)
EAFs	0.52 - 3.2	(Lee et al., 2005)
CR with bag filter	6.11*	(Wang et al., 2003a)
CR without bag filter	13.6*	(Wang et al., 2003a)

* Unit: μg I-TEQ/body

Emission Factors of PCDD/FS

Table 4 shows that the emission factors of PCDD/Fs herein were from 0.0433 (CK) to 18.7 μg I-TEQ/ton-feedstock (MSWI), whereas previous studies yielded 0.52 to 200 μg I-TEQ/ ton-feedstock (Wang et al., 2003a and 2003b). The PCDD/F emission factors of CR were 41.1 μg I-TEQ/ body herein, a value which is around three times greater than the value reported by Wang et al. (2003a). The PCDD/F emission factors of MSWI and MWI ranked second and third herein.

It is known that precursors are responsible for the formation of PCDD/Fs. Therefore, the control of feedstock components and the complete combustion of wastes are important in reducing the emission of PCDD/Fs from the stack-flue gas.

CONCLUSIONS

(1) The total PCDD/F I-TEQ concentrations in the stack gases of IWIa, IWIb, MSWI, MWI, cement kiln (CK) and crematory (CR) were 0.030, 0.137, 3.352, 0.168, 0.062, and 3.003 ng I-TEQ/Nm3, respectively. The total PCDD/F I-TEQ followed the order MSWI > CR > MWI > IWIb > CK > IWIa.

(2) The factor analysis results showed that CR and MSWI have similar fingerprints; similarly between IWIb and MWI3. The cluster analysis showed that if a vertical line is cut at a rescaled distance of four, then the PCDD/F congener profiles fall into four groups. These two analyses generally yielded consistent results.

(3) The indicators of PCDD/Fs of IWIa, IWIb, MSWI, MWI, and CR were very similar. They were OCDD, 1,2,3,4,6,7,8-HpCDF, 2,3,4,7,8-PeCDF, and 1,2,3,4,6,7,8-HpCDD, which were quite different from those of CK.

(4) The emission factors of PCDD/Fs herein were between 0.0433 (CK) and 18.7 (MSWI) μg I-TEQ/ton-feedstock.

Since precursors are responsible for the formation of PCDD/Fs, additional studies should be conducted to provide further understanding on their formation mechanisms during combustion-related processes.

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REFERENCES

Chen, S.-J., Lee, W.-S., Guo-Ping, C.-C., Wang, L.-C., Lee, W.-J., Kao, J.-H. and Hu, M.T.M.-T., (2004). Characterizing polychlorinated dibenzo-p-dioxins and dibenzofurans in the surrounding environment and workplace of a secondary aluminum smelter. *Atmospheric Environment.* 38(22): 3729-3732.

Dickson, L.-C., Lenoir, D. and Hutzinger, O. (1989). Surface-catalyzed formation of chlorinated dibenzodioxins and dibenzofurans during incineration. *Chemosphere.* 19: 277-282.

Dickson, L.-C., Lenoir, D. and Hutzinger, O. (1992). Quantitative comparison of de Nova and formation of polychlorinated dibenzo-p-dioxins under simulated municipal solid waste incinerator post-combustion conditions. *Environmental Science and Technology.* 26: 1822-1828.

Johnson, R.-A. and Wichern, D.-W. (2002). *Applied Multivariate Statistical Analysis.* 5th ed., Prentice-Hall, Inc., New Jersey, USA.

Lee, W.-S., Chang-Chien, G.-P., Chen, S.-J., Wang, L.-C., Lee, W.-J. and Wang, Y.-H., (2004). Removal of Polychlorinated Dibenzo-p-dioxins and Dibenzofurans in Flue Gases by Venturi Scrubber and Bag Filter. *Aerosol and Air Quality.* 4(1): 27-37.

Lee, W.-S., Guo-Ping, C.-C. and Wang, L.-C., (2005). Emissions of Polychlorinated Dibenzo-p-Dioxins and Dibenzofurans from Stack Gases of Electric Arc Furnaces and Secondary Aluminum Smelters. *Journal of the Air & Waste Management Association.* 55: 219-226.

Luijk, R., Akkerman, D.-M., Slot, P., Olie, K. and Kapteijn, F. (1994). Mechanism of formation of polychlorinated dibenzo-p-dioxins and dibenzofurans in the catalyzed combustion of carbon. *Environmental Science and Technology.* 28: 312-321.

Milligan, M.-S., Altwicker, E. (1993). The relationship between de Nova synthesis of polychlorinated dibenzo-p-dioxins and dibenzofurans and low-temperature carbon gasification in fly ash. *Environmental Science and Technology.* 27: 1595-1601.

Park, S., Kim, S.-J., Kim, K.S., Lee, D.S. and Kim, J.G., (2004). Influence of an Industrial Waste Incinerator as Assessed by the Levels and Congener Patterns of Polychlorinated Dibenzo-p-dioxins and Polychlorinated Dibenzofurans. *Environ. Sci. Technol.* 38: 3820-3826.

Tuppurainen, K., Asikainen, A., Ruokojärvi, P. and Ruuskanen , J., (2003). Perspectives on the Formation of Polychlorinated Dibenzo-p-dioxins and Dibenzofurans during Municipal Solid Waste (MSW) Incineration and Other Combustion Processes. *Accounts of Chemical Research.* 36 (9).

Wang, L.-C., Lee, W.-J., Lee, W.-S., Chang-Chien, G.-P. and Tsai, P.-J., (2003a). Characterizing the Emissions of Polychlorinated Dibenzo-p-dioxins and Dibenzofurans from Crematories and Their Impacts to the Surrounding Environment. *Environ. Sci. Technol.* 37(1): 62-67.

Wang, L.-C., Lee, W.-J., Tsai, P.-J., Lee, W.-S. and Chang-Chien, G.-P., (2003b). Emissions of polychlorinated dibenzo-p-dioxins and dibenzofurans from stack-flue gases of sinter plants. *Chemosphere.* 50(9): 1123-1129.

Wilkinson, L., Blank, G. and Gruber, C. (1996). *Desktop Data Analysis with SYSTAT.* Prentice-Hall, Inc., Upper Saddle River, NJ, USA.

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Influence of start-up on PCDD/F emission of incinerators

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Abstract

This study aims to evaluate the influence of start-up on polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans (PCDD/Fs) concentration in the stack flue gas of incinerators and its contributing PCDD/F emission. The PCDD/F emission of the first sample among three consecutive stack flue gas samples of five intermittent incinerators, which sampled at a stable combustion condition after start-up, is 2–3 times higher than the mean of the others. For verifying the PCDD/F characteristics of incinerators during start-up, one continuous MSWI was investigated for two years. The elevated PCDD/F emissions of the MSWI during start-up could reach 96.9 ng I-TEQ N m^{-3} and still maintained a high PCDD/F emission (40 times higher than the Taiwan emission limit) even 18 h after the injection of activated carbon, indicating the memory effect. Taking the MSWI for example, which consists of four incinerators, the estimated annual PCDD/F emission from normal operational conditions was 0.112 g I-TEQ. However, one start-up procedure can generate ~60% of the PCDD/F emissions for one whole year of normal operations. And the PCDD/F emission, which is the result of the start-ups of four incinerators, was at least two times larger than that of a whole year's normal operations, without consideration for the PCDD/F emission contributed by the long lasting memory effect.

Keywords: PCDD/Fs; Start-up; Incinerator; Memory effect

1. Introduction

After polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) were discovered in the flue gases and fly ash of municipal solid waste incinerators (MSWIs) in 1977 (Olie et al., 1977), PCDD/Fs have become a serious issue in many countries because of their toxicological effects and associated adverse health implications.

Most research investigated the PCDD/F characteristics and the induced hazardous effect of incinerators during normal operations. Till recently, several studies (Gass et al., 2002; Löthgren and van Bavel, 2005; Neuer-Etscheidt et al., 2006) have focused on the high PCDD/F emission during the start-up of incinerators. The start-up of continuous MSWIs is usually a cold start-up, which occurs after a plant revision and consists of the following procedures: (1) Oil burning with a maximum fuel feed rate until the suitable temperature for incineration (850 °C) is reached. (2) Start of waste feeding and increasing feed rate until design load is reached (Gass et al., 2002). However, for intermittent incinerators, the start-up procedure is a warm start-up, which can be characterized by the furnace's remaining temperature when it is started up again the next day.

Löthgren and van Bavel (2005) measured the PCDD/F levels after a polishing wet scrubber continuously for

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18 months using long-time sampling equipment at a hazardous waste incineration facility in Sweden. Each sampling period lasted two weeks. Two dramatic risings of TEQ levels were observed, both in the period just after a maintenance stop of the plant, from 0.02 to 0.25 ng TEQ m^{-3} and from 0.03 to 0.15 ng TEQ m^{-3}. Gass et al. (2002) reported raw flue gas PCDD/F concentrations during the start-up of a MSWI of up to 250 ng I-TEQ m^{-3} in the heat-up phase. In contrast, Neuer-Etscheidt et al. (2006) reported that PCDD/F I-TEQ crude gas concentrations during the heat-up period were a little lower than those during normal operations with waste as the fuel. One reason for this difference may be the state of cleaning of the boiler section. Immediately after waste was fed, PCDD/F concentrations (46 ng I-TEQ m^{-3}) in the crude gas increased by one order of magnitude compared to normal operating conditions (3–4 ng I-TEQ m^{-3}). Even for a ship's main engine, the highest PCDD/F emissions have been measured for the start-up samples (0.1–0.4 ng WHO-TEQ kW h^{-1} vs 0.03–0.1 ng WHO-TEQ kW h^{-1} during normal operations), which are characterized by relatively poor combustion conditions (also high CO emissions) (Cooper, 2005). However, still little research estimated the generated PCDD/Fs during start-up to what extent. Consequently, it is not yet possible to evaluate the exposures and potential health risks during these conditions (Mckone and Hammond, 2000).

In this study, five intermittently operating incinerators, including one industrial waste incinerator (IWI), one waste liquid incinerator (WLI) and three medical waste incinerators (MWIs) were measured for PCDD/Fs in the stack flue gases when combustion condition reached stabilization after start-up to evaluate the influence of memory effect caused by start-up on PCDD/F emission. For verifying the PCDD/F characteristics of incinerators during start-up, one large scale continuously operating MSWI was chosen to be investigated for two years. In the first year, a total of five start-up stack flue gas samples were collected. For more detailed characterization, the next year, a total of 10 stack flue gas samples were sampled and the sampling time was changed so it's more accordant with the start of waste feeding. Only the 2,3,7,8-PCDD/F congeners in the stack flue gases of the incinerator were measured because of their toxicities. The obtained results were not only compared to the typical concentrations during normal operations of the MSWI but also evaluated for the PCDD/F emission during start-up.

2. Experimental section

2.1. Basic information concerning the incinerators

The basic operation information concerning the intermittent incinerators, including one IWI, one WLI and three MWIs is described in Table 1. The feeding waste and air pollution control devices (APCDs) between these three categories of incinerators are not similar, but each kind of incinerator possesses its representation.

The continuously operating MSWI investigated for two years in this study consists of four 450 ton day^{-1}, two-stage, starved-air modular incinerators, each of which includes its own heat recovery system, dry scrubber, activated carbon injection, bag filter and stack. During start-up, the incinerators were preheated by burners operated with diesel.

2.2. PCDD/F sampling

On a usual day, the intermittent incinerators were started up in the morning and operated during the day. At night, the combustion chamber cooled down. However, in this study, the feeding waste was accumulated to enough for three consecutive 3 h stack flue gas samples during normal operations after start-up, which is characterized by the stable reading of combustion temperature and traditional pollutant, like CO concentration.

The sampling time of each stack flue gas sample of the MSWI during start-up was about 2–3 h and a total of five samples were collected in the first year. For more detailed characterization, the next year, the sampling time was changed to once an hour and a total of 10 stack flue gas samples were sampled.

The PCDD/F samples were collected isokinetically from the stack flue gas of the selected incinerators according to US EPA modified Method 23. The sampling train adopted in this study is comparable with that specified by US EPA

Table 1
Basic information concerning these five intermittent incinerators

Emission sources	IWI	WLI	MWI	MWI	MWI
Denotation	A	B	C	D	E
Feeding waste (kg h^{-1})	Industrial waste (420)	Waste liquid (200)	Infectious and pathological waste (400)	Infectious and pathological waste (300)	Infectious and pathological waste (300)
Auxiliary fuel (l h^{-1})	–	Diesel (0.1)	Diesel (22)	Diesel (21)	Diesel (19)
APCDs in sequence (operation temperature) (°C)	Cyclone (200) BF (160)	VS (90)	DS (250) ACI BF (150)	QC VS (90)	DS (250) ACI BF (160)
Mean stack flue gas flow (N m^3 h^{-1})	8500	2900	4500	4000	6000

Activated carbon injection: ACI; Bag filter: BF; Dry scrubber: DS; Quench chamber: QC; Venturi scrubber: VS.

Modified Method 5. Prior to sampling, XAD-2 resin was spiked with PCDD/F surrogate standards pre-labeled with isotopes, including $^{37}Cl_4$-2,3,7,8-TCDD, $^{13}C_{12}$-1,2,3,4,7,8-HxCDD, $^{13}C_{12}$-2,3,4,7,8-PeCDF, $^{13}C_{12}$-1,2,3,4,7,8-HxCDF and $^{13}C_{12}$-1,2,3,4,7,8,9-HpCDF. The recoveries of PCDD/F surrogate standards were 101–123%, and met the criteria within 70–130%. To ensure the free contamination of the collected samples, one trip blank and one field blank were also taken during the field sampling was conducted. Details are similar to that given in our previous work (Wang et al., 2003).

2.3. Analyses of PCDD/Fs

Analyses of stack flue gas followed the US EPA modified method 23. All chemical analyses were carried out by the Super Micro Mass Research and Technology Center at Cheng Shiu University – the accredited laboratory in Taiwan for PCDD/F analyses. Prior to analysis, each collected sample was spiked with a known amount of the $^{13}C_{12}$-labeled internal standard to the extraction thimble. Add toluene to fill the reservoir approximately 2/3 full. Adjust the heat source to cause the extractor to cycle three times per hour. After being extracted for 24 h, the extract was concentrated, treated with concentrated sulfuric acid, and then followed by a series of sample cleanup and fractionation procedures, including multilayer silica gel column, alumina column and activated carbon chromatography. The eluate was concentrated to approximately 1 ml and transferred to a vial. The concentrate was further concentrated to near dryness, using a stream of nitrogen. Immediately prior to analysis, the standard solution for recovery checking was added to the sample. The recoveries of PCDD/F internal standards for the tetra- through hexachlorinated homologues were between 65% and 98%, and met the criteria within 40–130%, while that for the hepta- and octachlorinated homologues were between 52% and 101%, and met the criteria within 25–130%.

A high-resolution gas chromatograph/high-resolution mass spectrometer (HRGC/HRMS) was used for PCDD/Fs analyses. The HRGC (Hewlett Packard 6970 Series gas, CA, USA) was equipped with a DB-5MS fused silica capillary column ($L = 60$ m, ID = 0.25 mm, film thickness = 0.25 μm) (J&W Scientific, CA, USA), and with a splitless injection. Helium was used as the carrier gas. However, if a valley between peaks is more than 25% of the lower of the two peaks for 2,3,7,8-TCDD and 2,3,7,8-TCDF, the column had to be changed to DB-225 to recheck isomer. The oven temperature program was set according to follows: begin at 150 °C (held for 1 min), then increased at 30 °C min^{-1} to 220 °C (held for 12 min), then increased at 1.5 °C min^{-1} to 240 °C (held for 5 min), and finally increased at 1.5 °C min^{-1} to 310 °C (held for 20 min). The HRMS (Micromass Autospec Ultima, Manchester, UK) was equipped with a positive electron impact (EI+) source. The analyzer mode of the selected ion monitoring (SIM) was used with resolving power at 10000. The electron energy and source temperature were specified at 35 eV and 250 °C, respectively. Details of analytical procedures are given in our previous work (Wang et al., 2003).

3. Results and discussion

3.1. Influence of memory effect resulted from start-up on PCDD/F emissions of the intermittent incinerators

The PCDD/F profiles of three consecutive stack flue gas samples of these five intermittent incinerators were illustrated in Fig. 1 and revealed that the PCDD/F emission of the first stack flue gas sample, which was sampled at a stable combustion condition after start-up, was considerably higher than that of the follow-up samples. For toxicity basis, the first stack flue gas sample was 2–3 times higher than the mean PCDD/F emission of the second and third one. Fig. 1 also revealed that the PCDD/F profiles of the three consecutive stack flue gas samples for each incinerator declined with time. The reason for this is that the memory effect that resulted from the high PCDD/F emission of the start-up contributed and changed the subsequent concentration and profiles significantly. But as time went by, the influence of the memory effect on the PCDD/F emission faded away.

The memory effect caused by the wet scrubber resulted from that PCDD/Fs are adsorbed on scrubber fillings (typically polypropylene (PP) plastics) and thus leading to unexpected PCDD/F releases when the plant is running under more stable conditions (Hunsinger et al., 1998; Adams et al., 2000; Takaoka et al., 2003). Compared to that caused by start-up procedure, the memory effect caused by the start-up can occur in all kinds of incinerators with different APCDs, not only for those with wet scrubbers, for examples, the incinerators in this study are no exception to this.

3.2. PCDD/F emission in the stack flue gases of the MSWI under normal conditions

For more understanding of the memory effect that resulted from the start-up, one continuous MSWI was chosen to characterize the PCDD/F emission during start-up. In the first instance, we need to establish the PCDD/F characteristics of the MSWI during normal operation.

The stack flue gases of the MSWI were sampled five times in different months under normal operation conditions and a total of 25 samples were collected for PCDD/F measurement. The mean PCDD/F concentration (normalized to the dry flue gas conditions of 273 K and 11% O_2) in the stack flue gas was 0.0358 ng I-TEQ N m^{-3} (range: 0.00273–0.0946 ng I-TEQ N m^{-3}, relative standard deviations (RSD): 66.4%), while their corresponding mean PCDD/F emission factor was 0.249 μg I-TEQ ton $waste^{-1}$ (RSD: 69.0%), which is close to that (mean: 0.251 μg I-TEQ ton $waste^{-1}$, range: 0.0512–0.561 μg I-TEQ ton $waste^{-1}$) of the other 13 MSWIs in Taiwan (Lee et al.,

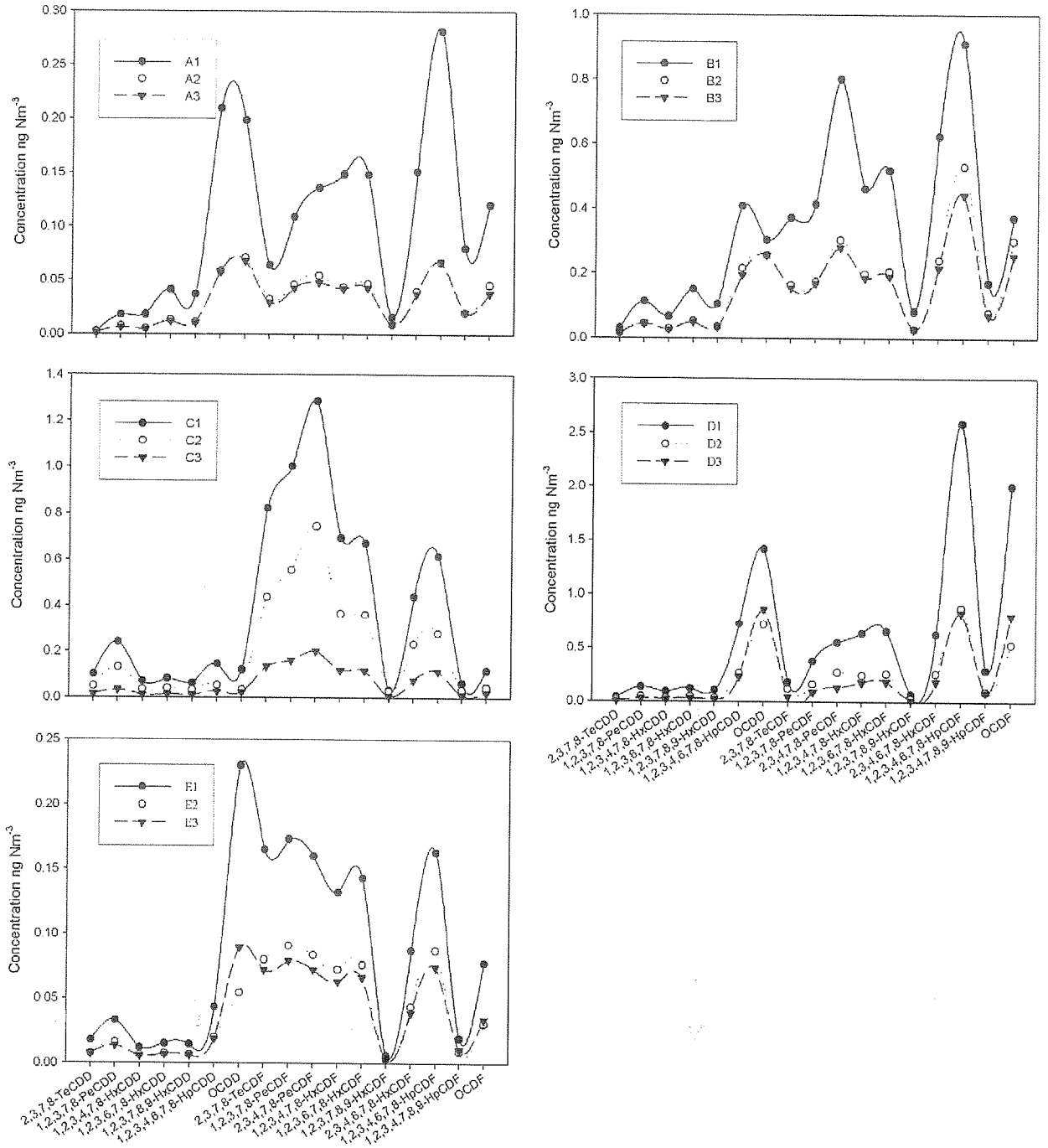


Fig. 1. The PCDD/F profiles of three consecutive stack flue gas samples of the five intermittent incinerators.

2005) and is comparable to that (0.17 μg I-TEQ ton waste^{-1}) of the MSWI (400 ton day^{-1}) in accordance with the best control technologies (Giugliano et al., 2002). Furthermore, five raw flue gas samples sampled in front of the activated carbon injection and bag filter were also measured for PCDD/Fs and the mean PCDD/F concentration was 0.610 ng I-TEQ N m^{-3} (range: 0.231–1.52 ng I-TEQ N m^{-3}, RSD: 86.1%).

According to statistical data, there were ~448 000 tonnes of waste incinerated by the MSWI investigated in this study in 2005. By directly adopting the mean emission factors that were obtained from this study (0.249 μg I-TEQ ton waste^{-1}), the total emission amount of PCDD/Fs from the MSWI is ~0.112 g I-TEQ yr^{-1}.

3.3. PCDD/F emission in the stack flue gases of the MSWI during start-up

Table 2 listed the operation conditions and their corresponding PCDD/F emission in the stack flue gases during

Table 2
PCDD/F emission in the stack flue gases of the MSWI during start-up in the first year's investigation

Sample denotations	Sampling day				
	Day one		Day two		
	a	b	c	d	e
Sampling time	15:30–17:40	19:25–20:55	23:40–3:15	10:10–12:35	13:42–16:10
Passed time after burner started (h)	5.5–7.7	9.4–11	13.7–17.3	24.2–26.6	28–30.2
Passed time after waste fed (h)	–	–	–	5.2–7.6	9–11.2
Auxiliary fuel ($l\,h^{-1}$)	660	830	650	4	7
Temperature of combustion chamber (°C)	310	490	850	900	920
Primary air flow rate ($N\,m^3\,h^{-1}$)	37500	34300	30900	50400	51000
Temperature in the inlet of superheater (°C)	190	240	360	490	500
Temperature in the inlet of economizer (°C)	150	210	300	330	340
O_2 Concentration after economizer (%)	22	22	22	11	7.3
Temperature of dry scrubber (°C)	130	160	210	240	230
Lime feeding rate ($m^3\,h^{-1}$)	0	0	0.6	2.2	4.0
Temperature of bag filter (°C)	120	150	180	160	150
Activated carbon feeding rate ($kg\,h^{-1}$)	0	0	6	10	10
CO concentration in the stack flue gas (ppm)	–	–	–	42	32
O_2 concentration in the stack flue gas (%)	18	17	14	8.3	8.7
PCDFs/PCDDs ratio	0.66	0.62	0.72	0.92	0.75
Total PCDD/Fs ($ng\,N\,m^{-3}$)	83.8	328	68.2	42.9	10.7
PCDFs/PCDDs TEQ ratio	3.0	2.1	2.4	2.6	2.4
Total I-TEQ ($ng\,I\text{-}TEQ\,N\,m^{-3}$)	2.09	12.2	3.34	2.28	0.507

–: No data.

the cold start-up of the MSWI in the first year's investigation. The PCDD/F concentrations (normalized to the dry flue gas conditions of 273 K) in the stack flue gases during the heat-up phase (samples a–c) were ranged at 2.09–12.2 ng I-TEQ N m^{-3} while those that were ranged at 0.507–2.28 ng I-TEQ N m^{-3} were observed 5 h after the waste feeding process had started.

For more detailed characterization, during the following year, the sampling time was changed to once an hour so it's more accordant with the start of waste feeding (see Table 3). The PCDD/F concentration in the stack flue gases during the heat-up phase (samples a–d) was between 5.16 and 41.5 ng I-TEQ N m^{-3}, which was higher than the first year's results (2.09–12.2 ng I-TEQ N m^{-3}). However, the drastic PCDD/F emissions in the stack flue gases during the heat-up phase of the two years' investigation both occurred at the combustion chamber's temperature reaching 490 °C. When the combustion chamber's temperature reaching 490 °C, the temperatures in the inlet of the superheater and economizer were between 210 °C and 290 °C, which were a little below the temperature region (250–450 °C) of the PCDD/F optimal formation. Otherwise, Clarke (2000), Gass et al. (2003) and Neuer-Etscheidt et al. (2006) reported that PCDD/F precursors (e.g., benzenes, phenols and chlorinated forms) are created in the furnace at the highest rate in these temperature regions. In fact, Benestad et al. (1990) reported that PAHs concentration during the start-up period was 2–16 times higher than that during normal operation for three small MSWIs. Yasuda and Takahashi (1998) also determined the PAHs emission of four MSWIs during start-up, burning, and burn-out and reported that during start-up conditions, PAH concentrations (7.7–248 μg N m^{-3}) have been shown to be 12–77 times higher during burning (0.1–63.2 μg N m^{-3}). Consequently, the drastic PCDD/F emissions occurred during the heat-up phase is more related to the operational temperature of the combustion chamber than to that of superheater and economizer.

But when the temperature of the combustion chamber raised from 490 °C to 850 °C, the PCDD/F emissions decreased from 12.2 ng I-TEQ N m^{-3} (no activated carbon injection) to 3.34 ng I-TEQ N m^{-3} (activated carbon feeding rate: 6 $kg\,h^{-1}$) during the heat-up phase in the first year's investigation. The injection of activated carbon and the elevated temperature of the combustion chamber, i.e. the more completely combustion condition, may be the reasons of drastic reduction of the PCDD/F emissions, however, due to the intervals of up to several hours where no samples were taken, which one is the most influence factor is needed to be clarified further.

In the first year, the temperature of the combustion chamber reached 490 °C after burning for 10 h with the average auxiliary fuel feeding rate of 710 $l\,h^{-1}$ and the average primary air of 35900 N $m^3\,h^{-1}$ while in the second year's investigation, reaching the same combustion condition with a higher average auxiliary fuel feeding rate of 1150 L h^{-1} and a less primary air of 27700 N $m^3\,h^{-1}$. The results revealed that the worse combustion efficiency occurred in the second year and might result in the PCDD/F concentration in the stack flue gases during the heat-up phase (5.16–41.5 ng I-TEQ N m^{-3}) which has a recording that is higher in the second year than that of the first year (2.09–12.2 ng I-TEQ N m^{-3}). Otherwise, Neuer-Etscheidt et al. (2006) proposed that the state of

Table 3
PCDD/F emission in the stack flue gases of the incinerator during start-up in the second year's investigation

Sample denotations	Sampling day									
	Day one			Day two						
	a	b	c	d	e	f	g	h	i	j
Sampling time	18:00–19:00	20:30–21:25	23:10–00:07	01:50–02:57	05:00–06:08	09:50–11:09	12:45–13:58	15:20–16:30	18:04–19:09	20:30–21:41
Passed time after burner started (h)	2–3	4.5–5.4	7.2–8.1	9.8–11	13–14.1	17.8–19.2	20.8–22	23.3–24.5	26.1–27.2	28.5–29.7
Passed time after waste fed (h)	–	–	–	–	1–2.1	5.8–7.2	8.8–10	11.3–12.5	14.1–15.2	16.5–17.7
Auxiliary fuel ($l\ h^{-1}$)	1100	1150	1300	1060	525	2	0	0	0	0
Temperature of combustion chamber (°C)	155	230	400	490	900	900	910	880	900	900
Primary air flow rate ($N\ m^3\ h^{-1}$)	32000	28700	26700	23200	53800	39100	39800	44100	48600	43900
Temperature in the inlet of superheater (°C)	140	190	220	290	360	460	490	490	500	500
Temperature in the Inlet of economizer (°C)	110	160	190	250	310	330	340	340	350	350
O_2 concentration after economizer (%)	19	19	17	17	7.0	9.2	9.1	10	8.6	8.8
Temperature of dry scrubber (°C)	90	140	180	210	210	220	230	230	230	230
Lime feeding rate ($m^3\ h^{-1}$)	0	0	0	0	0	0	0	0	0	0
Temperature of bag filter (°C)	80	130	170	170	160	160	150	150	150	150
Activated carbon feeding rate ($kg\ h^{-1}$)	0	0	0	0	6	6	6	6	6	6
CO concentration in the stack flue gas (ppm)	–	–	–	–	–	49	43	53	37	21
O_2 concentration in the stack flue gas (%)	19	19	13	9.2	8.9	9.1	8.3	8.9	10	8.8
PCDFs/PCDDs ratio	0.64	0.59	0.95	1.0	1.0	0.69	0.74	0.67	0.69	0.68
Total PCDD/Fs ($ng\ N\ m^{-3}$)	180	259	111	384	909	339	86.3	99.7	66.5	64.3
PCDFs/PCDDs TEQ ratio	4.1	3.4	4.0	1.7	2.0	1.9	2.0	2.0	2.1	2.0
Total I-TEQ ($ng\ I\text{-}TEQ\ N\ m^{-3}$)	6.19	8.64	5.16	41.5	96.9	23.1	6.18	6.41	4.30	4.19

–: No data.

cleaning of the boiler section could also resulted in the PCDD/F crude gas concentrations during the heat-up period ranging from a little lower than those during normal operations to 250 ng I-TEQ N m^{-3}, which was reported by Gass et al. (2002).

Another drastic elevated PCDD/F concentrations (from 41.5 to 96.9 ng I-TEQ N m^{-3}) in the stack flue gas occurred one hour after waste feeding (Table 3) and it declined to 23.1 ng I-TEQ N m^{-3} five hours after waste feeding, but still maintained high PCDD/F concentration, 4.19 ng I-TEQ N m^{-3}, 40 times higher than the Taiwan emission limit of 0.1 ng I-TEQ N m^{-3} even 18 h after the injection of activated carbon, indicating the long lasting memory effect. Neuer-Etscheidt et al. (2006) also observed that immediately after waste was fed, PCDD/F concentrations (46 ng I-TEQ m^{-3}) in the crude gas increased by one order of magnitude compared to normal operating conditions (3–4 ng I-TEQ m^{-3}). In this study, the raw gas in front of the APCD had not been sampled so that it is unclear that if the raw gas PCDD/F levels were still elevated at 18 h after the injection of activated carbon. However, the CO concentration (21 ppm, see Table 3) at that time was already in the range of normal operation condition, representing the well combustion condition and little PCDD/F precursors, like benzenes, phenols and PAHs formed, so we extrapolated that the high PCDD/F concentration, 4.19 ng I-TEQ N m^{-3} at 18 h after the injection of activated carbon, resulting from rather the memory effect than the incomplete combustion at that time.

During the heat-up phase, no activated carbon is injected prior to the bag filter in order to avoid ignitions of the activated carbon due to the high oxygen concentration in the flue gas (see Tables 2 and 3). Although it may be one of the reason that the PCDD/F concentration in the stack flue gases was between 5.16 and 41.5 ng I-TEQ N m^{-3}, however, by comparing to the PCDD/F concentration in the raw flue gases in front of the activated carbon injection and bag filter of the MSWI during normal operation (mean: 0.610 ng I-TEQ N m^{-3}, range: 0.231–1.52 ng I-TEQ N m^{-3}), it revealed that a high PCDD/F emission during the heat-up phase occurred due to the low furnace temperature and the poor combustion condition. The surfaces of the boiler and pipe were thus contaminated by the soot particles as well as hydrocarbons, which improved de novo synthesis of PCDD/F formation during the subsequent increase of the temperatures at these locations. Otherwise, the high PCDD/F emission occurred during the start-up procedure also increased the PCDD/F adsorption on the APCDs, like wet scrubber and bag filter and then released over a longer period of time.

3.4. PCDD/F emission quantity of the MSWI during start-up

The profile of the PCDD/F emission rate of stack flue gas with time during start-up of a two year investigation is illustrated in Fig. 2 and the shadowed areas represent

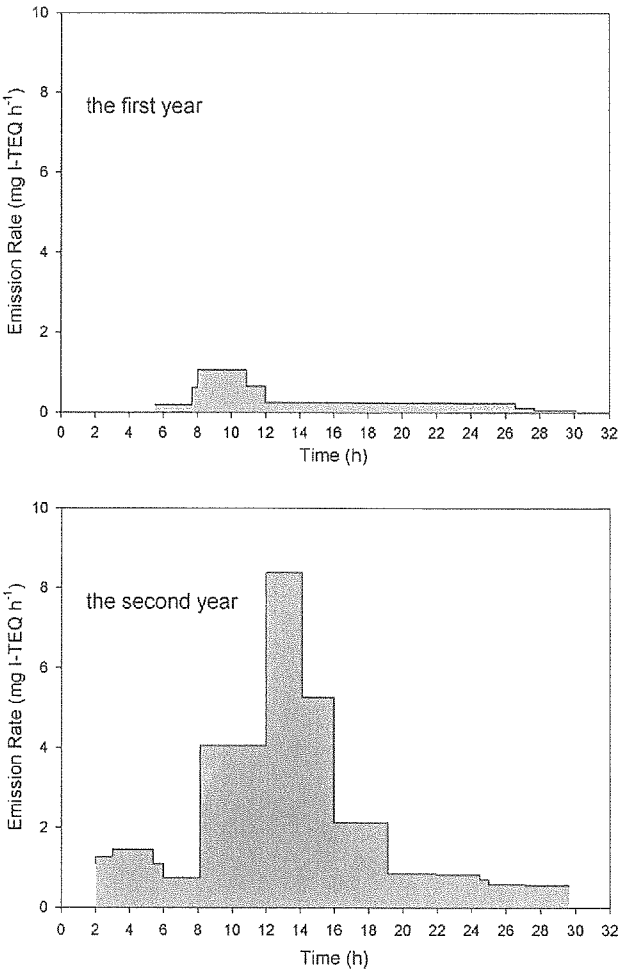


Fig. 2. PCDD/F emission rate from the stack flue gases of the MSWI during startup.

the PCDD/F emission quantity of MSWI during start-up. It revealed that one PCDD/F emission peak exists when the combustion chamber's temperature reaches 490 °C in both the first and second year. However, owing to the inexperience regarding the sampling strategy, another high PCDD/F emission peak resulted from the poor combustion when waste being fed was not observed in the first year's investigation. After changing the sampling time to be more accordant with the start of waste feeding, the PCDD/F profile clearly showed the phenomenon of PCDD/F generation and two high PCDD/F emission peaks with subsequently slow declination on emission in the second year's investigation. After calculating the shadowed areas, the estimated PCDD/F emission quantity during start-up was 0.0658 g I-TEQ in the second year's investigation.

Incinerators are usually at least shut-down and started-up once a year for maintenance. Taking the MSWI investigated in this study for example, which consists of four incinerators, the estimated annual PCDD/F emission from normal operational conditions was 0.112 g I-TEQ. However, one start-up procedure can generate ~60% of that and the PCDD/F emission quantity resulted from

the start-ups of four incinerators were at least two (=0.0658 × 4/0.112) times larger than that of a whole year's normal operations, without consideration for the PCDD/F emission contributed by the long lasting memory effect because in this study the sampling period ended two days after start-up.

According to the legislation of most countries, PCDD/F measurements of incinerators only have to be conducted once or twice annually and are usually under normal and good operational conditions. Well over a 1000-fold increase in PCDD/F concentration and a two-fold increase in annual PCDD/F emission during the start-up procedures. The days when a cold start-up occurs will thus be much more significant than on the other days of normal operations. After properly evaluating the real total PCDD/F emission, an effective risk management strategy can be developed by assessing the relationship between exposure and health.

4. Conclusions

The memory effect that resulted from the start-up caused the PCDD/F concentration of the first stack flue gas sample of the intermittent incinerators, which was sampled at a stable combustion condition after start-up, elevated as much as 2–3 times higher than the mean of the follow-ups. During the start-up procedure of the MSWI, two high PCDD/F emission peaks in the stack flue gas were observed. One occurred when the combustion chamber's temperature reached 490 °C and the other occurred at the moment when waste was first fed. The elevated PCDD/F concentrations could reach 96.9 ng I-TEQ N m^{-3} and still maintained a high PCDD/F concentration (40 times higher than the Taiwan emission limit) even 18 h after the injection of activated carbon, indicating the long lasting memory effect. The PCDD/F emission over several days resulted from the start-ups of the MSWI were at least two times larger than that from a whole year's normal operations, without consideration for the PCDD/F emission contributed by the subsequent memory effect.

The obtained results revealed that the intermittent incinerators, due to their design or operational characteristics, are prone to start-up procedure and should be replaced by continuous incinerators or the operation schedule from 8 h to 5 days a week should be changed to a continuous two day operation.

References

Adams, B., Buekens, A., Ex, W., Joannès, J., 2000. Dioxin emissions from a MSWI related to memory effects in a two stage wet scrubber. Organohalogen Compd. 46, 178–181.

Benestad, C., Hagen, I., Jebens, A., Oehme, M., Ramdahl, T., 1990. Emissions of organic micropollutants from discontinuously operated municipal waste incinerators. Waste Manage. Res. 8, 193–201.

Clarke, M.J. 2000. Characterization of cold start and upset conditions in municipal waste combustors. In: Proceedings of the Air & Waste Management Association, Salt Lake City, UT, USA.

Cooper, D.A., 2005. HCB, PCB, PCDD and PCDF emissions from ships. Atmos. Environ. 39, 4901–4912.

Gass, H.C., Lüder, K., Wilken, M., 2002. PCDD/F emissions during cold start-up and shut-down of a municipal waste incinerator. Organohalogen Compd. 56, 193–196.

Gass, H.C., Wilken, M., Lüder, K., 2003. Optimization of the start-up procedures in a municipal waste incinerator – impact on the emissions of dioxins and related compounds. Organohalogen Compd. 63, 25–28.

Giugliano, M., Cernuschi, S., Grosso, M., Miglio, R., Aloigi, E., 2002. PCDD/F mass balance in the flue gas cleaning units of a MSW incineration plant. Chemosphere 46, 1321–1328.

Hunsinger, H., Kreisz, S., Seifert, H., 1998. PCDD/F behavior in wet scrubbing systems of waste incineration plants. Chemosphere 37, 2293–2297.

Lee, W.S., Wang, L.C., Lee, W.J., Tsai, P.J., Chang, M.B., Chang-Chien, G.P., 2005. Major Emission Inventory of Polychlorinated Dibenzo-*p*-dioxins and Dibenzofurans in TaiwanTrends in Air Pollution Research. Nova Science Publishers, Inc., NewYork, USA.

Löthgren, C.J., van Bavel, B., 2005. Dioxin emissions after installation of a polishing wet scrubber in a hazardous waste incineration facility. Chemosphere 61, 405–412.

Mckone, T.E., Hammond, S.K., 2000. Managing the health impacts of waste incineration. Environ. Sci. Technol. A 34, 380A–387A.

Neuer-Etscheidt, K., Nordsieck, H.O., Liu, Y., Kettrup, A., Zimmermann, R., 2006. PCDD/F and other micropollutants in MSWI crude gas and ashes during plant start-up and shut-down processes. Environ. Sci. Technol. 40, 342–349.

Olie, K., Vermeulen, P.L., Hutzinger, O., 1977. Chlorodibenzo-*p*-dioxins and chlorodibenzofurans are trace components of fly ash and flue gas of some municipal incinerators in the Netherlands. Chemosphere 6, 455–459.

Takaoka, M., Liao, P., Takeda, N., Fujiwara, T., Oshita, K., 2003. The behavior of PCDD/Fs, PCBs, chlorobenzenes and chlorophenols in wet scrubbing system of municipal solid waste incinerator. Chemosphere 53, 153–161.

Wang, L.C., Lee, W.J., Lee, W.S., Chang-Chien, G.P., Tsai, P.J., 2003. Characterizing the emission of polychlorinated dibenzo-*p*-dioxins and dibenzofurans from crematories and their impacts to the surrounding environment. Environ. Sci. Technol. 37, 62–67.

Yasuda, K., Takahashi, M., 1998. The emission of polycyclic aromatic hydrocarbons from municipal solid waste incinerators during the combustion cycle. J. Air Waste Manage. Assoc. 48, 441–447.

Crematory Emissions Data

Outline of Referenced Sources:

This is an outline of key points in several documents found to have useful information on the matter of toxins in crematory emissions. The documents can be viewed by following the links in blue. Some of the documents referenced here have multiple source information. No representation here is meant to imply ownership of this information. Every attempt possible has been made to ensure that the authors of any material referenced here has been properly identified. For the sake of simplicity on this page, the source document has been linked. This information is for public service only, and nocrematory.com does not accept financial compensation or charge any fees for access to information. Please refer to these documents for verification of any facts or opinions represented on this page. Thank You!

1. **letter_from_CONGRESS-Kucinich_to_EPA-Jackson (http://no2crematory.files.wordpress.com/2011/01/letter_from_congress-kucinich_to_epa-jackson.pdf)**
Letter from US Congress written by Dennis Kucinich on Jan 15 2010, to Lisa P Jackson, US EPA, regarding mercury as a source of pollution through dental amalgam fillings. In this letter Kucinich tells the EPA that based on testimony from experts given to the Domestic Policy Subcommittee:
a. "EPA has underestimated amount of mercury pollution that dental amalgams accounts for, thereby rendering the problem a lower priority than it would otherwise be."
b. Estimated mercury emissions data came from one test at a single crematory 10 years ago, based on that data the estimate was 0.3 tons.
c. Based on 2.9 grams mercury per cremated person (average based on number of amalgam fillings) a better estimate is 3.3 tons, eleven times the level accounted for by EPA.
d. EPA is required by Clean Air Act to monitor emissions of mercury.
e. Congress requests EPA reevaluate and update mercury emissions from crematories, a devise a plan and timeline to address the problem in a timely fashion.

2. **EPA-response-_to-CONGRESS-Kucinich (http://no2crematory.files.wordpress.com/2011/01/epa-response-to-congress-kucinich.pdf)**
Letter from Arvin Ganesan, response from US EPA to US Congress.
a. EPA recognizes that pollution from all sources of mercury is a serious concern to human health and the environment.
b. Mercury is well-documented as a toxic, environmentally persistent substance that demonstrates the ability for bioaccumulation and atmospheric transportation on a local, regional, and global scale.
c. There is evidence that ... "serious health impacts from low-level exposure to mercury"
e. No state regulations on sale or distribution of dental amalgam. Knowledge of mercury in fillings may drive future declines in use; however the expense of non-mercury fillings can affect patients' preference for amalgam.
f. EPA acknowledges that mercury from amalgam is a source of controllable pollution; that mercury is released into the environment through cremation of bodies containing dental amalgam.
g. Worksheet from EPA in 2005 estimated that US emissions from crematoria to be 3,000 kilograms (6,613 lbs)
h. No federal or state regulations restrict mercury emissions from crematoria.
i. EPA intends to update mercury emissions, but "does not intend to regulate human crematoria at this time."
j. EPA has concluded that human crematories were not solid waste incinerators; therefore it was not appropriate to regulate them under Clean Air Act Section 129.
k. If in the future EPA concludes that crematoria should be regulated other Clean Air Act Authorities could be used.

3. **mercury_concentrations_[spike]_in_emissions (http://no2crematory.files.wordpress.com/2011/01/mercury-concentrations-spike-in-emissions.pdf)**
Mercury in Crematoria Using Flourescence Spectrometry - September 2010 study,: Corns, Dexter and Stockwell, P S Analytical, UK
a. Mercury emissions from crematoria are almost entirely due to the presence of amalgam fillings in the cadaver.
b. Leads to "significant variation in the concentration of mercury emitted during each cremation."
c. Testing was performed over the course of 4 separate cremations.
d. Mercury emissions in study had variance (ug/m-3 = micrograms per cubic meter) cremation #1 had 25.8 ug/m-3, #2 had 47.5 ug/m-3, #3 had 282.0 ug/m-3 and #4 had 1094.5 ug m-3 (THIS IS IMPORTANT. COMPARE TO ACTION SHEETS BELOW. These levels exceed the exposure levels by alarmingly high concentrations.)
e. Mercury is released in a spike about 40 minutes into the cremation, during the heating up process, as mercury reaches the temperature threshold at which it vaporizes.
f. Mercury released during a short period of time. (not gradually over time)

4. **tfacts46_ATSDR (http://no2crematory.files.wordpress.com/2011/01/tfacts46_atsdr.pdf)**
IndoorAIR_ATSDR (http://no2crematory.files.wordpress.com/2011/01/indoorair_atsdr.pdf)

ATSDR (Agency for Toxic Substances and Disease Registry) Suggested Action Levels for Indoor Mercury Vapors in Homes, and FACT SHEET
a. Primary route for entry is inhalation.
b. When heated, mercury becomes a colorless, odorless gas.
c. Methylmercury and Mercury vapors are more harmful than others, because mercury in these forms reaches the brain.
d. Can cause permanently damage the brain, kidneys and nervous systems.
e. Sensitive populations are those with developing nervous systems, including young children (under six) and the developing fetuses of pregnant women.
f. Action level is indoor air concentration which should prompt consideration of the need to implement a personal response by public health and environmental officials.
g. Indoor Air Concentration must be less than 1.0 ug/m3 for residential occupancy after a spill. (NIOSH)
h. OSHA has set limits of 0.1 mg/c3 organic mercury (milligrams per cubic meter) and 0.05 mg/c3 mercury vapors for 8 hr shifts and 40 hr work weeks.
i. 10 ug/m3 requires residents be isolated from exposure (ATSDR)
j. Pregnant women should consult physicians about mercury exposure

WIKI Page for Mercury Poisoning
Mercury_poisoning (http://no2crematory.files.wordpress.com/2011/01/mercury_poisoning.pdf)

5. **http://www.osha.gov/SLTC/healthguidelines/mercuryvapor/recognition.html (http://www.osha.gov/SLTC/healthguidelines/mercuryvapor/recognition.html)**
OSHA Guidelines for Mercury Vapor
1. Effects on Humans:
• Mercury vapor can cause effects in the:
• central and peripheral nervous systems,
• lungs, kidneys, skin and eyes in humans.
• It is also mutagenic and affects the immune system
• [Hathaway et al. 1991; Clayton and Clayton 1981; Rom 1992].
• Acute exposure to high concentrations of mercury vapor causes severe respiratory damage.
• chronic exposure to lower levels is primarily associated with:
• central nervous system damage
• [Hathaway et al. 1991].
• Chronic exposure to mercury is also associated with behavioral changes and alterations in peripheral nervous system [ACGIH 1991]. Pulmonary effects of mercury vapor inhalation include diffuse interstitial pneumonitis with profuse fibrinous exudation [Gosselin 1984]. Glomerular dysfunction and proteinuria have been observed mercury exposed workers [ACGIH 1991]. Chronic mercury exposure can cause discoloration of the cornea and lens, eyelid tremor and, rarely, disturbances of vision and extraocular muscles [Grant 1986]. Delayed hypersensitivity reactions have been reported in individuals exposed to mercury vapor [Clayton and Clayton 1981]. Mercury vapor is reported to be mutagenic in humans, causing aneuploidy in lymphocytes of exposed workers [Hathaway et al. 1991].

* Signs and symptoms of exposure</strong
• Acute exposure: chills, nausea, general malaise, tightness in the chest, chest pains, dyspnea, cough, stomatitis, gingivitis, salivation, and diarrhea [ACGIH 1991; Hathaway et al. 1991].
• Chronic exposure: Chronic exposure to mercury may result in weakness, fatigue, anorexia, weight loss, and disturbance of gastrointestinal function. A tremor may develop beginning with the fingers, eyelids, and lips which may progress to generalized trembling of the entire body and violent chronic spasms of the extremities. Parallel with development of the tremors, behavioral and personality changes may develop including increased excitability, memory loss, insomnia, and depression. The skin may exhibit abnormal blushing, dermographia, excessive sweating and irregular macular rashes. Severe salivation and gingivitis is also characteristic of chronic toxicity [Hathaway et al. 1991; Gosselin 1984].
• Another manifestation of chronic mercury exposure is characterized by apathy, anorexia, flush, fever, a nephrotic syndrome with albuminuria and generalized edema, diaphoresis, photophobia, insomnia and a pruritic and sometimes painful scaling or peeling of the skin of the hands and feet with bullous lesions [Gosselin 1984].

6. **Mercury_Emissions (http://no2crematory.files.wordpress.com/2011/01/mercury_emissions.pdf)**
John Reindl, Mercury Emissions from Crematoria (Reindl testified before the Domestic Policy Subcommittee referred to in the letter from Kucinich. The document referred to here is from his work in 2005. This is the data he shared during the portion of his testimony that dealt specifically on crematory emissions.
• Main source of mercury in crematory emissions from dental amalgams
• Cremation is third largest source of air emissions from (amalgam) products, 2436 kg a year in 2005
• Fate of emissions global
• Emissions mainly metallic vapor
• Control methodologies
• Removal of teeth before cremation , Stack controls: Co-flow filters, solid-bed filters, traditional gas scrubbing, honeycomb catalytic absorbers
• Barriers to control methodologies:
• Lack of recognition of need to control • For removing of teeth: cultural values for handling of corpse
• Costs and physical challenges of stack controls
• Industry tradition – only one crematorium in US has stack controls
• Information needs: more data on amount of mercury per cremation, more expertise among researchers, regulators

7. **Scientific Explanation for the Tri-State Incident (http://no2crematory.files.wordpress.com/2011/01/hgpressrelease.pdf)**
McCracken Poston, A scientific explanation for the events at Tri-State Crematory
1. Theory of an external, environmental cause claimed not only the literal sanity and judgment of Ray-Brent Marsch, but also the health and ultimately the life

itself of his father, Tommy Ray Marsch.
b. Suspect element is mercury
c. Ray-Brent Marsch cremated the first two-thirds of the bodies sent to him, the crematory was in somewhat of a functioning order, and there was obvious heavy particulate matter on the interior of the small crematory building due to inadequate ventilation and a breached stovepipe from the retort to the exterior.
d. Ray-Brent complained to his wife of headaches and seemingly minor body aches, and suffered from chronic insomnia. (symptoms of mercury toxicity)
e. Hair sample was taken two years after crematory discoveries and exposure to suspected mercury vapors had ended.
i. Hair sampled showed classic signature of mercury poisoning
ii. Alarmingly high levels of heavy metals
iii. Mineral transport impairment
iv. Dr Boyd Haley, University of Kentucky Scientist has signed affidavit supporting Poston's theory.
f. Failure was on the part of the governmental regulatory process in US and in the State of Georgia.
g. Little or no guidelines exist regarding proper ventilation or placement of crematories.
h. Case is over, no appeals for Marsch, paper written as an explanation for the loved ones & the curious.
More about Mercury Here:

http://no2crematory.files.wordpress.com/2011/01/mercury_specialreport.pdf

8. **Mercury.ToxicTimeBomb.Final (http://no2crematory.files.wordpress.com/2010/09/mercury-toxictimebomb-final.pdf)**
Ban Mercury Working group Mercury Exposure: The World's Toxic Time Bomb
a. Human health is compromised by significantly smaller concentrations than ever imagined.
b. WHO concluded that "a safe level of mercury exposure ... has never been established."
c. Less than 1/50th of a teaspoon mercury per 20-acre lake surface is enough to make fish in it unsafe for human consumption.
d. Methylmercury crosses blood-brain barrier
e. Mercury persists in the environment and can be tracked indoors on clothes and shoes, re-exposing residents in enclosed buildings continually.

9. **Texas Study Mercury-Autism Link (http://no2crematory.files.wordpress.com/2010/09/txstudy.pdf)**
University of Texas "Environmental mercury release, special education rates, and autism disorder: and ecological study of Texas"
a. Concluded that for every 1000 lbs of environmentally released mercury, there was a 43% increase in the rate of special ed services, 61% increase in the rate of autism. *(mercury has long been suspected as the main culprit in the dramatic rise in autism rates)*

10. **toxic emission from crematories[env.intl] (http://no2crematory.files.wordpress.com/2011/01/toxic_emission_from-_crematoriesenv-intl.pdf)**
Environment International "Toxic Emissions from Crematories: A review"
a. Crematories have been identified as sources of various environmental pollutants, being polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs), and mercury those raising most concern.
b. The pollutants emitted by the combustion of organic matter with presence of other trace elements are: combustion gases (NOx, CO, SO2, PM....), heavy metals, and polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs), among other persistent organic pollutants. Heavy metals and PCDD/Fs, stand ut because of their toxicity and capacity for bioaccumulation, which means potential risks for human health. Because of their toxicological properties, together with their persistence capacity, PCDD/Fs were listed by the Stockholm Convention on Persistent Organic Pollutants of 2001 as one of the "dirty dozen" pollutants whose levels should be significantly reduced.
c. As a result of the US Cremation Association's meeting with the US EPA in November 1991, it became known that the original regulations proposed for crematories were based on no actual test data. Dental amalgams are unstable at cremation temperatures (650–700 °C), ... the free mercury metal is highly volatile.
d. Concluded that threw was an increased risk of lethal congenital anomaly (specifically spina bifida and heart defects) in relation to proximity to incinerators, and an increased risk of stillbirth and anencephalus in relation to proximity to crematoriums.

11. **Characterizing the Emissions from Crematoria (http://no2crematory.files.wordpress.com/2010/09/crematory_toxins1.pdf)**
Environmental Science Technology "Characterizing the Emissions of Polychlorinated Dibenzo-p-dioxins and Dibenzofurans from Crematories and Their Impacts to the Surrounding Environment"
a. Two crematories in Taiwan were tested, one with no emissions controls, the other with a bag filter.
b. The one with a bag filter removed 55.1% of the dioxins/furans, but both crematories still emitted significant amount of both compounds.
c. Determined that crematories with a low stack and no pollution controls are more of a threat to the community, but even after filtration, dioxin and furan levels are still at concentration levels well above "safe" levels
d. US EPA has reported that there appears to be no "safe" level for dioxin exposure, and the levels of dioxin and dioxin-like chemicals found in the general U.S. population were "at or near levels associated with adverse health effects:
e. Stacks on tested crematories were 5 m (16.4') and 6 m (19.7')

12. **PennStateStudy-Crematory-property-values(2010) (http://no2crematory.files.wordpress.com/2011/01/pennstatestudy-crematory-property-values2010.pdf)**
PENN State/University of Wyoming Study: "The impact of crematory operations on adjacent residential values"
a. Study of residential house sales in Rawlins, Wyoming, was conducted to estimate the impact of an environmental shock from a new point source upon adjacent residential property values.
b. Data spans 27 months of house sales: 7 months before, and 20 months after the startup of crematory operations. Data based on actual home sales.
c. Studied both direction and wind patterns, determined downwind to be an important factor in study as it applies to atmospheric pollutants.
d. Rawlins City Planner issued a building permit to the subject mortuary to install a 40 ton, two-chamber, natural gas-fired Millennium II crematory in a vehicle storage garage adjacent to the mortuary building.
e. Controversy remains as to whether this Planner was authorized to issue a permit for this expanded, nonconforming use of an existing funeral home facility in an area zoned for residences.

f. Citizens began complaining to City and State authorities about the crematory with its glaring, all-night illumination, noise and – most notable – noxious odor, which permeated residents' houses, making them feel ill and 'devaluing' (Morton, 2005) their properties.
g. Wyoming Department of Environmental Quality ordered an emissions test and determined that the crematory had emissions comparable to its state permit request with several notable exceptions: annual ambient cadmium and dioxin/furan concentrations at the crematory property boundary exceeded National (and Wyoming) Air Quality Standards, by approximately 205% and 2200%, respectively (URS, 2006). Hydrogen chloride concentrations at this boundary exceeded the one-hour US Environmental Protection Agency's 'remediation goal' by 797%, with sulfur oxide, nitrogen oxide, chromium, and mercury concentrations being from 43 to 74% of the Agency's National Standard or remediation goal. Cadmium, chromium, dioxin/furans, hydrogen chloride and mercury are toxins for which any positive concentration may have human health impacts. (Wexler, 2005).

More links:

Crematory_toxins (http://no2crematory.files.wordpress.com/2011/01/crematory_toxins.pdf)

Cadmium_poisoning (http://no2crematory.files.wordpress.com/2010/09/cadmium_poisoning.pdf)

http://no2crematory.files.wordpress.com/2010/09/technical_background_report_mercury.pdf
(http://no2crematory.files.wordpress.com/2010/09/technical_background_report_mercury.pdf)

http://no2crematory.files.wordpress.com/2010/09/smoke.pdf (http://no2crematory.files.wordpress.com/2010/09/smoke.pdf)
(http://no2crematory.files.wordpress.com/2010/09/polychlorinated_dibenzofurans.pdf)

(http://no2crematory.files.wordpress.com/2010/09/polychlorinated_dibenzofurans.pdf)

http://no2crematory.files.wordpress.com/2010/09/polychlorinated_dibenzofurans.pdf
(http://no2crematory.files.wordpress.com/2010/09/polychlorinated_dibenzofurans.pdf)

http://no2crematory.files.wordpress.com/2010/09/polychlorinated_dibenzodioxins.pdf
(http://no2crematory.files.wordpress.com/2010/09/polychlorinated_dibenzodioxins.pdf)

http://no2crematory.files.wordpress.com/2010/09/organochloride.pdf (http://no2crematory.files.wordpress.com/2010/09/organochloride.pdf)

http://no2crematory.files.wordpress.com/2010/09/nitrogen_oxide.pdf (http://no2crematory.files.wordpress.com/2010/09/nitrogen_oxide.pdf)

http://no2crematory.files.wordpress.com/2010/09/nitric_oxide.pdf (http://no2crematory.files.wordpress.com/2010/09/nitric_oxide.pdf)

http://no2crematory.files.wordpress.com/2010/09/mercury_poisoning.pdf (http://no2crematory.files.wordpress.com/2010/09/mercury_poisoning.pdf)

http://no2crematory.files.wordpress.com/2010/09/lead_poisoning.pdf (http://no2crematory.files.wordpress.com/2010/09/lead_poisoning.pdf)

http://no2crematory.files.wordpress.com/2010/09/hydrogen_chloride.pdf (http://no2crematory.files.wordpress.com/2010/09/hydrogen_chloride.pdf)

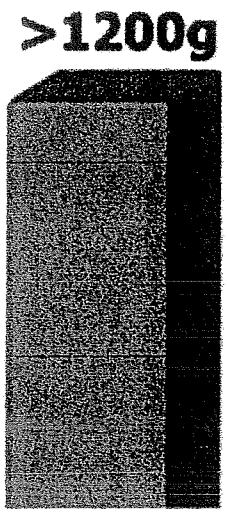
http://no2crematory.files.wordpress.com/2010/09/furans.pdf (http://no2crematory.files.wordpress.com/2010/09/furans.pdf)

http://no2crematory.files.wordpress.com/2010/09/formaldehyde.pdf (http://no2crematory.files.wordpress.com/2010/09/formaldehyde.pdf)

http://no2crematory.files.wordpress.com/2010/09/chromium_toxicity.pdf (http://no2crematory.files.wordpress.com/2010/09/chromium_toxicity.pdf)

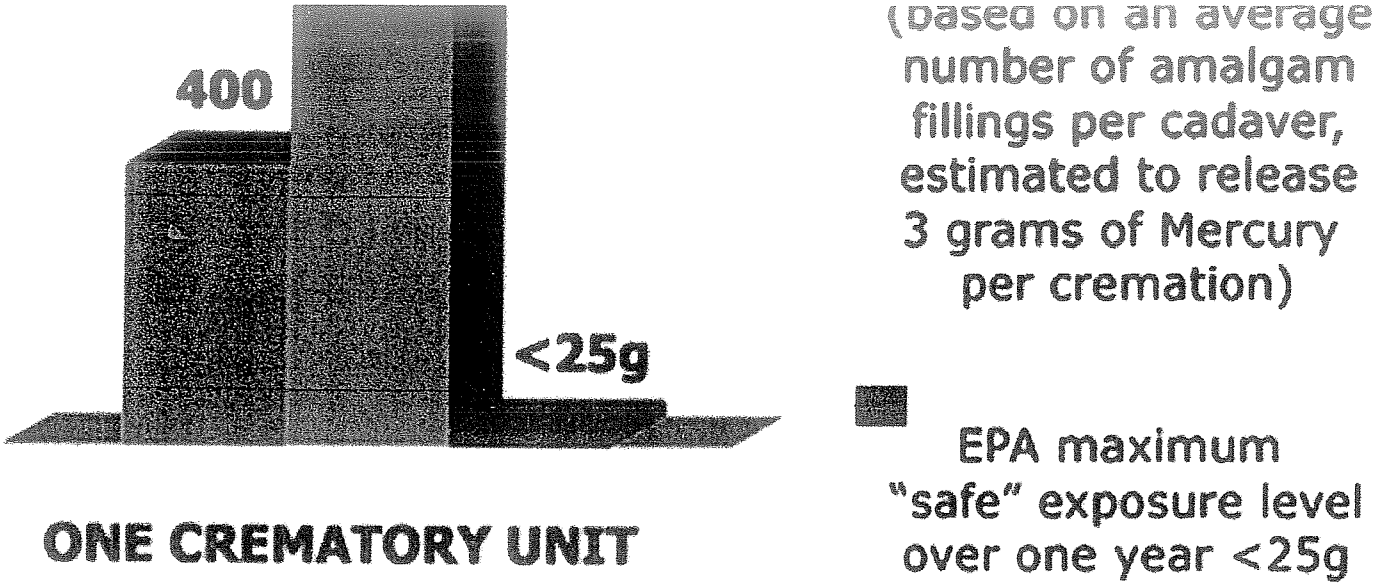
http://no2crematory.files.wordpress.com/2010/09/cadmium_poisoning.pdf (http://no2crematory.files.wordpress.com/2010/09/cadmium_poisoning.pdf)

What harm could one little crematory cause??



Average number of bodies cremated annually per crematory (Crematory Association of North America)

Grams of Mercury released annually



(http://no2crematory.files.wordpress.com/2011/01/mercury_impact_graph.jpg)

Mercury_Impact_Graph

The industry's data is mainly a mix of marketing materials and a few select tests on new crematories as they were being installed.

Rahill – Greening (http://no2crematory.files.wordpress.com/2011/01/rahill-greening.pdf)
Catholic Cremation A New Tradition In The Making (http://no2crematory.files.wordpress.com/2011/01/catholic-cremation_-a-new-tradition-in-the-making.pdf)
6655_narrative (http://no2crematory.files.wordpress.com/2011/01/6655_narrative.pdf)

Leave a Reply

You must be logged in (http://no2crematory.wordpress.com/wp-login.php?redirect_to=http%3A%2F%2Fno2crematory.wordpress.com%2Fthe-toxic-truth-data%2F) to post a comment.

ONE HUNDRED ELEVENTH CONGRESS

Congress of the United States

House of Representatives

COMMITTEE ON OVERSIGHT AND GOVERNMENT REFORM

2157 RAYBURN HOUSE OFFICE BUILDING

WASHINGTON, DC 20515–6143

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www.oversight.house.gov

January 15, 2010

The Honorable Lisa P. Jackson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue
Washington, DC 20460

Dear Administrator Jackson,

According to EPA, dentists historically use about 34 tons of mercury per year to place or replace dental mercury fillings in Americans. Mercury contained in the existing dental fillings of Americans is, in fact, one of the largest reservoirs of mercury in the United States. According to one estimate, dental mercury comprises over half of all mercury in use today, amounting to over 1000 tons.[1] Dental mercury is also a source of mercury pollution. EPA has developed several emission factors for three routes of atmospheric emissions of mercury related to dental use of mercury. They are: sewage sludge incineration, cremation of human remains containing mercury fillings, and direct air emissions from dental offices.

However, we are concerned that EPA has underestimated the amount of pollution that dental mercury accounts for, thereby rendering this problem a lower priority than it would otherwise be. On the basis of testimony our subcommittee has received, and the findings of our investigation, there appears to be ample justification for EPA to revise its emissions data. As you are fully aware, improving the accuracy of the emissions data is an important first step in improving regulation of dental mercury-source pollution. Yet even while the emission release data is being updated, we strongly urge you to require meaningful reductions of dental mercury into the water and air through goal-based regulatory controls, including mandatory employment of best management practices and amalgam separators, which is similar to what you required earlier when you were Commissioner of the New Jersey Department of Environmental Protection.

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[1] EPA International Mercury Market Study, cited in Mercury Policy Project, "Current Status of US Dental Mercury Reduction Initiatives," (Oct. 12, 2007).

Disparities in Data for Mercury Emissions Related to Dental Use of Mercury

At a November 14, 2007 hearing of the Domestic Policy Subcommittee, testimony from witnesses inside and outside the Agency revealed: 1) significant disparities between official EPA estimates and other, more recent emissions estimates, and 2) no emissions estimates for a number of significant additional pathways of dental mercury to the environment. For example, EPA has estimated airborne mercury attributable to wastewater sludge incineration to be 0.6 tons per year. Yet this figure, according to state regulatory officials and EPA itself, may significantly undercount sludge-related mercury pollution. In total, EPA earlier estimated mercury emissions from pathways related to dental use of mercury to be 1.5 tons per year, but in its March 6, 2008 response to my letter suggested that "...these emissions could be as much as two times higher..." Based on testimony provided to the subcommittee, a more realistic estimate for atmospheric emission of dental mercury could range from 7.1 tons per year to 9.4 tons per year, or up to six times official EPA estimates.[2]

The Northeast States for Coordinated Air Use Management (NESCAUM) estimated that mercury emissions from sludge incineration *in the northeast alone* amounted to 0.5 tons per year.[3] According to a northeast state official, "Sewage sludge incinerators were estimated to be the third largest point source of mercury emissions in the northeast prior to regional requirements that dentists use amalgam separators, and accounted for over 1,100 pounds of mercury or 12% of total emissions. This estimate did not include releases from wastewater or land applied sewage sludge, which would significantly increase the total."[4] Furthermore, EPA admits that its mercury emission data for sludge incineration is "poor," a deficiency it attributes to both the small number of facilities tested and the fact that these facilities were not a random sample of the industry.[5]

It also appears that EPA's estimate of mercury emissions from cremation significantly understates their actual magnitude. EPA estimated total mercury emitted as a byproduct of cremation of human remains to be around 0.3 tons per year. This official estimate is based

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[2] Testimony of Michael Bender, before the Domestic Policy Subcommittee (Nov. 14, 2007).

[3] NESCAUM, Inventory of Anthropogenic Emissions of Mercury, (Nov. 2005). Available at: http://www.nescaum.org/documents/inventory-of-anthroprogenic-mercury-emissions-in-the-northeast/

[4] Testimony of C. Mark Smith before the Domestic Policy Subcommittee, Oversight and Government Reform Committee, (Nov. 14, 2007).

[5] Emission Factor Documentation for AP-42 Section 2.2, Sewage Sludge Incineration, Office of Air Quality Planning and Standards, EPA, pp. 3-5 and 4-98, Jul. 1993. Available at: http://www.epa.gov/ttn/chief/ap42/ch02/bgdocs/ b02s02.pdf

entirely on a single test conducted at a single crematorium 10 years ago, and fails to explain the difference between the amount of mercury in fillings and the amount of mercury measured in crematorium emissions.[6] However, an article published in 2007 authored by an EPA environmental scientist estimates mercury emissions from cremation at about 3 tons per year, ten times the earlier EPA estimate.[7] According to the Cremation Association of America, there are about 1,900 crematoria in the US. Nationally, over 30% of Americans are now cremated, a figure that is anticipated to rise to 43% by 2025. The 1998 Northeastern States Mercury Study estimated that each person cremated had an average of 2.9 grams of mercury in fillings, and this figure is still widely considered to be in the right range.[8] From 2005 data, it is estimated that about 3.3 tons of mercury were emitted by crematoria that year.[9] In the model used, 25% of these emissions were assumed attached to particulates, which would settle to the ground locally and be classified as land deposition, and 75% assumed to be elemental mercury emissions to the atmosphere. Based on a literature review including ground deposition studies in New Zealand and Norway[10], it appears justifiable to allocate up to 90% of the mercury entering crematoria as emissions to the atmosphere, with some of the balance retained, at least temporarily, in combustion equipment and the stack.

During the next 25 years, emissions from crematoria are expected to rise considerably. There are two simultaneous trends contributing to this: a rise in the average number of fillings per person cremated (better dental health care has resulted in the retention of more teeth, and more fillings, as people age), and a rise in the number of cremations. This will only eventually be counter-balanced by the gradually increasing replacement of amalgam fillings with mercury-free alternatives.

EPA has not developed emissions factors for a number of additional pathways

As pointed out in a letter to EPA dated February 11, 2008, there are a number of other dental mercury-related air emissions for which EPA has not developed emission factors, including dental mercury:

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[6] Appendix A: NEI Nonpoint HAP Source Estimates – Human Cremation, Footnote 3.

[7] Alexis Cain et al. "Substance Flow Analysis of Mercury Intentionally Used in Products in the United States," Journal of Industrial Ecology, Volume 11, Number 3 (2007).

[8] J Reindl, Summary of References on Mercury Emissions from Crematoria, Dane County, (Nov. 3, 2008), available at: http://www.ejnet.org/crematoria/reindl.pdf

[9] A Cain, S Disch, C Twaroski, J Reindl and CR Case, Substance Flow Analysis of Mercury Intentionally Used in Products in the United States, Journal of Industrial Ecology, Volume 11, Number 3.

[10] *Id.*

- in sludge that is landfilled or spread on agricultural or forest land, or that is dried before it is used as fertilizer;
- in infectious and hazardous waste; in general municipal waste;
- in human respiration;
- in waste removed as grit and fines at wastewater treatment plants and disposed of in a variety of ways, and
- in combined sewer overflows.

EPA has Statutory Authority to Revise and Improve Emissions Data

As you know, EPA is required by the Clean Air Act (CAA) (codified at 42 U.S.C. §7401-7671) to monitor emissions and develop emission standards for a number of hazardous air pollutants, including mercury. Section 114 authorizes EPA to conduct inspections and to require monitoring at emission sources for developing emission standards, determining violations and "carrying out any provision of this Act." Section 130 requires the Agency to "permit any person to demonstrate improved emissions estimating techniques, and following approval of such techniques, the Administrator shall authorize the use of such techniques." Under the CAA, EPA is moving forward with MACT standards to control releases from coal-fired power plants. EPA has also recently published emission standards for mercury and other pollutants originating in hazardous waste incinerators, mercury-cell chlor-alkali plants, iron and steel foundries, and industrial boilers. But in spite of glaring deficiencies in its appreciation of the extent of emissions that relate to dental mercury, EPA has chosen not to revise and improve its data as the circumstances call for, and as the law requires.

Subcommittee Requests

We believe there is ample justification for EPA to invoke its authority to reevaluate and update its own emissions estimating techniques, and to consider the whole range of mercury emission sources attributable to dental applications. We call upon EPA to do so in a timely manner, and the Subcommittee looks forward to learning of EPA's work plan and schedule to address this issue.

To that end, we request that EPA provide the Subcommittee with a plan, complete with projected dates, by which it will reevaluate and update its own mercury emissions factors for wastewater sludge incineration and crematoria, and establish mercury emissions factors for: sludge that is landfilled or spread on agricultural or forest land, or that is dried before it is used as fertilizer; infectious and hazardous waste; general municipal waste; human respiration; waste removed as grit and fines at wastewater treatment plants and disposed of in a variety of ways, and combined sewer overflows. We request that EPA provide this plan to the Subcommittee **no later than Tuesday, February 16, 2010 at 5:00 p.m.**

The Oversight and Government Reform Committee is the principal oversight committee in the House of Representatives and has broad oversight jurisdiction as set forth in House Rule X.

If you have any questions regarding this request, please contact Jaron Bourke, Staff Director, at (202) 225-6427.

Sincerely,

Dennis J. Kucinich
Chairman
Domestic Policy Subcommittee

Diane E. Watson
Member
Domestic Policy Subcommittee

Mercury in Crematoria Using Atomic Fluorescence Spectrometry

Anthropogenic emission of mercury into the environment continues to cause concern. Increasing control of atmospheric mercury emissions is resulting in the gradual overall fall in mercury emissions. UK annual mercury emissions are estimated to have fallen from 40.7 t to 6.9 t between 1982 and 2002.[1] In contrast to the overall fall, mercury emissions from crematoria have increased significantly. One estimate shows annual mercury emissions from UK crematoria more than doubling from 0.36 t to 0.82 t between 1982 and 2002 with little change in the number of cremations performed.[2] The increase in emissions is readily attributable to the use of amalgam fillings and, due to better dental health, a large decrease in the number of people edentate at the time of death. Estimates of the mass of mercury present in an average cremation vary significantly, with estimates generally between 0.9 g and 3.0 g.[2]. The mass present depends on dental health practices, thus varying with time period and with country.

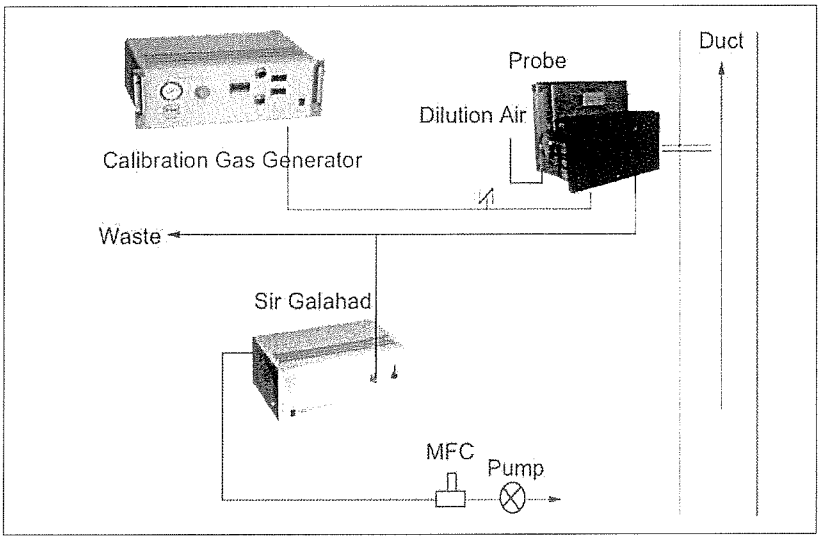


Figure 1: Schematic of apparatus

Mercury emissions during the cremation process are almost entirely due to the presence of amalgam fillings in the cadaver. This leads to very significant variation in the concentration of mercury emitted, in each cremation.

The P S Analytical Sir Galahad amalgamation-atomic fluorescence spectrometer is a proven technique for the determination of mercury in a wide range of gaseous media. This article summarizes the results of a study of mercury emissions on a single crematory stack at a UK crematorium and introduces a Hg CEM, specifically designed to continuously monitor mercury concentrations in cremation gases for regulatory purposes or online process control of mercury abatement equipment.

Instrumentation & Experimental

The PSA 10.680 system was designed to continuously monitor mercury emissions in incineration flue gas streams. For crematorium systems this consists of a PSA S123P100 heated dilution probe with pre-filter and thermo-catalyst. Sample gas was drawn into probe by an eductor-based dilution assembly and diluted 40:1 in compressed air. The probe is heated and all sample-wetted components are constructed with selected materials to avoid mercury losses. The diluted sample was drawn through the Sir Galahad amalgamation-AFS analyser by a mass flow controller (MFC) and pump assembly. The instrumentation was calibrated using the PSA 10.536 Calibration gas generator, calibration gas being introduced at the probe upstream of the filter. The apparatus is shown schematically in Figure 1 and a typical response in Figure 2.

To test for the presence of oxidised mercury in the flue gas, a side stream of the diluted sample gas was taken directly at the outlet to the probe and bubbled through an impinger containing 100 ml of 10 % potassium chloride solution connected as closely as possible to the sample probe. Flow control was by an MFC and pump downstream of the impinger. Sample was bubbled through the impinger for the duration of a cremation. This was repeated for the four cremations shown in Figure 2. The capture solutions were then treated with hydrochloric acid and potassium bromide-bromate and the mercury concentration determined by cold vapour AFS using the PSA 10.025 Millennium Merlin. Results are presented in Table 1.

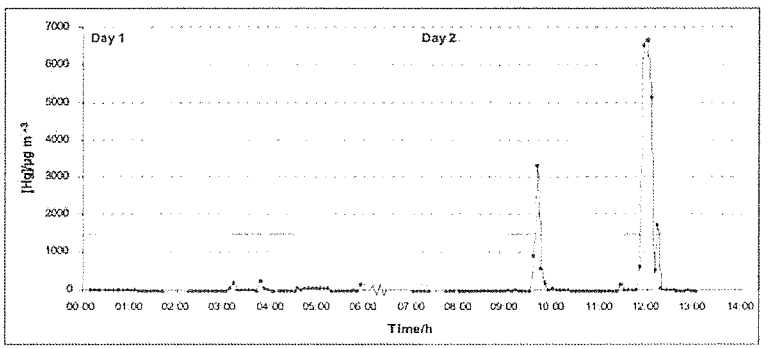


Figure 2: Typical mercury in crematory flue gas response. (Red lines indicate periods a cremation was in progress.)

Cremation No.	Mean Gas Phase Concentration		
	$[Hg^{2+}]$ /µg m^{-3}	$[Hg^{total}]$ /µg m^{-3}	Max. Oxidised mercury (%)
1	19.6	25.8	75
2	27.3	47.5	57
3	100.4	282.0	36
4	314.8	1094.5	29

Table 1: Speciation analysis for the four cremations

Discussion

The results shown in Figure 2 demonstrate the variability in mercury concentration that occurs in crematoria flue gases. Only minimal information is available about the deceased, making predictions of the mercury concentration for a particular cremation difficult.

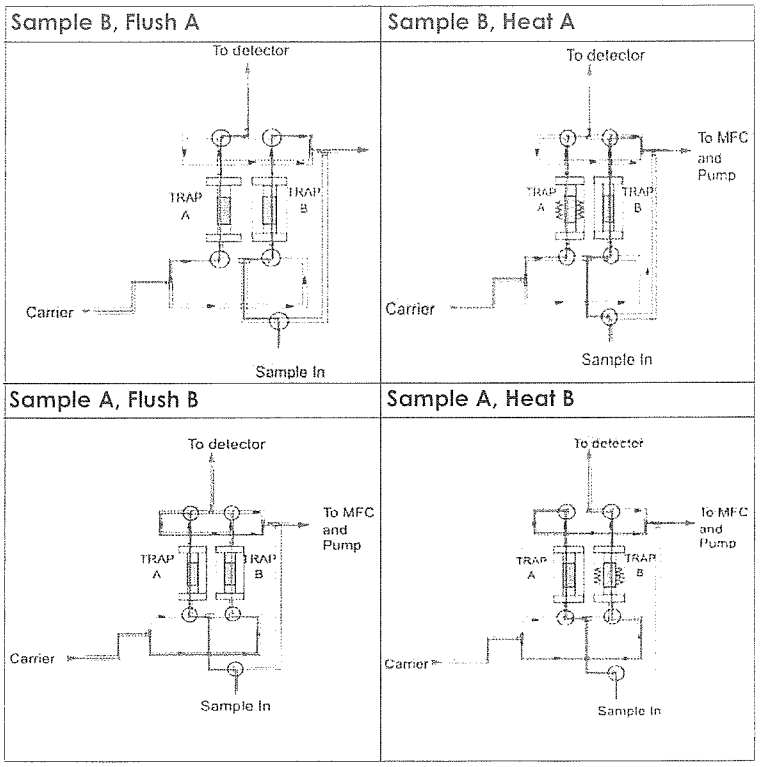


Figure 3: Schematic Diagram of the Sir Galahad

In the example presented here low levels of mercury observed for the first two cremations suggest that the deceased were edentate or had no amalgam fillings, whereas the 3rd and 4th cremations show high levels of mercury consistent with several amalgam fillings. Where mercury was present, it is emitted in a fairly short time period, approximately 40 min into the cremation cycle, this is consistent with the mercury remaining in the fillings until it evaporates during the combustion process. This tight time span of mercury removal is significant for any mercury abatement process employed, as the removal system must be able to handle spikes in concentration of several mg m^{-3}, with only background levels between spikes. Thus, process control of the abatement system with feedback from a mercury monitoring system could result in significant cost savings compared to running the abatement system continuously.

It is not possible in this case to estimate the mass of mercury emitted in each cremation as the volumetric flow in the flue had not been characterised. The data in Table 1 show that significant levels of oxidised mercury were present in the flue gas. The variation in oxidation may be due to differing levels of oxidising components, believed to be principally HCl, between the different cremations. That at least a quarter of the mercury present was oxidised has implications for any mercury abatement system due to the different chemistries of elemental and oxidised mercury. Oxidised forms of mercury are also water soluble and are therefore more likely to present a localized pollution of mercury during periods of precipitation compared to elemental mercury.

The various factors discussed above were accounted for when the Crematorium Hg CEM was designed. This system is described below:

AUTHOR DETAILS
Warren T Corns, Matthew A Dexter and Peter B Stockwell
P S Analytical, UK
Web: www.psanalytical.com

PSA Crematorium Hg CEM

The PSA Crematorium Hg Continuous Emission Monitor (CEM) consists of two principal components; a dilution probe (S123P100) and an analyser unit 10.665/10.680. The sample probe is designed to require minimal maintenance and to be as small and unobtrusive as possible for installation in awkward locations.

For system calibration and validation test purposes, elemental mercury calibration gas generated within the CEM is automatically introduced to the system upstream of the probe filter at flow rates suitable to totally displace the sample stream.

The diluted preconditioned sample is continuously delivered to the PSA 10.525F Continuous Sampling Sir Galahad analyser where the mercury concentration is continuously determined by amalgamation atomic fluorescence spectroscopy in less than 3 minutes per sample (a result is reported every 90 s). A carrier gas of compressed air is required.

Amalgamation-Atomic Fluorescence Spectroscopy

Hg is determined by the Sir Galahad analyser using amalgamation-atomic fluorescence spectrometry. Mercury is captured on the Amasil™ gold substrate by amalgamation, pre-concentrating the mercury and separating it from the sample matrix and thus overcoming potential interferences. Sample flow onto the Amasil tube is controlled by an MFC and pump arrangement to deliver an accurate sample volume to the analyser. Two Amasil™ tubes are used in parallel such that one tube is sampling while the other is being desorbed to ensure that any transient mercury spikes are determined. A photograph and schematic flow diagram is shown in Figures 3.

After sampling, the mercury collected is thermally desorbed into a carrier gas stream and subsequently delivered to a non dispersive atomic fluorescence spectrometer where mercury is detected at 253.7 nm. The combination of AFS with amalgamation offers excellent selectivity, an absolute detection limit of 0.1 pg and up to 8 orders of magnitude linearity.

Automation and System Integration

The system is designed to operate with minimal user intervention. The system can be operated unattended for several months, with internal QC checks performed automatically to ensure data validity. The system can readily be integrated with other data handling and control equipment using a variety of different communication protocols.

Conclusion

The mercury emissions from a crematory have been studied by amalgamation-atomic fluorescence spectroscopy. Mercury is emitted in a short period approximately 40 min into the cremation. The concentration emitted varies significantly but can be as high as several mg m^{-3}. Both elemental and ionic mercury are emitted during the cremation. The ratio of the two forms depends on the total level of mercury being emitted.

The PSA Crematorium Hg CEM has been presented, this system is specifically designed to determine mercury in crematorium flue gas for process control or regulatory purposes. The system is designed to operate with minimal user intervention.

References

1. National Atmospheric Emission Inventory, Emission Statistics by UNECE Source Category: Inventory Year 2006, AEA Technology, August 2008 (accessed online www.naei.org.uk Feb 2009)
2. N. Passant, Review of emission factors for mercury emitted from cremation, AEA Technology, June 2004

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

MERCURY
CAS # 7439-97-6

Agency for Toxic Substances and Disease Registry ToxFAQs April 1999

This fact sheet answers the most frequently asked health questions (FAQs) about mercury. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to mercury occurs from breathing contaminated air, ingesting contaminated water and food, and having dental and medical treatments. Mercury, at high levels, may damage the brain, kidneys, and developing fetus. This chemical has been found in at least 714 of 1,467 National Priorities List sites identified by the Environmental Protection Agency.

What is mercury?
(Pronounced mŭr'kyə-rē)

Mercury is a naturally occurring metal which has several forms. The metallic mercury is a shiny, silver-white, odorless liquid. If heated, it is a colorless, odorless gas.

Mercury combines with other elements, such as chlorine, sulfur, or oxygen, to form inorganic mercury compounds or "salts," which are usually white powders or crystals. Mercury also combines with carbon to make organic mercury compounds. The most common one, methylmercury, is produced mainly by microscopic organisms in the water and soil. More mercury in the environment can increase the amounts of methylmercury that these small organisms make.

Metallic mercury is used to produce chlorine gas and caustic soda, and is also used in thermometers, dental fillings, and batteries. Mercury salts are sometimes used in skin lightening creams and as antiseptic creams and ointments.

What happens to mercury when it enters the environment?

- Inorganic mercury (metallic mercury and inorganic mercury compounds) enters the air from mining ore deposits, burning coal and waste, and from manufacturing plants.
- It enters the water or soil from natural deposits, disposal of wastes, and volcanic activity.
- Methylmercury may be formed in water and soil by small organisms called bacteria.
- Methylmercury builds up in the tissues of fish. Larger and older fish tend to have the highest levels of mercury.

How might I be exposed to mercury?

- Eating fish or shellfish contaminated with methylmercury.
- Breathing vapors in air from spills, incinerators, and industries that burn mercury-containing fuels.
- Release of mercury from dental work and medical treatments.
- Breathing contaminated workplace air or skin contact during use in the workplace (dental, health services, chemical, and other industries that use mercury).
- Practicing rituals that include mercury.

How can mercury affect my health?

The nervous system is very sensitive to all forms of mercury. Methylmercury and metallic mercury vapors are more harmful than other forms, because more mercury in these forms reaches the brain. Exposure to high levels of metallic, inorganic, or organic mercury can permanently damage the brain, kidneys, and developing fetus. Effects on brain functioning may result in irritability, shyness, tremors, changes in vision or hearing, and memory problems.

Short-term exposure to high levels of metallic mercury vapors may cause effects including lung damage, nausea,

vomiting, diarrhea, increases in blood pressure or heart rate, skin rashes, and eye irritation.

How likely is mercury to cause cancer?

There are inadequate human cancer data available for all forms of mercury. Mercuric chloride has caused increases in several types of tumors in rats and mice, and methylmercury has caused kidney tumors in male mice. The EPA has determined that mercuric chloride and methylmercury are possible human carcinogens.

How can mercury affect children?

Very young children are more sensitive to mercury than adults. Mercury in the mother's body passes to the fetus and may accumulate there. It can also can pass to a nursing infant through breast milk. However, the benefits of breast feeding may be greater than the possible adverse effects of mercury in breast milk.

Mercury's harmful effects that may be passed from the mother to the fetus include brain damage, mental retardation, incoordination, blindness, seizures, and inability to speak. Children poisoned by mercury may develop problems of their nervous and digestive systems, and kidney damage.

How can families reduce the risk of exposure to mercury?

Carefully handle and dispose of products that contain mercury, such as thermometers or fluorescent light bulbs. Do not vacuum up spilled mercury, because it will vaporize and increase exposure. If a large amount of mercury has been spilled, contact your health department. Teach children not to play with shiny, silver liquids.

Properly dispose of older medicines that contain mercury. Keep all mercury-containing medicines away from children.

Pregnant women and children should keep away from rooms where liquid mercury has been used.

Learn about wildlife and fish advisories in your area from your public health or natural resources department.

Is there a medical test to show whether I've been exposed to mercury?

Tests are available to measure mercury levels in the body. Blood or urine samples are used to test for exposure to metallic mercury and to inorganic forms of mercury. Mercury in whole blood or in scalp hair is measured to determine exposure to methylmercury. Your doctor can take samples and send them to a testing laboratory.

Has the federal government made recommendations to protect human health?

The EPA has set a limit of 2 parts of mercury per billion parts of drinking water (2 ppb).

The Food and Drug Administration (FDA) has set a maximum permissible level of 1 part of methylmercury in a million parts of seafood (1 ppm).

The Occupational Safety and Health Administration (OSHA) has set limits of 0.1 milligram of organic mercury per cubic meter of workplace air (0.1 mg/m^3) and 0.05 mg/m^3 of metallic mercury vapor for 8-hour shifts and 40-hour work weeks.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological profile for mercury. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Suggested Action Levels for Indoor Mercury Vapors in Homes

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

General Exposure Assessment Considerations: The primary route of entry for metallic mercury is by inhalation; ingestion and skin absorption of this form of mercury is usually not biologically significant. Sensitive populations to mercury exposure are those with developing central nervous systems, including young children and the fetuses of women who are pregnant. Other individuals of potential concern are those with pre-existing kidney conditions, usually at exposures to much higher concentrations than the first group. The specific exposure of these groups in any given situation should be considered when assessing the need for any given response action. Specific concerns are mentioned in the tables below. If there is any doubt, responders should consult with state or local public health officials before deciding on a course of action. Responders may also contact ATSDR at 404-639-0615, 24 hours a day.

Exposure Assumptions for Different Settings: For the purposes of this document, the residentially exposed population includes infants, small children, and pregnant women presumed to have inhaled mercury for a period up to 24 hours per day, 7 days per week potentially for months or even years. Occupational or commercial settings include those individuals that are primarily healthy adults exposed up to 8-10 hours per day, 40 hours per week, with transient exposures by sensitive populations (e.g., a retail establishment or schools). The concentrations provided as suggested action levels are for comparison to the environmental data collected in affected residences and workplaces.

Background: An *action level* is an indoor air concentration of mercury vapor, which should prompt consideration of the need to implement a recommended response by public health and environmental officials. The various suggested action levels provided in this document are intended as recommendations, not as regulatory values or cleanup values, although some may correspond to present or future values adopted by regulatory authorities. The suggested action levels presented in this document recognize that an individual must be exposed to a sufficient concentration over some specific period of time in order for mercury vapor to cause adverse health effects. The suggested action levels also recognize that while individual susceptibility may vary, developing fetuses and young children under six years old are generally at higher risk than others of incurring adverse health effects from exposure to mercury vapor. If the indoor air concentration corresponding to any suggested action level is exceeded, then a potential health risk may be present, and responders should evaluate the exposures at that location and consider implementing appropriate protective measures to reduce or eliminate the risk.

The suggested action levels presented here are based on data available in ATSDR's Toxicological Profile for Mercury (1999) or in the Hazardous Substance Databank of the Toxicology Data Network at the National Library of Medicine. ATSDR has also made use of additional data collected by the US Environmental Protection Agency (US EPA) and of specific experiences at other sites. Other factors considered in the development include available information on normal background levels and analytical detection limits of various techniques for evaluating airborne contamination. Any information specific to the exposures at any given location as described below should also be considered before implementing a response action.

These suggested action levels are extrapolated from health guidance values (HGVs) independently developed by two federal agencies, ATSDR and EPA. These HGVs are based on both animal studies and human epidemiology studies that detail the health effects of inhalation of mercury-contaminated air. ATSDR has developed a chronic Minimal Risk Level (MRL) of 0.2 ug/m3 that is based on a 1983 study of workers exposed to an average Lowest Observed Adverse Effect Level (LOAEL) of 26 ug/m3 over an average of 15 years. This workplace average exposure was adjusted from a 40 hour per week exposure to a 168 hour per week exposure (i.e., 24 hours/day, 7 days/week) and then divided by an uncertainty factor of 30 to account for the use of the LOAEL and the different sensitivities of individuals. In addition, EPA has used the same study to develop a Reference Concentration (RfC) of 0.3 ug/m3, using different assumptions and uncertainty factors. ATSDR considers the RfC and the Chronic MRL to be the same value for all practical purposes. A MRL, then, is defined as an estimate of the daily exposure level to a hazardous substance (in this case, metallic mercury) that is likely to be without appreciable risk of adverse, non-cancer health effects (metallic mercury is not considered to be a carcinogenic substance) over a specific exposure route and duration of exposure. For further information, see Section 2.5, Chapter 7, and Appendix A of the ATSDR Tox Profile and the EPA's Integrated Risk Information System (IRIS) on the Internet at **www.epa.gov/iriswebp/iris/index.html**

Suggested Action Levels for Mercury (CAS # 7439-97-6)

Indoor Air Concentration (ug/m 3)	Use of the Action Level	Method of Analysis *	Reference
<1.0	Level acceptable for occupancy of any structure after a spill (also called the residential occupancy level)	NIOSH 6009 or Real-time air monitoring with Lumex or equivalent	ATSDR HGVs 1999. EPA/IRIS
10	Isolate residents from the exposure	Real-time air monitoring instrument (i.e., Jerome™ or Lumex meter equivalent)	ATSDR, 1999.
10	Acceptable level in a modified test procedure to allow personal effects to remain in the owner's possession	Real-time air monitoring instrument (i.e., Jerome™ or Lumex meter equivalent)	
3.0	Re-occupancy after a spill of an occupational or commercial setting where mercury is not usually handled.	NIOSH 6009 or equivalent	ATSDR HGVs 1999. EPA/IRIS
25	Occupational settings where mercury is handled. ♂	Real-time air monitoring instrument (i.e., Jerome™ or Lumex meter equivalent)	HSDB, 1999
25	Response Worker Protective Equipment Upgrade. ♂	Real-time air monitoring instrument (i.e., Jerome™ or Lumex meter equivalent)	29 CFR 1910.120; 40 CFR 311; NIOSH, 1987
10,000	IDLH. Response Workers Protective Equipment upgrade.	Real-time air monitoring instrument (i.e., Jerome™ or Lumex meter equivalent)	29 CFR 1910.120; 40 CFR 311;NIOSH 1987

*** Environmental analysis should be in accordance with the requirements specified by environmental authorities. When real-time air monitoring instruments are specified in this table, laboratory analysis may be substituted at the discretion of the risk managers involved in the event. Operation of real-time instruments should be in accordance with manufacturer's instructions.**

♂ Women workers in these settings who are pregnant or attempting to become pregnant should consult their physicians regarding their mercury exposure.

Occupational Safety and Health Guideline for Mercury Vapor

DISCLAIMER:

These guidelines were developed under contract using generally accepted secondary sources. The protocol used by the contractor for surveying these data sources was developed by the National Institute for Occupational Safety and Health (NIOSH), the Occupational Safety and Health Administration (OSHA), and the Department of Energy (DOE). The information contained in these guidelines is intended for reference purposes only. None of the agencies have conducted a comprehensive check of the information and data contained in these sources. It provides a summary of information about chemicals that workers may be exposed to in their workplaces. The secondary sources used for supplements 111 and 1V were published before 1992 and 1993, respectively, and for the remainder of the guidelines the secondary sources used were published before September 1996. This information may be superseded by new developments in the field of industrial hygiene. Therefore readers are advised to determine whether new information is available.

Introduction | Recognition | Controls | References

Introduction

This guideline summarizes pertinent information about mercury vapor for workers and employers as well as for physicians, industrial hygienists, and other occupational safety and health professionals who may need such information to conduct effective occupational safety and health programs. Recommendations may be superseded by new developments in these fields; readers are therefore advised to regard these recommendations as general guidelines and to determine whether new information is available.

Recognition

SUBSTANCE IDENTIFICATION

* Formula
Hg

* Structure
(For Structure, see paper copy)

* Synonyms
None reported.
* Identifiers

1. CAS No.: 7439-97-6
2. RTECS No.: OV4550000
3. Specific DOT number: None
4. Specific DOT label: None

* Appearance and odor

Mercury vapor is the vapor generated from elemental liquid mercury or compounds of mercury. No information is available on the appearance or odor of mercury vapor.

CHEMICAL AND PHYSICAL PROPERTIES

* Physical data

1. Atomic weight: 200.59
2. Boiling point: Not applicable.
3. Specific gravity: Not applicable.
4. Vapor density: Data not available.
5. Melting/Freezing point: Not applicable.
6. Vapor pressure: Not applicable.
7. Solubility: Not applicable.
8. Evaporation rate: Not applicable.

* Reactivity

1. Conditions contributing to instability: None reported.
2. Incompatibilities: None reported.
3. Hazardous decomposition products: Not applicable.
4. Special precautions: None reported.

* Flammability

The National Fire Protection Association has not assigned a flammability rating to mercury vapor.

1. Flash point: Not applicable.
2. Autoignition temperature: Not applicable.
3. Flammable limits in air: Not applicable.
4. Extinguishant: Use an extinguishant that is suitable for the materials involved in the surrounding fire.

Fires involving mercury vapor should be fought upwind from the maximum distance possible. Isolate the hazard area and deny access to unnecessary personnel. Firefighters should wear a full set of protective clothing and self-contained breathing apparatus when fighting fires involving mercury vapor.

EXPOSURE LIMITS

* OSHA PEL

The current Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL) for mercury vapor is 0.1 milligram per cubic meter (mg/m(3)) of air as a ceiling limit. A worker's exposure to mercury vapor shall at no time exceed this ceiling level.

* NIOSH REL

The National Institute for Occupational Safety and Health (NIOSH) has established a recommended exposure limit (REL) for mercury vapor of 0.05 mg/m(3) as a TWA for up to a 10-hour workday and a 40-hour workweek. NIOSH also assigns a "Skin" notation, which indicates that the cutaneous route of exposure, including mucous membranes and eyes, contributes to overall exposure [NIOSH 1992].

* ACGIH TLV

The American Conference of Governmental Industrial Hygienists (ACGIH) has assigned mercury vapor a threshold limit value (TLV) of 0.025 mg/m(3) as a TWA for a normal 8-hour workday and a 40-hour workweek and considers mercury vapor an A4 substance (not classifiable as a human carcinogen). The ACGIH also assigns a "Skin" notation to mercury vapor [ACGIH 1994, p. 25].

* Rationale for Limits

The NIOSH limit is based on the risk of central nervous system damage, eye, skin, and respiratory tract irritation [NIOSH 1992].
The ACGIH has not published documentation for the current TLV for mercury vapor. The 1991 Documentation of Threshold Limit Values (6th edition) discusses the basis for the prior TLV of 0.05 mg/m(3), but does not discuss the current TLV for mercury vapor [ACGIH 1991, p. 881].

HEALTH HAZARD INFORMATION

* Routes of Exposure

Exposure to mercury vapor can occur through inhalation, and eye or skin contact.

* Summary of toxicology

1. Effects on Animals: Mercury vapor can damage the kidneys, liver, brain, heart, lungs and colon in experimental animals. It is also mutagenic and can affect the immune system. Rabbits exposed for a single 4 hour period to mercury vapor at a concentration of 28.8 mg/m(3) developed severe damage to the kidneys, liver, brain, heart, lungs, and colon [Clayton and Clayton 1981]. Rabbits exposed to 0.86 mg/m(3) for 6 weeks had significant brain and kidney damage, which resolved on cessation of exposure. Exposure to 6 mg/m(3) mercury vapor caused severe damage to the kidney, heart, lung, and brain of rabbits; however, dogs exposed to 0.1 mg/m(3) for 83 weeks had no microscopic indication of tissue damage [Clayton and Clayton 1981]. Mercury may injure the kidneys through an autoimmune mechanism [ACGIH 1991]. Mercury was mutagenic in eukaryotic cells [ACGIH 1991].
2. Effects on Humans: Mercury vapor can cause effects in the central and peripheral nervous systems, lungs, kidneys, skin and eyes in humans. It is also mutagenic and affects the immune system [Hathaway et al. 1991; Clayton and Clayton 1981; Rom 1992]. Acute exposure to high concentrations of mercury vapor causes severe respiratory damage, while chronic exposure to lower levels is primarily associated with central nervous system damage [Hathaway et al. 1991]. Chronic exposure to mercury is also associated with behavioral changes and alterations in peripheral nervous system [ACGIH 1991]. Pulmonary effects of mercury vapor inhalation include diffuse interstitial pneumonitis with profuse fibrinous exudation [Gosselin 1984]. Glomerular dysfunction and proteinuria have been observed mercury exposed workers [ACGIH 1991]. Chronic mercury exposure can cause discoloration of the cornea and lens, eyelid tremor and, rarely, disturbances of vision and extraocular muscles [Grant 1986]. Delayed hypersensitivity reactions have been reported in individuals exposed to mercury vapor [Clayton and Clayton 1981]. Mercury vapor is reported to be mutagenic in humans, causing aneuploidy in lymphocytes of exposed workers [Hathaway et al. 1991].

* Signs and symptoms of exposure

1. Acute exposure: Acute inhalation of mercury vapor may result in toxicity similar to metal fume fever including chills, nausea, general malaise, tightness in the chest, chest pains, dyspnea, cough, stomatitis, gingivitis, salivation, and diarrhea [ACGIH 1991; Hathaway et al. 1991].
2. Chronic exposure: Chronic exposure to mercury may result in weakness, fatigue, anorexia, weight loss, and disturbance of gastrointestinal function. A tremor may develop beginning with the fingers, eyelids, and lips which may progress to generalized trembling of the entire body and violent chronic spasms of the extremities. Parallel with development of the tremors, behavioral and personality changes may develop including increased excitability, memory loss, insomnia, and depression. The skin may exhibit abnormal blushing, dermographia, excessive sweating and irregular macular rashes. Severe salivation and gingivitis is also characteristic of chronic toxicity [Hathaway et al. 1991; Gosselin 1984]. Another manifestation of chronic mercury exposure is characterized by apathy, anorexia, flush, fever, a nephrotic syndrome with albuminuria and generalized edema, diaphoresis, photophobia, insomnia and a pruritic and sometimes painful scaling or peeling of the skin of the hands and feet with bullous lesions [Gosselin 1984].

EMERGENCY MEDICAL PROCEDURES

* Emergency medical procedures: [NIOSH to supply]

Rescue: Remove an incapacitated worker from further exposure and implement appropriate emergency procedures (e.g., those listed on the Material Safety Data Sheet required by OSHA's Hazard Communication Standard [29 CFR 1910.1200]). All workers should be familiar with emergency procedures, the location and proper use of emergency equipment, and methods of protecting themselves during rescue operations.

EXPOSURE SOURCES AND CONTROL METHODS

The following operations may involve mercury and lead to worker exposures to the vapor of this substance:

- The mining, production, and transportation of mercury
- The mining and refining operations for gold and silver ores
- Use in thermometers, manometers, barometers, gauges, and valves
- Use in amalgams for dentistry, preservatives, heat transfer technology, pigments, catalysts, and in lubricating oils

Methods that are effective in controlling worker exposures to mercury vapor, depending on the feasibility of implementation, are as follows:

- Process enclosure
- Local exhaust ventilation
- General dilution ventilation
- Personal protective equipment

Workers responding to a release or potential release of a hazardous substance must be protected as required by paragraph (q) of OSHA's Hazardous Waste Operations and Emergency Response Standard [29 CFR 1910.120].

Good sources of information about control methods are as follows:

1. ACGIH [1992]. Industrial ventilation--a manual of recommended practice. 21st ed. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.
2. Burton DJ [1986]. Industrial ventilation--a self study companion. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.
3. Alden JL, Kane JM [1982]. Design of industrial ventilation systems. New York, NY: Industrial Press, Inc.
4. Wadden RA, Scheff PA [1987]. Engineering design for control of workplace hazards. New York, NY: McGraw-Hill.
5. Plog BA [1988]. Fundamentals of industrial hygiene. Chicago, IL: National Safety Council.

Controls

MEDICAL SURVEILLANCE

OSHA is currently developing requirements for medical surveillance. When these requirements are promulgated, readers should refer to them for additional information and to determine whether employers whose employees are exposed to mercury vapor are required to implement medical surveillance procedures.

* Medical Screening

Workers who may be exposed to chemical hazards should be monitored in a systematic program of medical surveillance that is intended to prevent occupational injury and disease. The program should include education of employers and workers about work-related hazards, early detection of adverse health effects, and referral of workers for diagnosis and treatment. The occurrence of disease or other work-related adverse health effects should prompt immediate evaluation of primary preventive measures (e.g., industrial hygiene monitoring, engineering controls, and personal protective equipment). A medical surveillance program is intended to supplement, not replace, such measures. To detect and control work-related health effects, medical evaluations should be performed (1) before job placement, (2) periodically during the term of employment, and (3) at the time of job transfer or termination.

* Pre-placement medical evaluation

Before a worker is placed in a job with a potential for exposure to mercury vapor, a licensed health care professional should evaluate and document the worker's baseline health status with thorough medical, environmental, and occupational histories, a physical examination, and physiologic and laboratory tests appropriate for the anticipated occupational risks. These should concentrate on the function and integrity of the eyes, skin, respiratory system, central and peripheral nervous systems, and kidneys. Medical surveillance for respiratory disease should be conducted using the principles and methods recommended by the American Thoracic Society.

A pre-placement medical evaluation is recommended to assess medical conditions that may be aggravated or may result in increased risk when a worker is exposed to mercury vapor at or below the prescribed exposure limit. The health care professional should consider the probable frequency, intensity, and duration of exposure as well as the nature and degree of any applicable medical condition. Such conditions (which should not be regarded as absolute contraindications to job placement) include a history and other findings consistent with diseases of the eyes, skin, respiratory system, central and peripheral nervous systems, or kidneys.

* Periodic medical evaluations

Occupational health interviews and physical examinations should be performed at regular intervals during the employment period, as mandated by any applicable Federal, State, or local standard. Where no standard exists and the hazard is minimal, evaluations should be conducted every 3 to 5 years or as frequently as recommended by an experienced occupational health physician. Additional examinations may be necessary if a worker develops symptoms attributable to mercury vapor exposure. The interviews, examinations, and medical screening tests should focus on identifying the adverse effects of mercury vapor on the eyes, skin, respiratory system, central and peripheral nervous systems, or kidneys. Current health status should be compared with the baseline health status of the individual worker or with expected values for a suitable reference population.

* Termination medical evaluations

The medical, environmental, and occupational history interviews, the physical examination, and selected physiologic or laboratory tests that were conducted at the time of placement should be repeated at the time of job transfer or termination to determine the worker's medical status at the end of his or her employment. Any changes in the worker's health status should be compared with those expected for a suitable reference population.

* Biological monitoring

Biological monitoring involves sampling and analyzing body tissues or fluids to provide an index of exposure to a toxic substance or metabolite. No biological monitoring test acceptable for routine use has yet been developed for mercury vapor. However, total inorganic mercury can be measured in the urine by preshift sampling. A mercury level of 35 micrograms per gram of creatinine should be used as the biological exposure index. In addition, total inorganic mercury can also be measured in the blood by sampling at the end of shift at the end of the workweek. A mercury level of 15 micrograms per liter of blood should be used as the biological exposure index.

WORKPLACE MONITORING AND MEASUREMENT

Determination of a worker's exposure to airborne mercury vapor is made using a Hydrar or Hopcalite tube (200 mg section), SKC brand with a prefilter/cassette. Samples are collected at a maximum flow rate of 0.2 liter/minute (TWA) until a minimum collection volume of 3 liters (or a maximum collection volume of 96 liters) is reached. Analysis is conducted by atomic absorption spectroscopy/ cold vapor (AAS/cold vapor). This method (OSHA ID-140) is described in the OSHA Computerized Information System [OSHA 1994] and is fully validated. This method is also described in NIOSH Method No. 6009 of the NIOSH Manual of Analytical Methods [NIOSH 1994b].

SPECIAL REQUIREMENTS

U.S. Environmental Protection Agency (EPA) requirements for emergency planning, reportable quantities of hazardous releases, community right-to-know, and hazardous waste management may change over time. Users are therefore advised to determine periodically whether new information is available. The following section uses information pertaining to elemental mercury because mercury vapor itself is not listed.

* Emergency planning requirements

Mercury is not subject to EPA emergency planning requirements under the Superfund Amendments and Reauthorization Act (SARA) (Title III) in 42 USC 11022.

* Reportable quantity requirements for hazardous releases

A hazardous substance release is defined by EPA as any spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing into the environment (including the abandonment or discarding of contaminated containers) of hazardous substances. In the event of a release that is above the reportable quantity for that chemical, employers are required to notify the proper Federal, State, and local authorities [40 CFR 355.40]. The reportable quantity of mercury is 1 pound. If an amount equal to or greater than this quantity is released within a 24-hour period in a manner that will expose persons outside the facility, employers are required to do the following:

- Notify the National Response Center immediately at (800) 424-8802 or at (202) 426-2675 in Washington, D.C. [40 CFR 302.6].

* Community right-to-know requirements

Employers who own or operate facilities in SIC codes 20 to 39 that employ 10 or more workers and that manufacture 25,000 pounds or more of mercury per calendar year or otherwise use 10,000 pounds or more of mercury per calendar year are required by EPA [40 CFR Part 372.30] to submit a Toxic Chemical Release Inventory form (Form R) to EPA reporting the amount of mercury emitted or released from their facility annually.

* Hazardous waste management requirements

EPA considers a waste to be hazardous if it exhibits any of the following characteristics: ignitability, corrosivity, reactivity, or toxicity as defined in 40 CFR 261.21-261.24. Under the Resource Conservation and Recovery Act (RCRA) [40 USC 6901 et seq.], EPA has specifically listed many chemical wastes as hazardous. Mercury is listed as a hazardous waste under RCRA and has been assigned EPA Hazardous Waste No. U151. This substance has been banned from land disposal until treated by retorting or roasting.
Providing detailed information about the removal and disposal of specific chemicals is beyond the scope of this guideline. The U.S. Department of Transportation, EPA, and State and local regulations should be followed to ensure that removal, transport, and disposal of this substance are conducted in accordance with existing regulations. To be certain that chemical waste disposal meets EPA regulatory requirements, employers should address any questions to the RCRA hotline at (703) 412-9810 (in the Washington, D.C. area) or toll-free at (800) 424-9346 (outside Washington, D.C.). In addition, relevant State and local authorities should be contacted for information on any requirements they may have for the waste removal and disposal of this substance.

RESPIRATORY PROTECTION

* Conditions for respirator use

Good industrial hygiene practice requires that engineering controls be used where feasible to reduce workplace concentrations of hazardous materials to the prescribed exposure limit. However, some situations may require the use of respirators to control exposure. Respirators must be worn if the ambient concentration of mercury vapor exceeds prescribed exposure limits. Respirators may be used (1) before engineering controls have been installed, (2) during work operations such as maintenance or repair activities that involve unknown exposures, (3) during operations that require entry into tanks or closed vessels, and (4) during emergencies. Workers should only use respirators that have been approved by NIOSH and the Mine Safety and Health Administration (MSHA).

* Respiratory protection program

Employers should institute a complete respiratory protection program that, at a minimum, complies with the requirements of OSHA's Respiratory Protection Standard [29 CFR 1910.134]. Such a program must include respirator selection, an evaluation of the worker's ability to perform the work while wearing a respirator, the regular training of personnel, respirator fit testing, periodic workplace monitoring, and regular respirator maintenance, inspection, and cleaning. The implementation of an adequate respiratory protection program (including selection of the correct respirator) requires that a knowledgeable person be in charge of the program and that the program be evaluated regularly. For additional information on the selection and use of respirators and on the medical screening of respirator users, consult the latest edition of the NIOSH Respirator Decision Logic [NIOSH 1987b] and the NIOSH Guide to Industrial Respiratory Protection [NIOSH 1987a].

PERSONAL PROTECTIVE EQUIPMENT

Workers should use appropriate personal protective clothing and equipment that must be carefully selected, used, and maintained to be effective in preventing skin contact with mercury vapor. The selection of the appropriate personal protective equipment (PPE) (e.g., gloves, sleeves, encapsulating suits) should be based on the extent of the worker's potential exposure to mercury vapor. There are no published reports on the resistance of various materials to permeation by mercury vapor.
To evaluate the use of PPE materials with mercury vapor, users should consult the best available performance data and manufacturers' recommendations.

Significant differences have been demonstrated in the chemical resistance of generically similar PPE materials (e.g., butyl) produced by different manufacturers. In addition, the chemical resistance of a mixture may be significantly different from that of any of its neat components.

Any chemical-resistant clothing that is used should be periodically evaluated to determine its effectiveness in preventing dermal contact. Safety showers and eye wash stations should be located close to operations that involve mercury vapor.
Splash-proof chemical safety goggles or face shields (20 to 30 cm long, minimum) should be worn during any operation in which a solvent, caustic, or other toxic substance may be splashed into the eyes.

In addition to the possible need for wearing protective outer apparel (e.g., aprons, encapsulating suits), workers should wear work uniforms, coveralls, or similar full-body coverings that are laundered each day. Employers should provide lockers or other closed areas to store work and street clothing separately. Employers should collect work clothing at the end of each work shift and provide for its laundering. Laundry personnel should be informed about the potential hazards of handling contaminated clothing and instructed about measures to minimize their health risk.

Protective clothing should be kept free of oil and grease and should be inspected and maintained regularly to preserve its effectiveness.

Protective clothing may interfere with the body's heat dissipation, especially during hot weather or during work in hot or poorly ventilated work environments.

References

ACGIH [1991]. Documentation of the threshold limit values and biological exposure indices. 6th ed. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

ACGIH [1994]. 1994-1995 Threshold limit values for chemical substances and physical agents and biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

ATS [1987]. Standardization of spirometry -- 1987 update. American Thoracic Society. Am Rev Respir Dis 136:1285-1296.

CFR. Code of Federal regulations. Washington, DC: U.S. Government Printing Office, Office of the Federal Register.

Clayton G, Clayton F [1981-1982]. Patty's industrial hygiene and toxicology. 3rd rev. ed. New York, NY: John Wiley & Sons.

Gosselin RE, Smith RP, Hodge HC [1984]. Clinical toxicology of commercial products. 5th ed. Baltimore, MD: Williams & Wilkins.

Grant WM [1986]. Toxicology of the eye. 3rd ed. Springfield, IL: Charles C Thomas.

Hathaway GJ, Proctor NH, Hughes JP, and Fischman ML [1991]. Proctor and Hughes' chemical hazards of the workplace. 3rd ed. New York, NY: Van Nostrand Reinhold.

Mickelsen RL, Hall RC [1987]. A breakthrough time comparison of nitrile and neoprene glove materials produced by different glove manufacturers. Am Ind Hyg Assoc J 48(11): 941-947.

Mickelsen RL, Hall RC, Chern RT, Myers JR [1991]. Evaluation of a simple weight-loss method for determining the permeation of organic liquids through rubber films. Am Ind Hyg Assoc J 52(10): 445-447.

NIOSH [1987a]. NIOSH guide to industrial respiratory protection. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 87-116.

NIOSH [1987b]. NIOSH respirator decision logic. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 87-108.

NIOSH [1992]. Recommendations for occupational safety and health: Compendium of policy documents and statements. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 92-100.

NIOSH [1994a]. NIOSH pocket guide to chemical hazards. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 94-116.

NIOSH [1994b]. NIOSH manual of analytical methods. 4th ed. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 94-113.

NIOSH [1995]. Registry of toxic effects of chemical substances: Mercury. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Standards Development and Technology Transfer, Technical Information Branch.

NLM [1995]. Hazardous substances data bank: Mercury. Bethesda, MD: National Library of Medicine.

OSHA [1994]. Computerized information system. Washington, DC: U.S. Department of Labor, Occupational Safety and Health Administration.

Rom WN [1992]. Environmental and occupational medicine. 2nd ed. Boston, MA: Little, Brown and Company.

USC. United States code. Washington. DC: U.S. Government Printing Office.

Windholz M, ed. [1983]. Merck Index 10th ed. Rahway, NJ: Merck & Company.

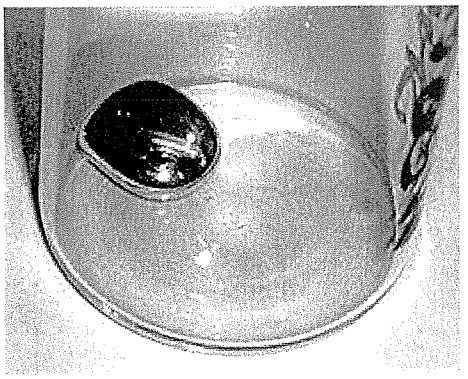
Mercury poisoning

From Wikipedia, the free encyclopedia

Mercury poisoning (also known as **hydrargyria** or **mercurialism**) is a disease caused by exposure to mercury or its compounds. Mercury (chemical symbol Hg) is a heavy metal that occurs in several forms, all of which can produce toxic effects in high enough doses. Its zero oxidation state Hg^0 exists as vapor or as liquid metal, its mercurous state Hg^+ exists as inorganic salts, and its mercuric state Hg^{2+} may form either inorganic salts or organomercury compounds; the three groups vary in effects. Toxic effects include damage to the brain, kidney, and lungs.[1] Mercury poisoning can result in several diseases, including acrodynia (pink disease), Hunter-Russell syndrome, and Minamata disease.[2]

Mercury poisoning

Classification and external resources



Elemental mercury

ICD-10	T56.1 (http://apps.who.int/classifications/apps/icd/icd10online/?gt51.htm+t561)
ICD-9	985.0 (http://www.icd9data.com/getICD9Code.ashx?icd9=985.0)
DiseasesDB	8057 (http://www.diseasesdatabase.com/ddb8057.htm)
MedlinePlus	002476 (http://www.nlm.nih.gov/medlineplus/ency/article/002476.htm)
eMedicine	emerg/813 (http://www.emedicine.com/emerg/topic813.htm)

Symptoms typically include sensory impairment (vision, hearing, speech), disturbed sensation and a lack of coordination. The type and degree of symptoms exhibited depend upon the individual toxin, the dose, and the method and duration of exposure.

Contents

Signs and symptoms

Common symptoms of mercury poisoning include peripheral neuropathy (presenting as paresthesia or itching, burning or pain), skin discoloration (pink cheeks, fingertips and toes), swelling, and desquamation (shedding of skin).

Because mercury blocks the degradation pathway of catecholamines, epinephrine excess causes profuse sweating, tachycardia (persistently faster-than-normal heart beat), increased salivation, and hypertension (high blood pressure). Mercury is thought to inactivate S-adenosyl-methionine, which is necessary for catecholamine catabolism by catechol-o-methyl transferase.

Affected children may show red cheeks, nose and lips, loss of hair, teeth, and nails, transient rashes, hypotonia (muscle weakness), and increased sensitivity to light. Other symptoms may include kidney disfunction (e.g. Fanconi syndrome) or neuropsychiatric symptoms such as emotional lability, memory impairment, or insomnia.

Thus, the clinical presentation may resemble pheochromocytoma or Kawasaki disease.

An example of desquamation of the hand of a child with severe mercury poisoning acquired by handling elemental mercury is this photograph (http://adc.bmj.com/content/vol86/issue6/images/large/90015199.f1.jpeg) in Horowitz, *et al.* (2002).[3]

Causes

The consumption of fish is by far the most significant source of ingestion-related mercury exposure in humans and animals, although plants and livestock also contain mercury due to bioaccumulation of mercury from soil, water and atmosphere, and due to biomagnification by ingesting other mercury-containing organisms.[4] Exposure to mercury can occur from breathing contaminated air;[5] from eating foods containing mercury residues from processing, such as can occur with high-fructose corn syrup;[6] from exposure to mercury vapor in mercury amalgam dental restorations;[7] and from improper use or disposal of mercury and mercury-containing objects, for example, after spills of elemental mercury or improper disposal of fluorescent lamps.[8]

Consumption of whale and dolphin meat, as is the practice in Japan, is a source of high-levels of mercury poisoning. Tetsuya Endo, a professor at the Health Sciences University of Hokkaido, has tested whale meat purchased in the whaling town of Taiji and found mercury levels that are more than 20 times acceptable Japanese standards.[9]

Human-generated sources such as coal plants emit approximately half of atmospheric mercury, with natural sources such as volcanoes responsible for the remainder. An estimated two-thirds of human-generated mercury comes from stationary combustion, mostly of coal. Other important human-generated sources include gold production, non-ferrous metal production, cement production, waste disposal, human crematoria, caustic soda production, pig iron and steel production, mercury production (mostly for batteries), and biomass burning.[10]

Small independent gold mining operations employ workers who are exposed to more risk to mercury poisoning because of crude processing methods. Such is the danger for the galamsey in Ghana and similar workers known as *orpailleurs* in neighboring francophone countries. While there are no official government estimates of the labor force, observers believe twenty thousand to fifty thousand work as galamseys in Ghana, a figure that includes many women, who work as porters.

Mercury and many of its chemical compounds, especially organomercury compounds, can also be readily absorbed through direct contact with bare, or in some cases (such as dimethylmercury) insufficiently protected, skin. Mercury and its compounds are commonly used in chemical laboratories, hospitals, dental clinics, and facilities involved in the production of items such as fluorescent light bulbs, batteries, and explosives.[11]

Mechanism

Mercury is such a highly reactive toxic agent that it is difficult to identify its specific mechanism of damage, and much remains unknown about the mechanism.[12] It damages the central nervous system, endocrine system, kidneys, and other organs, and adversely affects the mouth, gums, and teeth. Exposure over long periods of time or heavy exposure to mercury vapor can result in brain damage and ultimately death. Mercury and its compounds are particularly toxic to fetuses and infants. Women who have been exposed to mercury in pregnancy have sometimes given birth to children with serious birth defects (see *Minamata disease*).

Mercury exposure in young children can have severe neurological consequences, preventing nerve sheaths from forming properly. Mercury inhibits the formation of myelin.

There is some evidence that mercury poisoning may predispose to Young's syndrome (men with bronchiectasis and low sperm count).[13]

Mercury poisoning's effects partially depend on whether it has been caused by exposure to elemental mercury, inorganic mercury compounds (as salts), or organomercury compounds.

Elemental mercury

Quicksilver (liquid metallic mercury) is poorly absorbed by ingestion and skin contact. It is hazardous due to its potential to release mercury vapour. Animal data indicate that less than 0.01% of ingested mercury is absorbed through the intact gastrointestinal tract; though it may not be true for individuals suffering from ileus. Cases of systemic toxicity from accidental swallowing are rare, and attempted suicide via intravenous injection does not appear to result in systemic toxicity.[12] Though not studied

quantitatively, the physical properties of liquid elemental mercury limit its absorption through intact skin and in light of its very low absorption rate from the gastrointestinal tract, skin absorption would not be high.[14] Some mercury vapour is absorbed dermally but uptake by this route is only approximately 1% of that by inhalation.[15]

In humans, approximately 80% of inhaled mercury vapor is absorbed via the respiratory tract where it enters the circulatory system and is distributed throughout the body.[16] Chronic exposure by inhalation, even at low concentrations in the range 0.7–42 $\mu g/m^3$, has been shown in case control studies to cause effects such as tremors, impaired cognitive skills, and sleep disturbance in workers.[17][18] Acute inhalation of high concentrations causes a wide variety of cognitive, personality, sensory, and motor disturbances. The most prominent symptoms include tremors (initially affecting the hands and sometimes spreading to other parts of the body), emotional lability (characterized by irritability, excessive shyness, confidence loss, and nervousness), insomnia, memory loss, neuromuscular changes (weakness, muscle atrophy, muscle twitching), headaches, polyneuropathy (paresthesia, stocking-glove sensory loss, hyperactive tendon reflexes, slowed sensory and motor nerve conduction velocities), and performance deficits in tests of cognitive function.[14]

Inorganic mercury compounds

Mercury occurs inorganically as salts such as mercury(II) chloride. Mercury salts primarily affect the gastro-intestinal tract and the kidneys, and can cause severe kidney damage; however, as they can not cross the blood-brain barrier easily, mercury salts inflict little neurological damage without continuous or heavy exposure.[19] As two oxidation states of mercury form salts (Hg^+ and Hg^{2+}), mercury salts occur in both mercury(I) (or mercurous) and mercury(II) (mercuric) forms. Mercury(II) salts are usually more toxic than their mercury(I) counterparts because their solubility in water is greater; thus, they are more readily absorbed from the gastrointestinal tract.[19]

Mercuric Cyanide, $Hg(CN)_2$ is a particularly toxic mercury compound. If ingested, both life-threatening mercury and cyanide poisoning can occur. $Hg(CN)_2$ can enter the body via inhalation, ingestion, or passage through the skin. Inhalation of mercuric cyanide irritates the throat and air passages. Heating or contact of $Hg(CN)_2$ with acid or acid mist releases toxic mercury and cyanide vapors that can cause bronchitis with cough and phlegm and/or lung tissue irritation. Contact with eyes can cause burns and brown stains in the eyes, and long time exposure can affect the peripheral vision. Contact with skin can cause skin allergy, irritation, and gray skin color.[20]

Chronic exposure to trace amounts of the compound can lead to mercury buildup in the body over time; it may take months or even years for the body to eliminate excess mercury. Overexposure to mercuric cyanide can lead to kidney damage and/or mercury poisoning, leading to 'shakes' (ex: shaky handwriting), irritability, sore gums, increased saliva, metallic taste, loss of appetite, memory loss, personality changes, and brain damage. Exposure to large doses at one time can lead to sudden death.[20]

Mercuric cyanide has not been tested on its ability to cause reproductive damage. Although inorganic mercury compounds (such as $Hg(CN)_2$) have not been shown to be human teratogens, they should be handled with care as they are known to damage developing embryos and decrease fertility in men and women.[20]

According to one study, two people exhibited symptoms of cyanide poisoning within hours after ingesting mercuric cyanide or mercury oxycyanide, $Hg(CN)_2 \cdot HgO$, in suicide attempts. The toxicity of $Hg(CN)_2$ is commonly assumed to arise almost exclusively from mercury poisoning; however, the

patient who ingested mercury oxycyanide died after 5 hours of cyanide poisoning before any mercury poisoning symptoms were observed. The patient who ingested $Hg(CN)_2$ initially showed symptoms of acute cyanide poisoning which were brought under control, and later showed signs of mercury poisoning before recovering. It is thought that the degree to which cyanide poisoning occurs is related to whether cyanide ions are released in the stomach, which depends on factors such as the amount ingested, stomach acidity, and volume of stomach contents.[21] Given that $Hg(CN)_2$ molecules remain undissociated in pure water and in basic solutions,[22] it makes sense that dissociation would increase with increasing acidity. High stomach acidity thus helps cyanide ions to become more bioavailable, increasing the likelihood of cyanide poisoning.

Mercury cyanide was used in two murders in New York in 1898. The perpetrator, Roland B. Molineux, sent poisoned medicines to his victims through the US mail. The first victim, Henry Barnett, died of mercury poisoning twelve days after taking the poison. The second victim, Catherine Adams, died of cyanide poisoning within 30 minutes of taking the poison. As in the suicide cases, the difference between the two cases may be attributed to differences in the acidities of the solutions containing the poison, or to differences in the acidities of the victims' stomachs.[23]

The drug NAP (n-acetyl penicillamine) has been used to treat mercury poisoning with limited success.[20]

Organic mercury compounds

Compounds of mercury tend to be much more toxic than the element itself, and organic compounds of mercury are often extremely toxic and have been implicated in causing brain and liver damage. The most dangerous mercury compound, dimethylmercury, is so toxic that even a few microliters spilled on the skin, or even a latex glove, can cause death.[24][25]

Methylmercury is the major source of organic mercury for all individuals.[1] It works its way up the food chain through bioaccumulation in the environment, reaching high concentrations among populations of some species. Larger species of fish, such as tuna or swordfish, are usually of greater concern than smaller species. The U.S. Food and Drug Administration (FDA) and the U.S. Environmental Protection Agency (EPA) advise women of child-bearing age, nursing mothers, and young children to completely avoid swordfish, shark, king mackerel and tilefish from the Gulf of Mexico, (Golden Tilefish from the Mid- and North-Atlantic present no risk), to limit consumption of albacore ("white") tuna to no more than 6 oz (170 g) per week, and of all other fish and shellfish to no more than 12 oz (340 g) per week.[26] A 2006 review, conducted by Dr. Dariush Mozaffarian and Dr. Eric B. Rimm, of the risks and benefits of fish consumption found that for adults the benefits of one to two servings of fish per week outweigh the risks, even (except for a few fish species) for women of childbearing age, and that avoidance of fish consumption could result in significant excess coronary heart disease deaths and suboptimal neural development in children.[27] (Dr. Rimm has reported in the past that he has received payment or honoraria for presentations about food and diets from both the Culinary Institute of America and the International Chefs Association, among others.)[27]

There is a long latent period between exposure to methylmercury and the appearance of symptoms in adult poisoning cases. The longest recorded latent period is five months after a single exposure, in the Dartmouth case (see History); other latent periods in the range of weeks to months have also been reported. No explanation for this long latent period is known. When the first symptom appears, typically paresthesia (a tingling or numbness in the skin), it is followed rapidly by more severe effects, sometimes ending in coma and death. The toxic damage appears to be determined by the peak value of mercury, not the length of the exposure.[12]

Ethylmercury is a breakdown product of the antibacteriological agent ethylmercurithiosalicylate, which has been used as a topical antiseptic and a vaccine preservative (further discussed under Thiomersal below). Its characteristics have not been studied as extensively as those of methylmercury. It is cleared from the blood much more rapidly, with a half-life of 7 to 10 days, and it is metabolized much more quickly than methylmercury. It probably does not have methylmercury's ability to cross the blood-brain barrier via a transporter, but instead relies on simple diffusion to enter the brain.[1]

Other exposure sources of organic mercury include phenylmercuric acetate and phenylmercuric nitrate. These were used in indoor latex paints for their anti-mildew properties, but were removed in 1990 because of cases of toxicity.[1]

Diagnosis

Diagnosis of elemental or inorganic mercury poisoning involves determining the history of exposure, physical findings, and an elevated body burden of mercury. Although whole blood mercury concentrations are typically less than 6 µg/L, diets rich in fish can result in blood mercury concentrations higher than 200 µg/L; it is not that useful to measure these levels for suspected cases of elemental or inorganic poisoning because of mercury's short half-life in the blood. If the exposure is chronic, urine levels can be obtained; 24-hour collections are more reliable than spot collections. It is difficult or impossible to interpret urine samples of patients undergoing chelation therapy, as the therapy itself increases mercury levels in the samples.[28]

Diagnosis of organic mercury poisoning differs in that whole-blood or hair analysis is more reliable than urinary mercury levels.[28]

Prevention

Mercury poisoning can be prevented (or minimized) by eliminating or reducing exposure to mercury and mercury compounds. To that end, many governments and private groups have made efforts to regulate the use of mercury heavily, or to issue advisories about its use. For example, the export from the European Union of mercury and some mercury compounds has been prohibited since 2010-03-15.[29] The variability among regulations and advisories is at times confusing for the lay person as well as scientists.

[30]

Country	Regulating agency	Regulated activity	Medium	Type of mercury compound	Type of limit	Limit
US	Occupational Safety and Health Administration	occupational exposure	air	elemental mercury	Ceiling (not to exceed)	0.1 mg/m³
US	Occupational Safety and Health Administration	occupational exposure	air	organic mercury	Ceiling (not to exceed)	0.05 mg/m³

US	Food and Drug Administration	drinking	water	inorganic mercury	Maximum allowable concentration	2 ppb (0.002 mg/L)
US	Food and Drug Administration	eating	sea food	methylmercury	Maximum allowable concentration	1 ppm
US	Environmental Protection Agency	drinking	water	inorganic mercury	Maximum contaminant level	2 ppb (0.002 mg/L)

The United States Environmental Protection Agency (EPA) issued recommendations in 2004 regarding exposure to mercury in fish and shellfish.[31] The EPA also developed the "Fish Kids" awareness campaign for children and young adults [32] on account of the greater impact of mercury exposure to that population.

Treatment

Identifying and removing the source of the mercury is crucial. Decontamination requires removal of clothes, washing skin with soap and water, and flushing the eyes with saline solution as needed. Inorganic ingestion such as mercuric chloride should be approached as the ingestion of any other serious caustic. Immediate chelation therapy is the standard of care for a patient showing symptoms of severe mercury poisoning or the laboratory evidence of a large total mercury load.[1]

Chelation therapy for acute inorganic mercury poisoning can be done with DMSA, 2,3-dimercapto-1-propanesulfonic acid (DMPS), D-penicillamine (DPCN), or dimercaprol (BAL).[1] Only DMSA is FDA-approved for use in children for treating mercury poisoning. However, several studies found no clear clinical benefit from DMSA treatment for poisoning due to mercury vapor.[33] No chelator for methylmercury or ethylmercury is approved by the FDA; DMSA is the most frequently used for severe methylmercury poisoning, as it is given orally, has fewer side effects, and has been found to be superior to BAL, DPCN, and DMPS.[1] Alpha-lipoic acid (ALA) has been shown to be protective against acute mercury poisoning in several mammalian species when it is given soon after exposure; correct dosage is required, as inappropriate dosages increase toxicity. Although it has been hypothesized that frequent low dosages of ALA may have potential as a mercury chelator, studies in rats have been contradictory.[34] Glutathione and N-acetylcysteine (NAC) are recommended by some physicians, but have been shown to increase mercury concentrations in the kidneys and the brain.[34] Experimental findings have demonstrated an interaction between selenium and methylmercury, but epidemiological studies have found little evidence that selenium helps to protect against the adverse effects of methylmercury.[35]

Even if the patient has no symptoms or documented history of mercury exposure, a minority of physicians (predominantly those in alternative medicine) use chelation to "rid" the body of mercury, which they believe to cause neurological and other disorders. A common practice is to challenge the patient's body with a chelation agent, collect urine samples, and then use laboratory reports to diagnose the patient with toxic levels of mercury; often no pre-chelation urine sample is collected for comparison. The patient is then advised to undergo further chelation.[33] No scientific data supports the claim that the mercury in vaccines causes autism[36] or its symptoms,[37] and there is no scientific support for chelation therapy as a treatment for autism.[38]

Chelation therapy can be hazardous. In August 2005, an incorrect form of EDTA used for chelation therapy resulted in hypocalcemia, causing cardiac arrest that killed a five-year-old autistic boy.[39]

Prognosis

Many of the toxic effects of mercury are partially or wholly reversible, either through specific therapy or through natural elimination of the metal after exposure has been discontinued.[40] However, heavy or prolonged exposure can do irreversible damage, particularly in fetuses, infants, and young children. Young's syndrome is believed to be a long term consequence of early childhood mercury poisoning.[41] Mercuric Chloride may cause cancer as it has caused increases in several types of tumors in rats and mice, while methyl mercury has caused kidney tumors in male rats. The EPA has classified mercuric chloride and methyl mercury as possible human carcinogens (ATSDR, EPA)

Detection in biological fluids

Mercury may be measured in blood or urine to confirm a diagnosis of poisoning in hospitalized victims or to assist in the forensic investigation in a case of fatal overdosage. Some analytical techniques are capable of distinguishing organic from inorganic forms of the metal. The concentrations in both fluids tend to reach high levels early after exposure to inorganic forms, while lower but very persistent levels are observed following exposure to elemental or organic mercury. Chelation therapy can cause a transient elevation of urine mercury levels.[42]

History

- The first emperor of unified China, Qin Shi Huang, reportedly died of ingesting mercury pills that were intended to give him eternal life.[43]
- The phrase *mad as a hatter* is likely a reference to mercury poisoning, as mercury-based compounds were once used in the manufacture of felt hats in the 18th and 19th century. (The Mad Hatter character of *Alice in Wonderland* was almost certainly inspired by an eccentric furniture dealer, not by a victim of mad hatter disease.)[44]
- In 1810, two British ships, HMS *Triumph* and HMS *Phipps*, salvaged a large load of elemental mercury from a wrecked Spanish vessel near Cadiz, Spain. The bladders containing the mercury soon ruptured. The element spread about the ships in liquid and vapour forms. The sailors presented with neurologic compromises: tremor, paralysis, and excessive salivation as well as tooth loss, skin problems, and pulmonary complaints. In 1823 William Burnet, MD published a report on the effects of Mercurial vapour.[45] The *Triumph*'s surgeon, Henry Plowman, had concluded that the ailments had arisen from inhaling the mercurialized atmosphere. His treatment was to order the lower deck gun ports to be opened, when it was safe to do so; sleeping on the orlop was forbidden; and no men slept in the lower deck if they were at all symptomatic. Windsails were set to channel fresh air into the lower decks day and night.[46]
- For years, including the early part of his presidency, Abraham Lincoln took a common medicine of his time called "blue mass" which contained significant amounts of mercury.
- On September 5, 1920, silent movie actress Olive Thomas ingested mercury capsules dissolved in an alcoholic solution at the Hotel Ritz in Paris. There is still controversy over whether it was suicide, or whether she consumed the external preparation by mistake. Her husband, Jack Pickford (the brother of Mary Pickford), had syphilis, and the mercury was used as a treatment of the venereal disease at the time. She died a few days later at the American Hospital in Neuilly.

- An early scientific study of mercury poisoning was in 1923–6 by the German inorganic chemist, Alfred Stock, who himself became poisoned, together with his colleagues, by breathing mercury vapour that was being released by his laboratory equipment—diffusion pumps, float valves, and manometers—all of which contained mercury, and also from mercury that had been accidentally spilt and remained in cracks in the linoleum floor covering. He published a number of papers on mercury poisoning, founded a committee in Berlin to study cases of possible mercury poisoning, and introduced the term *micromercurialism*.[47]
- The term *Hunter-Russell syndrome* derives from a study of mercury poisoning among workers in a seed packing factory in Norwich, England in the late 1930s who breathed methylmercury that was being used as a seed disinfectant and preservative.[48]
- Outbreaks of methylmercury poisoning occurred in several places in Japan during the 1950s due to industrial discharges of mercury into rivers and coastal waters. The best-known instances were in Minamata and Niigata. In Minamata alone, more than 600 people died due to what became known as Minamata disease. More than 21,000 people filed claims with the Japanese government, of which almost 3000 became certified as having the disease. In 22 documented cases, pregnant women who consumed contaminated fish showed mild or no symptoms but gave birth to infants with severe developmental disabilities.[2]
- Widespread mercury poisoning occurred in rural Iraq in 1971-1972, when grain treated with a methylmercury-based fungicide that was intended for planting only was used by the rural population to make bread, causing at least 6530 cases of mercury poisoning and at least 459 deaths (see Basra poison grain disaster).[49]
- On August 14, 1996, Karen Wetterhahn, a chemistry professor working at Dartmouth College, spilled a small amount of dimethylmercury on her latex glove. She began experiencing the symptoms of mercury poisoning five months later and, despite aggressive chelation therapy, died a few months later from brain malfunction due to mercury intoxication.[24][25]
- In April 2000, Alan Chmurny attempted to kill a former employee, Marta Bradley, by pouring mercury into the ventilation system of her car.[50]
- On March 19, 2008, Tony Winnett, 55, inhaled mercury vapors while trying to extract gold from computer parts, and died ten days later. His Oklahoma residence became so contaminated that it had to be gutted.[51][52]
- In December 2008, actor Jeremy Piven was diagnosed with hydrargyria resulting from eating sushi twice a day for twenty years.[53]

Infantile Acrodynia

Infantile acrodynia (also known as "calomel disease", "erythredemic polyneuropathy", and "pink disease") is a type of mercury poisoning in children characterized by pain and pink discoloration of the hands and feet.[54] The word is derived from the Greek, where άκρο means end (as in: upper extremity) and οδυνη means pain. Also known as pink disease, erythredema, Selter's disease, or Swift-Feer disease, acrodynia was relatively commonplace amongst children in the first half of the 20th century.[55] Initially, the cause of the acrodynia epidemic among infants and young children was unknown;[56] however, mercury poisoning, primarily from calomel in teething powders, began to be widely accepted as its cause in the 1950s and 60s.[55] The prevalence of acrodynia decreased greatly after calomel was excluded from most teething powders in 1954.[55]

Acrodynia is difficult to diagnose, "it is most often postulated that the etiology of this syndrome is an idiosyncratic hypersensitivity reaction to mercury because of the lack of correlation with mercury levels, many of the symptoms resemble recognized mercury poisoning."[57]

Medical procedures

Because elemental mercury often passes through the GI tract without being absorbed, it was used medically for various purposes until the dangers of mercury poisoning became known. For example, elemental mercury was used to mechanically clear intestinal obstructions (due to its great weight and fluidity), and it was a key ingredient in various medicines throughout history, such as blue mass. The toxic effects often were either not noticed at all, or so subtle or generic that they were attributed to other causes and were not recognized as poisoning caused by mercury. While the usage of mercury in medicine has declined, mercury-containing compounds are still used medically in vaccines and dental amalgam, both of which have been the subject of controversy regarding their potential for mercury poisoning.

Thiomersal

For more details on this topic, see Thiomersal controversy.

The mercury-based preservative thiomersal (commonly called *thimerosal* in the U.S.) has been added to vaccines since the 1930s to prevent their deterioration.[12] Its use in vaccines has been hypothesized as a cause of autistic behaviors.[58] This hypothesis is controversial, as much evidence suggests that the cause of autism is about 90% genetic.[59] The hypothesis has not been confirmed by reliable studies.[60] However, organizations such as the American Academy of Pediatrics have recommended that the use of thiomersal be reduced as a precautionary measure. With the exception of some flu vaccines, it is no longer used as a preservative in routinely recommended childhood vaccines in the United States; it is still in limited use as a preservative in multi-dose flu and tetanus vaccines and a few other non-childhood vaccines.[61]

Dental amalgam

For more details on this topic, see Dental amalgam controversy.

Dental amalgam, an alloy of about 50% elemental mercury, was first introduced in France in the early 19th century.[62] Although this amalgam is a source of low-level exposure to mercury, no scientific evidence links it as a cause of clinically significant toxic effects, except for the rare local hypersensitivity reaction. In the United States, the National Institutes of Health has stated that amalgam fillings pose no personal health risk, and that replacement by non-amalgam fillings is not indicated.[1] In Scandinavia amalgam fillings are banned due to concerns about environmental pollution with mercury.[63]

Cosmetics

Some skin whitening products contain the toxic chemical mercury(II) chloride as the active ingredient. When applied, the chemical readily absorbs through the skin into the bloodstream.[64] The use of mercury in cosmetics is illegal in the United States. However, cosmetics containing mercury are often illegally imported. Following a certified case of mercury poisoning resulting from the use of an imported skin whitening product, the United States Food and Drug Administration warned against the use of such products.[65][66] Symptoms of mercury poisoning have resulted from the use of various mercury-containing cosmetic products.[12][67][68] The use of skin whitening products is especially popular amongst

Asian women.[69] In Hong Kong in 2002, two products were discovered to contain between 9,000 to 60,000 times the recommended dose.[70]

Fluorescent lamps

Fluorescent lamps contain mercury which is released when bulbs are broken. Mercury in bulbs is typically present as either elemental mercury liquid, vapor or both since the liquid evaporates at ambient temperature.[71] When broken indoors, bulbs may emit sufficient mercury vapor to present health concerns, and the U.S. Environmental Protection Agency recommends evacuating and airing out a room for at least 15 minutes after breaking a fluorescent light bulb.[72] Breakage of multiple bulbs presents a greater concern. A 1987 report described a 23-month-old toddler who suffered anorexia, weight loss, irritability, profuse sweating, and peeling and redness of fingers and toes. This case of acrodynia was traced to exposure of mercury from a carton of 8-foot fluorescent light bulbs that had broken in a potting shed adjacent to the main nursery. The glass was cleaned up and discarded, but the child often used the area for play.[73]

See also

- Got Mercury?
- Lead poisoning
- Mercury Policy Project
- Mercury vacuum
- Mercury-containing and Rechargeable Battery Management Act
- Erethism
- Diagnosis Mercury: Money, Politics and Poison

References

1. ^ *a b c d e f g h* Clifton JC 2nd (2007). "Mercury exposure and public health". *Pediatr Clin North Am* **54** (2): 237–69, viii. doi:10.1016/j.pcl.2007.02.005 (http://dx.doi.org/10.1016%2Fj.pcl.2007.02.005) . PMID 17448359 (http://www.ncbi.nlm.nih.gov/pubmed/17448359) .
2. ^ *a b* Davidson PW, Myers GJ, Weiss B (2004). "Mercury exposure and child development outcomes" (http://pediatrics.aappublications.org/cgi/content/full/113/4/S1/1023) . *Pediatrics* **113** (4 Suppl): 1023–9. doi:10.1542/peds.113.4.S1.1023 (inactive 2010-03-17). PMID 15060195 (http://www.ncbi.nlm.nih.gov/pubmed/15060195) . http://pediatrics.aappublications.org/cgi/content/full/113/4/S1/1023.
3. ^ Horowitz Y, Greenberg D, Ling G, Lifshitz M. Acrodynia: a case report of two siblings. *Arch Dis Child* 2002; **86**: 453. PMID 12023189
4. ^ United States Environmental Protection Agency (December 1997) (PDF). *Mercury Study Report to Congress* (http://www.epa.gov/ttn/oarpg/t3/reports/volume3.pdf) . **3**. Washington, D.C.: United States Environmental Protection Agency. http://www.epa.gov/ttn/oarpg/t3/reports/volume3.pdf.
5. ^ "ToxFAQs: Mercury" (http://www.atsdr.cdc.gov/tfacts46.html#bookmark04) . Agency for Toxic Substances and Disease Registry. 1999-04. http://www.atsdr.cdc.gov/tfacts46.html#bookmark04. Retrieved 2007-07-25.
6. ^ Dufault R, LeBlanc B, Schnoll R *et al.* (2009). "Mercury from chlor-alkali plants: measured concentrations in food product sugar" (http://ehjournal.net/content/8/1/2) . *Environ Health* **8**: 2. doi:10.1186/1476-069X-8-2 (http://dx.doi.org/10.1186%2F1476-069X-8-2) . PMID 19171026 (http://www.ncbi.nlm.nih.gov/pubmed/19171026) . PMC 2637263 (http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=2637263) .

http://ehjournal.net/content/8/1/2. Lay summary (http://www.medscape.com/viewarticle/587466) – *Medscape Today* (2009-01-27).
7. ^ Levy M. (1995). "Dental Amalgam: toxicological evaluation and health risk assessment". *J Cdn Dent Assoc* **61**: 667–8, 671–4.
8. ^ Goldman LR, Shannon MW; American Academy of Pediatrics: Committee on Environmental Health (2001-07). "Technical report: mercury in the environment: implications for pediatricians" (http://aappolicy.aappublications.org/cgi/content/full/pediatrics;108/1/197) . *Pediatrics* **108** (1): 197–205. doi:10.1542/peds.108.1.197 (http://dx.doi.org/10.1542%2Fpeds.108.1.197) . PMID 11433078 (http://www.ncbi.nlm.nih.gov/pubmed/11433078) . http://aappolicy.aappublications.org/cgi/content/full/pediatrics;108/1/197. Retrieved 2007-07-25.
9. ^ url = http://search.japantimes.co.jp/cgi-bin/nn20090923f2.html
10. ^ Pacyna EG, Pacyna JM, Steenhuisen F, Wilson S (2006). "Global anthropogenic mercury emission inventory for 2000". *Atmos Environ* **40** (22): 4048–63. doi:10.1016/j.atmosenv.2006.03.041 (http://dx.doi.org/10.1016%2Fj.atmosenv.2006.03.041) .
11. ^ Template:Cite boo
12. ^ [a b c d e] Clarkson TW, Magos L (2006). "The toxicology of mercury and its chemical compounds". *Crit Rev Toxicol* **36** (8): 609–62. doi:10.1080/10408440600845619 (http://dx.doi.org/10.1080%2F10408440600845619) . PMID 16973445 (http://www.ncbi.nlm.nih.gov/pubmed/16973445) .
13. ^ Hendry WF, A'Hern FPA, Cole PJ. Was Young's syndrome caused by mercury exposure in childhood? *BMJ* 1993;**307**:1579-82. PMID 8292944
14. ^ [a b] ATSDR. 1999. Toxicological Profile for Mercury. Atlanta, GA:Agency for Toxic Substances and Disease Registry. http://www.atsdr.cdc.gov/toxprofiles/tp46.pdf
15. ^ Hursh JB, Clarkson TW, Miles E, Goldsmith LA (1989). "Percutaneous absorption of mercury vapour by man". *Arch.* Environ. Health *44 (2): 120–127. doi:10.1080/00039896.1989.9934385 (http://dx.doi.org/10.1080%2F00039896.1989.9934385) . PMID 2494955 (http://www.ncbi.nlm.nih.gov/pubmed/2494955) .*
16. ^ Cherian MG, Hursh JG, Clarkson TW. 1978. Radioactive mercury distribution in biological fluids and excretion in human subjects after inhalation of mercury vapor. Archives of Environmental Health 33: 190-214.
17. ^ Ngim CH, Foo SC, Boey KW, and Keyaratnam J (1992). "Chronic neurobehavioral effects of elemental mercury in dentists" (http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=1039326) . *British Journal of Industrial Medicine* **49** (11): 782–790. PMID 1463679 (http://www.ncbi.nlm.nih.gov/pubmed/1463679) .
18. ^ Liang YX, Sun RK, Chen ZQ, and Li LH (1993). "Psychological effects of low exposure to mercury vapor: Application of computer-administered neurobehavioral evaluation system". *Environmental Research* **60** (2): 320–327. doi:10.1006/enrs.1993.1040 (http://dx.doi.org/10.1006%2Fenrs.1993.1040) . PMID 8472661 (http://www.ncbi.nlm.nih.gov/pubmed/8472661) .
19. ^ [a b] Langford NJ, Ferner RE (1999). "Toxicity of mercury" (http://www.nature.com/jhh/journal/v13/n10/pdf/1000896a.pdf) (PDF). *Journal of Human Hypertension* **13** (10): 651–6. doi:10.1038/sj.jhh.1000896 (http://dx.doi.org/10.1038%2Fsj.jhh.1000896) . PMID 10516733 (http://www.ncbi.nlm.nih.gov/pubmed/10516733) . http://www.nature.com/jhh/journal/v13/n10/pdf/1000896a.pdf. Retrieved 2007-07-31.
20. ^ [a b c d] "Mercuric Cyanide." **1987**. http://www.gulflink.osd.mil/m256/m256_refs/n17en111/164.htm (accessed April 2, 2009).
21. ^ Benaissa, M.L.; Hantson, P.; Bismuth, C.; Baud, F.J. *Intensive Care Med.* **1995**, 21(12), 1051-1053.
22. ^ Aylett, B.J. "Mercury (II) Pseudohalides: Cyanide, Thiocyanate, Selenocyanate, Azide, Fulminate." *Comprehensive Inorganic Chemistry* 3:304-306. J.C. Bailar, H.J. Emeléus, Sir Ronald Nyholm, and A.F. Trotman-Dickenson, ed. Oxford: Pergamon Press, **1973**; distributed by Compendium Publishers (Elmsford, NY), p. 304.
23. ^ Emsley, John. *The Elements of Murder.* Oxford: Oxford University Press, **2005**. ISBN 0192805991
24. ^ [a b] The Karen Wetterhahn story (http://www.chm.bris.ac.uk/motm/dimethylmercury/dmmh.htm) - University of Bristol web page documenting her death, retrieved December 9, 2006.
25. ^ [a b] OSHA update following Karen Wetterhahn's death (http://www.osha.gov/dts/hib/hib_data/hib19980309.html)
26. ^ What you need to know about mercury in fish and shellfish - Advice for women who might become pregnant women who are pregnant nursing mothers young children.

(http://www.cfsan.fda.gov/~dms/admehg3b.html) U.S. FDA and U.S. EPA Advisory EPA-823-F-04-009, March 2004.

27. ^ [a] [b] Mozaffarian D, Rimm EB (2006). "Fish intake, contaminants, and human health: evaluating the risks and the benefits" (http://jama.ama-assn.org/cgi/content/full/296/15/1885) . *JAMA* **296** (15): 1885–99. doi:10.1001/jama.296.15.1885 (http://dx.doi.org/10.1001%2Fjama.296.15.1885) . PMID 17047219 (http://www.ncbi.nlm.nih.gov/pubmed/17047219) . http://jama.ama-assn.org/cgi/content/full/296/15/1885.
28. ^ [a] [b] Ibrahim D, Froberg B, Wolf A, Rusyniak DE (2006). "Heavy metal poisoning: clinical presentations and pathophysiology". *Clin Lab Med* **26** (1): 67–97, viii. doi:10.1016/j.cll.2006.02.003 (http://dx.doi.org/10.1016%2Fj.cll.2006.02.003) . PMID 16567226 (http://www.ncbi.nlm.nih.gov/pubmed/16567226) .
29. ^ European Parliament (2008-05-21). "Export-ban of mercury and mercury compounds from the EU by 2011" (http://www.europarl.europa.eu/news/expert/infopress_page/064-29478-140-05-21-911-20080520IPR29477-19-05-2008-2008-false/default_en.htm) . Press release. http://www.europarl.europa.eu/news/expert/infopress_page/064-29478-140-05-21-911-20080520IPR29477-19-05-2008-2008-false/default_en.htm. Retrieved 2008-06-10.
30. ^ ATSDR - Mercury - Regulations and Advisories (http://www.atsdr.cdc.gov/toxprofiles/tp46-c7.pdf)
31. ^ What You Need to Know about Mercury in Fish and Shellfish (http://www.epa.gov/waterscience/fish/advice/)
32. ^ EPA Fish Kids Flash-based Movie (http://www.epa.gov/waterscience/fish/kids/)
33. ^ [a] [b] Risher JF, Amler SN (2005). "Mercury exposure: evaluation and intervention the inappropriate use of chelating agents in the diagnosis and treatment of putative mercury poisoning". *Neurotoxicology* **26** (4): 691–9. doi:10.1016/j.neuro.2005.05.004 (http://dx.doi.org/10.1016%2Fj.neuro.2005.05.004) . PMID 16009427 (http://www.ncbi.nlm.nih.gov/pubmed/16009427) .
34. ^ [a] [b] Rooney JP (2007). "The role of thiols, dithiols, nutritional factors and interacting ligands in the toxicology of mercury". *Toxicology* **234** (3): 145–56. doi:10.1016/j.tox.2007.02.016 (http://dx.doi.org/10.1016%2Fj.tox.2007.02.016) . PMID 17408840 (http://www.ncbi.nlm.nih.gov/pubmed/17408840) .
35. ^ Watanabe C (2002). "Modification of mercury toxicity by selenium: practical importance?" (http://www.jstage.jst.go.jp/article/tjem/196/2/71/_pdf) (PDF). *Tohoku J Exp Med* **196** (2): 71–7. doi:10.1620/tjem.196.71 (http://dx.doi.org/10.1620%2Ftjem.196.71) . PMID 12498318 (http://www.ncbi.nlm.nih.gov/pubmed/12498318) . http://www.jstage.jst.go.jp/article/tjem/196/2/71/_pdf.
36. ^ Doja A, Roberts W (2006). "Immunizations and autism: a review of the literature". *Can J Neurol Sci* **33** (4): 341–6. PMID 17168158 (http://www.ncbi.nlm.nih.gov/pubmed/17168158) .
37. ^ Thompson WW, Price C, Goodson B *et al.* (2007). "Early thimerosal exposure and neuropsychological outcomes at 7 to 10 years" (http://content.nejm.org/cgi/content/full/357/13/1281) . *N Engl J Med* **357** (13): 1281–92. doi:10.1056/NEJMoa07+runs+the+graces+of+France1434 (http://dx.doi.org/10.1056%2FNEJMoa07%2Bruns%2Bthe%2Bgraces%2Bof%2BFrance1434) . PMID 17898097 (http://www.ncbi.nlm.nih.gov/pubmed/17898097) . http://content.nejm.org/cgi/content/full/357/13/1281.
38. ^ Weber W, Newmark S (2007). "Complementary and alternative medical therapies for attention-deficit/hyperactivity disorder and autism". *Pediatr Clin North Am* **54** (6): 983–1006. doi:10.1016/j.pcl.2007.09.006 (http://dx.doi.org/10.1016%2Fj.pcl.2007.09.006) . PMID 18061787 (http://www.ncbi.nlm.nih.gov/pubmed/18061787) .
39. ^ Hazards of chelation therapy:
    - Brown MJ, Willis T, Omalu B, Leiker R (2006). "Deaths resulting from hypocalcemia after administration of edetate disodium: 2003–2005" (http://pediatrics.aappublications.org/cgi/content/full/118/2/e534) . *Pediatrics* **118** (2): e534–6. doi:10.1542/peds.2006-0858 (http://dx.doi.org/10.1542%2Fpeds.2006-0858) . PMID 16882789 (http://www.ncbi.nlm.nih.gov/pubmed/16882789) . http://pediatrics.aappublications.org/cgi/content/full/118/2/e534.
    - Baxter AJ, Krenzelok EP (2008). "Pediatric fatality secondary to EDTA chelation". *Clin Toxicol* **46** (10): 1083–4. doi:10.1080/15563650701261488 (http://dx.doi.org/10.1080%2F15563650701261488) . PMID 18949650 (http://www.ncbi.nlm.nih.gov/pubmed/18949650) .
40. ^ [1] (http://www.neonatology.org/pdf/pinkdisease.pdf)
41. ^ Hendry WF, A'Hern RP, Cole PJ (1993). "Was Young's syndrome caused by exposure to mercury in childhood?" (http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=1697782) . *BMJ*

307 (6919): 1579–82. doi:10.1136/bmj.307.6919.1579 (http://dx.doi.org/10.1136%2Fbmj.307.6919.1579) . PMID 8292944 (http://www.ncbi.nlm.nih.gov/pubmed/8292944) .
42. ^ R. Baselt, *Disposition of Toxic Drugs and Chemicals in Man*, 8th edition, Biomedical Publications, Foster City, CA, 2008, pp. 923-927.
43. ^ Zhao HL, Zhu X, Sui Y (2006). "The short-lived Chinese emperors". *J Am Geriatr Soc* **54** (8): 1295–6. doi:10.1111/j.1532-5415.2006.00821.x (http://dx.doi.org/10.1111%2Fj.1532-5415.2006.00821.x) . PMID 16914004 (http://www.ncbi.nlm.nih.gov/pubmed/16914004) .
44. ^ Waldron HA (1983). "Did the Mad Hatter have mercury poisoning?" (http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=1550196) . *Br Med J (Clin Res Ed)* **287** (6409): 1961. doi:10.1136/bmj.287.6409.1961 (http://dx.doi.org/10.1136%2Fbmj.287.6409.1961) . PMID 6418283 (http://www.ncbi.nlm.nih.gov/pubmed/6418283) .
45. ^ An Account of the Effect of Mercurial Vapours on the Crew of His Majesty's Ship Triumph, in the year 1810. By Wm. Burnet, M.D. one of the Medical Commissioners of the Navy, formerly Physician and Inspector of Hospitals to the Mediterranean Fleet.
46. ^ Michael J. Doherty MD: The Quicksliver Prize: Mercury vapor poisoning aboard HMS Triumph and HMS Phipps (2003).
47. ^ Stock A (1926). "Die Gefaehrlichkeit des Quecksilberdampfes". *Zeitschrift für angewandte Chemie* **39**: 461–466. doi:10.1002/ange.19260391502 (http://dx.doi.org/10.1002%2Fange.19260391502) .
48. ^ Hunter D, Bomford RR, Russell DS (1940). "Poisoning by methylmercury compounds". *Quart. J. Med.* **9**: 193–213.
49. ^ Engler R (April 27 1985). "Technology out of Control" (http://www.questia.com/PM.qst?a=o&d=5002117801) . *The Nation* **240**. http://www.questia.com/PM.qst?a=o&d=5002117801.
50. ^ Vargas JA (2007-01-26). "'Mad Scientist': On Court TV, Fatal Chemistry" (http://www.washingtonpost.com/wp-dyn/content/article/2007/01/25/AR2007012502091.html) . The Washington Post. http://www.washingtonpost.com/wp-dyn/content/article/2007/01/25/AR2007012502091.html. Retrieved 2007-01-28.
51. ^ Swearengin M (2008-04-01). "Man dies from mercury poisoning after trying to extract gold". *Durant Daily Democrat*.
52. ^ (Associated Press) (2008-04-01). "Colbert man dies from mercury poisoning" (http://www.tulsaworld.com/news/article.aspx?articleID=20080401_12_80377) . *Tulsa World*. http://www.tulsaworld.com/news/article.aspx?articleID=20080401_12_80377. Retrieved 2008-04-20.
53. ^ Tiffany McGee (2009-01-15). "Jeremy Piven Explains His Mystery Ailment" (http://www.people.com/people/article/0,,20252763,00.html) . *People*. http://www.people.com/people/article/0,,20252763,00.html. Retrieved 2009-01-15.
54. ^ James WD, Berger TG, Elston DM (2006). *Andrews' diseases of the skin: clinical dermatology* (10th ed.). Saunders. p. 134. ISBN 0-7216-2921-0.
55. ^ [a] [b] [c] Dally A (1997). "The rise and fall of pink disease". *Soc Hist Med* **10** (2): 291–304. doi:10.1093/shm/10.2.291 (http://dx.doi.org/10.1093%2Fshm%2F10.2.291) . PMID 11619497 (http://www.ncbi.nlm.nih.gov/pubmed/11619497) .
56. ^ No Given Author or Editor. (1935). *Everybody's Family Doctor*. London, UK: Odhams Press LTD. pp. 16.
57. ^ Ford M, Delaney KA, Ling L, Erickson T (2000). *Clinical Toxicology* (1st ed.). Saunders. ISBN 0-7216-5485-1.
58. ^ Mutter J, Naumann J, Schneider R, Walach H, Haley B (2005). "Mercury and autism: accelerating evidence?" (http://www.uniklinik-freiburg.de/iuk/live/forschung/publikationen/Mutter_Autism_NEL.pdf) (PDF). *Neuro Endocrinol Lett* **26** (5): 439–46. PMID 16264412 (http://www.ncbi.nlm.nih.gov/pubmed/16264412) . http://www.uniklinik-freiburg.de/iuk/live/forschung/publikationen/Mutter_Autism_NEL.pdf. Retrieved 2007-08-15.
59. ^ Freitag CM (2007). "The genetics of autistic disorders and its clinical relevance: a review of the literature" (http://www.nature.com/mp/journal/v12/n1/full/4001896a.html) . *Mol Psychiatry* **12** (1): 2–22. doi:10.1038/sj.mp.4001896 (http://dx.doi.org/10.1038%2Fsj.mp.4001896) . PMID 17033636 (http://www.ncbi.nlm.nih.gov/pubmed/17033636) . http://www.nature.com/mp/journal/v12/n1/full/4001896a.html.
60. ^ Rutter M (2005). "Incidence of autism spectrum disorders: changes over time and their meaning". *Acta Paediatr* **94** (1): 2–15. doi:10.1080/08035250410023124 (http://dx.doi.org/10.1080%2F08035250410023124) . PMID 15858952 (http://www.ncbi.nlm.nih.gov/pubmed/15858952) .

61. ^ "Mercury and vaccines (thimerosal)" (http://www.cdc.gov/od/science/iso/concerns/thimerosal.htm) . Centers for Disease Control and Prevention. http://www.cdc.gov/od/science/iso/concerns/thimerosal.htm. Retrieved 2007-12-25.
62. ^ Ferracane JL (2001). *Materials in Dentistry: Principles and Applications* (2nd ed.). Lippincott Williams & Wilkins. p. 3. ISBN 0781727332.
63. ^ Edlich RF, Cochran AA, Cross CL, Wack CA, Long WB, Newkirk AT (2008). "Legislation and informed consent brochures for dental patients receiving amalgam restorations". *Int J Toxicol* **27** (4): 313–6. doi:10.1080/10915810802366851 (http://dx.doi.org/10.1080%2F10915810802366851) . PMID 18821394 (http://www.ncbi.nlm.nih.gov/pubmed/18821394) .
64. ^ Counter SA (December 16, 2003). *Whitening skin can be deadly* (http://www.boston.com/news/globe/health_science/articles/2003/12/16/whitening_skin_can_be_deadly/) . The Boston Globe. http://www.boston.com/news/globe/health_science/articles/2003/12/16/whitening_skin_can_be_deadly/.
65. ^ "FDA Proposes Hydroquinone Ban" (http://www.medicinenet.com/script/main/art.asp?articlekey=64167) . http://www.medicinenet.com/script/main/art.asp?articlekey=64167.FDA bans hydroquinone in skin whitening products
66. ^ "NYC Health Dept. Warns Against Use of "Skin-lightening" Creams Containing Mercury or Similar Products Which Do Not List Ingredients" (http://www.nyc.gov/html/doh/html/pr/pr008-05.shtml) . January 27, 2005. http://www.nyc.gov/html/doh/html/pr/pr008-05.shtml.
67. ^ Counter SA, Buchanan LH (PDF). *Mercury exposure in children: a review* (http://www.state.nj.us/health/eoh/cehsweb/kiddiekollege/documents/counter04_mercuryexpochildren.pdf) . http://www.state.nj.us/health/eoh/cehsweb/kiddiekollege/documents/counter04_mercuryexpochildren.pdf.
68. ^ Mahaffey KR. "Dynamics of Mercury Pollution on Regional and Global Scales" (http://www.springerlink.com/content/w245027uu23r4381/) . http://www.springerlink.com/content/w245027uu23r4381/.
69. ^ In a survey, 28% of Koreans and 50% of Philippians say that they use skin whitening products."Skin lightening in Asia? A bright future?" (http://www.synovate.com/knowledge/infact/issues/200406/) . http://www.synovate.com/knowledge/infact/issues/200406/.
70. ^ Bray M (2002-05-15). *SKIN DEEP: Dying to be white* (http://edition.cnn.com/2002/WORLD/asiapcf/east/05/13/asia.whitening/) . CNN. http://edition.cnn.com/2002/WORLD/asiapcf/east/05/13/asia.whitening/. Retrieved 2010-05-12.
71. ^ Aucott M, McLinden M, Winka M (2003). "Release of mercury from broken fluorescent bulbs". *J Air Waste Manag Assoc* **53** (2): 143–51. PMID 12617289 (http://www.ncbi.nlm.nih.gov/pubmed/12617289) .
72. ^ "Spills, disposal and site cleanup" (http://epa.gov/hg/spills/) . U.S. Environmental Protection Agency. 2009-07-13. http://epa.gov/hg/spills/. Retrieved 2009-06-30.
73. ^ Tunnessen WW Jr, McMahon KJ, Baser M (1987). "Acrodynia: exposure to mercury from fluorescent light bulbs". *Pediatrics* **79** (5): 786–9. PMID 3575038 (http://www.ncbi.nlm.nih.gov/pubmed/3575038) .

External links

- Hazardous Substances: Mercury (http://www.dmoz.org/Health/Occupational_Health_and_Safety/Hazardous_Substances/Mercury/) at the Open Directory Project
- Toxic Substances: Mercury (http://www.dmoz.org/Science/Environment/Environmental_Health/Toxic_Substances/Mercury/) at the Open Directory Project

- http://www.epa.gov/ttn/oarpg/t3/reports/volume5.pdf|Mercury study report to congres
- http://www.atsdr.cdc.gov/tfacts46.html|Agency for Toxic substances and Disease registry

Categories: Drug eruptions | Element toxicology | Mercury (element) | Toxic effects of substances chiefly nonmedicinal as to source

Mercury Emissions from Crematoria

Great Lakes Binational Toxic Strategy
December 6, 2005

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Source and Quantity

Main source of mercury is dental amalgam

US flow model has cremation as third largest source of air emissions of mercury from products at 2436 kg a year in 2005

Canadian 2001 report to UNEP puts air emission of mercury at 6% of total from incineration; 120 kg a year in 1995

Data have high levels of uncertainty

Background Data

Nearly 1,900 crematoria in US. Canadian number not known

710,000 cremations in US in 2004, just under 30% of all deaths

Mississippi:	< 9% of deaths
Hawaii:	68% of deaths

121,000 cremations in Canada, 56% of deaths

Background Data (cont'd)

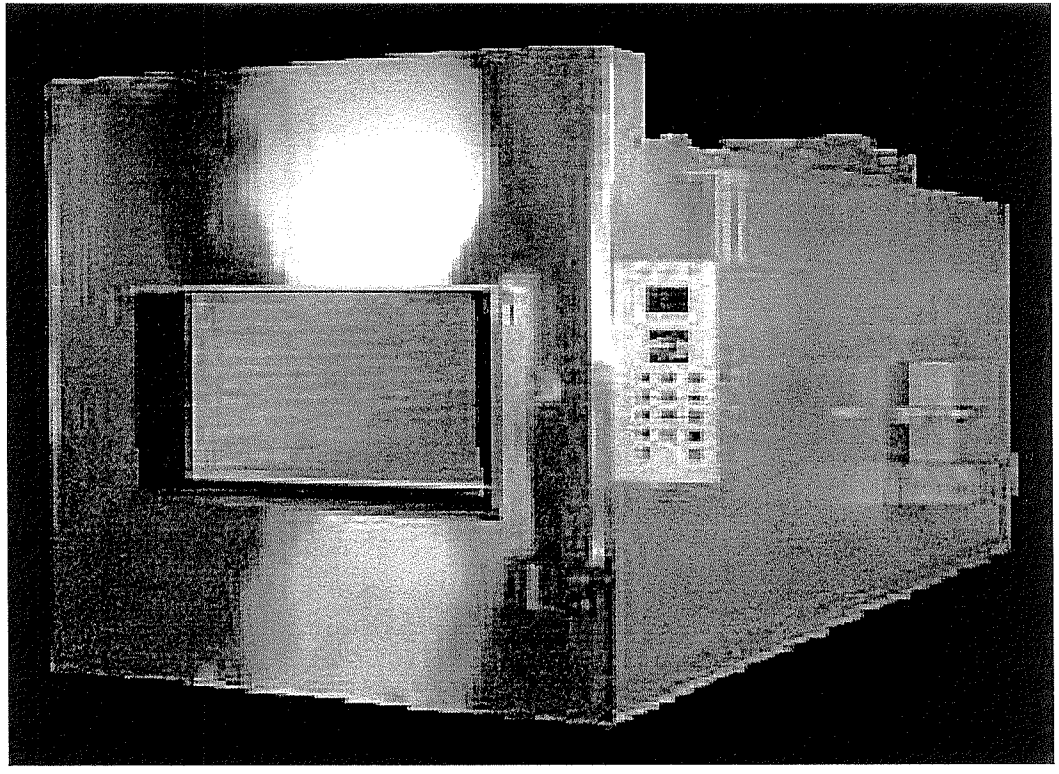
Rate of use of crematoria varies widely

Delaware: 137 cremations per unit

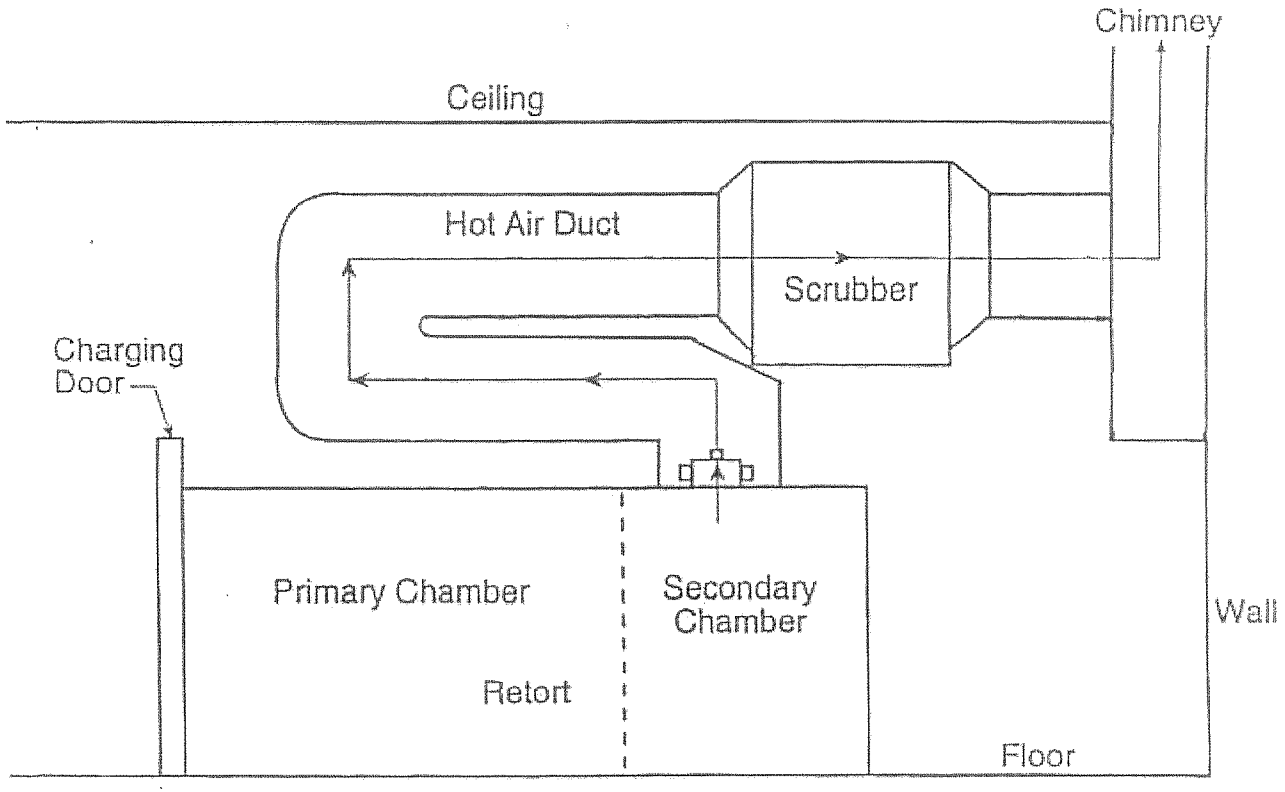
Maine: 1,059 cremations per unit

Use rate affects economics of scale, including for control systems

Typical Crematorium



Crematorium Schematic (with Scrubber)



Outside View of Cremation Area

Cremation Process

Corpse put in cardboard container on gurney

Pacemakers, jewelry and possibly other products removed

Fed into primary chamber, temperature raised to 1650 F (900 C)

Process lasts 2-3 hours

Environmental Fate of Emissions

Effects are probably most global; emissions appear to be mostly (if not all) in a metallic form

Swiss study found elevated levels in soil; New Zealand and Norwegian data show the opposite

Emission Standards

No known standards in North America

European standards include:

- Concentration limits of 0.050 to 0.2 mg/Nm^3
- Total limit of 150 mg per 4 cremations
- BAT without specific numerical standards

Control Methodologies

Removal of restored teeth prior to cremation

Stack controls

- Co-flow filters
- Solid-bed filters
- Traditional gas scrubbing
- Honeycomb catalytic absorbers

Barriers to Control Methodologies

Lack of recognition of the need to control

For removal prior to cremation, cultural values for the handling of corpses

Costs and physical challenges of stack controls

Industry tradition – only one crematorium in the US is said to have stack controls

Recent Legislation in the US

Washington state in 2003 – exemption industry in comprehensive legislation

In 2005, Maine and Minnesota had bills introduced, failed in both states

Data Uncertainties, Future Forecasts

There are significant uncertainties in North American data – few studies exist

North American demographics may be different than European for restoration sizes, composition and number

Forecast for the future is for an increase in emissions for the next several decades, followed by a decrease

Recommended Informational Needs

Amount of mercury released per cremation

Mass balance – air, ash, deposited on crematorium surfaces

Speciation of air emissions

More expertise among researchers, regulators

For a Copy of the Literature Review

Electronic or paper version available

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PRESS RELEASE
FOR IMMEDIATE RELEASE
February 7, 2007

(All comments below, except those within the attached affidavit, may be attributed to McCracken Poston, attorney for Ray-Brent Marsh.)

A SCIENTIFIC EXPLANATION FOR THE EVENTS AT TRI-STATE CREMATORY

As we approach the five-year anniversary of the disturbing discoveries of hundreds of uncremated bodies on the grounds of the former Tri-State Crematory in Noble, Georgia, one persistent question has continued. For five years the hundreds of family members affected by the infamous inactions of Ray-Brent Marsh have been left without an answer to the understandable question: "*Why?*" What was the reason that the tragic circumstances ever took place?

I had also struggled with this question, even as I worked with my client, Ray-Brent Marsh, who seems to be the most unlikely person to have deceived so many people, including those closest to him – his wife and family. Until now a reason has never been offered as to why he failed to cremate the approximately three hundred thirty-three bodies of the approximately one thousand that he took in, or why he undertook the even more difficult task of concealing the uncremated bodies on the property. In court when entering his guilty pleas he expressed great remorse, but even in accepting responsibility at his sentencing was unable to articulate an explanation when directly asked by the Court.

Immediately after getting involved in the case, I began to explore any possible theories that might explain this bizarre behavior. It should be first noted that even the Chief Medical Examiner of the State of Georgia, Dr. Kris Sperry, eliminated all of the more disturbing possibilities, ruling out necrophilia or any other wrongful abuses of bodies as are sometimes present in other criminal cases involving human corpses. Dr. Sperry noted that in Noble the bodies were simply not cremated, and ultimately many were buried or left in vaults together with other bodies in a gargantuan effort to conceal Ray-Brent's failure to cremate them. Dr. Sperry has gone on to enjoy a popular role in the medical examiner lecture circuit on the subject of the Tri-State Crematory, but in those talks has offered no more than the very *unscientific* speculation that my client must have been "*lazy*," even though the effort to conceal hundreds of dead bodies obviously took a great deal of effort.

Furthermore, there seemed to be very little financial incentive for the defendant to fail to cremate. For the hundreds or even thousands of dollars paid to area funeral homes to cremate each body, Brent Marsh was in turn was never paid by those funeral homes more than two-hundred and fifty dollars. The extremely unusual nature of the offense, as well as the difficulty involved in its concealment for over six years, led me to believe a neurological disorder of some sort was involved.

As the evidence was examined, one theory began to emerge as an explanation for not only Ray-Brent's bizarre behavior, but amazingly also shed light on the multiple

medical issues that plagued Brent's father, Tommy Ray Marsh, who built and ran the Tri-State Crematory from 1982 until his deteriorating medical condition forced his retirement in 1996. The senior Mr. Marsh died in 2003, having never recovered from his conditions.

Ultimately, I ceased my daily investigation and intensive work on the development of this theory when the criminal cases came to an early end. The defense won a critical pre-trial effort when the Georgia Supreme Court agreed to bring into appellate scrutiny some of the state's prosecution legal theories, and this event brought the district attorney and the defense together to the negotiating table, where we ultimately reached an agreement to dispose of all the cases. Ray-Brent himself wanted to end the misery that his charges caused for his family, as well as that of the hundreds of other families affected. Still, at the sentencing he could never adequately explain to anyone why he felt all of this had happened, *even though the failure to be able to do so could have jeopardized the court's acceptance of the plea deal.*

Even though the criminal cases were at a conclusion, I continued to be nagged by the notion that there were still no answers as to why this happened. There are some who were victims of the tragedy who have found forgiveness in their heart toward my client. But there are others, victims or just the curious, who continue to express other views. Most difficult to tolerate are the persistent unsolicited "man on the street" theories that are constantly offered to me when the subject of the case comes up, many of which involve either ignorantly conceived opinions involving racism or misguided and inaccurate conspiracy theories that allege multiple participants, again often couched in

racist terms. Years after we put the case to rest for history to judge, I continued to hear from almost anyone and everyone who had an opinion, and I have and always will reject any offered theories that involve derogatory racial stereotypes (sometimes even stated directly to me) as a legitimate conclusion to be reached. But I realize now that the need for a plausible explanation is what has invited the rank speculation, both good and bad.

Sometime after the end of the criminal case I again continued to work on the emerging theory that an external, environmental cause claimed not only the literal sanity and judgment of Ray-Brent Marsh, but also the health and ultimately the life itself of his father, Tommy Ray Marsh. The suspect element is **mercury** (listed on the Periodic Table as ***Hg***).

Mercury is a naturally occurring element, but it well established to be present in high concentrations in the cremation process due to the decades-old practice of mercury dental amalgam being used in patients, who pass away and their bodies are made subject to cremation. It well established that Tommy Ray Marsh never failed to cremate a body of the thousands given to him from 1982 until his failing heath, including strokes, neuropathy, senility, and "Parkinson-like" symptoms (all classic mercury poisoning symptoms) forced him to retire in 1996. He never recovered and died in 2003.

It is also known that Ray-Brent Marsh, who returned home from college to take over the crematory when his father could no longer work, successfully cremated two-thirds of the bodies given to him. It was also shown to the world by the state

investigators and other witnesses that the crematory was in somewhat of a functioning order, but it was also obvious that the heavy particulate matter of cremated bodies coated the *interior* of the small crematory building due to the inadequate ventilation and breached stovepipe from the retort to the exterior of the structure. When I first took this case and began to ask questions, Ray-Brent's wife could only tell me that her husband had long suffered from chronic insomnia and had only complained of headaches and other seemingly minor body aches. Remarkably, all of these are also symptoms of mercury toxicity.

The neurological and psychological effects of mercury exposure are well known, and have been documented in history. The documented mental illness noted among the Huguenot craftsmen of the eighteenth century who used mercury in the process of hat making made the "Mad Hatter" a character of science, a character in our literary world and now of our modern reference. In the nineteenth century, the Medical Society of New Jersey again noted the multiple and bizarre symptoms of mercury-poisoned hat makers in 1860. A wide array of physiological and neurological problems were documented in these and other studies of victims of mercury exposure and toxicity.

In 2004, I ordered a simple mercury test kit for Ray-Brent Marsh to submit a hair sample, without telling him what he was being tested for. This was over two years after the state's discoveries at Tri-State Crematory – and over two years since he was last exposed to the suspected source of mercury vapors. When the results came back, I at first almost dismissed the report and began to question my own theories about the effect of

mercury on the Marsh men. The 2004 hair test revealed the element mercury itself within "normal" ranges, but there were alarmingly high values for aluminum, antimony, arsenic, cadmium, lead, nickel and tin. Some of these other metals were *three to eight times* over the reference range of members of the healthy adult population. An advisory form letter came with the results suggesting that the subject of the test consult a doctor due to the unusual values represented in the test.

Only when I later researched and learned about the *mechanism* of mercury exposure and the proper *interpretation* of hair tests were my suspicions confirmed. A characteristic signature of mercury toxicity is that the element interferes with the results of some testing, often causing unusual values of other minerals. For example, a more recent exposure to mercury vapor would have likely caused a higher reading of mercury itself in the hair test, but if the test is taken after a long passage of time from the exposure the way to determine the signature of mercury toxicity in the body is to look for the biochemical aberrations in the body that mercury *causes*. The scientific term for this interruption in the process is called "impaired mineral transport," where mercury interferes with the ability of cells to move other minerals in and out selectively. In Ray-Brent's test, the mercury value result was normal, but the alarmingly high values of the other minerals are a classic signature of mercury toxicity. Research has shown that in some hair tests of known victims of mercury exposure, the test for mercury itself often reads low but the effect of mercury causes other metals to show unusually high values. Impaired mineral transport indicates mercury poisoning is likely. It is hoped that more independent testing may be done to support these findings.

I believe that Ray-Brent Marsh, while living and working at the crematory, became a modern-day "Mad Hatter". Fortunately for him, the element mercury over time leeches from the body of the victims who inhale it, and there should be no long-term neurological effects. For Ray-Brent's father, however, the element took a physiological path, and possibly eventually took his life.

Since the end of the criminal case I continued to consult an expert on the subject of mercury toxicity, Dr. Boyd Haley, who has given an affidavit concerning his opinion that mercury toxicity can explain the events at Tri-State Crematory. Interestingly, Dr. Haley reveals why the men at the site were affected while the women who lived nearby were not: That the effects of mercury are enhanced by testosterone, and somewhat limited by estrogen. I have attached a copy of his affidavit. Dr. Haley received no payment for sharing his professional scientific opinions.

The crematory building itself has been destroyed, per federal court order, but it was well documented in photographs and by witnesses who personally viewed the unhealthy conditions within the structure. The facility was in a poor condition of repair. These conditions included the well-documented layer of incinerated particulate matter within the structure and the inadequate crematory retort ventilation that caused it, evidenced by the many holes and separations in the system which allowed the burned particulate into the interior of the structure where over a period of twenty years the father and son consecutively worked in the building and lived near the site.

It should be noted that in Scotland and other European countries there are requirements that mercury amalgam-filled teeth be extracted from a corpse before subjecting the body to the cremation process. The existence of mercury vapor being released in the cremation process is well documented in the scientific community. Also well documented in science and history are the multiple physiological and neurological affects that this dangerous and elusive neurotoxin can create within the human body, a toxin which can affect two or more victims in entirely different ways.

What has failed in part is the governmental regulatory process in the United States and in the State of Georgia. For the latter I can even blame myself in part, as a former member of the Georgia General Assembly that voted to temporarily exempt this crematory from regulation (for eighteen months) in 1992, but even then there was no concern or regulation about the smokestack emissions from such facilities. Even today there are little or no guidelines concerning the proper ventilation or the proper location placement of crematories.

This is a cautionary tale for the ages. Tommy Ray Marsh and his son Ray-Brent Marsh appear to be victims in their own right of the disaster in Noble. I hope that the release of this information can give some comfort to those who were most deeply affected by the disaster, and who have been looking for answers.

There will likely be some who are openly skeptical of any studies revealed by a criminal defense lawyer; however, one must remember that the case is over and there is no appeal from the conviction of Ray-Brent Marsh for failing to cremate the bodies at the Tri-State Crematory. It is my intention to reveal information developed in the defense of the case to offer an explanation for the bizarre events of over five years ago, in hopes to give a most wanted and even *demanded* answer to the affected families and to the community. It is an opinion based in science that I stand by and am confident will not be contradicted. I ask that any and all in the community consider these facts before making their own final judgment of about Ray-Brent Marsh and the events at Tri-State Crematory. If a better explanation is to be had, it is simply not to be found.

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Mercury Exposure:
The World's Toxic Time Bomb

A Report by:
Ban Mercury Working Group

Prepared for the 22nd
United Nations Environment Programme
Governing Council Meeting
Nairobi, Kenya

A Call To Action

In the last century mercury levels in the global environment have tripled as a result of increased pollution from industrial, occupational, medicinal and domestic uses. This alarming, yet under-publicized, rise in mercury pollution has exponentially increased the risk of dangerous and deadly exposure to all peoples, wildlife and ecosystems, and threatens the long-term security of fish as one of the world's most important protein sources.

Mercury concentrations in the environment are now on the verge of exceeding a threshold that endangers the citizenry of every continent. Major food sources have already been contaminated; children are poisoned by excessive thimerosal vaccine schedules; indigenous groups from Baffin Island to the Guyana Shield risk losing irreplaceable food staples in fragile subsistence economies; and millions of people breathe mercury vapor into their lungs everyday through dental mercury-fillings.

No single country can resolve the mercury problem on its own. There are alternatives to mercury, but there is no alternative to international cooperation.

Economically viable alternatives to mercury exist for almost every known human use. Control technologies and conservation strategies are available that could reduce releases from the largest source of mercury pollution—coal-fired power plants.[1] Nevertheless, global releases of mercury to the environment are growing dramatically. Recognizing the immediate global threat, in September 2002 at the United Nations Environment Program (UNEP) Global Mercury meeting in Geneva, 150 experts concluded *"there is sufficient evidence of significant global adverse impacts to warrant international action to reduce the risks to human health and the environment arising from the release of mercury into the environment."*

Over the past half-century, numerous large scale exposure epidemics in places like Japan, Iraq, South Africa, India and Peru have provided the scientific community with all too many opportunities to study the effects of methylmercury on human health. This body of research has clarified what many had long feared: human health is compromised by significantly smaller concentrations of mercury than ever imagined. Accordingly, our understanding of so-called safe exposure levels has become more precise. In 1991, the World Health Organization concluded that *a safe level of mercury exposure below, which no adverse effects occur, has never been established.*[2]

Developed countries are increasingly concerned of the risk to their children of neurological and developmental defects from mercury passing through the placental and blood-brain barrier during pregnancy. For people in developing countries—particularly gold miners, subsistence fishers and indigenous communities—the impact of mercury is very real and more than just a concern. The developing world experiences a disproportionate mercury pollution burden from industrialized nations exporting their excess elemental mercury, outdated industrial processes and mercury-containing products to nations with weaker environmental regulations, awareness of how harmful mercury is, and human rights protection.

To avert this impending global mercury crisis, concrete and binding international action must be developed to coordinate and harmonize action at the local, national and regional levels. Only a binding international instrument can require equal responsibility to all state-actors and prevent the unjust transfer of mercury from the developed to the developing world. Voluntary and aspirational international targets are insufficient: *no single country can resolve the mercury problem on its own. There are alternatives to mercury, but there is no alternative to international cooperation.*

Mercury pollution compromises the most basic human rights—life, clean food and water, work in safe environments, environmental health, and the rights of indigenous peoples to preserve traditional ways of life and foodways. These basic rights are threatened by the buildup of mercury dental fillings, vaccines, and in fish, and the transference of mercury from richer, developed countries to poorer, less developed nations.

In order to create a healthy and equitable living environment for future generations, we must stop the circle of poison that mercury use and pollution perpetuates, and take immediate steps to limit human exposure. As the authors of the UNEP Global Mercury Assessment Report point out, despite remaining data gaps in our understanding of how mercury negatively affects human and environmental health, *international actions to address the global mercury problem should not be delayed.*

In response to this growing ecological and health crisis, this report of the BAN-HG Working Group provides an overview of the toxicological impact of methylmercury, and highlights five primary sources of human exposure to mercury: 1) Consumption of Contaminated Fish; 2) Occupational Exposure; 3) Dental Amalgam; 4) Vaccines; and 5) Domestic Use. It also lays out a framework of recommendations for addressing exposure pathways to mercury through international agreements, coupled with actions at the national and local community levels.

Introduction: Background Toxicology

Mercury occurs in three basic forms: elemental (metallic), inorganic and organic mercury. Metallic mercury is poorly absorbed in the digestive track, but enters the body via inhalation.[3] Exposure to high levels of elemental mercury vapor can result in severe neurological disorders; metallic mercury is transformed into methylmercury—sometimes years after its initial release.[4]

While most mercury released into the environment is in the form of elemental or inorganic mercury, it is organic mercury—in particular, methlymercury—that poses the greatest threat to people and wildlife. A potent neurotoxin, exposure to methylmercury impairs the brain, kidneys and liver, and causes developmental problems, reproductive disorders, disturbances in sensations, impairment of speech and vision, hearing and walking difficulties, mental disturbances, and death.[5] Methylmercury concentrates in fish tissue, becoming increasingly potent in predatory fish and fish-eating mammals, sometimes reaching toxic levels over a million times greater than the surrounding waters.[6]

At present global mercury loading rates, the equivalent of less than 1/50th of a teaspoon of mercury per 20 acre lake surface is enough to make fish unfit for human consumption.[7] In Sweden, for instance, fish in 50 percent of the country's 100,000 lakes have mercury levels exceeding WHO limits, and in 10 percent of the lakes levels are double the recognized limits. Once

Recommendations For Action

The BAN-HG Working Group invites the Governing Council to consider the following recommendations:

1. Convene an open-ended ad hoc working group with a mandate to propose international action to reduce releases of mercury to the environment, with a mandate to consider all measures to reduce or eliminate releases of mercury to the environment.

2. Adopt as its goal, the virtual elimination of all uses and releases of human-induced mercury pollution, including the development and implementation of national and regional action plans and agreements that aim to reduce or eliminate all mercury release to all media, to the maximum extent possible, within a specified time.

3. Develop and promote the creation of an international inventory to account for and monitor mercury emissions, sources, uses, imports and exports.

4. Develop an international binding instrument that contains the following:

   a) Strict control measures on the global trade of mercury, mercury wastes, and technologies, and prevention of mercury trafficking from developed to developing countries;

   b) Permanent retirement of all existing civil and military mercury stockpiles, including chlor-alkali mercury and the Defense National Logistics Agency stocks;

   c) Promotion of mercury-free alternatives in the small-scale gold mining sector;

   d) Return of mercury to countries of origin for permanent storage;

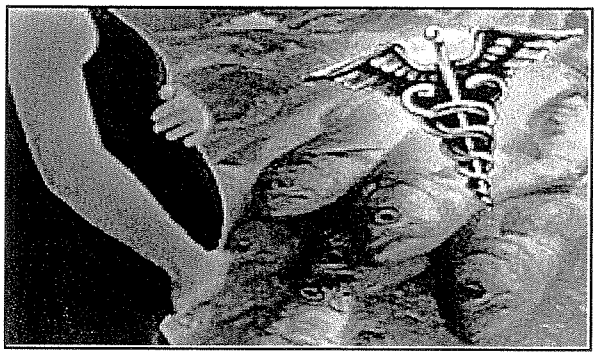
   e) Funding mechanism for the rehabilitation of communities and environments negatively affected by industrial processes knowingly transferred from developed nations to less developed countries in Asia, Africa, Latin America, Central Asia and Eastern Europe; included in this should be technical and financial assistance to developing countries to support efforts to reduce releases of mercury to the environment and substitute use of mercury with safer alternatives; and

   f) End of government subsidies for primary mining of mercury, and an effective strategy for managing byproduct mercury produced in the metals mining industry, including gold mining.

ingested by people, methylmercury is rapidly absorbed through the gastrointestinal tract, and it easily penetrates the blood-brain and placental barriers in humans, allowing passage of mercury from a pregnant woman to her fetus.

I. Fish: A Toxic Mercury Time Bomb

The mercury exposure route of greatest concern to the international community is the contamination of the world food supply. Recognizing the increasing risks of consuming mercury-contaminated freshwater fish and seafood, a recent European Commission report says that, "exposure to methyl mercury via diet is the critical mercury problem for Europe, the reduction of potential exposure to this Hg species should be the focus for the steps to be taken in Europe..."[8]



Through regular fish consumption in their diet, entire populations are exposed to methylmercury. Virtually 100 percent of the mercury found in such seafood as tuna, king mackerel, swordfish, and shark, is methylmercury. More than one billion people around the world rely on fish and other vulnerable seafood as their primary protein source. Indeed, global fish consumption is at record levels, reaching 121 million tons in 1996—making fish a more important global staple than beef, pork and poultry. In the developing world, the dietary importance of fish is even more profound. Fish provides as much as 25 percent of all animal protein in Asia, and 17 percent in Africa, while in many indigenous communities fish is at the center of centuries-old subsistence economies.

The most recent US Centers for Disease Control data indicates that 8 percent of US women of childbearing age have unsafe mercury levels, translating into over 300,000 children born each year in the US at risk of exposure to mercury.[9] Eating fish during pregnancy and through nursing exposes infants to dangerous levels of mercury. Daily consumption of as little as 60 grams—or just 2.5 ounces— of fish can exceed the safe levels set for mercury exposure of the average woman.

Both pre- and post-natal mercury exposure from fish is linked to impaired development of the infant's nervous system.[10] A 1997 population study conducted in the Faroe Islands demonstrated that children born to mothers who consumed mercury-contaminated whale meat during pregnancy, exhibited cognitive delays and irregular cardiovascular development.[11] Concentrations of mercury in cord blood among Faroe Islands children who were exclusively breast-fed averaged about four times the recommended exposure limit recommended by the U.S. EPA.

Indeed, global fish consumption is at record levels, reaching 121 million tons in 1996—making fish a more important global staple than beef, pork and poultry.

Several recent advisories have been issued in Europe and the US to protect developing infants from methylmercury poisoning. The recent European Commission report acknowledges, "dietary restriction with respect to fish with high levels of MeHg should be advised for pregnant women."[12] And in May 2002, Britain's Food Standards Agency began advising pregnant women, women intending to become pregnant, and children less than 16 years of age, to avoid eating swordfish, shark, and marlin because of high mercury levels. Forty-one American states have issued fresh water fish advisories, and 10 now advise women and children to limit consumption of canned fish.

In the U.S., fish consumption—particularly canned tuna—is thought to be the main culprit for the 7-8 percent of women between the ages of 15-44 who have excessive mercury levels in their bodies. According to one U.S EPA scientist, canned tuna is a threat not because its mercury levels are so high, but because people consume so much of it that even at the relatively low average exposure rate of 0.2 ppm, canned tuna is still likely the largest source of mercury exposure.

Adults in fish-eating indigenous communities frequently consume as much as of 40 to 60 micrograms of mercury per day from predatory fish contaminated by pollution....

From the Arctic to the Amazon, mercury's propensity to bioaccumulate in the environment is particularly threatening to indigenous communities. Adults in fish-

eating indigenous communities frequently consume as much as of 40 to 60 micrograms of mercury per day from predatory fish contaminated by pollution from artisanal gold mining —about 6 to 10 times the international average consumption. In one Inuit community in Baffin Island, Canada, more than 50 percent have mercury levels in their daily diet of seal, walrus and narwhal blubber that exceed the WHO's guidelines for tolerable daily intake. People with the highest intake have mercury levels six times higher than the provisional tolerable weekly intake of mercury.[13] Meanwhile, among the Wayana in French Guiana, close to 60 percent of the community exceeded WHO safe limits.[14] And roughly 14 percent of the fish taken from the heavily mined Caroni River surpassed safe levels.[15]

A recent Finnish study links cardiovascular risks to mercury exposure through contaminated fish. Among middle-aged men in Finland, patients who consumed greater than 30 g/day fish had 56% higher mean hair Hg (mercury) content than people who consumed less than 30 g/day of fish. The higher consumption and subsequent higher hair-mercury levels were associated with a 2-fold increase of risk of acute myocardial infarction and coronary heart disease.[16]

II. Occupational Exposure: Protecting Workers' Rights

Countless people throughout the world are put in harm's way as a result of regular occupational exposure to mercury. Mercury use spans many industries and continents, including chlor-alkali production, thermometer factories, primary mercury mining, gold, silver, lead, copper and nickel production, dental clinics, and coal-fired power plants.

The world's most severe mercury-related occupational exposure crisis is happening far from the focus of any media attention. Millions of people engaged in small-scale—or artisanal—gold mining use mercury to extract gold from unwanted sediment. Bonded gold-mercury amalgam is then heated with an intense flame to burn off mercury, directly exposing miners and bystanders to deadly elemental vapors. As much as 95 percent of all the mercury used in artisanal gold mining is lost to the environment. This mercury methylates after mixing with organic matter—bioaccumulating in fish and contaminating precious food supplies. Although the informal nature of this industry makes accurate numbers difficult to ascertain, in Brazil 130 tons of mercury per year are released into local rivers for every 90 tons of gold produced from artisanal gold mining.[17]

...faced with stringent environmental standards in developed countries, multinational companies regularly shift their mercury-based operations to the developing world, where they literally get away with murder....

Weak enforcement of labor rights in developing countries places a disproportionate burden of mercury pollution on their people. These communities are faced with an exploitative paradigm of profound poverty and official disregard for basic human rights protection and respect for human dignity. When faced with stringent environmental standards in developed countries, multinational companies regularly shift their mercury-based operations to the developing world, where they literally get away with murder, sacrificing human life in the name of the bottom line.

Lenient environmental laws in much of the developing world means that even in more formal industrial sectors occupational exposure to mercury is a persistent threat to human health. According to environmental and community groups in Kodaikanal, a hill station in southern India, 10 people died and dozens others were poisoned from mercury inhalation at the local thermometer factory—run by a subsidiary of the multinational giant Unilever. The company—which has since closed the factory but denies all allegations of personal injury to workers—employed no occupational safety measures, even though mercury levels inside the plant were reportedly 600-times greater than internationally accepted safety limits.

While the workers' rights case against Unilever rages on, a similar pattern can be detected in the now decade old case against British operated Thor Chemicals in KwaZulu-Natal, South Africa. Workers were systematically exposed

to elemental mercury for over a decade before the government finally stepped in and shut down the plant. At least two workers died as a result of occupational exposure, while dozens more reported the typical symptoms of mercury poisoning, such as nervous disorders, infertility, and madness.

Nevertheless, mercury-related occupational exposure issues are not limited to developing countries. The US Department of Labor's Mine Safety and Health Administration reports that 12.5 percent of workers tested at gold and silver mines—where thousands of tons of "byproduct" mercury are produced—showed dangerous levels of mercury in their bodies.[18] In 50 percent of these cases mercury levels were more than twice the permissible limit, while some workers' mercury levels were 50 times safe limits. Mine workers' families were also found to be at risk from trace mercury entering their homes attached to items of clothing worn at the mines.

People working in resource extractive industries are not the only employees exposed to mercury. Dentists and dental clinic employees are another high-risk group of workers. Mercury dental amalgam is generally heated in the dental office in order to extract silver, volatilizing elemental mercury vapor, and exposing workers via the skin and the lungs.[19] A recent Scottish study revealed high rates of kidney disease and memory disorders among dentists whose urine samples contained four-times the normal level of mercury.[20]

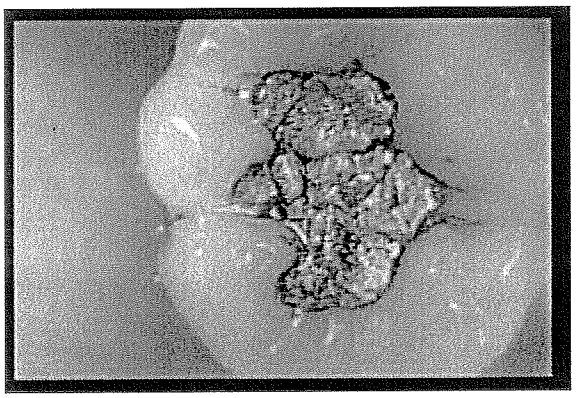
III. Dental Amalgam

The WHO[21] and several US federal and health and research agencies,[22] confirm that dental amalgam—an inexpensive alloy of silver, copper, tin and 50 percent mercury—is the largest source of human exposure to elemental mercury for those who have dental amalgam.[23] The lungs rapidly absorb 75-85% of elemental mercury vapors coming from dental amalgam.[24] Recent research confirms that mercury escapes from dental amalgam and is converted to methylmercury after combining with bacteria in the mouth.[25] Laboratory tests have shown that the average person with dental amalgam gets 10 times as much daily mercury exposure as the average person without amalgam fillings. Depending on the number of amalgam surfaces in a person's mouth, average daily absorption of mercury is between 3 and 17 micrograms of mercury.[26]

Dental amalgam is the predominant mercury source in wastewater systems. In addition to exposing dental industry workers directly to mercury vapors, waste mercury from clinics accounts for 40 percent of the mercury load in U.S. sewer systems—three times the pollution from the next largest contributor. Mercury in wastewater systems makes its way back into the environment by passing through sewage treatment plants and leaching from landfills and sites where sewage sludge is applied for agricultural use, into rivers, lakes, ocean and groundwater. It is directly distributed to the atmosphere as air emissions when sludge is incinerated.

The WHO and several US federal and health and research agencies, confirm that dental amalgam...is the largest source of human exposure to elemental mercury....

The governments of Sweden, Germany, Denmark, Norway, Finland, Austria, and Canada have taken steps to significantly reduce dental mercury release and limit or phase out mercury use in amalgams, especially among sensitive populations including pregnant women, children and those with impaired kidney functions. Nevertheless,



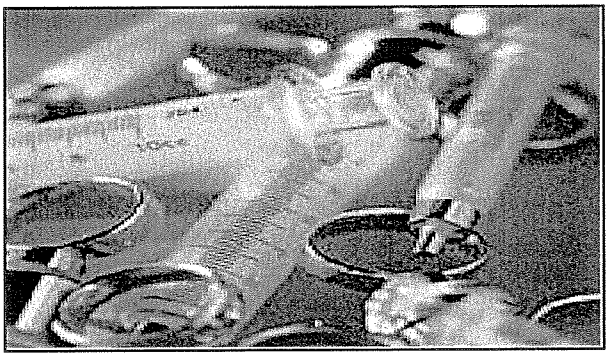
the mainstream US dental establishment continues the unabated use of dental-mercury amalgam.

In 2001, U.S. dental clinics used 44 metric tons of mercury to make 100 million amalgamated fillings—an increase of three tons from 1999. Meanwhile, insurance companies perpetuate this exposure crisis by only covering the cost of cheaper mercury fillings, despite recent findings that blood mercury levels from dental amalgam can be as high as 20 micrograms per liter—more than twice the mean concentration for blood. Ironically, two industries—the dental and insurance industry—that exist for the sake of serving the public health and protecting people from

harm, are knowing contributors to one of the world's critical mercury exposure crises.

IV. Thimerosal

Thimerosal, a mercury-containing preservative, was first added to vaccines in the 1930s to protect against bacterial contamination. A proprietary formulation of the Eli Lilly Company, thimerosal is composed of nearly 50 percent mercury, and metabolizes to ethyl mercury and thiosalicylate. Although ethyl mercury toxicity has not yet been thoroughly evaluated, its composition is very close to methylmercury.[27]



In 1999, European regulatory agencies and the US FDA agreed that exposure risks warranted removing single-dose mercury-containing vaccines from the market as soon as possible.[28] Based on EU and US calculations, the cumulative impact of mercury-vaccines on a six-year-old child exceeds the acceptable reference dose level set by EPA. Until recently, all pediatric diphtheria-tetanus-pertussis (DTP and DTaP), hepatitis B (hepB), Hib (haemophilus influenzae type b), meningococcal vaccines, and some rabies and pneumococcal vaccines, manufactured and used in the U.S. contained thimerosal.

Until the 1980's, pre-school children received only one mercury-containing vaccine (DTP) in the U.S. Six other mercury-free vaccines gave a total of 23 doses. But over the last two decades, administering vaccines to infant children has multiplied exponentially, and in 1988, four new doses of a mercury-containing vaccine (Hib) were added to the routine childhood vaccination schedule in the U.S. This was followed in 1991 by three doses of mercury-containing hepatitis B vaccine, first given in the newborn nursery at birth. By 1999, before the FDA and EPA told U.S. drug companies to remove the mercury preservative from all pediatric vaccines, the U.S. Centers for Disease Control directed pediatricians to inject all young children with 30 doses of 11 different vaccines in the first 18 months of life, when children are most susceptible to neuro-developmental disruption caused by mercury poisoning.[29]

High mercury levels detected in hair and blood samples of autism patients[30] have contributed to mounting evidence that childhood exposure to thimerosal in vaccines is linked to the onset of autism, as well as other cognitive disorders such as attention deficit disorder and speech/language delay.[31] And studies initiated by the FDA demonstrate that the cumulative impact on a six-month-old infant from mercury-containing vaccines exceeds the acceptable reference-dose-level established by US EPA and upheld by the US National Academy of Sciences in their July 2000 report. Until 2000, a 6-month old infant undergoing standard pediatric vaccination recommendations in the U.S. would have received 187.5 mcg of mercury, almost three times the calculated exposure limit of 65 micrograms, based on EPA guideline of 0.1 microgram/kg/day.[32] Despite current recommendations, an infant may still receive excessive levels of mercury if given some brands of Hib, hepB and pneumococcal vaccines.

Although most vaccines in the U.S. are now available without thimerosal, pharmaceutical companies continue to sell mercury-based vaccines—including DTP, hepB and Hib—to developing countries where mercury ingestion guidelines are less stringent or non-existent. Sixty percent of thimerosal-containing DTP world vaccine supply is produced locally outside of the U.S. and used in developing countries.[33] The WHO guideline used for thimerosal exposure from vaccines in countries worldwide is five times higher than the safety limit recommended by the U.S. EPA, and higher than those established by the Agency for Toxic Substances Disease Registry[34] and the FDA.

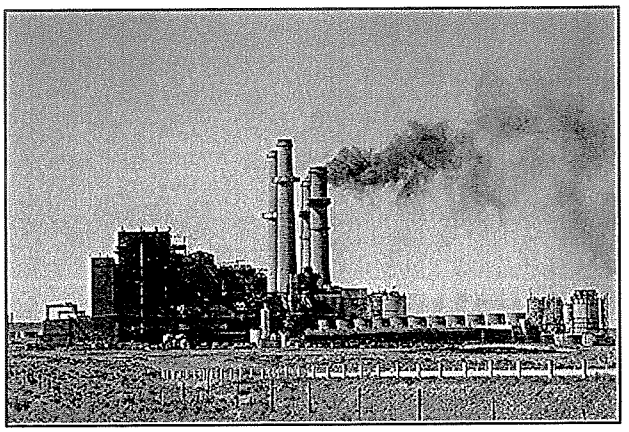
Although most vaccines in the US are now available without thimerosal, pharmaceutical companies continue to sell mercury-based vaccines—including DTP, hepB and Hib—to developing countries where mercury ingestion guidelines are less stringent or non-existent.

Infants who are vaccinated in developing countries typically receive 150-175 micrograms of mercury by 18 months.[35] And even though the WHO has recognized the potential side effects of thimerosal since 1990,[36] the recommended vaccine schedules from African countries includes large quantities of mercury from DTwP, hepB and Hib vaccines.

Although some developing countries can only afford routine vaccines of DTP or DTwP, those children vaccinated in such a country would still be exposed to levels of mercury exceeding the U.S. EPA safety guideline. WHO's Joint Expert Committee on Food reaffirmed that the mercury exposure limit of 3.3 micrograms per kilogram per week should be reduced by a factor of 5 for pregnant women and nursing mothers. However, it was not made as a specific official recommendation.[37]

V. Domestic Mercury Use

Domestic sources of mercury such as thermostats, thermometers, manometers, batteries, light switches, fluorescent lights, pesticides, gas regulators, and ritual uses of mercury among Afro-Caribbean peoples, all expose people to mercury vapors in their homes.[38] According to a recent study, as much as 10 percent of U.S. households may have mercury exposure levels that exceed the worker safety standard established by OSHA-PEL—a relatively conservative risk level targeted for healthy adults rather than more at-risk segments of the population like children and pregnant women.[39] Mercury may also be transported home by children returning from school science labs, and by people who work with mercury-bearing equipment at medical facilities.[40] As noted earlier, employees at thermometer plants or gold mines frequently track mercury home on shoes and clothing.



Once mercury enters the domicile, it can be difficult to remove—exposing people to volatile vapors over extended periods with little hope for remediation. Mercury vapors can remain for months or years on furniture, carpet, floors and walls, and is tracked and transferred easily from shoes, personal items and clothing. In modern "tight" buildings, vapors can also be trapped for long periods of time, continually re-exposing inhabitants.

Some Mercuric Facts on Coal

Humans have mined and used mercury throughout the world for more than 2000 years. But widespread mercury emissions from fossil-fuel energy production, mining and industrial practices like chlorine production have increased mercury pollution 300 percent since the beginning of the industrial age 250 years ago. While total mercury emissions in North America and Europe have decreased since about 1990, expanding global coal-use is creating unprecedented mercury pollution levels.

Worldwide, 2500 tons of mercury are emitted from human activities each year.[i] Fifty-percent of all U.S. mercury emissions are from coal-fired power plants. China and India account for about half of the world's anthropogenic mercury emissions. In Asia, coal burning accounts for 42 percent of mercury emissions;[ii] in eastern Africa and the former Soviet Union coal accounts for 40 percent.[iii]

Over the next two decades, total coal consumption is expected to double to 10 billion tons per year. Nearly 50 percent of this increase is will come from China, while 15 percent will be from the U.S. and 7 percent from India. Without the employment of effective control strategies or an increased emphasis on cleaner fuels (such as natural gas), renewable energy (e.g. wind, biomass, and solar) and conservation and efficiency improvements, expanding coal use will dramatically increase worldwide mercury emissions.[iv]

[i] Environmental Protection Agency (US). Mercury study report to Congress. Washington;EPA. Pub.No.: EPA/600/P-97/002Ab.

[ii] Pacyna,E.G., & Pacyna, J.M., Global Emission of Mercury from Anthopogenic Sources in 1995, Norwegian Institute for Air Research, P.O. Box 100, 2027 Kjeller, Norway.

[iii] Pirrone,N.,Keeler,G.J.,and Nriagu,J.O., "Regional Differences in Worldwide Emissions of Mercury to the Atmosphere," Atmospheric Environment Vol.30,No.17,pp 2981,2987, 1996.

[iv] Miller,S., Dunham,G., and Olson,E., "Worlwide Mercury Control Strategy for Coal," Mercury as a Global Pollutant-5th International Conference, May 23-28, 1999, Rio de Janeiro, Brazil.

In parts of the developing world—notably China—coal is used for domestic functions such as heating and cooking, and is burned in simple household stoves, exposing people directly to emissions of mercury and other toxic metals and organic compounds. The US Geological Survey reports that hundreds-of-millions of people in rural China commonly burn raw coal in unvented stoves, and use coal briquettes to dry corn and other foods.[41] This type of coal-use poses an extremely high risk because the coal typically has higher mercury concentrations than coal burned in a U.S. or European power plant *(see sidebar page 7)*. For instance, in Guizhou Province in southwest China—where domestic coal consumption is commonplace—mercury levels in coal were measured as high as 55 ppm, approximately 200 times the average mercury concentration for U.S. coals.

To Conclude...

The tripling in mercury levels in the global environment for the past 100 hundred years has resulted in increased risks to all peoples, wildlife and ecosystems, and threatens the future viability of fish as one of the world's most important protein sources.

Mercury from a variety of industrial, occupational, household and health care uses—as well as local and global mercury sources—poses further exposure risks to millions of people around the globe each day.

Various large-scale exposure epidemics over the last 50 years have demonstrated the devastating impacts of severe mercury poisonings. From Minimata, Japan to Choropampa, Peru and across the world over, direct human exposure to mercury has injured and killed tens-of-thousands of people, devastating generations of survivors, wrecking communities, and ruining contaminated sites for decades.

People in developing countries-- and in particular gold miners, subsistence fishers and their families-- are disproportionately impacted by mercury, due to their economic and cultural situations and lack of awareness of the exposure risks posed by mercury. Mercury's propensity to bioaccumulate and persist in the environment is particularly threatening to indigenous communities from the Arctic—where atmospheric deposition from coal burning in industrialized countries accumulates in fish and mammals—to the Amazon, where mercury releases from small-scale gold mining is threatening critical ecosystems.

These are the worrisome facts, chronicled in the brutal history of mercury. The world's people have a right to ask, how many more must be poisoned by mercury before nation's unite to stop the toxic trade, use and release of mercury forever? Unless an alternate path is clearly articulated, increasing fossil-fuel generation, gold-mining, mercury cell chlor-alkali production, waste disposal, and new industrial and domestic uses for mercury will push the planet beyond the thresholds for living beings.

Clearly, to avert a global mercury catastrophe, concrete and binding international action must be developed to coordinate and harmonize action at the local, national and regional levels in order to protect children and future generations from mercury exposure - the world's toxic time bomb. *Written by: Shefa Siegel, Lori Stratton, Michael Bender, and Richard Gutierrez, Cover Photo: Copyright Eugene Smith.*

Ban-Hg-Wg

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[1] Bureau of Waste Prevention, Division of Planning and Evaluation, Massachusetts Executive Office of Environmental Affairs and Department of Environmental Protection, "Evaluation of the Technological and Economic Feasibility of Controlling and Eliminating Mercury Emissions from the Combustion of Solid Fossil Fuel, Pursuant to 310 CMR 7.29-Emissions Standards for Power Plants, Dec 2002.
[2] World Health Organization (WHO), 1991, Environmental Health Criteria 118, Inorganic Mercury, WHO, Geneva.
[3] Carpi, A. The Toxicology of Mercury, City College of New York. P. 2, 1998.
[4] Veiga, M.M., & Hinton, J., Abandoned Artisanal Gold Mines in the Brazilian Amazon: A Legacy of Mercury Pollution, Dept of Mining and Mineral Process Engineering, University of British Columbia, Vancouver, BC, VST 1Z4 Canada.
[5] Environmental Protection Agency (US). Mercury study report to Congress. Washington;EPA. Pub.No.: EPA/600/P-97/002Ab.
[6] Zillious, E.J., Porcella, D.B., Benoit, J.M., "Mercury Cycling and Effects in Freshwater Wetland Ecosystems," Environmental Toxicology and Chemistry 12 (1993), pp.2245-2264.
[7] Swain, E., Slide Presentation on Sources of Mercury Deposition in Upper Midwest, Minnesota Pollution Control Agency, Apr 3, 1997.
[8] See http://europa.eu.int/comm/environment/air/background.htm#mercury.
[9] Bolger and Schwetz N. Engl. J. Med., 2002, 347(2): 1735-1736.
[10] Sorenson N, Murata K, Budtz-Jorgenson E, Weihe P, Grandjean P. Prenatal methylmercury exposure as a cardiovascular risk factor at seven years of age. Epidemiol 1999; 10:370-5.
[11] Grandjean et. al., 1997.
[12] See http://europa.eu.int/comm/environment/air/background.htm#mercury.
[13] "Pollutants threaten Artic wildlife, Inuit," SeaWeb Ocean Update, September 1997.
[14] 10 micrograms per gram of hair.
[15] The U.S. local and Brazilian safety limit—500 nanograms/gram wet weight (0.5 microgram per gram).
[16] Salonen et al., 1995.
[17] Raloff, 2001.
[18] Out of 690 samples taken at 72 gold and silver mines across the US, there were 86 cases of overexposure to mercury.
[19] International Dental Journal (1988) 38, 191-192. Recommendations on dental mercury hygiene: Revision of FDI Technical Report No. 7.
[20] Ritchie et al., 2002.
[21] World Health Organization, Environmental Health Criteria 118, Inorganic Mercury, WHO, Geneva, Switzerland,1991.
[22] Agency for Toxic Substances and Disease Registry, US Public Health Service, Toxicological Profile for Mercury. 1999; Kingman A., et al, National Institute of Dental Research, "Mercury concentrations in urine and blood associated with amalgam exposure in the US military population," Dent Res. 77(3);461-71, 1998; National Research Council, Toxicological Effects of Methylmercury, pp.41 and 304-332: Risk Characterization and Public Health Implications, National Academy Press, 2000.
[23] Clarkson et. al., 1988.
[24] NAS/NRC 2000 report, p. 46.
[25] Leistevuo J. Et al, Dental amalgam fillings and the amount of organic mercury in human saliva, Caries Res May-Jun:35(3):163-166, 2001; Sellars, WA, Sellars, R., University of Texas Southwestern Medical School, "Methyl mercury in dental fillings in the human mouth," Journal of Nutritional & Environmental Medicine, 6(1):33-37, 1996.
[26] "Concise International Chemical Assessment Document No. 50: Elemental mercury and inorganic mercury compounds: Human health aspects (www.who.int/pcs/cicad/summaries/cicad_50.html), September 2002, based on the "Toxicological profile for mercury (update) published by the Agency for Toxic Substances and Disease Registry of the US Department of Health and Human Services (ATSDR) in 1999.

[27] There is a difference of one carbon-hydrogen molecule. Institute of Medicine News Conference Oct. 1, 2001; Clarkson, 2002.
[28] "Thimerosal in Vaccines: A Joint Statement of the American Academy of Pediatrics and the Public Health Service," Morbidity and Mortality Weekly Report, July 9, 1999 (/48(26);p563-565)
[29] WHO Database for AMRO (2003), Clarkson, 2002.
[30] Bernard et. al., 2000; VOSI, 2001.
[31] Grandjean et. al., 1998.
[32] 75 micrograms from 3 doses of DTaP, 75 micrograms from 3 doses of Hib, and 37.5 micrograms from three doses of Hep B vaccine. See table of "thimerosal content in some U.S. vaccines " in AAP, 1999, interim report; United States schedule, Tables 1 and 2.
[33] WHO position paper, Weekly Epidemiological Record, No.2, January 14, 2000
[34] ATSDR, 1999.
[35] This assumes that measles, polio, BCG, and yellow fever vaccines do not contain mercury because they are live vaccines.
[36] Schumacher, 1999.
[37] Pless, 1999.
[38] Elemental forms of mercury exposure in homes from consumer products, health and beauty aids, ritualistic, religious and cultural uses are well documented; see RUMTF, USEPA July 2001.
[39] US OSHA-PE regulation.
[40] ATDSR, 1999.
[41] "China and U.S. Geological Survey...Working Together on Environmental Issues." USGS News Release, June 23, 1998.