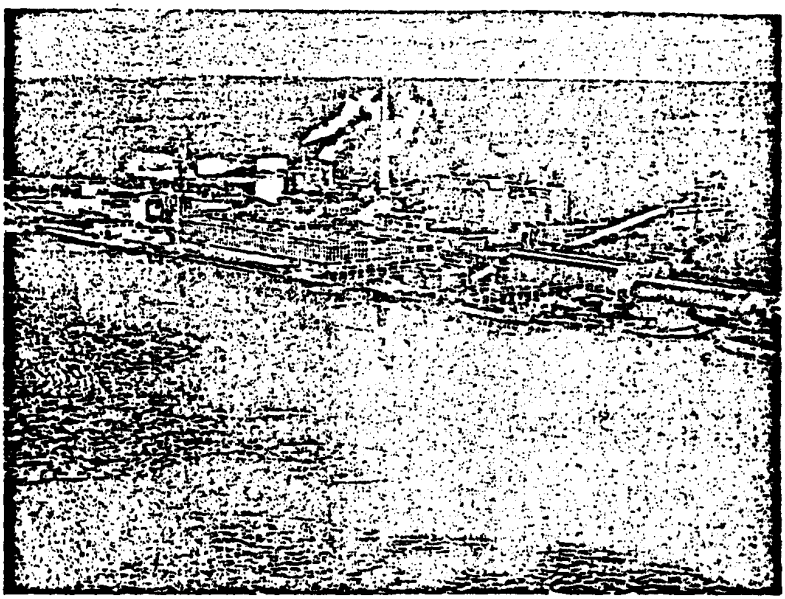
ON THE BRINK OF CATAS



Enso-Gutzeit Oy's Kaukopää Mills is one of the largest forest industry production facilities in Finland.

BY VEIKKO RUOHIO

MONS 003421

ROPHE



Veikko Ruohio is safety manager at Esso-Gutzeit Oy's Kaukopää Mills.

EXPLOSION IN THE CAPACITOR ROOM

A little after midnight the night shift supervisor of the paperboard line heard two successive loud explosions from the mill's electrical switchgear room. He notified the fire brigade and the mill electrician on duty from the line's control room. Soon after this, heavy smoke forced the paperboard line supervisor and the personnel on duty in the adjoining pulp tank room to vacate the control room.

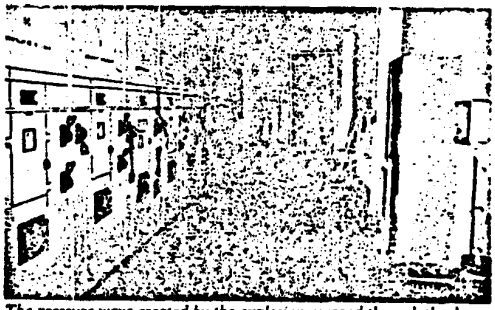
The fire brigade made a swift response, but since no flames could be seen, it took almost half an hour to pinpoint the site of the explosion, a capacitor bank behind a 10 kV switchgear facility.

INITIAL UNCERTAINTY ABOUT THE TOXIC EFFECTS OF PCBS

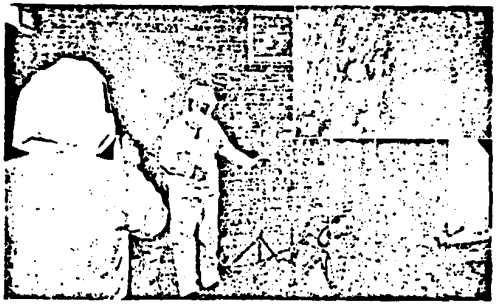
Oil containing PCBs is used in capacitor banks as a dielectric and coolant. In the accident at Kaukopää, the gasified liquid surged with explosive force into the open. A couple of hours after the explosion ten people were taken to hospital. They were all suffering from the same symptoms: nausea, heavy perspiration and headache. By this time it was clear that the gasified liquid contained PCBs, but



The oil containing PCB which leaked onto the floor from the damaged capacitor banks was cleaned up using plastic buckets and absorbent.



The pressure wave created by the explosion escaped through the door from the capacitor facility seen in the background into the 10 kV switchgear room seen in the foreground. The areas from which samples were taken can be seen in the soot covering the floor.



Airborne PCB concentrations were measured almost constantly during the first few days. The picture shows a measuring team looking for a suitable location for one of the suction pumps used in the sampling. The PCB and furan samples from surfaces were collected from areas measuring 10 cm x 10 cm using cotton wadding dipped in acetone or alcohol.

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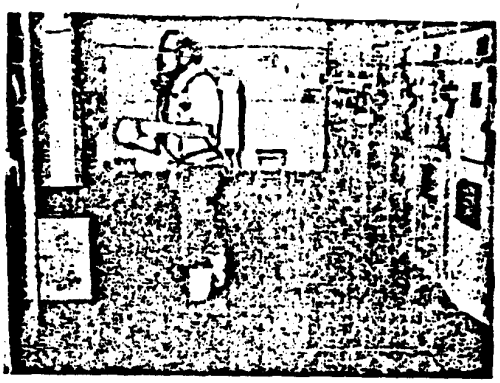
no one on the site was familiar with the effects of exposure to PCBs or with its treatment. Initial information about the potential effects of exposure to the gas was found in the library of the mill's doctor.

As the night when the explosion occurred was warm, those exposed to the reaction products of PCBs waited for treatment on the hospital grounds. Their symptoms disappeared in the fresh air.

The first symptoms may have been caused by exposure to chlorine and hydrochloric acid, which had also been produced in the decomposition of PCBs.

As soon as the first symptoms were reported, the fire brigade closed off the contaminated area with warning signs. Because of the initial uncertainty about the effects of exposure to PCBs, several mill employees moved about in the contaminated area without proper personal protection. The total number of people who had been in the contaminated area and who therefore had at least a theoretical chance of slight exposure was estimated at 150, among them industrial hygienists and cleaning personnel. All these people were later interviewed. Their movements and lengths of stay in the contaminated area were noted and samples of their blood were taken. Follow-up studies have indicated that the exposure to PCBs and its reaction products was at a low level, very likely thanks to the speedy action taken immediately after the incident.



The importance of skin protection was not fully understood immediately after the accident.



Scaffolding and plastic sheet was used to create a makeshift shelter for donning protective wear and equipment.

INFORMATION STARTS TRICKLING IN

The first outside expert to reach the site of the explosion was an industrial hygienist from the Lappeenranta Regional Institute of Occupational Health. He initiated gas concentration sampling during the early morning hours. The results, which indicated that the level of PCB concentrations in the form of gas in the contaminated area was sufficient to require personal protection, were received on the afternoon of the day after the explosion. Two chemical safety experts from Industrial Insurance arrived on the morning following the explosion. They brought with them literature on PCBs acquired from abroad and on the hazards of its decomposed elements. On the afternoon of the same day samples of soot were flown to the Chemical Instrument Center of The University of Helsinki for analysis. The preliminary results indicating the presence of dibenzofurans were received on the evening of the same day. The capacitor manufacturer arrived at the mill at about the same time, bringing with him information on the construction of the capacitor banks and on the amounts of PCBs used in them.

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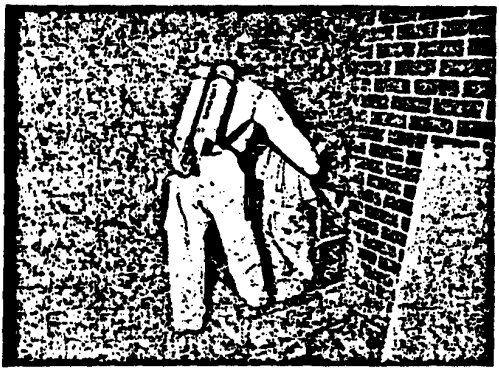
CLEAN-UP AND MEASUREMENTS HAND IN HAND

A large number of samples were analyzed to establish the size of the contaminated area and the concentrations of haz-



Change of clean-up shifts. The two "spacemen" on the right-hand side have just returned from a one-hour clean-up shift while the two men on the left are getting ready for their turn on duty.



Protective clothing and equipment were used in areas which had been cleaned up until sufficiently low concentrations were registered.

ardous substances. The Institute of Occupational Health undertook to measure the PCB concentrations, and the Chemical Instrument Center of the University of Helsinki the furan concentrations. The extent of the measurements performed is shown by the fact that the number of PCB concentrations analyzed amounted to twenty and the number of individual samples taken went far beyond 200.

The clean-up operations, which were started without delay, were performed by an outside company. The immediate problem was to train the clean-up personnel. The importance of skin protection and the use of self-contained breathing apparatus were emphasized.

The clean-up work focused first on the area where the pulp tanks were located because they are of crucial importance to the operation of the board machines. The operation of this department requires the continual presence of personnel, whereas occasional work on electrical installations can well be done wearing protective clothing and using protective devices.

The pulp tank department, including the control room, underwent thorough cleaning, which consisted of alternating vacuum cleaning and washing. In less than a week, the concentrations in that area had fallen below hazardous levels, and the department was out of danger.

Measurements indicated that vacuuming, water blasting and wiping with alcohol were sufficient for areas with low concentrations and for painted metal surfaces. The PCBs adhered so strongly to concrete floors in areas which contained electrical installations and to plastered and brick walls that the only solution was to resurface them with epoxy paint. During the first few days there was uncertainty about the level of PCB concentrations that should be reached in the clean-up, as no official maximum allowable concentration of PCBs in air or on surfaces had yet been established in Finland or elsewhere. The maximum allowable concentration of airborne PCBs without protection, issued three days after the incident by the local labor protection district, was 10 $\mu g/m^3$. The maximum allowable concentrations of PCBs on surfaces, which were obtained from the local labor protection district one month later, were 100 $\mu g/m^2$ for PCBs, 100 ng/m^2 for furans as a whole and 10 ng/m^2 for 2, 3, 7, 8 tetrachlorodibenzofuran. No instructions have been issued as to how samples from surfaces are to be taken, an omission which has prompted some debate.

WHAT REALLY HAPPENED?

Capacitor bank explosions do not occur everyday. Fuse burns in elements inside capacitors are not rare, but an explosion which destroys an entire capacitor bank, requires the presence of several simultaneous factors. At Kaukopää these factors may have included increased use of thyristors with the acquisition of a new paper machine, exceptional distribution in the electrical network and a long period

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of warm weather. The combined effect of these factors could have set off a number of element fuse burns. The unbalance current protection device was defective, and as a result, one capacitor unit blew up. Before the overcurrent relay was able to disconnect the supply of electricity to the defective capacitor bank, voltage had increased considerably as a result of harmonics. This caused a chain reaction that destroyed yet other capacitor units.

The capacitor bank consists of 96 capacitor units. Two of them were destroyed by the electric arc, another two units were destroyed mechanically, and a further 11 units were damaged by pressure. The overcurrent relays cut off the supply of electricity to the capacitor bank two seconds after the electric arc.

The incident immediately stopped the operation of three board machines, one paper machine, an external pulper station and some machinery used in the conversion of paperboard. Because of the contamination in the area housing the pulp tanks, pulp production had to be halted the day after the accident. This led to considerable losses in production.

Two board machines were back in operation within three days thanks to a by-pass arrangement. Other affected production machinery was operating at full capacity within seven days of the explosion.

NATIONWIDE CONCERN

As soon as investigations had indicated that a risk of similar accidents exists wherever capacitors and transformers containing PCBs are used, the matter became a topic of national concern. A quick survey showed that there are nearly 100,000 PCB-filled capacitors and 250 PCB-filled transformers in Finland.

At the moment, the situation is under control. An ample supply of guidance material has been issued by both insurance companies and manufacturers. Public discussion in the aftermath of the accident has been extensive, and the participants have included numbers of private organizations and public bodies. The media have spared no effort in covering issues involving PCBs. If there are any people still working in electrical engineering, fire protection, or industrial safety today who are unaware of the discussion on PCBs, they must be deaf and blind.

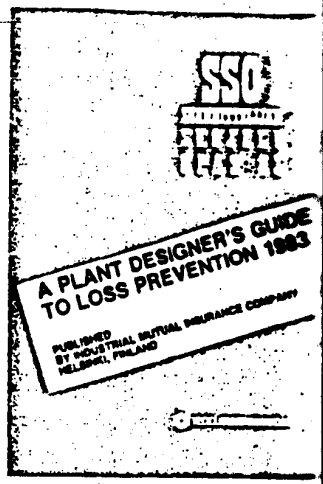
Regardless of the intensity of the discussion, the approach to the issue has been basically sound. Various committees have investigated risks involved in the use of equipment containing PCBs and the reduction of such risks. The Labor Protection Fund has received grant applications which contain proposals for developing ways to measure PCB concentrations, the concentrations of its reaction products, and investigation of their effects. In summary, one can say that the warm August night shook the nation.

A Guide to Loss Prevention

The scope of industrial safety in Finland has expanded at a fast rate. Consequently, the number of laws, statutes, regulations and instructions, and the volume of research have grown steadily. As a result, even safety professionals have found it hard to keep up with the ever-increasing wealth of reference material.

The situation has been particularly difficult for plant designers who are expected to design safe plants, machinery, and equipment but who seldom know where to find the relevant information.

Years ago, the participants at a safety meeting sponsored by Industrial Insurance expressed the wish that Industrial Insurance compile a list of laws, statutes, regulations, and instructions pertaining to industrial safety. The decision was taken to pool efforts and resources and, under the leadership of Industrial Insurance, to compile a guidebook containing the principal sources of information on industrial safety and fire protection. Two working groups were set up for the task: one for industrial safety, the other for fire protection.



PROTECTION CANNOT BE REDUCED

Perhaps the most difficult task to be faced in accidents of this type is how to motivate staff to use protective wear and equipment. This problem is enhanced when the substance against which protection is required cannot be seen, smelled, or felt.

At Kaukopää, familiarity with protection required in the use of chlorine bleaching turned out to be helpful. Because of the risk of accidental chlorine emissions, the mill has to maintain a sufficient stock of protective wear, self-contained breathing apparatus and gas masks. Employees must also be trained to wear protective clothing on the job. The outside clean-up personnel had to be given this training, and they required frequent forceful reminding about the importance of personal pro-

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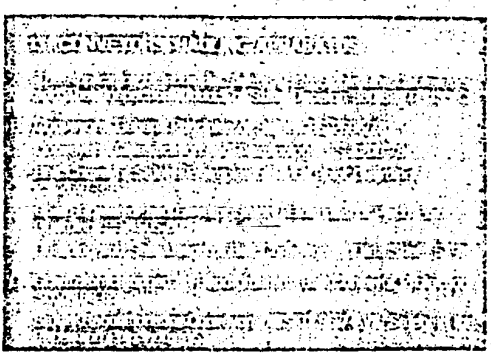
Since the first edition of the Guide in 1977, two supplements have been issued, in 1978 and 1980. It was initially agreed that a revised edition should be published five years later, in 1982. The bulk of the practical revision was in the hands of loss prevention experts from Industrial Insurance with the help of a supporting organization consisting of active safety and fire protection professionals representing Industrial Insurance's policyholders.

A Plant Designer's Guide to Loss Prevention is a source book, a bibliographic work of reference listing relevant items of legislation, statutes, government decisions, regulations and instructions. Loss prevention is divided into four subfields:
- industrial safety
- fire protection
- environmental protection
- civil defense.

The same major headings are used in the industrial safety and fire protection sections. Letter symbols (P = Fire Protection, T = Industrial Safety) keep information in the two subfields distinct from each other.

Information on each subject is subdivided into three sections: section A lists laws, statutes, government decisions, regulations, and instructions; section B lists Finnish Standards, data sheets issued by insurance companies, and other similar items; section C lists lectures, articles in journals, short reports, and studies published outside Finland (e.g. NSC Data Sheets).

Although the compilation aims at practicality and simplicity, as is reflected in the choice of entries, the formulation of each entry, and the disposition of the Guide, Industrial Insurance recommends a short introductory meeting with the users of the Guide. The charge to policyholders for the Guide covers only the printing costs. The Guide is available to non-policyholder enterprises, organizations, and individuals at a slightly higher price.



A sample page from the Guide.

tection. The main point in protection against PCBs and its reaction products is the protection of skin and the respiratory organs. Great care must also be taken not to spread the toxic substances to non-contaminated areas.

Disposable overalls and hoods, rubber boots, plastic gloves and disposable footwear covers turned out to be handy basic protective wear. Disposable overalls were sufficient alone in low concentrations, while higher concentrations required additional protective overalls developed for civil defense use.

The disposable hood gave good protection to hair and neck, and the filter-equipped facial mask could also be worn under it. Immediately after use the disposable overalls, hoods, gloves and footwear covers were placed in special trash bags for hazardous waste. The light protective overalls developed for civil defense use, the self-contained breathing apparatus and the gas masks were washed with the utmost care before reuse. The extent of the Kaukopää incident is also demonstrated by the following statistics on the materials required in the clean-up, measuring, inspection and related work:

Item	Used
respiratory filters	3,300
disposable overalls	4,900
protective hoods	1,200
footwear covers	4,100
disposable gloves	2,800
glass jars (for samples)	600
foam sealant	129 kg
absol absorbent	4,000
civil defense overalls in use at one time (in laundry about 350 pairs)	93
compressed air cylinders (filled)	2,000
paint for resurfacing	2,300 l

Follow-up examinations have shown no cause for alarm. As indicated above, the number of people with possible exposure to PCBs was about 150. As no medical treatment was known for such exposure, medicinal carbon was prescribed. If taken in large amounts, it speeds up the removal of toxic substances from the body. The amount of medicinal carbon taken was considerable, as the daily dose for each exposed person was 50 g for one week.

In addition to the ten people exposed to PCBs and taken to the hospital, five other exposed employees showed some similar symptoms of exposure soon after the accident. This group of 15 has been under closer supervision. So far analyses of blood, fat tissue and other samples have not revealed anything alarming. Follow-up studies of this group are still in progress.

MONS 003426

P.J. Landrigan

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CHAPTER 9B1

Yusho

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9B1.1. Introduction

'Yusho' is a mass food poisoning caused by ingestion of a commercial brand of rice oil contaminated with polychlorinated biphenyls (PCBs), polychlorinated dibenzofurans (PCDFs) and polychlorinated quaterphenyls (PCQs), as revealed by the latest investigations (Tsukamoto et al., 1969; Nagayama et al., 1975; Miyata et al., 1978; Masuda and Kuratsune, 1979; Yamaryo et al., 1979). The poisoning occurred in Western Japan in 1968, involving more than 1,600 people, the majority of whom were residents of Fukuoka and Nagasaki prefectures (Ibayashi, 1979).

9B1.2. Results of epidemiologic and related studies

9B1.2.1. Descriptive epidemiologic studies

Early in October 1968, a newspaper announced an epidemic of a strange disease characterized by severe acne-like eruptions in Fukuoka prefecture, suggesting the ingestion of a brand of rice oil produced by K company (abbreviated as K rice oil) as its possible cause. We soon started an extensive epidemiologic investigation in order to clarify the cause of the disease which was diagnosed as chloracne by dermatologists (Kuratsune et al., 1969, 1972; Goto and Higuchi, 1969). Early subjective symptoms experienced by Yusho patients are listed in Table 9B1.1.

Examination of 325 early patients soon demonstrated very unique characteristics of the distribution of the disease, that is, their distinct familial aggregation and their marked temporal clustering that nearly all of them had been affected between February and October 1968, with a distinguished peak of occurrence in summer

Kimbrough (ed.) Halogenated biphenyls, terphenyls, naphthalenes, dibenzodioxins and related products
© *Elsevier/North-Holland Biomedical Press 1980*

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TABLE 9B1.1
Frequency of early symptoms complained of by Yusho patients (Kuratsune et al., 1969, 1972)

Symptoms	Percent	
	Males (n = 89)	Females (n = 100)
Dark-brown nail pigmentation	83.1	75.0
Distinctive hair follicles	64.0	56.0
Increased sweating at the palms	50.6	55.0
Acneform skin eruptions	87.6	82.0
Red plaques on the limbs	20.2	16.0
Itching	42.7	52.0
Pigmentation of the skin	75.3	72.0
Swelling of the limbs	20.2	41.0
Stiffened soles of the feet and palms of the hands	24.7	29.0
Pigmented mucous membrane	56.2	47.0
Increased eye discharge	88.8	83.0
Hyperemia of the conjunctiva	70.8	71.0
Transient visual disturbance	56.2	55.0
Jaundice	11.2	11.0
Swelling of the upper eyelids	71.9	74.0
Feeling of weakness	58.4	52.0
Numbness of the limbs	32.6	39.0
Fever	16.9	19.0
Hearing difficulties	18.0	19.0
Spasms of the limbs	7.9	8.0
Headaches	30.3	39.0
Vomiting	23.6	28.0
Diarrhea	19.1	17.0

followed by its spontaneous sharp decline. Examination of the geographical distribution of the patients, however, did not indicate any common socioeconomic or environmental features which might be associated with the disease. Both sexes had been equally affected. Age- and sex-specific incidence rates indicated that all age groups were affected, although a considerably lower risk was apparent for both males and females over 60 years of age.

9B1.2.2. Analytical epidemiologic studies

In order to examine the suspicion that K rice oil might be involved in the disease, a thorough investigation was undertaken with regard to the cooking oil that the patients had used. It was discovered that all the patients had used K rice oil either in canned form or in bottled form, and regardless of where they lived, 166 of 170 patients who had used only canned K rice oil had used a very specific one, viz. that produced or shipped by the company on February 5 and 6, 1968. Investigation also revealed that for 143 of 155 patients who had used bottled K rice oil only, their use of the bottled K rice oil shipped between February 5 and 15, 1968 was highly probable. Furthermore, an attack rate as high as 63.9% was calculated for those who

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had consumed the above specific canned rice oil. It was also shown that 113 persons from 29 households who had regularly used K rice oil, but were not considered to have used the above specifically dated ones, were free of the disease.

In a case-control study, 121 pairs of patients and their randomly selected healthy controls matched individually by age, sex and place of residence to the patients were asked 60 questions concerning their occupation, medical background, health status, habits, custom, diet and other characteristics of their daily lives. Only one of these factors tested, namely the habit of "eating fried foods or tempura almost every day", was significantly commoner among the cases than among the controls. In an additional case-control study, cooking oils used were examined from 68 households with Yusho patients and from 207 control households without such patients being matched by place of residence to each of the case households. A highly significant difference was noted between the two groups only in regard to their regular use of rice oil, as shown in Table 9B1.2.

Thus, all these epidemiological approaches unanimously indicated that Yusho was probably caused by ingestion of the specific K rice oil.

9B1.2.3. Toxic agents in K rice oil

After an investigation for about 2 weeks, the Chemical Study Group on Yusho found that the canned K rice oil produced or shipped on February 5, 1968 and used by some of the patients had been contaminated with a large amount of Kanechlor 400, a brand of PCBs of chlorine content 48%, produced by Kanegafuchi Chemical Industry Company (Tsukamoto et al., 1969). Since no analytical methods specific for PCBs were available at that time, the concentration of Kanechlor 400 in the oil was estimated from its organic chlorine content to be 2,000–3,000 ppm. It was also demonstrated that the oil did not contain any significant amounts of Cu, Ni, Zn, Co, As, Hg, chlorinated naphthalenes, or pentachlorophenol. This contamination of the rice oil with PCBs was limited to the products from February 5, 1968, to the middle of the same month, with the highest contamination on February 5 and 6

TABLE 9B1.2
Results of case–control study on fat and oils used.

Fat or oil	Cases[a]		Controls[b]	
	No. of households	(%)	No. of households	(%)
Butter	35	50.7	105	50.7
Margarine	44	63.8	127	61.4
Sesame oil	21	30.5	85	41.1
Rapeseed oil	10	14.5	77	37.2
Rice bran oil	66	95.7	64	30.9
Lard	12	17.4	38	18.4
Other oils	13	18.8	117	56.5

a. 69 patient households.
b. 207 non-patient households.

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(Tsukamoto et al., 1969; Kuratsune et al., 1972). Components of Kanechlor 400 were also readily found in various tissues of patients. Thus, the results of the chemical analyses were completely consistent with the epidemiological findings.

Kanechlor 400 had been used by K company for heating the rice oil over 200°C at a reduced pressure of 3–4 mmHg in a metal container in order to remove odorous matters from it at the final process of the production. It is generally believed that Kanechlor must have leaked from the heating pipe and contaminated the processed oil, but the real mechanism of the contamination does not seem to have been solved yet.

Three samples of the canned K rice oil used by the patients were recently reanalyzed by us for PCBs by using the government standard analytical method for PCBs, finding approximately 1,000 ppm PCBs in each of them (Nagayama et al., 1975, 1976). This concentration was much lower than was originally estimated from the content of organic chlorine. There must be a fairly large amount of chlorine-containing compounds other than PCBs in the oil. Thus, we analyzed the above samples of the oil for PCDFs and polychlorinated dibenzodioxins.

As shown in Table 9B1.3, the oil contained on average 5 ppm of PCDFs, about 250 times the concentration (0.020 ppm) expected from the concentration of PCDFs in the unused Kanechlor 400 (Nagayama et al., 1975, 1976). This finding was also confirmed by Miyata et al. (1977). This marked increase of PCDFs in the rice oil relative to its PCBs content could be ascribed to the fact that the oil had been contaminated with Kanechlor 400 heated at higher than 200°C to be used as the heat-transferring agent for a prolonged period of time, since PCBs could be converted to PCDFs by heating (Miyata and Kashimoto, 1978). The deodorizing process to which the rice oil was subjected, as already mentioned, is also probably connected with this relative increase of PCDFs.

According to Rappe et al. (1979), the Yusho oil contains more than 40 PCDF isomers, including as their major components highly toxic 2,3,7,8-tetra- and 2,3,4,7,8-pentachlorodibenzofurans. In view of the well known high toxicity of these PCDFs and their strong inducing activity of drug-metabolizing enzymes, the

TABLE 9B1.3
Concentrations of PCBs and PCDFs and their ratios in various materials.

Materials	PCBs (ppm[b])	PCDFs (ppm[b])	PCBs/PCDFs
Kanechlor-400 (unused)	1,000,000	ca. 20	50,000
Yusho oils[a]	ca. 1,000	5	200
Yusho patients[c]			
Adipose tissue	1.3	0.009	144
Liver	0.05	0.013	4

a. Produced on February 5 or 6, 1968.
b. On whole base.
c. Two patients who died in 1969 and one patient who died in 1972 were included.

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above fact seems to be important. Contrary to PCDFs, no polychlorinated dibenzodioxins were detected in the Yusho oil.

Although the presence of PCDFs cannot be overlooked, they cannot represent the whole of the unidentified organo-chlorine compounds in the oil, because their concentration is small. There must be still other organo-chlorine compounds in the oil. Miyata et al. (1978) and Kamps et al. (1978) found that the toxic rice oil produced or shipped on February 5 and 6, 1968, contained PCQs. Their concentration was as high as that of PCBs, that is, 866 ppm according to the former authors. Thus, it became clear that the major components of the unidentified organo-chlorine compounds in the oil were PCQs. Although the toxicity of PCQs is not clear yet, the possible significance of the above facts should not be dismissed.

9B1.2.4. Consumption of the oil by patients and dose–response relationship

Selecting 146 patients who were known to have used the specific canned K rice oil of February 5 and 6, 1968, we made a rough estimate of their consumption of the oil, disregarding age, sex, amount of food intake and possible losses of oil during and after cooking (Yoshimura, 1971). The average personal oil consumption was calculated to be about 800 ml which should have contained about 2 g of PCBs if the oil was assumed to have contained 2,000–3,000 ppm of Kanechlor 400 according to the original determinations. Furthermore, a dose–response as well as a dose–effect relationship were clearly seen between the intake of the toxic rice oil and the incidence of the poisoning and between the intake and the clinical severity of the poisoning.

As already mentioned, it was later shown that the PCB concentration of the oil was much lower than was originally estimated and the oil contained toxic agents other than PCBs in a significant amount. Consequently, our original data were reanalyzed, using somewhat different assumptions from those used in the former analysis, and taking the age, sex and food intake of the patients into consideration (Hayabuchi et al., 1979). The total amounts of PCBs, PCDFs and PCQs taken by the patients were, on the average, 633, 3.4 and 596 mg, respectively, or 157, 0.9 and 148 μg/kg/day, respectively. The length of latent period of the poisoning was on average 71 days, with a range from 20 to 190 days. The amounts of PCBs, PCDFs and PCQs taken during the latent period were calculated to be, on average, 466, 2.5 and 439 mg, respectively. The smallest amounts of PCBs, PCDFs and PCQs taken by a patient during the latent period were estimated to be 111, 0.6 and 105 mg, respectively, and the smallest doses 29, 0.16 and 27 μg/kg/day, respectively.

A highly significant negative correlation was seen between the latent period and the amount of oil consumed per kg per day. Another notable fact was that the clinical severity of the patients showed a close positive correlation with the total amounts of the oil consumed but not with the amount of oil consumed per kg per day in adult males and females aged 20 to 44 years (Hayabuchi et al., 1979). This observation seems important because it may indicate a general rule that exposure to a toxic agent with a strong persistency in the body is better represented by the total dose taken than by the dose per kg per day as commonly practised.

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9B1.2.5. Deaths among Yusho patients

According to Urabe (1974), a total of 22 deaths occurred among 1,200 Yusho patients up to September 13, 1973. Malignant tumors accounted for 41% of the deaths. Among these deaths from malignant neoplasms, two were from liver cancer. One of these cases also had a stomach cancer. The deaths from liver cancer occurred within 3 years after exposure. In 1977, an additional autopsied case of liver cancer was reported. At present, it has not been established what the mortality rate of cancer among Yusho patients is. No reliable analysis of deaths has been made by following the patients' illness for an adequately long period of time.

9B1.3. Clinical features of Yusho

9B1.3.1. PCBs, PCDFs and PCQs in tissues of Yusho patients

As shown in Table 9B1.4 the PCB concentrations in the adipose tissues of 3 Yusho patients who were examined soon after they had become ill were very high as compared to controls. Most of these PCBs retained in the tissues were eliminated relatively rapidly. The concentrations of PCBs in the adipose tissues of 3 other patients who died one year later were much lower than those of the above cases. The

TABLE 9B1.4
Concentration of PCBs in tissues of patients with Yusho or other diseases.

Case	Sex	Age	Time of death or surgical operation	PCB concentration (ppm)[b]			
				Adipose tissue		Liver	
				Whole base	Fat base	Whole base	Fat base
1	F	still-born	Oct. 1968	0.02	0.1	0.07	
2	M	high-school student	Nov. 1968	76 (face) 13 (abdomen)			
3,4	M,F	adult	Nov. 1968	32,46 (cheese-like substance from acneform (eruptions)			
5	M	17	July 1969	1.3	3.7	0.14	9.5
6	M	25	July 1969	2.8	15.1	0.2	10.4
7	M	73	Nov. 1969	3.8	8.4	0.07	3.1
8	F	48	Dec. 1970	0.7	0.9	0.07	1.3
9	M	46	May 1972	4.3	6.5	0.08	8.4
10	F	33	Sept. 1972	1.9	2.9		
11	M	72	Apr. 1975	0.2	0.4	0.04	3.0
12	M	59	Mar. 1977	1.2	1.4	0.006	
13	M	69	Sept. 1977	0.8	1.1	0.02	
Controls[a]	M,F	0–61	1973	av. 1.0	av. 2.3	av. 0.05	

a. 31 patients with diseases other than Yusho.
b. Calculated as Kanechlor-500 + Kanechlor-600 (1:1).

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es.

	Liver	
Fat base	Whole base	Fat base
0.1	C.0:	

3.7	0.14	9.5
15.1	0.2	10.4
8.4	0.07	3.1
0.9	0.07	1.3
6.5	0.08	8.4
2.9		
0.4	0.04	3.0
1.4	0.006	
1.1	0.02	
:v. 2.3	av. 0.05	

concentrations of PCBs in subsequently deceased patients were also low, hardly different from those of the controls. A similar trend of PCB concentration was also noted for the liver.

Table 9B1.5 shows the concentrations of PCBs in the blood of the Yusho patients, normal persons, and workers of a chemical industry (Masuda et al., 1974b; Hasegawa et al., 1972; Okumura et al., 1979). It is clear that the PCB levels of the patients were still higher than those of normal persons more than 5 years after the poisoning but not remarkably so. This is more evident if these figures are compared with those of workers occupationally exposed to PCBs. It is interesting to note that most of these Yusho patients were still suffering from dermal signs and various symptoms peculiar to Yusho, while the majority of the workers were free of such symptoms in spite of their extremely high blood PCB levels. These facts indicate that various signs and symptoms complained of by Yusho patients can hardly be explained by the current levels of PCBs in their bodies alone.

Quantitatively speaking, the PCBs retained in the bodies of the Yusho patients are now close to those in normal persons, as already mentioned. We noticed in 1973, however, that they differed qualitatively in a great deal. Fig. 9B1.1 shows the peculiar gas-chromatographic pattern of the PCBs retained in the tissues and blood of the patients. When 41 patients with Yusho visiting our university hospital were examined, 24 (58.5%) of them showed the so-called type A gas-chromatographic pattern which was characteristic of severe cases of Yusho, while 15 (36.6%) showed the type B pattern, which is an intermediate one between the type A and the normal one, and only 2 (4.9%) showed the normal pattern commonly seen in the general population (Masuda et al., 1974a). These peculiar gas-chromatographic patterns of PCBs persist and can still be seen in most of the Yusho patients even now, greatly facilitating the differential diagnosis of Yusho. Table 9B1.6 shows that the composition of PCB isomers present in the blood of Yusho patients is quite different from

TABLE 9B1.5
PCBs in the blood of patients with Yusho and others.

Subjects	No. of subjects	Year	PCBs (ppb)[a]	
			Mean ± S.D	Range
Yusho patients	41	1973	7	2–26[d]
	116	1974	7.0 ± 5.4[c]	1–31[c]
	79	1976	6.2 ± 4.7	1–32[c]
	92	1977	5.3 ± 3.7	1–21[c]
Normal persons	37	1972	3	1–7[d]
Workers[b]	23	1972	364 ± 262	60–920

a. Whole base.
b. Workers engaged in the production of Kanechlor-200–600 inhaling the air containing 0.05–0.2 mg/m³ of PCBs (Kanechlor-300 + Kanechlor-400 (1:1)). Two of them showed dermal signs (Hasegawa et al., 1972).
c. Okumura et al. (1979).
d. Masuda et al. (1974b).

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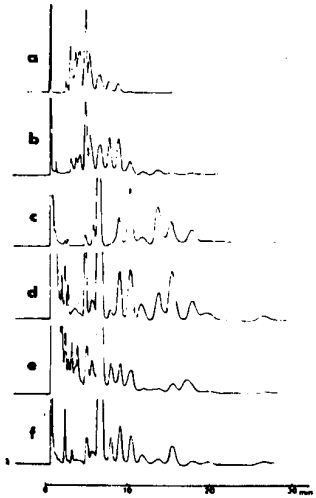

Fig. 9B1.1. Gas-chromatograms of PCBs in various specimens. a, unused Kanechlor-400; b, PCBs in the toxic K rice oil; c, PCBs from the blood of a Yusho patient, pattern A; d, PCBs from the blood of a Yusho patient, pattern B; e, PCBs from the blood of a Yusho patient, pattern C; f, PCBs from the blood of a normal person.

TABLE 9B1.6
Concentrations of PCB isomers in the blood of Yusho patients and normal persons.

PCB isomers	Concentration in blood (ppb)[a]	
	Yusho patients (n = 9)	Normal persons (n = 7)
2,4,5,3',4'-Pentachloro-	0.13 ± 0.05	0.22 ± 0.09
2,3,4,3',4'-Pentachloro-	n.d.[b]	0.05 ± 0.02
2,4,5,2',4',5'-Hexachloro-	0.52 ± 0.22	0.15 ± 0.07
2,3,4,2',4',5'-Hexachloro-	0.65 ± 0.21	0.17 ± 0.07
2,3,4,5,3',4'-Hexachloro-	0.28 ± 0.07	0.04 ± 0.02
2,3,4,5,2',4',5'-Heptachloro-	0.44 ± 0.15	0.12 ± 0.07
2,3,4,5,2',3',4'-Heptachloro-	0.28 ± 0.09	0.11 ± 0.04

a. Mean ± Standard deviation.
b. n.d. = less than 0.01 ppb.

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normal persons. It may be noteworthy that 2,3,4,5,3',4'-hexachlorobiphenyl, which is known to have a strong inducing activity of drug-metabolizing enzymes, is present in the patients' blood at a much higher level than in the blood of normal persons (Kuroki and Masuda, 1977).

A significant positive association was noted between the gas-chromatographic patterns or concentrations of PCBs in the blood of Yusho patients and their serum triglyceride concentrations, severity of dermal lesions, ocular signs, pigmentation of the oral mucosa, respiratory symptoms, and subjective symptoms such as headache and paresthesia of the limbs (Okumura et al., 1974; Kōda and Masuda, 1975; Ohnishi and Yoshimura, 1977; Fukuyama et al., 1977; Shigematsu et al., 1977; Iwashita et al., 1977).

As shown in Table 9B1.3 the adipose tissue and the liver of Yusho patients who died in 1969 or 1972 contained on average 0.009 ppm and 0.013 ppm of PCDFs, respectively. According to Miyata et al. (1977), tissue samples of the omentum and the liver of normal subjects contained at most 45 and 17 ppt of PCDFs, respectively. It is clear, therefore, that Yusho patients contained a far larger amount of PCDFs in the tissues than do normal persons. For PCB concentrations, however, there were no such great differences at all. It is also very interesting that in Yusho patients the PCDF concentrations were very close to those of PCBs in the liver and much closer than they were in the adipose tissue. This relative abundance of PCDFs in the liver was also noted in normal persons (Miyata et al., 1977). Rappe et al. (1979) analyzed the liver tissues from 2 Yusho patients, finding 9 isomers of PCDFs with 4–6 chlorine atoms. Most of the PCDFs present in the Yusho oil were not detected in the liver, but highly toxic 2,3,7,8-tetrachloro- and 2,3,4,7,8-pentachlorodibenzofurans that were the major PCDFs in the Yusho oil were also identified in the liver as its major components of PCDFs, together with 1,2,3,4,7,8-hexachlorodibenzofuran. These researchers noted that none of the isomers retained in the liver had two vicinal hydrogenated C atoms in either of the two carbon rings of the dibenzofuran system. Position of hydrogenated C atoms seems to be closely associated with the retention of PCDFs.

Recently, we were informed that Drs. H. Miyata and T. Kashimoto at the Osaka Prefectural Institute of Public Health succeeded in determination of PCQs in the blood and other tissues of Yusho patients at a similar level to that of PCBs. This suggests an urgent necessity to investigate the toxicity of these compounds.

Taking all the above findings into account, we have to realize the complex nature of Yusho which will not be explained by ingestion of PCBs alone. The fact that a considerable number of patients have suffered from severe clinical manifestations without quick improvements for a prolonged period of time even after their PCB levels in the blood and tissues had decreased nearly to background levels may be associated with the highly persistent toxic compounds other than PCBs, still retained in the patients' bodies.

9B1.3.2. Clinical symptoms and manifestations

Clinical features of Yusho are well summarized in the diagnostic criteria for Yusho

iused Kanechlor-400; b,
nt, pattern A; d, PCBs
od of a Yusho patient,

rsons.

l (ppb)[a]

Normal persons (n = 7)
0.22 ± 0.09
0.05 ± 0.02
0.15 ± 0.07
0.17 ± 0.07
0.04 ± 0.02
0.12 ± 0.07
0.11 ± 0.04

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TABEL 9B1.7
The diagnostic criteria for Yusho (revised in October, 1972; translated by Kuratsune).

Yusho is considered as an acute or subacute poisoning with PCBs. The general symptoms currently seen are retarded growth, neuroendocrine disturbances, phenomenon of enzyme induction, disorders in the respiratory system, and disturbed lipid metabolism. As the local symptoms, acneform eruptions and pigmentation as cutaneomucosal lesions and ocular symptoms are seen.

1. Conditions of attack
   Fact of ingestion of Kanemi rice oil contaminated with PCBs and familial occurrence seen in most cases.

2. General symptoms
   a. Subjective symptoms
      1. general fatigue
      2. heavy headedness and headache
      3. inconstant abdominal pain
      4. feeling of numbness and pain at the limbs
      5. swelling and pain at the joints
      6. cough and sputum
      7. changes in menstruation
   b. Objective signs
      1. bronchitis-like signs
      2. sensory neuropathy
      3. bursitis
      4. inhibition in growth and abnormal teeth in children
      5. Small-For-Dates baby and pigmentation of the entire skin of newborns
   c. Results from clinical examination
      1. unusual patterns and concentration of PCBs in blood
      2. increase of neutral lipids in blood
      3. anemia, lymphocytosis, hypoalbuminemia
      4. reduced conduction velocity of the sensory nerve and adrenocortical hypofunction

3. Cutaneomucosal signs
   a. Acneform eruptions
      Black comedones and acneform eruptions which are seen at the face, buttocks and other intertriginous sites and their suppurative tendency
   b. Pigmentation
      Pigmentation of the face, palpebral conjunctiva, gingiva, and nails of the fingers and toes
   c. Ocular signs
      Swelling and hypersecretion of the Meibomian gland and palpebral edema

as revised in 1972 (Urabe, 1974) (Table 9B1.7). The criteria were further revised in 1977 but were essentially the same as the former ones (Sugiyama, 1977).

9B1.3.2.1. Dermal signs. The most notable manifestations of Yusho are dermal lesions such as marked enlargement and elevation of the follicular orifice, comedo formation, acneform eruptions, hyperpigmentation and hyperkeratosis (Goto and Higuchi, 1969; Urabe and Koda, 1976; Asahi et al., 1979). The acneform eruptions

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develop in the face, ear lobe, retroauricular region, external ear canal, axillae, trunk and external genitalia. Marked enlargement and elevation of the follicular orifice with hyperkeratosis and formation of keratotic plugs occurs in the axillae, groin, glenoid regions, trunk, thigh, and outer aspect of the forearm. Dark colored pigmentation of the gingivae, lips, oral mucosa and nails is frequently seen among Yusho patients. The above dermal lesions improved fairly well in light cases in a few years after the intoxication but in severe cases there was a strong tendency for cyst formation which prevented their cure for many years.

9B1.3.2.2. Ocular signs. In the acute phase of Yusho, most patients showed severe ocular signs. The most prominent ones were hypersecretion of the meibomian glands and abnormal pigmentation of the conjunctiva. The meibomian glands swelled like cysts and were filled with yellow infarct-like contents. The pigmentation of the conjunctiva was examined by electron microscopy and considered to be due to melanin deposits. No particular lesion was noted in the intraocular tissues (Ikui et al., 1969). The above signs have well subsided during the past 10 years but 84% of the 75 patients examined recently still showed abnormal changes of the meibomian glands, such as their irregular arrangements, atrophy, secretion of cheese-like material, infarction and lithiasis, and 43% showed pigmentation of the conjunctivae and eyelids. Sixty-four percent of the patients also complained of eye discharge (Kohno and Yamana, 1979).

9B1.3.2.3. Neurological signs. Most Yusho patients complained of various neurological symptoms such as headache, numbness at the limbs, hypoesthesia and neuralgic limbs. Electroencephalographic examinations did not show any significant changes which might indicate some central nervous damage (Nagamatsu and Kuroiwa, 1971). However, a reduced sensory nerve conduction velocity of the radial and/or sural nerve was observed in 9 out of 23 cases examined soon after the poisoning, while a reduced motor nerve conduction velocity of the ulnar and tibial nerves was seen only in two cases (Kuroiwa et al., 1969). Thus, it seemed certain that the sensory fibers were preferentially affected in Yusho. These findings were well in accord with the fact that the motor functions were intact in all the cases examined. No follow-up study has been made for the above disturbed function of the sensory nerves by re-examining the conduction velocity, but according to the authors, the lesions seem to have disappeared relatively soon after exposure as indicated by the sequence of related symptoms and manifestations.

9B1.3.2.4. Endocrine signs. One or two years after the onset of the poisoning, laboratory tests for endocrine functions were made for the patients. Rapid ACTH test performed in 86 patients gave no evidence of marked abnormalities of the adrenocortical function (Watanabe et al., 1971). Determination of 17-ketosteroids and 17-hydroxycorticosteroids in urine was made in 95 Yusho patients by Nagai et al. (1971). About 40% of the patients showed an elevated excretion of these

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steroids, as possibly caused by the poisoning. Kusuda (1971) observed irregular menstrual cycles in about 60% of the female patients and abnormal patterns of the basal body temperature (mainly inadequate luteal phase and prolonged low phase) in 85% of them, which suggested the existence of corpus luteum insufficiency and retarded follicular maturation. Urinary excretion of estrogens and pregnanediol was also found to be low in the patients. All the above facts indicate that endocrine disorders were present in some patients although not extremely severe.

9B1.3.2.5. Respiratory signs. Shigematsu et al. (1971) reported that about 40% of the patients complained of persistent cough with sputum production, suffering from chronic bronchitis. Most of such patients were afebrile, with little change in leucocyte count or erythrocyte sedimentation rate, but secondary respiratory infections were often observed. Their serum IgA and IgM, especially the former, were lower. PCBs were proved to be present in the sputa from these patients with respiratory troubles (Kojima, 1971; Shigematsu et al., 1977). The respiratory symptoms seem in general to be improving, but extremely slowly as shown by a follow-up study of 79 cases for about 5 years by Shigematsu et al. (1977) which indicated that 26, 21, 38 and 15% of the cases were free of the symptoms with aggravated, unchanged or improved symptoms in 1972–73, respectively, while the corresponding proportions were 15, 16, 49 and 19% in 1974–75.

9B1.3.2.6. Changes of the liver. Contrary to our expectation, Yusho patients have hardly shown any abnormalities of the liver in gross appearance or in routine function tests such as bilirubinemia, SGOT, SGPT, BSP retention and some others. However, a marked proliferation of the smooth endoplasmic reticulum and a distinct reduction of the rough endoplasmic reticulum were noted by electron-microscopy of liver biopsy specimens from a patient (Hirayama et al., 1969). These phenomena were considered to indicate a stimulated induction of drug-metabolizing enzymes occurring in the liver of Yusho patients, as expected from the observations of animals administered PCBs (Nishihara and Yamamoto, 1972). Alterations of the mitochondria such as morphological heterogenecity, and frequent appearance of giant mitochondria and inclusion bodies which are probably connected with the disturbed metabolism of fatty acids were also observed in the liver (Yamamoto et al., 1971). Thus, morphologically speaking, the liver of Yusho patients cannot be said to be intact, although hardly any notable changes were detected by light microscopy.

9B1.3.2.7. Hyperglyceridemia. In the early stage of the poisoning, markedly elevated serum triglyceride was one of the few abnormal laboratory findings peculiar to the disease. Uzawa et al. (1969) observed serum triglyceride ranging from 200 to 600 mg/dl in 12 out of 24 cases, while the serum total cholesterol remained unchanged and phospholipids tended to be somewhat lowered. This hyperglyceridemia persisted for several years in most of the patients. According to Okumura et

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al. (1979), 24 Yusho patients who have regularly been examined every year during the past ten years have showed their serum triglyceride levels lowered to the normal range, approximately since 1973 in females and since 1975 in males.

9B1.3.2.8. Babies born to patients and to non-affected wives of patients and babies breast-fed by patients. Eleven wives with Yusho and two unaffected wives of patients delivered 11 live and two stillborn babies in 1968, 10 of whom had unusually dark-brown colored skin with a marked increase in melanin pigment in the basal cells of the epidermis (Taki et al., 1969; Yamaguchi et al., 1971; Funatsu et al., 1971). A similar pigmentation of the gingivae and nails and increased discharge of eyes were also noted in most of these cases. A chemical analysis of tissue specimens from one of the stillborn babies showed, however, that the concentration of PCBs in the skin, mesenteric fatty tissue and liver were not high at all, being 0.11, 0.02 and 0.07 ppm on a wet-weight basis, respectively (Table 9B1.4). As the babies grew older, the skin pigmentation gradually faded. Yoshimura (1974) reported a mother with Yusho who delivered 3 babies with dark-brown colored skin within 3 years after exposure. The last baby's skin was only slightly colored. He also reported a particular case of a Yusho baby affected by the ingestion exclusively of the milk from his mother with Yusho.

Yusho is one of the most typical man-made health hazards. Unfortunately, no effective treatments have yet been developed for it, except for the fasting therapy which seems to be fairly effective according to Imamura's observations (Imamura, 1975). Such a drastic treatment, however, requires great caution in application. Not many of the patients can readily be subjected to it. Although a general tendency of spontaneous recovery can be seen in most of the patients, it is often too slow and too vague, leaving a considerable number of patients still far from complete recovery even after more than ten years after the onset of the poisoning. Further efforts on a world-wide basis to accelerate the recovery of these patients are highly desirable.

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DETERMINATION OF POLYCHLORINATED DIBENZOFURANS IN TISSUES OF PATIENTS WITH 'YUSHO'

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Abstract— Tissues of patients with 'Yusho' and of persons not suffering from this disease were analysed for polychlorinated biphenyls (PCBs) and polychlorinated dibenzofurans (PCDFs) by column chromatography, gas chromatography, and gas chromatography–mass spectrometry. Both PCBs and PCDFs were identified in the tissues of patients with Yusho, while only PCBs were detected in the tissues of the other persons studied. In contrast to the PCBs, which were found far more in the adipose tissue than in the liver, PCDFs occurred in very similar concentrations in these two types of tissue. The persisting PCDFs in the tissues of Yusho patients were almost exclusively penta and hexachloro derivatives.

INTRODUCTION

Polychlorinated biphenyls (PCBs) are known to accumulate in the human body as a result of their ingestion in foods, mainly fish. Since polychlorinated dibenzofurans (PCDFs) have been reported to contaminate commercial PCB formulations (Bowes, Mulvihill, DeCamp & Kende, 1975; Bowes, Mulvihill, Simoneit, Burlingame & Risebrough, 1975; Nagayama, Kuratsune & Masuda, 1976; Nagayama, Masuda & Kuratsune, 1975; Roach & Pomerantz, 1974; Vos, Koeman, van der Maas, ten Noever de Brauw & de Vos, 1970) the human body may also be contaminated with PCDFs, which are more toxic than PCBs. PCBs are known to have been the cause of a mass food poisoning which affected more than 1000 persons in western Japan in 1968 (Kuratsune, Yoshimura, Matsuzaka & Yamaguchi, 1972; Tsukamoto *et al.* 1969). The disease is called 'Yusho' i.e. oil disease, because the patients were proved to have consumed a commercial rice oil contaminated with a large amount of Kanechlor-400 (KC-400), a Japanese PCB formulation. Recently, a sample of unused KC-400 and samples of the rice oil consumed by patients with Yusho were found to be contaminated with 20 and 5 ppm PCDF, respectively, indicating that the patients had ingested PCDFs with the PCBs (Nagayama *et al.* 1975 & 1976).

All these findings made it imperative to analyse tissues of unaffected persons and of patients with Yusho for their possible content of PCDFs. This paper describes the main results of such an investigation.

EXPERIMENTAL

Materials analysed. Samples of the liver, mesenteric adipose tissue and a few other organs of three Yusho patients who had died were supplied from the Department of Pathology, School of Medicine, Yamaguchi University, and the Department of Pathology, Faculty of Medicine, Kyushu University. These tissues had been preserved in 4% formaldehyde solution after resection. Cases 1 and 2 had died in 1969, the year after the outbreak of Yusho, while case 3 had died in 1972. From the severity of dermal lesions, case 2 had been identified as grade 4 according to the criteria established by Goto & Higuchi (1969) and case 3 as grade 3, while case 1 was judged to be grade 3 or 4 from an examination of his clinical records. The similarly preserved adipose tissue and livers of two persons without Yusho who died in accidents were also obtained for analysis from the Department of Forensic Medicine, Faculty of Medicine, Kyushu University.

Chemicals. *n*-Hexane, ethanol, potassium hydroxide, anhydrous sodium sulphate and silica gel, all reagent-grade, were purified by the methods described previously (Nagayama *et al.* 1975). Reagent-grade carbon tetrachloride and methylene chloride were fractionally redistilled. Alumina for column chromatography (about 300 mesh) was obtained from Wako Pure Chemical Industry, Ltd., Osaka, and activated by heating at 150°C for 12 hr before every use. PCDF for use as a standard was synthesized by chlorination of dibenzofuran (Nishizumi, Kuratsune & Masuda, 1975) and consisted mainly of tetra- and pentachlorodibenzofurans. 2,3,7,8-Tetrachlorodibenzofuran was kindly donated by Dr. J. G. Vos, National Institute for Public Health, Bilthoven, The Netherlands, and by Dr. I. H. Pomerantz, Food and Drug Administration, Washington, DC, USA. KC-500 was supplied by Kanegafuchi Chemical Industry, Japan.

Analytical procedure. The official standard analytical method for PCB established by the Ministry of Health and Welfare, Tokyo (Kawashiro *et al.* 1972) and the separation method for PCDFs and PCBs (Nagayama *et al.* 1975) were used to fractionate PCBs and PCDFs from tissue samples and to separate the PCBs and PCDFs in the fraction. Samples (about 5–180 g) were first homogenized with *n*-hexane (150–400 ml) and anhydrous sodium sulphate (20 g) in a Waring blender. The *n*-hexane solutions were pooled and evaporated to dryness to yield fatty residues, which were saponified with 1 N-KOH–ethanol

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solution (50 ml). The *n*-hexane extract of the KOH–ethanol solutions was concentrated and then chromatographed on a column of silica gel (2 g) eluted with *n*-hexane (150 ml). The *n*-hexane eluate containing PCBs and PCDFs was further fractionated on a column of activated alumina (3 g) using *n*-hexane–carbon tetrachloride (4:1, v/v; 60 ml) and *n*-hexane–methylene chloride (4:1, v/v; 20 ml) as successive eluants. PCBs were expected to be contained in the former eluate and PCDFs in the latter. These eluates were evaporated, and the residues were redissolved in *n*-hexane and subjected first to gas chromatography (ECD–GC) in a Beckman GC 72-5 fitted with an electron-capture detector and a glass column (2 mm × 2 m) containing Chromosorb W AW-DMCS (100–120 mesh) coated with 2% SE-30 (column temp. 200°C, carrier gas helium, flow rate 30 ml/min) and subsequently to gas chromatography–mass spectrometry (GC–MS) using a type D-100 instrument (Japan Electron Optics Laboratory, Tokyo) and a column similar to that used for ECD–GC, the ionizing energy being 25 eV and the ionizing current 0·3 mA.

PCBs were estimated quantitatively by means of the official standard analytical method already mentioned (Kawashiro *et al.* 1972), while PCDFs were estimated by comparing the heights of the gas-chromatographic peaks with those of synthesized PCDF, the assumption being made that all the PCDF isomers isolated from tissue samples had the same peak height sensitivity regardless of the number and sites of chlorine substitution. This assumption can be justified on two counts. First we had found in quantitative studies of PCDF in Kanechlors and 'Yusho oil' that the use of this assumption gave figures very close to those obtained by the perchlorination method (Nagayama *et al.* 1975 & 1976). The latter method could not be used in the present analyses because the PCDF levels in the tissue samples were much lower than those in Kanechlors or 'Yusho oil'. Secondly, as will become apparent later, the patterns of gas-chromatographic peaks were similar for PCDFs isolated from a variety of tissues taken from the Yusho patients, indicating that a fairly accurate comparison could be made of PCDF levels in various tissue on the basis of this assumption.

RESULTS

Gas chromatograms of PCB and/or PCDF fractions of the liver of a patient (case 1) and of the toxic rice oil, before and after separation on an alumina column, are shown in Fig. 1, chromatograms A, B and C indicating that a good separation of PCB and PCDF was achieved.

Comparison of chromatograms C and D shows that there is a distinct difference in the peak pattern between PCDF obtained from the liver of the patient and that from the toxic rice oil. The former consisted almost exclusively of peaks 4', 6' and 7', which coincided exactly in retention times with peaks 4, 6 and 7, respectively, in chromatogramr D. Peak 3 of chromatogram D had exactly the same retention time as that of 2,3,7,8-tetrachlorodibenzofuran, but was hardly seen in chromatogram C. A similar lack of peaks corresponding to peaks 1, 2 and 5 was also noted in chromatogram C.

Each peak in chromatogram C was examined by GC–MS and the components of peaks 4' and 6' were identified as pentachlorodibenzofurans, and that of peak 7' as hexachlorodibenzofuran. The mass spectrum of peak 6' is shown in Fig. 2, together with the mass spectrum of a synthesized pentachlorodibenzofuran, which showed the same retention time as peak 6'. These two mass spectra are considered to be practically the same. The gas chromatograms of other tissue samples of Yusho patients were almost the same as chromatogram C in Fig. 1.

Table 1 summarizes the results of analyses of various tissues from patients and from accident victims. With the exception of the spleen of case 2, all the tissues taken for analysis from Yusho patients contained PCDFs. The adipose tissues and livers of the accident victims contained PCBs but did not contain any detectable amount of PCDFs (detection limit 0·05 ppb). Table 1 also shows another interesting fact

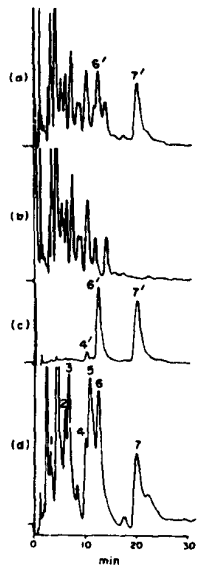

Fig. 1. Gas chromatograms of PCB and/or PCDF fractions obtained from the liver of a patient with Yusho (case 1) and from the toxic rice oil: (a) PCB + PCDF fraction from the liver of a patient with Yusho; (b) PCB fraction separated from A; (c) PCDF fraction separated from A; (d) PCDF fraction from the rice oil. The peaks were identified by GC–MS as (1) $C_{13}H_2OCl_5$; (2–3) $C_{12}H_4OCl_4$; (4–6, 4', 6') $C_{12}H_3OCl_5$; (7, 7') $C_{12}H_2OCl_6$. Column, 2% SE-30; detector, ECD.

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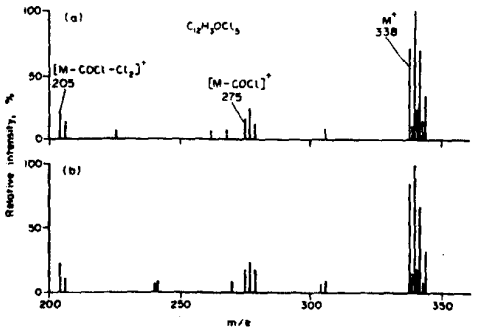

Fig. 2. Mass spectra of (a) gas-chromatographic peak 6' of PCDF fraction from the liver of a patient (case 1) with Yusho and (b) peak 6 of synthesized PCDF.

in that in all three patients concentrations of PCDF in relation to PCB were much higher in the liver than in the adipose tissue. The concentrations of PCDF on whole basis were particularly high in the liver in cases 1 and 2. This pattern of distribution contrasted sharply with that of the PCBs which showed far higher concentrations in the adipose tissue than in the liver. Analysis of the kidneys from cases 2 and 3 showed that PCDF concentrations in this organ were very much lower than those found in the liver.

DISCUSSION

In contrast to the situation with PCBs (Burse, Kimbrough, Villanueva, Jennings, Linder & Sovocool, 1974; Curley, Burse, Grim, Jennings & Linder, 1971; Grant, Phillips & Villeneuve, 1971; Platonow, Liptrap & Geissinger, 1972), the tissue distribution of PCDFs has received little attention. This study seems to be the first to identify PCDFs in the tissues of patients with Yusho and to disclose the peculiarity of their tissue distribution, although no definite conclusions can yet be drawn from our findings because of the limited number of observations made. It is noteworthy, in this connexion, that polychlorinated dibenzo-p-dioxins, which are closely related to PCDFs in chemical structure, also have a definite tendency to accumulate in the livers of animals (Allen, Van Miller & Norback, 1975; Firestone, Flick, Rees & Higginbotham, 1971; Norback, Engblom & Allen, 1975; Van Miller, Marlar & Allen, 1976; Williams, Cunningham & Blanchfield, 1972). As indicated ear-

Table 1. *Concentrations of PCB and PCDF in the tissues of male patients with Yusho and of male accident victims not affected by the disease*

Case no.	Age (yr)	Time of death	Tissue	Conc (ppm) of PCB: In whole tissue	PCB: Calc on tissue fat	PCDF: In whole tissue	PCDF: Calc. on tissue fat	PCDF concn / PCB concn × 100
				Yusho patients				
1	13	July 1969	Adipose	1·4	3·4	0·013	0·03	0·9
			Liver	0·05	4·7	0·025	2·3	50
2	25	July 1969	Adipose	1·3	8·5	0·006	0·04	0·5
			Liver	0·06	5·6	0·010	1·1	17
			Kidney	0·13	6·6	0·0005	0·03	0·4
			Spleen	0·02	8·5	ND	ND	—
3	46	May 1972	Adipose	1·2	2·1	0·007	0·001	0·6
			Liver	0·03	3·5	0·003	0·3	10
			Kidney	0·03	1·4	0·0002	0·007	0·7
			Spleen	0·02	1·9	TR	TR	—
			Lung	0·01	2·2	TR	TR	—
				Accident victims				
4	52	March 1975	Adipose	1·0	1·4	ND	ND	—
			Liver	0·08	1·3	ND	ND	—
5	58	March 1975	Adipose	0·40	0·7	ND	ND	—
			Liver	0·02	1·0	ND	ND	—

TR = 0·00005–0·0001 ppm ND = None detectable (< 0·00005 ppm)

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lier, all the three cases analysed were in the early convalescent stage as far as the Yusho incident was concerned, and the current distribution of residual PCDFs and PCBs in the bodies of survivors more than 8 years after the poisoning may differ considerably from that observed in this study. It is highly desirable that the current distribution pattern should be clarified soon, because it is closely connected with the medical treatment of the surviving patients.

In contrast to cases 1 and 2, fairly accurate information is available concerning the consumption of toxic rice oil by case 3. From an earlier epidemiological study (T. Yoshimura, unpublished data, 1973), case 3 is known to have consumed approximately 560 ml of a canned rice oil containing about 1000 ppm PCB (KC-400) and 5 ppm PCDF over a period of 140 days. Therefore, the total amounts of PCB and PCDF ingested are estimated to have been approximately 560 and 2·8 mg, respectively. On the other hand, it is calculated that the whole liver of patient no. 3, weighing 2070 g at autopsy, contained 62 μg PCB and 6·2 μg PCDF. Thus, 0·22% of the PCDF ingested apparently remained in the liver of this patient when he died in May 1972, approximately 44 months after use of the toxic rice oil had been discontinued. The corresponding figure for PCB was 0·011%, much smaller than that for PCDF. At the present time, it is hard to comment with any certainty on the possible significance of these figures, because adequate information on the intestinal absorption rate and toxicity of PCDF in man and the possible metabolic conversion of PCBs to PCDFs is lacking. It may be said at least, however, that PCDFs deserve more attention in attempts to improve our understanding of Yusho.

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June 24, 1982

Document Control Officer (TS-793)
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Re: Docket Number OPTS-62015B;
Reply Comments on Notice of
Proposed Rulemaking Concerning
Polychlorinated Biphenyls (PCBs);
Use in Electrical Equipment,
47 Federal Register 17,246 (April 22, 1982)

Dear Sir/Madam:

These reply comments are submitted to the Environmental Protection Agency ("EPA") on behalf of the National Electrical Manufacturers Association ("NEMA"), the principal national trade association of the electrical manufacturing industry. NEMA's reply comments address the following topics considered at EPA's informal hearing on the above-proposed rule: (1) the assessment of "insignificant exposure"; (2) the likelihood of exposure to polychlorinated dibenzofurans ("PCDFs") as a result of the continued use of PCB-containing electrical equipment; (3) the electrical manufacturing industry's ability to replace all existing Large High and Large Low Voltage Capacitors containing PCB dielectric fluid ("Large PCB Capacitors") in ten years; (4) the 50 parts per million ("ppm") regulatory cutoff as it applies to the use of PCB-containing electrical equipment; and (5) various additional questions directed by the hearing panel to NEMA.

1. Insignificant Exposure

Section 6(e)(2)(A) of the Toxic Substances Control Act ("TSCA") prohibits PCB uses except in a "totally enclosed

manner." Section 6(e)(2)(C) provides that "'totally enclosed manner' means any manner which will ensure that any exposure of human beings or the environment to a polychlorinated biphenyl will be insignificant as determined by the Administrator by rule." (Emphasis added.) In other words, "totally enclosed manner" must be defined in terms of "insignificant exposure" and it is the Administrator's duty to determine what constitutes an "insignificant exposure" to PCBs.

In both the proposed and final PCB Ban Rules, EPA defined "insignificant exposure" as no exposure, or exposure "not measurable or detectable by any scientifically acceptable analytical method." 40 CFR 761.3(h)(h). This stringent determination was based in large measure on EPA's findings that any PCB exposure may have adverse effects and poses a significant risk to the health of man. These findings, in turn, were fundamentally based on the toxicological and human health effects information developed in the mid-1970's during the rulemaking to establish toxic pollutant effluent standards for PCBs under Section 307(a) of the Federal Water Pollution Control Act.

Since that time, however, much additional scientific information about the human health effects of PCBs has become available, including several reports of studies of heavily exposed (blood PCB levels ranging from 10 parts per billion to 3330 parts per billion) worker populations. Since some of the worker populations studied included employees at facilities owned and operated by NEMA members, NEMA has maintained a keen interest in understanding as much as possible about the human health effects of PCBs. As a party to Environmental Defense Fund, Inc. v. Environmental Protection Agency, 636 F.2d 1267 (D.C. Cir. 1980), NEMA also recognized EPA's obligation to assess the risk to human health of any PCB exposures that may result from the continued use of PCB-containing electrical equipment; i.e., to determine "insignificant exposure" in this rulemaking.

In order to draw EPA's attention to important recent scientific information concerning the human health effects of PCBs, NEMA, together with the Utility Solid Waste Activities Group ("USWAG") of the Edison Electric Institute ("EEI"), contracted with the firm of Drill, Friess, Hayes, Loomis and Shaffer, Inc. ("DFHLS") to conduct a study to examine the toxicological and epidemiological literature on PCBs ("DFHLS study"). Both NEMA and EEI/USWAG have submitted the DFHLS study to the docket.

NEMA and EEI/USWAG also subsequently asked DFHLS to comment on EPA's memorandum dated April 12, 1982 from Dr. I. Baumel (TS-796) to Dr. M. Halper (B-798) entitled "Proposed Rule on Polychlorinated Biphenyls (PCBs) Use in Electrical Equipment." NEMA encloses a copy of DFHLS' comments, which have also been separately submitted to the docket.

Both the DFHLS study and the enclosed comments contain numerous citations to the original data and studies that were reviewed. (Likewise, NEMA's previous submissions to this docket and to Docket No. OPTS-62018 contain numerous citations to the original data and studies on which NEMA based its opinions regarding the human health effects of PCBs.) As stated at the informal hearing, NEMA urges EPA's review of both the DFHLS study and comments and the original scientific data.

Indeed, Section 6(e) requires EPA to review and weigh such scientific data and to make a determination of "insignificant exposure" based on it. NEMA respectfully submits that recitation of virtual truisms (i.e., "the risks posed from exposure to PCBs are reduced as exposure to PCBs is reduced") and broad-brush characterizations and dismissals of the scientific information (i.e., "the Agency does not agree that this information [e.g., the DFHLS study] proves that PCBs do not pose any serious risks to human health or the environment") do not constitute a determination of "insignificant exposure" (and/or "unreasonable risk" and/or de minimis risk) based on substantial evidence.

2. PCDFs

At the informal hearing, Dr. Ian Webber representing RTE Corporation asserted that concentrations of PCDFs in askarel transformers1/ increase with time in service. Ms. Juliann Sum representing Local 1245 of the International Brotherhood of Electrical Workers asserted that continued industrial use of PCB-containing electrical equipment may result in occupational exposures to higher concentrations of PCDFs than were present in the contaminated rice oil causing

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1/ When used in these comments, the term "askarel transformer" refers to those transformers designed and manufactured to contain a fire resistant liquid insulating coolant (askarel) with between 60% (600,000 ppm) and 100% (1,000,000 ppm) of the commercial PCB products. The term "PCB Transformer" as defined at 40 CFR § 761.3(y) includes all askarel transformers (as defined above) plus transformers filled with mineral oil but contaminated with PCBs at concentrations of 500 ppm or greater.

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Yusho disease. By contrast, NEMA has consistently maintained that (1) there is no evidence that the conversion of PCBs into PCDFs occurs in PCB-containing electrical equipment; and (2) the causative circumstances for the production of high levels of PCDFs in the Yusho incident do not exist in the United States today. As discussed below, available data support NEMA's position on both points.

Samples of unused Aroclor-trademarked commercial PCB products manufactured by Monsanto have been analyzed for PCDF concentration. The values usually reported have been between 0.1 to 2.0 ppm, although higher values (up to 5.6 ppm) have been reported as well.2/

Samples of used askarel taken from United States and Canadian-manufactured transformers have also been analyzed for PCDF concentration. For instance, Moseley et al., from the National Institute for Occupational Safety and Health analyzed both bulk samples of used askarel and air in a transformer maintenance workplace, finding trace tetrachlorodibenzofuran ("TCDF") concentrations (0.013-0.116 ppm) in the fluids and no detectable concentration in the air.3/ Albro and Parker from the National Institute of Environmental Health Sciences have characterized two samples of used askarel.4/ Fluid A, obtained from a transformer of early manufacture but unknown extent of use,5/ contained 0.7 ppm PCDF. Fluid B, a bulk sample of used askarel obtained from a transformer maintenance operation,5/ contained 4.7 ppm PCDF.6/

2/ Samples of unused commercial PCB products manufactured by foreign producers have also been analyzed and found to contain considerably higher PCDF concentrations. See Exhibit A. (All exhibits are attached at the end of these comments.)

3/ Moseley, C.L., Geraci, C.L. and Burg, J. (1982) Polychlorinated Biphenyl Exposure in Transformer Maintenance Operations. J. Amer. Ind. Hyg. Assoc., 43, 170-174.

4/ Albro, P.W. and Parker, C.E. (1980) General Approach to the Fractionation and Class Determination of Complex Mixtures of Chlorinated Aromatic Compounds. J. Chrom., 197, 155-169.

5/ Private communication, P. W. Albro (1982).

6/ The PCDFs found in Fluid B consisted principally of octachlorodibenzofuran (4.5 ppm), and no detectable TCDF.

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At the informal hearing, Dr. Webber referred to the unpublished work of Chittim et al., who reported TCDF concentrations ranging from ca. 0.005 to 5.0 ppm in used askarel. Chittim et al., concluded that (1) TCDF concentration increases with time in service, although there is no linear relationship; and (2) electrical sparks and/or failure do not increase PCDF concentration, most likely because of the short duration of the sparks or events causing failure.7/

Several points should be kept in mind when evaluating the work of Chittim et al.: (1) the TCDF concentrations in twenty-seven (27) of twenty-nine (29) samples of used askarel analyzed contained 1.1 ppm TCDF or less;8/ (2) the analytical work was performed in 1979 using gas chromatography with a packed column, a less sensitive analytical methodology than is generally used today; and (3) it does not appear that the values reported had been corrected to take into account possible interference by polychlorinated diphenyl ethers ("PCDPEs"), which co-elute with PCDFs.

NEMA also is aware of additional, more recent analyses of used askarel for PCDFs. Specifically, a year ago the General Electric Company ("GE") submitted a sample of used askarel obtained from the Canadian General Electric Company to both a university-affiliated and an industrial laboratory for PCDF analysis. Using "state of the art," high resolution gas chromatography/mass spectroscopy ("GC/MS"), the industrial laboratory found PCDFs on the order of 1.0 ppm or lower. The university-affiliated laboratory found no

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7/ Chittim, B. G., Clegg, B. S., Safe S. N., and Hutzinger, O. (1979) Chlorinated Dibenzofurans and Dibenzo-P-Dioxins: Detection and Quantitation in Electrical Equipment and Their Formation During the Incineration of PCBs, Draft Report prepared by Wellington Science Associates, Inc. under Contract No. OSS78-00067, Environmental Protection Service, Department of Fisheries and Environment (Canada).

8/ See Exhibit B, captioned "Table 6: Transformer Data, Askarel Composition and TCDF Concentrations", and excerpted from the Draft Report of Chittim et al., ibid. Samples 4-1 and 4-2 from "very old" (circa 1947) transformers contained 4.7 and 2.2 ppm TCDF respectively. Note that (1) Chittim et al., elsewhere acknowledged the lack of maintenance and operating records for the transformers sampled; and (2) even these "high" values are not appreciably higher than concentrations reported for some samples of unused Aroclors. (See Exhibit A.)

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PCDFs at detection limits of 10.0 ppm for 4-Cl and 5-Cl, and 1.0 ppm for 6-Cl, 7-Cl, and 8-Cl isomers.

Subsequently, GE submitted four (4) additional samples of used askarel to the same industrial laboratory for PCDF analysis. These samples had been obtained from in-service distribution transformers at a GE transformer manufacturing facility in the United States during the course of normal maintenance activities.9/ The following table describes each sample and sets forth the PCDF concentrations found:

Concentration of PCDFs in Used Askarel Transformer Fluids

Sample Number	Type of Transformer from Which Sample Obtained	Date Installed	PCDF (ppm) Total	% PCB 10/
#3	Single Phase - 100KVA 2300 vt -- 115/230 115 gallons fluid	1940	1.1	56.4
#23	3 phase - 1250KVA 1380 vt -- 480Y/277 490 gallons fluid	1947	0.62-0.82	66.2
#44	2 phase - 375KVA 2300 vt -- 550 31.5 gallons	1938	1.84-1.86	60.1
#45	Single phase -100KVA 2300 vt -- 120/240 42 gallons	1939	0.64-0.86	66.8
"Pure Aroclor 1254" (control)			0.62-0.82	

The industrial laboratory estimated accuracy at ± 30%, but emphasized that PCDPE interference could be responsible for larger errors; therefore, these values should be regarded

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9/ That is, sampling is routinely done on an annual basis as part of the normal maintenance program for in-plant transformers. Approximately one quart of fluid is removed and tested for a variety of properties such as dielectric strength, color, dissipation factor and chloride analysis.

10/ There is no record of retrofill for the four transformers sampled. This is consistent with the percentage of PCB (Aroclor 1260) in each of the four samples.

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as maximum concentrations.11/ Note that the values range from 0.62 to 1.86 ppm PCDF, indicating that PCDF concentrations in these forty-year old, used askarels are comparable to the PCDF concentrations reported for unused Aroclors.

Nor should these analytical results be surprising, given the conditions of temperature and residence time reported in the literature as required for the conversion of PCBs into PCDFs. That is, Buser has reported that both Aroclors 1254 and 1260 heated for one minute in air to 550°-650°C yield about 1.0-3.0% PCDF; and heated for one minute to 700-850°C, PCBs were completely destroyed and no PCDFs were observed.12/ Morita has reported that Aroclor 1248 heated for one week at 300°C in the presence of oxygen yields about 0.2% PCDF; and heated for one week at 300°C in the presence of air yields about 0.05% PCDF. Morita also concluded that temperatures over 270°C are necessary to initiate the transformation, and that PCDFs decompose at temperatures over 330°C.13/ The instability of PCDFs at their formation temperatures is undoubtedly the reason that their highest yields are limited to a few percent.

These conditions of temperature and time simply do not exist in in-service askarel transformers and capacitors. That is, transformers usually operate at temperatures of 100° - 130°C and transformer "hot spots" typically are no greater than 135°C. Occasionally, there are short circuits, with temperatures reaching 200° - 250°C for a few minutes. In the very rare case of an arc where a transformer is destroyed, maximum temperatures may reach 1000°C, but failure occurs within fractions of a second.

11/ That is, PCDPEs present in a GC/MS analysis will appear as PCDFs since they decompose in the mass spectrometer to yield the same fragments. As a result, the ethers must be scrupulously separated from the furans before introduction into the mass spectrometer. This understanding of PCDPE interference in PCDF analysis has developed only recently, principally as a result of the work of Albro and Parker.

12/ Buser, H.R., Bosshardt, H.P. and Rappe, C. (1978) Formation of Polychlorinated Dibenzofurans (PCDFs) From the Pyrolysis of PCBs. Chemosphere, 7, 109.

13/ Morita, M., Nakagawa, J., and Rappe, C. (1978) Polychlorinated Dibenzofuran (PCDF) Formation from PCB Mixture by Heat and Oxygen, Bull. Environm. Contam. Toxicol., 19, 665-670.

Capacitors typically operate below 100°C. In those capacitor failures resulting in rupture, heating and internal arcing may have occurred prior to rupture; however, it is questionable whether there is sufficient time at temperature or oxygen for PCDF formation.

New information has also added to our understanding of Yusho (Japan) disease, cited by Ms. Sum as a basis for grave concern regarding current United States occupational exposures to PCBs. This incident involved human ingestion of rice oil containing approximately 1000 ppm PCBs, 1000 ppm polychlorinated quaterphenyls ("PCQs") and 5 ppm PCDFs.

The rice oil contaminant has been identified as fluid that leaked from a heat exchanger originally filled with a Japanese commercial PCB product, Kanechlor 400. The quantities of actual PCBs ingested by Yusho victims (average intake of 466 mg) were much lower than PCB body burdens exhibited by asymptomatic, heavily exposed capacitor workers, but were accompanied by comparable quantities of PCQs, which are of unknown toxicological activity, and an average of 2.5 mg of PCDFs, which are more toxic than PCBs.14/ The PCQs and PCDFs are believed to have been formed from high temperature thermal oxidation and condensation of PCBs in the heat exchanger. Recent findings from animal tests and follow-up studies on Yusho patients have linked Yusho disease symptoms with exposure to unusually large amounts of PCDFs and, possibly, PCQs. See the DFHLS Study at 26, and references cited therein.

14/ Ms. Sum consistently equates the toxicity of 2,3,7,8-TCDF and 2,3,7,8-tetrachlorodibenzodioxin ("TCDD"), and further asserts that 2,3,7,8-TCDF is "about 1 million times more poisonous than PCB's in terms of laboratory experiments." T-618. In fact, on the basis of a species-by-species comparison of LD-50, 2,3,7,8-TCDF is approximately 1/10 to 1/700 as toxic as 2,3,7,8-TCDD. Moreover, 2,3,7,8-TCDF is only 40 to 150 times more toxic in the guinea pig (among the more susceptible species) than is 3,4,3',4'-tetrachlorobiphenyl (the correspondingly most toxic PCB isomer). (Ms. Sum appears to have compared the LD-50 for 2,3,7,8-TCDF in the guinea pig to the LD-50 for commercial PCB products in mice and rats. This is somewhat like comparing apples with oranges.) See Goldstein, J. A. (1980) Structure-Activity Relationships for the Biochemical Effects, Chapter 6, Halogenated Biphenyls, Terphenyls, Napthalenes, Dibenzo-dioxins and Related Products, R. D. Kimbrough, Ed., Elsevier Press, Amsterdam.

There are no reports of Yusho disease in the United States and there is virtually no possibility of such an event arising in the future. The Kanechlor 400 involved in the Yusho incident contained PCDF concentrations (based on PCBs) 250 times greater than the concentrations reported for unused Kanechlor 400 (20 ppm) and 2500 to 5000 times the usual concentrations (1-2 ppm) in unused Aroclors. (See (Exhibit A.) Assuming a PCDF concentration in Aroclors of 2 ppm and a PCDF toxicity threshold dose of 0.5 mg, a mimimum 250g of Aroclor would have to be absorbed - a highly unlikely event - before Yusho disease symptoms would appear. Moreover, as noted previously, conversion of PCBs to PCDFs and PCQs, requires heating to over 270°C in the presence of air.

There is no evidence that this conversion occurs in the use, maintenance or repair of PCB-containing electrical equipment. Since November 1, 1979, heat transfer systems employed in the manufacture or processing of food, drugs or cosmetics in the United States have not been authorized to contain heat transfer fluid with PCB concentrations over 50 ppm. 40 CFR 761.31(d)(3). Any industrial heat transfer system that ever contained PCBs has been drained and refilled at least three times since November 1, 1979 with fluids containing less than 50 ppm PCB. 40 CFR 761.31(d)(1) and (2).

3. Capacitor Phase-Out

Questions were raised at the informal hearing about the ability of the electrical equipment manufacturers to meet the increased demand that would result from EPA's proposed ten-year phase-out of Large PCB Capacitors. NEMA reiterates its position that (1) there should be no ban; (2) an absolute minimum of ten years is required for such a changeout; and (3) the success of any such changeout depends on a relatively steady rate of equipment replacement. NEMA can think of no "command and control" regulation that EPA might successfully impose to insure such a steady rate.

4. The 50 ppm Regulatory Cutoff

There is a need for a ppm regulatory cutoff insofar as the PCB Ban Rule is concerned unless (1) EPA determines - as NEMA believes it should - that the use of PCBs in electrical equipment is use in a "totally enclosed manner"; or (2) EPA deletes the assumption - as EEI/USWAG has urged - that all oil-filled transformers, voltage regulators, switches, electromagnets and cable are PCB-Contaminated Electrical Equipment unless tested or otherwise verified and found not

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to contain between 50 and 500 ppm PCB. Moreover, EPA's retention of the cutoff is supported by substantial evidence.

Regulation under Section 6(e) of electrical equipment containing less than 50 ppm PCBs would yield but de minimis health or environmental benefits. First, it can not be overemphasized that 50 ppm represents almost a 20,000-fold decrease in PCB concentration below that of the commercial PCB products to which asymptomatic capacitor workers were exposed for many years. By any measure, this is a truly remarkable "margin of safety."

Second, indiscriminate disposal represents virtually the only conceivable means of widespread human or environmental exposure to electrical fluids containing less than 50 ppm PCBs. But disposal of such "waste oil" is regulated already pursuant to TSCA or the Resource Conservation and Recovery Act ("RCRA"), or soon will be regulated under RCRA. Since whatever unreasonable risk may be presented by electrical fluids containing PCBs in concentrations below 50 ppm is therefore amply regulated,15/ it is NEMA's view that duplicative regulation of such fluids pursuant to Section 6(e) would be confusing and unnecessarily burdensome to both the regulated community and EPA. Such regulation would afford no additional protection to human health or the environment, and would run counter to Section 9(b) of TSCA, which expresses Congress' intent that TSCA not be used when other statutes are sufficient to regulate a particular risk.

5. Question Raised by the Hearing Panel

Q: What inconsistency does NEMA see between EPA's conditions for the use authorizations and the definition of a leak as disposal?

A: The proposed conditions for the use authorizations for PCB Transformers, Large PCB Capacitors and PCB-Contaminated Electrical Equipment should define the equipment owner's total obligation. An owner who complies in good faith with these conditions should not also be subject to penalties for improper disposal in the event a leak nonetheless occurs. This is especially so in the case of any leaks from PCB-Contaminated Electrical Equipment, where inspections for leaks are not even required in

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15/ So that there may be no misunderstanding, NEMA stresses again its belief that PCBs in concentrations below 50 ppm present no unreasonable risk of injury to health or the environment.

view of the equipment's miniscule PCB content. Yet such leaks as might occur constitute illegal disposal (or so NEMA reads the proposed rule).

Q: What are NEMA's recommendations?

A: NEMA recommends (1) revising the definition of "leak" or "leaking" to mean "any instance in which a PCB Article, PCB Container or PCB Equipment has any quantity of PCBs running off or about to run off any portion of its external surface;" and (2) making no change in the definition of "disposal" or the spills provision of the disposal requirements.

Q: Is there any correlation between capacitor size and location?

A: NO.

Q: Is there any correlation between capacitor cost and size?

A: Yes, the larger the capacitor the more costly.

Q: Is there any correlation between capacitor location and age?

A: NO.

Q: Is there any correlation between capacitor failure rate and location?

A: NO.

Q: How long does it take for a manufacturer to provide capacitors once an order has been placed?

A: The time will vary depending on the plant's workload. (No generalization may be made.)

Q: How many units are typically included in a single order?

A: This will vary anywhere from a single unit to more than one thousand units. (No generalization may be made.)

Q: What percentage of new orders are attributable to failure, obsolescence or new applications?

A: Statistics are unavailable.

Q: What data does NEMA have as to the fluids, effectiveness and costs of available capacitor protective devices?

A: NEMA believes these kind(s) of questions can best be answered by the users of such devices.

Q: Are capacitors shelf items or custom designed?

A: Both, depending on customer requirements.

Q: Does NEMA have engineering data to show failure, leakage and rupture rates for electrical equipment?

A: NO.

Q: Do engineering specifications for transformers and capacitors vary greatly among manufacturers, by customer industry or by application (i.e. indoor/outdoor)?

A: Engineering specifications depend on customer requirements.

Sincerely,

Daniel K. Shipp
Vice President,
Public Affairs

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Concentrations of Chlorinated Dibenzofurans in Commercial PCB Products

PCB	4-Cl	5-Cl	6-Cl	Total
Aroclor 1016 (1977)[a]	ND	ND	ND	--
Aroclor 1016[b]	ND	ND	ND	--
Aroclor 1242[b]	0.07	0.03	.003	0.15
Aroclor 1242[f]	2.3	2.2	ND	4.5
Aroclor 1254 (1969)[a]	0.1	0.2	1.4	1.7
Aroclor 1254 (1970)[a]	0.2	0.4	0.9	1.5
Aroclor 1254[c]	0.02	0.2	0.4-0.6	0.8
Aroclor 1254[f]	0.1	3.6	1.9	5.6
Aroclor 1260 (1969)[a]	0.1	0.4	0.5	1.0
Aroclor 1260 (lot AK3)[a]	0.2	0.3	0.3	0.8
Aroclor 1260[d]	0.3	1.0	1.1	3.8*/
Aroclor 1260[f]	0.8	0.9	0.5	2.2
Clophen A60[a]	1.4	5.0	2.2	8.4**/
Clophen T64[d]	0.3	1.73	2.5	5.4*/
Phenoclor DP-6[a]	0.7	10.0	2.9	13.6
Prodelec 3010[d]	1.1	0.4	0.1	2.0*/
Kanechlor 400[e,f]				ca 20.0

*/ Totals include quantities of 3-Cl and 7-Cl isomers analysed for and reported only in reference d.

**/ Total as reported in the original.

References to Table

a. Bowes, G.W., Mulvihill, M.J., Simoneit, B.R.T., Burlingame, A.L. & Risebrough, R.W. (1975) Identification of chlorinated dibenzofurans in American polychlorinated biphenyls. Nature (Lond.), 256, 305-307.

b. Albro, P.W. & Parker, C.E. (1979) Comparison of the Compositions of Aroclor 1242 and Aroclor 1016. J. Chrom., 169, 161-166.

c. Bell, R.A. (1981) General Electric Company, unpublished results. These results represent maximum values because of possible chlorinated diphenyl ether interference.

d. Rappe, C. & Buser, H.R. (1980) Chemical Properties and Analytical Methods, Chapter 2, Halogenated Biphenyls, Terphenyls, Napthalenes, Dibenzodioxins and Related Products, R.D. Kimbrough, Elsevier Press, Amsterdam.

e. Nagayama, J., Kuratsune, M., and Masada, Y. (1976) Determination of chlorinated dibenzofurans in Kanechlors and 'Yusho Oil', Bull. Environ. Contamin. Toxicol. 15, 9-13.

f. Morita, M., Nakagawa, J., Akiyama, K., Nimura, S. and Isono, N. (1977) Bull. Environ. Contamin. Toxicol. 18, 67-73.

Table 6

Table 6: Transformer Data, Askarel Composition and TCDF Concentration

Sample	Installation	Manufacturer	Dielectric Strength (kv)	Power (kva)	Volume (litres)	kva/volume	1,3,5-Cl_3Bz	1,2,4-Cl_3Bz	1,2,3-Cl_3Bz	1,2,3,5- & 1,2,4,5-Cl_4Bz	1,2,3,4-Cl_4Bz	1,2,3,4,5-Cl_5Bz	PCBz/PCB ratio MONS 003461	Aroclor	TCDF
Group 1															
1-1	1957	FP	36	1500	1888	0.79	tr	26.4	6.8	1.0	14.4	2.4	51.0/49.0	1260	0.780
1-2	1961	FP	36	1500	2093	0.72	tr	28.4	6.6	tr	15.2	3.4	53.6/46.4	1260	1.110
1-3	1963	FP	36	1500	1251	1.19	tr	27.8	6.2	tr	14.0	2.6	50.6/49.4	1260	1.020
1-4	1963	FP	36	1500	1251	1.19	tr	28.8	6.4	tr	14.4	2.8	52.4/47.6	1260	0.790
1-5	1963	FP	36	1500	1274	1.17	tr	28.4	8.0	tr	2.2	tr	38.6/61.4	1260	0.350
1-6	1965	FP	29	1500	1151	1.30	tr	28.0	7.6	tr	1.6	ND	37.2/62.8	1260	0.290
1-7	1970	FP	30	1500	1765	0.85	tr	22.6	7.0	tr	tr	ND	29.6/70.4	1254	0.550
1-8	1970	FP	22	1500	1490	1.01	tr	21.2	6.8	tr	tr	ND	28.0/72.0	1254	0.470
1-9	1971	FP	28	1500	1490	1.01	tr	21.2	7.8	tr	tr	ND	29.0/71.0	1254	0.290
1-10	1974	FP	28	1500	1490	1.01	ND	21.6	6.8	ND	ND	ND	28.4/71.6	1254	0.051
1-11	NEW	FP	-	1500	-	-	tr	20.8	9.8	tr	tr	ND	30.6/69.4	1254	0.041
Group 2															
2-1	1967	CGE	33	500	725	0.69	ND	21.4	5.6	tr	14.8	3.4	45.2/54.8	1260	0.030
2-2	1967	CGE	31	1000	938	1.07	tr	26.2	7.2	tr	14.8	3.2	44.3/55.7	1260	0.004
2-3	1967	CGE	36	1000	770	1.30	tr	26.0	7.0	tr	14.4	3.2	50.6/49.4	1260	0.007
2-4	1967	CGE	35	866	1268	1.47	tr	26.4	6.4	tr	13.4	2.8	49.0/51.0	1260	0.010
2-5	1965	FP	36	700	660	1.06	tr	27.6	7.8	tr	tr	tr	35.4/64.6	1260	0.176
2-6	1965	FP	36	1500	1251	1.20	tr	27.0	5.8	tr	13.6	2.6	49.0/51.0	1260	0.435
2-7	1965	FP	36	2500	2084	1.20	tr	27.8	6.6	tr	1.0	tr	35.4/64.6	1260	0.110
2-8	1965	FP	36	2500	1092	2.29	tr	28.4	8.2	tr	tr	tr	36.6/63.4	1260	0.141
Group 3															
3-1	1967	CGE	33	500	725	0.69	ND	21.4	5.6	tr	14.8	3.4	45.2/54.8	1260	0.419
3-2	1967	CGE	41	1000	770	1.30	tr	26.0	7.0	tr	14.4	3.2	50.6/49.4	1260	0.341
3-3	1967	W	41	750	883	0.85	tr	28.2	8.0	tr	tr	tr	36.2/63.8	1260	0.444
3-4	1967	W	41	1200	1087	1.10	tr	27.4	7.8	tr	tr	tr	35.2/64.8	1260	0.426

cont'd...

Table 6 (continued)

Sample	Installation	Manufacturer	Dielectric Strength (kv	Power (kva)	Volume (litres)	kva/volume	$1,3,5\text{-}Cl_3Bz$	$1,2,4\text{-}Cl_3Bz$	$1,2,3\text{-}Cl_3Bz$	$1,2,3,5\text{-}$ & $1,2,4,5\text{-}Cl_4Bz$	$1,2,3,4\text{-}Cl_4Bz$	$1,2,3,4,5\text{-}Cl_5Bz$	PCBz/PCB ratio	Arocior	MONS 003462 TCDF
3-5	1967	FP	31	750	829	0.90	tr	28.0	7.8	tr	tr	tr	32.8/67.2	1260	0.168
3-6	1967	FP	36	1000	838	1.19	tr	25.0	7.8	tr	tr	tr	32.8/67.2	1260	0.179
Group 4															
4-1	1947	CGE	-	56	660	0.08	ND	24.8	7.2	tr	14.2	3.0	49.2/50.8	1260	4.730
4-2	1947	CGE	-	10,000	6,900	1.45	ND	25.2	6.6	tr	14.0	2.6	48.4/51.6	1260	2.201
4-3	1965	AD	-	-	-	-	ND	27.0	5.2	tr	14.4	3.6	50.2/49.8	1260	0.453
4-4	1973	WA	-	-	-	-	ND	27.4	5.2	tr	14.8	4.0	51.4/48.6	1260	0.403
4-5	'new'	CGE	-	-	-	-	tr	35.4	13.4	tr	10.0	ND	58.8/41.2	1254	< 0.001
4-6	'new'	W	-	-	-	-	ND	20.2	10.0	tr	ND	ND	30.2/69.8	1254	0.014
4-7	'new'	FP	-	-	-	-	tr	20.8	9.8	tr	tr	ND	30.6/69.4	1254	0.041

Table 6 : Legend

- **Samples:**

  Group 1 (1-1, 1-2, etc.) - variable time-in-service.
  Group 2 - 2-1 to 2-4 - CGE Transformers; variable kva/volume ratio
  - 2-5 to 2-6 - FP Transformers; variable kva/volume ratio
  Group 3 - variable fluid type - 3-1 and 3-2 Pyranol
  3-3 and 3-4 Chlorextol
  3-5 and 3-6 Inerteen
  Group 4 - 4-1 and 4-2 - 'very old' transformers; large variation in kva relative to fluid volume.
  - 4-3 and 4-4 - transformers which had failed in service
  AD - Adelaide Street (Toronto Hydro)
  WA - Walkerton, Ont. (Canada Packers)
  - 4-5, 4-6 and 4-7 - new Pyranol, Inerteen and Chlorextol.

- **Manufacturer:** (fluid type)

CGE	- Canadian General Electric	(Pyranol)
FP	- Ferranti-Packard	(Chlorextol)
W	- Westinghouse	(Inerteen)
AD	- Brown Boveri	(Pyroclor)
WA	- Foster Electric	(Inerteen)

- 1,3,5-Cl_3Bz - 1,3,5-trichlorobenzene content (± 0.05% w/w)
- 1,2,4-Cl_3Bz - 1,2,4-trichlorobenzene content (± 0.05% w/w)
- 1,2,3-Cl_3Bz - 1,2,3-trichlorobenzene content (± 0.05% w/w)
- 1,2,3,5- & 1,2,4,5-Cl_4Bz - 1,2,3,5- & 1,2,4,5-tetrachlorobenzene content (± 0.05% w/w)
- 1,2,3,4-Cl_4Bz - 1,2,3,4-tetrachlorobenzene content (± 0.05% w/w)
- 1,2,3,4,5-Cl_5Bz - 1,2,3,4,5-pentachlorobenzene content (± 0.05% w/w)
- TCDF (ppm) - concentration of 2,3,7,8-tetrachlorodibenzofuran in askarel (ppm)

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