

PX 2097

A Wire Reclamation Incinerator as a Source of Environmental Contamination with Tetrachlorodibenzo-p-dioxins and Tetrachlorodibenzofurans

DANIEL O. HRYHORCZUK, M.D.
University of Illinois School of Public Health
and Section of Occupational Medicine
Cook County Hospital
Chicago, Illinois

WILLIAM A. WITHROW
Illinois Pollution Control Board
Chicago, Illinois

CAROLYN S. HESSE, M.S.
United States Environmental
Protection Agency
Chicago, Illinois

VAL R. BEASLEY, D.V.M.
College of Veterinary Medicine
University of Illinois
Urbana, Illinois

ABSTRACT. The authors investigated an outbreak of unusual illnesses in humans and horses residing within 1.3 km of a wire reclamation incinerator. The study included site visits; medical and veterinary examinations; analyses of furnace ash, fly ash, soil, and biologic samples for air residues. Tetrachlorodibenzo-*p*-dioxins (TCDDs) and tetrachlorodibenzofurans (TCDFs) were discovered in furnace ash, fly ash, soil, horse fat, and horse liver samples.

INCINERATION is a commonly used method for the recovery of metals from wire scrap. The combustible portion of wire insulation can be comprised of a wide variety of materials, including rubber, paper, cotton, asphalt-impregnated fabrics, silk, and a large variety of plastics, such as polyethylene, polypropylene, and polyvinyl chloride.¹ Older cables can contain chlorinated naphthalenes and polychlorinated biphenyls.² Thermal degradation of insulated electrical wire without proper air pollution control equipment can lead to the emission of particulates, hydrocarbons, metals, halides, carbon monoxide, nitrogen oxides, and sulfur oxides.^{1,3}

Recent studies have shown that highly toxic polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) can be emitted by municipal and

industrial incinerators, fossil-fueled power plants, and industrial heating facilities. Olie⁴ identified several PCDDs and PCDFs in samples of fly ash from three municipal incinerators in the Netherlands. Buser and Bosshardt⁵ determined that the total concentrations of PCDDs and PCDFs in fly ash from a Swiss municipal incinerator were 0.2 parts per million (ppm) and 0.1 ppm, respectively; the total concentrations of PCDDs and PCDFs in fly ash from an industrial heating facility were 0.6 ppm and 0.3 ppm, respectively. Dow⁶ found PCDDs in parts per billion (ppb) levels in particulate matter from the air emissions of a stationary tar burner, rotary kiln incinerator, and a fossil-fueled powerhouse. Kimble and Gross⁷ found no tetrachlorodibenzo-*p*-dioxins (TCDDs) in fly ash collected from the stack of a commercial coal-fired power plant, whereas Tiernan et al.⁸ detected TCDDs in emissions from a municipal incinerator. The origin of PCDDs and PCDFs in the airborne particulates from these combustion processes is still unclear. Recent studies⁹⁻¹² have shown that chlorinated phenols, polychlorinated biphenyls, chlorinated diphenyl ethers, and chlorobenzenes can convert to PCDDs and PCDFs in thermal processes. As insulated electrical wire and cable may contain some of these precursors, incineration of these materials might lead to the formation and emission of PCDDs and PCDFs. Our investigation of an outbreak of unusual illnesses in humans and horses residing within 1.3 km of a wire

reclamation incinerator included analyses of furnace ash, fly ash, soil, and horse fat and liver samples for TCDDs and tetrachlorodibenzofurans (TCDFs).

Description of the Incident

From 1976 to 1980, citizens of a rural, midwestern community filed numerous complaints with their state Environmental Protection Agency regarding a wire reclamation facility located within 1.3 km of their homes. They claimed that incinerators at the facility were continuously emitting dense, malodorous smoke which they believed posed a hazard to their health and property. The complaints were supported by photographs and a daily emission log kept by one of the citizens. Several complained of burning eyes, sore throat, headache, dizziness, and nausea temporally related to the emissions.

The proprietor of a horse boarding stable and riding school stated that from 1976 to 1980, fourteen of her horses had died of unexplained causes. The affected horses suffered chronic weight loss, hair loss, and thickened skin on the dorsal aspect of their bodies; some developed paresis in the rear and edema of the distal parts of the limbs. Horses which grazed in the adjacent pasture were the most severely affected.

The wire reclamation facility incinerated wire, cable, and X-ray film to recover copper, silver, and lead. In 1979, the wire reclamation facility had increased operations from one to three incinerators; the new incinerators increased their processing capacity from 45 to 82 tons of scrap per month. Though the incinerators were equipped with afterburners, officials of the Environmental Protection Agency doubted that they were operating properly, if at all. The manager of the facility admitted that there was a smoke problem from the incineration of 350 tons of scrap power plant cable. He described the cable as being a 6-in diameter copper wire core, wrapped in oil-saturated paper, and covered by a lead sleeve. Subsequent to a preliminary investigation, the wire reclamation facility was closed by temporary injunction in April, 1980.

METHODS

The state Environmental Protection Agency requested the assistance of a health effects specialist from the U.S. Environmental Protection Agency (U.S. EPA), a medical toxicologist, and a veterinary toxicologist to investigate alleged health effects from pollutants emitted by the wire reclamation incinerator. Members of this team visited the site, interviewed the citizens and manager of the incinerator, collected samples for air pollutant residues, and examined the affected people and animals.

We selected the sampling sites based on the predicted behavior of the incinerator's plume, which was estimated using an atmospheric dispersion model and wind rose.^{13,14} The dispersion model predicted the *X* max, i.e., the distance to the point where the plume touches the ground with maximum concentration. The wind rose predicted the sector within the *X* max area which would be most heavily impacted by the plume. The most distal *X* max and most heavily impacted sector are shown in Figure 1. The sampling scheme consisted of several soil samples,

scrapings from two of the three stacks, and scrapings from the inside of all three furnaces. Water samples were obtained from private wells in the area. All samples were collected in June and July of 1980.

The soil sample sites are shown in Figure 1. Site No. 1 was located 50 m east of the stacks. This sample consisted of the top 5 cm of soil. Site No. 2 was located 10 m south of the facility on a spot where oil had been spilled. This sample consisted of the top 3 cm of soil. Site No. 3 was located 215 m east of the facility in the yard of one of the affected families. A core soil sample to a depth of 20 cm was collected for inorganics; a surface sample to a depth of 4 cm was collected for organics. Site No. 4 was located 1 km northeast of the incinerator in the pasture where the affected horses had grazed; both a core soil sample to a depth of 20 cm and a surface soil sample to a depth of 3 cm were collected. Site No. 5 was located 6.4 km northeast of a facility in a relatively undisturbed wooded area. A surface soil sample to a depth of 3 cm and a core soil sample to a depth of 20 cm were collected from this site to serve as controls.

Fly and furnace ash samples were collected from the top of the stacks and from the incinerator furnaces, respectively. A fire department snorkel was used to obtain samples from the top of the stacks. The material scraped from the stack of incinerator No. 1 was a reddish-brown scale. In stack No. 2, a pale green powder was removed from the base of the spark arrester. Access to stack No. 3 was blocked by a utility pole and a mobile home. Samples collected inside the incinerators were from the knockdown chamber, the ledge at the base of the stack, and the wall of the primary chamber, in incinerators No. 1, 2, and 3, respectively.

All samples were placed in new glass jars and refrigerated. Aluminum foil was placed between the sample and lid of the jar. Samples for inorganics; polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBBs), pesticides, and phthalates; and nonvolatile organics were analyzed by the U.S. EPA Central Regional Lab according to their standard operating procedures: CRL method No. 571-598, No. 198-207, and No. 625, respectively.¹⁵⁻¹⁷ Veterinary samples for PCBs and PBBs were analyzed by gas liquid chromatography at the Regional Veterinary Diagnostic Laboratory in Centralia, Illinois. Analyses for TCDDs and TCDFs were performed by Dr. M. Gross at the Midwest Center for Mass Spectrometry at the University of Nebraska using a gas chromatography/high resolution mass spectrometry procedure, as described in a previous work.⁷

Citizens who complained of adverse health effects were offered medical examinations in the Occupational Medicine Clinic at a nearby county hospital. The examinations included a medical history, review of medical records, physical examination, complete blood count (CBC), serum chemistry screen, liver profile, urinalysis, pulmonary function tests, blood lead, free erythrocyte protoporphyrin (FEP), and serum PCB level. One patient (N.C.) had an extensive neurologic work-up before being examined at the Occupational Medicine Clinic, the results of which are also presented.

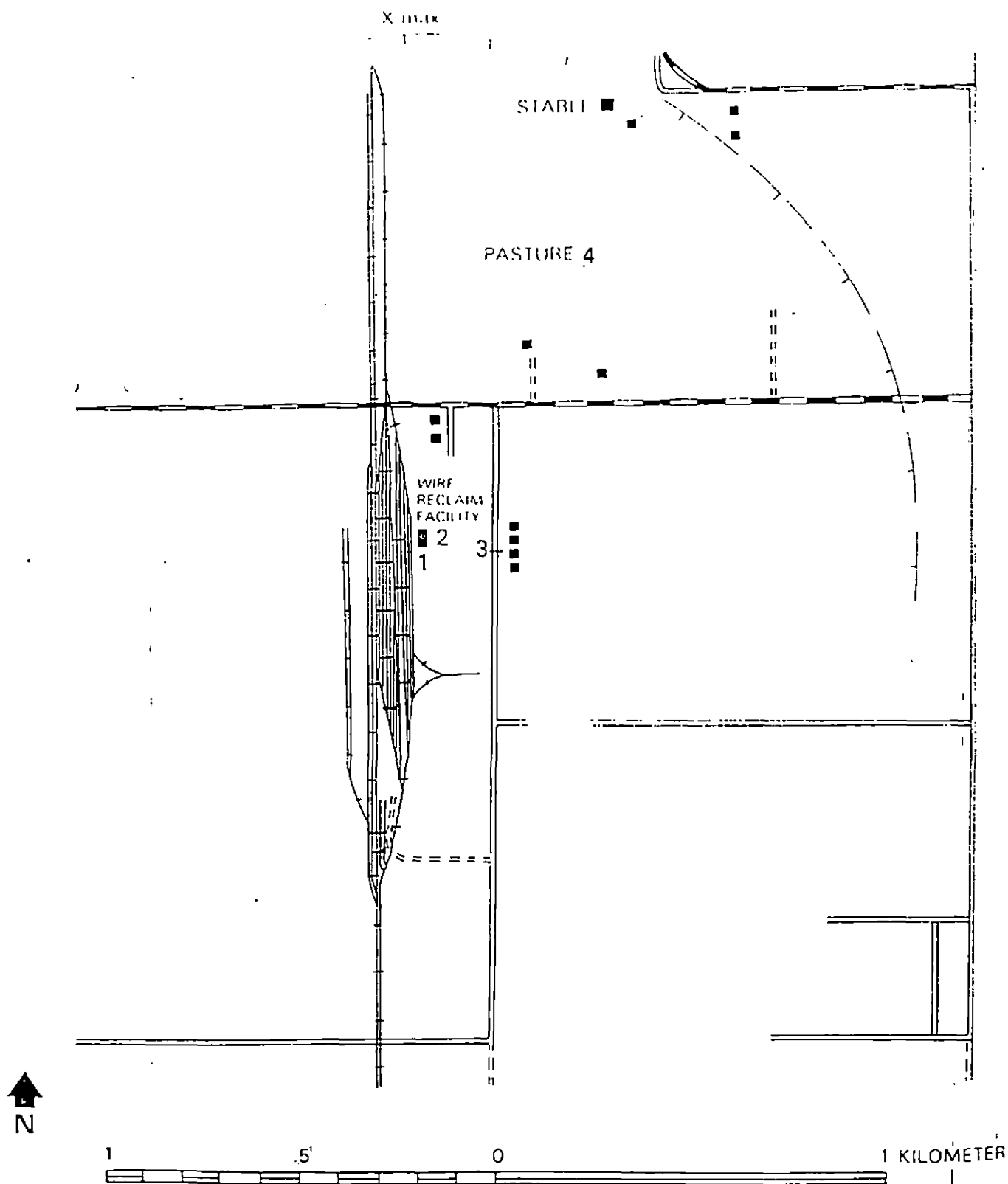


Fig. 1. Map of area including most heavily impacted sector (dashed lines) and soil sample sites 1-4.

The veterinary toxicologist examined and obtained a blood lead level on a dog kept in a home located 225 m east of the incinerator. Bone lead content was measured in two rabbits taken in the field between the home and

the incinerator. A fat sample for PCBs and PBBs analyses was obtained from a chicken raised nearby. Calves, chickens, and horses were examined at a small farm located 0.5 km from the incinerator. Frozen liver from two

Table 1. Levels of Selected Inorganics in Soil, Stack, and Furnace Samples

Sample	Inorganic Parameters (ppm)			
	Copper	Lead	Silver	Chloride
<i>Soil samples</i>				
1	83	110	ND (<.3)†	25
2	16,000	148,000	14	32
3*	18	68	ND (<.3)	118
4*	11	37	ND (<.3)	94
5*	19	50	ND (<.3)	45
<i>Stack samples</i>				
1	39,000	37,000	46	NA‡
2	138,000	179,000	44	NA
<i>Furnace samples</i>				
1	53,000	31,000	58	85,500
2	12,000	11,000	6	185
3	2,600	6,300	55	131

*Core samples.

†Not detected at the detection limit indicated in parentheses.

‡Not analyzed.

calves previously butchered were collected and analyzed for PCBs, PBBs, arsenic, lead, copper, and molybdenum. Fresh eggs were collected and analyzed for PCBs and PBBs. A live chicken was sacrificed for pathologic examination. The liver was analyzed for levels of lead, PCBs, and PBBs; fixed lung tissue was analyzed for metals using energy dispersive X-ray analysis.

The veterinary toxicologist examined the horses, feed, and pasture at the horse boarding stable located 1.3 km northeast of the incinerator. Blood, fecal, skin, and hair samples were collected. Blood tests included a complete blood count, serum chemistry screen, liver profile, and blood lead. A post-mortem examination was performed on an 18-yr-old mare at the state university's Laboratory of Diagnostic Veterinary Medicine. Fat and liver samples were frozen in methylene chloride-rinsed aluminum foil and sent to Dr. Gross for TCDD and TCDF analyses.

RESULTS

Pollutant residues. The levels of copper, lead, silver, and chloride in the soil, stack, and furnace samples are presented in Table 1. The levels of copper, lead, and silver in the furnaces and stacks reflect their use as metal recovery incinerators. The high level of chloride from furnace No. 1 suggests that material incinerated there had contained an appreciable amount of chlorine. Soil sample No. 2, located 10 m south of the incinerator, contained the highest soil levels of copper, lead, and silver. The on-site soil samples—Nos. 1 and 2—contained higher levels of copper and lead than did the off-site and control samples.

The levels of organic compounds in the soil, furnace, and stack samples are presented in Table 2. Several polycyclic aromatic hydrocarbons, usually associated with combustion, are present in all three furnace samples, with the highest levels and the greatest variety from furnace No. 1. Many of these compounds (phenanthrene, anthracene, fluoranthene, and pyrene) were also found in soil sample No. 1, collected 50 m east of the facility. Soil sample No. 2, collected 10 m south of the facility from an area where oil had been spilled, contained 6.9 ppm of PCBs. Low levels of several phthalates, commonly used as plasticizers, were found in soil sample No. 3. Soil sample No. 4, collected from the horse pasture, contained 0.06 ppm of DDT. Except for elevated iron and total solids, the water samples had no abnormal values beyond the maximum contaminant level for public water supplies.

Total TCDD and total TCDF levels in soil sample No. 1, soil sample No. 4, furnace No. 2, stack No. 2, horse fat, and horse liver are presented in Table 3. The highest levels of TCDDs and TCDFs were found in the stack sample. Lower levels were found in the furnace and in soil sample No. 1. TCDDs and TCDFs were not detected in soil sample No. 4. TCDFs were found in both horse fat and liver. TCDDs were found in the horse fat, but not in the horse liver. No known dioxin-contaminated pesticides had been used in the area.

Medical examinations. Thirteen people residing within 1.3 km of the incinerator complained to state Environmental Protection Agency officials of adverse health effects. Five people agreed to be examined in April and May of 1980.

N.C., a 27-yr-old male, and his wife (C.C.) had resided approximately 220 m east of the incinerator since 1978. The patient complained of eye and throat irritation, dizziness, and nausea when he was exposed to smoke emitted by the incinerator. In May, 1979, he had an episode of transient neurologic dysfunction characterized by mild weakness of the right upper extremity and face along with blurred vision. In December, 1979, he had the onset of oscillopsia and was noted to have bilateral nystagmus. On physical examination the patient was found to have acne vulgaris, upbeating nystagmus in all directions of gaze, mild diminishment of alternate movement rate in the right upper extremity, and a right Babinsky sign. The following laboratory tests were within normal limits: CBC, serum chemistry group screen, serum triglycerides, serum protein electrophoresis, sed rate, syphilis serology, L.E. clot test, urinalysis, blood lead, FEP, serum PCB, and head CT scan. Lumbar puncture showed normal total protein and IgG with a normal cell count. Brainstem auditory evoked responses were normal bilaterally, but pattern shift visual evoked responses were abnormal.

C.C., a 28-yr-old female, complained of irritation of the eyes and throat, dizziness, and nausea when exposed to smoke from the incinerator. She complained of a gradual onset of frontal headache and transient visual disturbances since February, 1979. These symptoms persisted after the birth of a healthy daughter in May, 1979. On physical examination, the patient was found to have a maculopapular rash localized in the right scapular area and a III/VI systolic murmur heard at the apex and left parasternal border.

Laboratory studies, including a CBC, urinalysis, liver profile, triglycerides, urinalysis, blood lead, FCP, serum PCB, and pulmonary function tests were within normal limits.

S.D., a 52-yr-old female, had owned horse boarding stables and a riding school approximately 1.3 km north-

east of the incinerator since 1966. During the past 2 yr the patient complained of excessive tearing, rhinitis, episodic throat and chest tightness, occipital headache, and transient left-sided paresthesias. She also complained of generalized weakness, loss of appetite, and joint stiffness. On

Table 2.—Levels of Organic Compounds in Soil, Stack, and Furnace Samples

Organic Parameters	Samples								
	Soil			Stack			Furnace		
	#1	#2	#3*	#4*	#5*	#1	#2	#1	#2
Pesticides [ppm (detection limit < 1 ppm)] †	— ‡	—	—	0.06 §	—	—	—	—	—
PBBs [ppm (detection limit < .5 ppm)]	—	—	—	—	—	—	—	—	—
PCBs [ppm (detection limit ~ .5 ppm)]	—	6.9 /	—	—	—	—	—	—	—
Chemicals detected from organic scans (ppm):									
Hexachlorobenzene	—	—	—	—	—	—	—	23.8	—
Phenanthrene & anthracene	1.4	—	—	0.3	—	4.3	—	481.5	9.1
Methyl phenanthrene & methyl anthracene	—	—	—	—	—	—	—	0.4	—
Fluoranthene	0.3	—	—	—	—	0.1	—	39.3	—
Pyrene	0.3	—	—	—	—	—	—	37.8	—
Napthalene	—	—	0.1	0.1	—	—	—	—	—
Methyl naphthalene	0.1	—	—	—	—	—	—	—	0.2
2-phenyl naphthalene	—	—	—	—	—	—	—	1.4	—
Hexachlorostyrene	—	—	—	—	—	—	—	4.1	—
9H-fluoren-9-one	—	—	—	—	—	—	—	6.3	—
9, 10-anthracenedione	—	—	—	—	—	—	—	.14	—
7-(1, 1-dimethyl) 2, 3 dihydro-3, 3-dimethyl-1 H-inden-1-one	—	—	—	—	—	—	—	3.5	—
2, 6-dimethyl-2, 5-heptadiene-4-one	—	—	—	—	—	—	—	43	—
3, 5, 5-trimethyl-2-cyclohexen-1-one	—	—	—	—	—	—	—	130	—
3 hexen-2-one	—	—	—	—	—	6	—	—	—
2, 5 hexanedione	—	—	—	—	—	—	—	—	0.7
Dodecanoic acid	—	—	—	—	—	—	—	—	1.3
Hexadecanoic acid	1.5	—	—	—	—	—	—	—	1
Octadecanoic acid	0.9	—	—	—	—	—	—	—	3.8
Phenol	1.2	—	—	0.1	0.2	—	—	—	—
1-Hexacosanol	3.6	110	—	—	—	—	—	—	—
1-Hexacosene	5.8	—	—	—	—	—	—	—	—
3-Hexenal	—	64	—	—	—	—	—	—	—
Methylchlorocyclo-hexane (2 isomers)	—	—	—	—	—	—	—	61	—
N-nitrosodiphenyl-amine	—	—	0.4	—	—	—	—	—	—
Di-n-butyl phthalate	—	—	1.8	—	—	—	—	—	—
Bis (2-ethylhexyl) phthalate	—	—	#	—	—	0.1	—	—	1.5
Butylbenzyl phthalate	—	—	—	—	—	—	—	—	0.6
Diethyl phthalate	—	—	—	—	—	0.2	—	—	—
Unidentified silicone compounds	—	—	30 (6 cmpds)	1.5	9.6 (5 cmpds)	—	—	—	—
Other hydrocarbons	—	400	—	—	—	—	1.3	—	—
Other unknown compounds	—	370 (3 cmpds)	—	—	—	—	—	61 (2 cmpds)	—

*Surface samples.

†Pesticides analyzed for were: chlordane, endrin, pp'-DDT, toxaphene, heptachlor, lindane, dieldrin, and methoxychlor.

‡Compound was not detected.

§The pesticide detected was DDT.

/Arochlor 1254.

≠Level detected was not sufficiently high to quantitate.

physical examination the patient was found to have rosacea. Laboratory studies, including CBC, urinalysis, serum chemistry screen, blood lead, and FEP were within normal limits.

L.G., a 24-yr-old male, had resided with his wife (F.G.) approximately 225 m east of the incinerator since 1976. He complained of eye irritation when exposed to smoke from the incinerator, but had no other symptoms. On physical examination he was found to have an erythematous macular rash over his upper chest and neck. The patient had an elevated cholesterol level of 256 mg/100 ml and elevated triglyceride level of 319 mg/100 ml. Other laboratory tests, including serum chemistry screen, blood lead, FEP, serum PCB, and urinalysis were within normal limits.

F.G., a 24-yr-old female, complained of frequent frontal headache, blurry vision, nervousness, and an intermittent sore throat. Her past medical history was significant in that she had viral B hepatitis with four recurrences of jaundice since June, 1979. On physical examination she was found to have a macular rash over her chest and back. Laboratory tests, including a CBC, liver profile, urinalysis, blood lead, FEP, and serum PCB were within normal limits.

Veterinary examinations. Lead levels in all animal samples were within normal limits. No PCBs or PBBs were detected in the fat or liver of the various animal species at a 0.1 ppm limit of detection. The dog was found to be clinically normal. While calves at the small farm had not grown as well as previously owned calves, their rations contained a fairly poor quality hay and shelled, but uncracked corn. The two horses received adequate rations and were kept in a barn at night and suffered no noteworthy illness. The chickens were normal in outward appearance. The necropsied chicken had no significant gross lesions. On histopathology, there were accumulations of dark, irregularly shaped masses, primarily within macrophages, around airways; there were also microfocal lymphoid aggregates adjacent to the airways.

Energy dispersive X-ray analysis of the chicken's lung revealed elevated levels of aluminum, palladium, titanium, and silver. The copper content of the calves' livers was low at 6 to 16 ppm.

We could not determine whether the horses kept at the boarding stable located 1.3 km northeast of the incinerator had been receiving adequate feed. At the time of the first visit, the pasture appeared overgrazed, the oats were of poor quality, and the hay in feed bunks was in short supply. At a subsequent visit, however, the feed was plentiful and of good quality. The horses most severely affected and all those that died had been allowed to remain in the pasture located 1 km northeast of the incinerator at all times. Horses kept indoors were either unaffected or much less affected.

Several of the horses present were noticeably underweight. Three had alopecia on their dorsal aspects with exfoliation and hyperemic margins; large hair-containing crusts could be peeled away. Culture failed to recover organisms apart from *Alternaria*, a contaminant. One extremely underweight animal had paraphimosis. Three of five animals' fecal samples revealed only small strongyles from

200 to 400 eggs/g. The horse identified as the "broodmare" had small strongyles, as well as *Strongylus edentatus* and *Triodontophorus* sp. with an egg count of 1700 eggs/g. Egg counts are difficult to interpret because of the temporal variability of egg shedding by intestinal parasites. There was no clinical correlation between the animals' conditions and fecal egg counts. Complete blood counts on three animals revealed no abnormalities. Serum profiles revealed consistent decreases in albumin.

Post-mortem examination of a mare that died in June, 1980, revealed a tear in the colon near the cecum with extensive secondary peritonitis. She had appeared in good flesh at the time of the initial visit, although she had been among the severely affected original group. She had given birth to a blind foal and a stunted foal. Her fecal samples had revealed small strongyles, *Strongylus edentatus*, and *Strongylus vulgaris* with an egg count of 2200 eggs/g. On post-mortem, there were pathologic anterior mesenteric artery changes consistent with *Strongylus vulgaris* larval migration. There was a fibrous band extending from the tip of the cecum to the right body wall compressing the colon. Histopathology revealed foci of macrophage accumulation in portal areas of the liver. There was mineralization of the renal tubular cells and slight thickening of Bowman's capsules and glomerular tufts. Forty-five parts per trillion (ppt) of total TCDD was detected in the mare's fat; no TCDD was detected in the mare's liver. One hundred sixty-five ppt of total TCDF was detected in the mare's fat and 57 ppt of total TCDF was detected in the mare's liver.

DISCUSSION

Our study demonstrates that a wire reclamation incinerator can be a source of environmental contamination with TCDDs and TCDFs. These compounds may have entered the incinerator intact in the fuel or charge. An alternative explanation is that they were formed in the complex chemistry of combustion from precursors found in wire insulation. Human and animal exposure to airborne TCDDs and TCDFs appears to have been very low, as indicated by the

Table 3.—Levels of Tetrachlorodibenzo-p-Dioxins (TCDDs) and Tetrachlorodibenzofurans (TCDFs) in Soil, Furnace, Stack, Horse Fat, and Horse Liver Samples

Sample	Total TCDDs (ppt)	Total TCDFs (ppt)
Soil No. 1	21	230
Soil No. 4*	ND (< 3 ppt)†	ND (< 3 ppt)†
Furnace No. 2	58	730
Stack No. 2	410	11,600
Horse fat	45	165
Horse liver	ND (< 6 ppt)‡	57

*Surface sample.

†Not detected at a detection limit of 3 ppt.

‡Not detected at a detection limit of 6 ppt.

trace concentrations of these residues in the ash, soil, and horse tissue samples.

The most toxic of the 22 TCDD and 38 TCDF isomers are 2, 3, 7, 8 TCDD and 2, 3, 7, 8 TCDF. The LD₅₀'s of these compounds are in the order of micrograms per kilogram in many animal species. 2, 3, 7, 8 TCDD is a multi-system toxin, teratogen, fetotoxin, and carcinogen.¹⁸ The dose-response relationships of these compounds in humans and horses are largely unknown. As we were only able to measure total TCDDs and TCDFs, we do not know whether the highly toxic 2, 3, 7, 8 isomers were present in our samples.

We concluded that many of the acute human health effects in the present case were caused by exposure to air pollutants commonly found in the smoke emitted by improperly controlled wire reclamation incinerators. These pollutants include carbon monoxide, halides, nitrogen oxides, sulfur oxides, and other pyrolysis products.^{1,2} There was no evidence of lead, copper, or silver poisoning in the humans or animals. We could not establish a definite association between the human health effects and the exposure to trace amounts of TCDDs and TCDFs. One person had longstanding acne vulgaris, but neither he nor any of the others had a condition resembling chloracne. While visual disturbances, nystagmus, and polyneuropathy have been previously described in persons exposed to 2, 3, 7, 8 TCDD,¹⁸ the present exposure appears to have been much lower than in the previous cases.

The presence of trace amounts of TCDDs and TCDFs in the horse tissue is evidence of exposure rather than intoxication. The absence of TCDDs and TCDFs in the pasture soil sample suggests that inhalation may have been an important route of exposure. There are no data on the kinetics, metabolism, and critical concentrations of TCDDs and TCDFs in horses. The symptoms in the affected horses were remarkably similar to those described in a previous TCDD poisoning episode.^{19,20} In the previous episode, however, the horses had been exposed to soil contaminated with part per million levels of TCDDs; no tissue levels of TCDD were available from those horses for comparison.

TCDDs and TCDFs have previously been detected in the fly ash of municipal and industrial incinerators.^{4-6,8} Operators of wire reclamation facilities should be aware that the potential exists for the emission of these compounds from wire reclamation incinerators. Data collected to date on the toxic, carcinogenic, teratogenic, and fetotoxic potential of the 2, 3, 7, 8 isomers warrant concern over trace environmental contamination with TCDDs and TCDFs.

• • • • •

The authors wish to acknowledge the support of the U.S. Environmental Protection Agency Central Regional Laboratory, the Illinois Environmental Protection Agency Laboratory, and Cook County Hospital.

Submitted for publication July 2, 1981; accepted for publication August 22, 1981.

Requests for reprints should be sent to: Daniel Hryhorczuk, M.D., Section of Occupational Medicine, 720 S. Wolcott, Chicago, IL 60612.

• • • • •

REFERENCES

1. Danielson, J.A. 1973. Air Pollution Engineering Manual, Publication No. AP-40. Washington, D.C.: U.S. Environmental Protection Agency.
2. Kimbrough, R.D. 1972. Toxicity of chlorinated hydrocarbons and related compounds. *Arch Environ Health* 25: 125-31.
3. Petkus, E.J.; Thorp, W.T.; Kimura, G.; and Linna, L.W. 1979. Chloride and Heavy Metal Emissions from Wire Burning Incinerators. Paper presented at the 72nd Annual Meeting of the Air Pollution Control Association held June 24-29, 1979, Cincinnati, OH.
4. Olie, L.; Vermeulen, P.L.; and Hutzinger, O. 1977. Chlorodibenzo-p-dioxins and chlorodibenzofurans are trace components of fly ash and flue gas of some municipal incinerators in the Netherlands. *Chemosphere* 8: 455-59.
5. Buser, H.R., and Bosshardt, H.P. 1978. Polychlorinated dibenzo-p-dioxins, dibenzofurans, and benzenes in the fly ash of municipal and industrial incinerators. *Mitt Gebiete Lebensm Hyg* 69: 191-99.
6. Dow Chemical Company. 1978. *The Trace Chemistries of Fire—A Source of and Routes for Entry of Chlorinated Dioxins into the Environment*. The Chlorinated Dioxins Task Force, the Michigan Division, Dow Chemical (U.S.A.).
7. Kimble, B.J., and Gross, M.L. 1980. Tetrachlorodibenzo-p-dioxin quantitation in stack-collected coal fly ash. *Science* 207:59-61.
8. Tiernan, T.O.; Sulch, J.G.; Van Ness, G.F.; and Dryden, J. 1980. *Analyses of Industrial Samples for Tetrachlorodibenzo-p-dioxins (TCDD's)*, Contract No. 68-03-2830. Cincinnati, OH: U.S. Environmental Protection Agency.
9. Buser, H.R.; Bosshardt, H.P.; and Rappe, C. 1978. Formation of polychlorinated dibenzofurans (PCDFs) from the pyrolysis of PCBs. *Chemosphere* 1: 109-19.
10. Rappe, C.; Markland, S.; Buser, H.R.; and Bosshardt, H.P. 1978. Formation of polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs) by burning or heating chlorophenates. *Chemosphere* 7: 269-81.
11. Buser, H.R. 1979. Formation of polychlorinated dibenzofurans (PCDFs) and dibenzo-p-dioxins (PCDDs) from the pyrolysis of chlorobenzenes. *Chemosphere* 6: 415-24.
12. Rappe, C.; Buser, H.R.; and Bosshardt, H.P. 1979. *Polychlorinated Dibenzo-p-Dioxins (PCDDs) and Dibenzofurans (PCDFs): Occurrence, Formation, and Analysis of Environmentally Hazardous Compounds*, Paper presented at the CIPAC Symposium, June 5-6, 1979, Baltimore, MD.
13. Turner, B.D. 1970. *Workbook of Atmospheric Dispersion Estimates*, Publication No. AP-26. Washington, D.C.: U.S. Environmental Protection Agency.
14. Vik, L.F., and Byers, M.E. 1978. *Illinois Department of Transportation Air Quality Manual*. Springfield, IL: Illinois Department of Transportation.
15. U.S. Environmental Protection Agency, Central Regional Laboratory. 1980. *Determination of Total Metals in Sediments and other Solids-Total Metals*, CRL Method No. 571-598. Chicago, IL: U.S. Environmental Protection Agency.
16. U.S. Environmental Protection Agency, Central Regional Laboratory. 1980. *Method for Analyses of PCBs, Pesticides and Phthalates in Soils and Bottom Sediments*, CRL Method No. 198-207. Chicago, IL: U.S. Environmental Protection Agency.
17. U.S. Environmental Protection Agency, Central Regional Laboratory. 1980. *Standard Operating Procedure for GC/MS/DS Analysis of Non-Volatile Organic Compounds*, based on Method No. 625 Fed. Reg. 44: 233, December 3, 1979. Chicago, IL: U.S. Environmental Protection Agency.
18. Esposito, M.P.; Tiernan, T.O.; and Dryden, F.E. 1980. *Dioxins*, Publication No. 600/2-80-197. Cincinnati, OH: U.S. Environmental Protection Agency.
19. Coleman, D.C.; Kimbrough, R.D.; Liddle, J.A.; Cline, R.E.; Zack, M.M.; Barthel, W.F.; Koehler, R.C.; and Phillips, P.L. 1975. Tetrachlorodibenrodioxin: An accidental poisoning episode in horse arenas. *Science* 188: 738-40.
20. Kimbrough, R.D.; Carter, D.C.; Liddle, J.A.; Cline, R.E.; and Phillips, P.E. 1977. Epidemiology and pathology of a tetrachlorodibenrodioxin poisoning episode. *Arch Environ Health* 32: 77-86.