The Nováky Chemical Plant (Novácke chemické závody) as a source of mercury and organochlorine contaminants to the Nitra River, Slovakia

Iryna Labunska, Kevin Brigden, David Santillo
& Ruth Stringer

Greenpeace Research Laboratories
Technical Note 01/2002

June 2002

Table of contents

Publ: Greenpeace Research Laboratories, Department of Biological Sciences, University of Exeter, Exeter EX4 4PS, UK

Summary

The Nováky Chemical Plant (Novácke chemické závody), situated on the banks of the Nitra River 140 km north east of Bratislava, Slovakia, manufactures a wide range of chemicals and chemical products, including trichloroethene, PVC resin and finished PVC building products. The plant gained notoriety as the source of numerous case studies of liver cancer in workers exposed to vinyl chloride monomer (VCM), the building block of PVC, in the 1950s and 60s, and more recently the plant has been identified as one of Slovakia's pollution "hotspots".

Greenpeace collected samples in the vicinity of the Nováky plant in February 2002 in order to characterise in more detail the waste streams generated by the plant. Analyses confirmed the presence of high levels of the toxic metal mercury (up to 112 ug/l) and organochlorine contaminants in what appeared to be the main wastewater discharge to the Nitra River (to the western side of the plant). Concentrations of trichloroethene and tetrachloroethene were particularly high (21-28 mg/l and 11-14 mg/l respectively). Levels of VCM (1.9-2.5 mg/l) exceeded that which would be allowable for plants manufacturing PVC within the OSPAR (North East Atlantic) catchments. Toxic and persistent chlorinated benzenes and chlorinated butadienes were also present in the wastewater and associated sediments.

Similarly, heavily chlorinated benzenes and butadienes were a conspicuous component of solid waste collected from an open lagoon situated on the banks of the Nitra River, close to the north-western boundary. With mercury at 197 mg/kg, these solid deposits could act as an additional point source of hazardous chemicals to the Nitra and the wider environment. Indeed, a second wastewater discharge, smaller than that originating directly from the plant on the western boundary, was identified immediately below this sedimentation lagoon. A sample of wastewater from this discharge yielded a similar range of contaminants, although both mercury and the volatile organochlorine compounds were present at lower levels.

Chlorinated dioxins and furans, a common component of wastes generated by the chlorine chemical and PVC industry, were detected in both the sediments collected from the main discharge channel and in the solid waste from the lagoon, albeit at relatively low concentrations (15-18 ng/kg, parts per trillion, ppt)

Taken together, these data confirm that the Nováky Chemical Plant remains an important point source of mercury and toxic organochlorines to the Nitra River system. Given that many of the organochlorine compounds are volatile, it is likely that discharges result also in substantial losses of these harmful compounds to the atmosphere. Given that the generation of complex chlorinated waste streams is an inevitable consequence of chemical manufacture based on chlorine, including the production of PVC, the only manner in which such releases can ultimately be addressed will be to substitute current processes and products with non-chlorinated alternatives.

Introduction

The Nováky Chemical Plant (Novácke chemické závody), located 140km north-east of Bratislava, is one of Slovakia's key chemical manufacturing sites, engaged in production of a wide range of organic and inorganic products, and with a primary focus on chlorinated industrial chemicals and polyvinyl chloride (PVC) plastic. The plant generates its own supply of chlorine from electrolysis of brine and produces technical chlorine gas, trichloroethene, PVC resin and hydrogen chloride (HCl) for onward sale. In addition, the plant has the capacity to manufacture some finished PVC products, including flexible flooring, rigid window profiles and other PVC building products. Other listed products include chlorinated paraffins, as well as a range of non-chlorinated glycols and polyols (NCZ 2000).

According to company literature, the first developments of the Nováky site began in the mid 1930s, with an electrolysis plant being installed in 1940. PVC production began in the late 1940s/early 1950s and expanded rapidly over the following decades. The plant extended into finished PVC products in the 1960s and both resin and product manufacture continued to expand through the 1970s. According to company literature, current activities centre on manufacture of caustic soda and technical gases, organic chemicals based on acetylene and chlorine, PVC resin and construction products and polyvinyl acetate.

PVC is one of the mostly widely used thermoplastics in the world, with global production for 1999 estimated at almost 25 million tonnes (Stringer & Johnston 2001). PVC is made from vinyl chloride, also called vinyl chloride monomer (VCM), which in turn is manufactured by chlorinating either acetylene or ethylene. According to company information (NCZ 2000), the Nováky plant has the capacity for both processes, buying in ethylene but also generating acetylene as a by-product of calcium carbide production. VCM is a highly toxic compound, which is also explosive if mixed with air.

Beyond Slovakia, the Nováky plant is perhaps best known for its notoriety as a source of case studies of human liver cancer (angiosarcoma) resulting from prolonged worker exposure to VCM, primarily during the 1950s and 60s (Bátora *et al*. 1998). Such cases led over the years to improved monitoring and control of VCM exposure in the workplace. Nevertheless, concerns also relate to the Nováky plant as a substantial point source of pollutants to the atmosphere and to the Nitra River system. The plant is located adjacent to the Nitra River, part of the Váh river basin, and discharges liquid wastes directly to the Nitra channel. For plants such as Nováky, engaged in the manufacture of trichloroethene, PVC and other chlorinated organic compounds, the by-production of a range of chlorinated hydrocarbon contaminants is unavoidable. Accordingly, such plants commonly discharge effluents containing complex mixtures of organochlorine chemicals, many of which are acutely and/or chronically toxic and some of which are highly persistent in the environment once released.

According to the Danube Pollution Reduction Programme Final Report (DPRP 1999), which identified the Nováky plant as a pollution "hotspot", there are two wastewater outfalls to the Nitra from the plant. The first of these apparently carries wastewaters containing calcium chloride, calcium hydroxide and chlorinated hydrocarbons, following their passage through sedimentation tanks and subsequent neutralisation with hydrochloric acid. A second discharge, located further downstream, is identified as carrying wastes from a mechanical and biological waste water treatment plant (WWTP), including sewage from the plant and from the community of Nováky itself, stormwater run-off and cooling water exiting from oil traps. The precise nature of the wastes directed to the WWTP from the plant is not known. However, from the

limited information available it is appears that this second discharge is by far the greater source of chlorinated hydrocarbon contaminants from the plant to the Nitra River (estimated at 449 tonnes per year, compared to 19.4 tonnes from the designated chlorinated hydrocarbon waste stream).

Data collated for the Danube Pollution Reduction Programme (DPRP 1999) also indicate that discharges from the Nováky Chemical Plant are resulting in substantial elevations in concentration in the Nitra River for a range of pollutants, including chloride, mercury and chlorinated hydrocarbons. Indeed, mercury concentrations in river water 6km downstream from the plant have been recorded to be more than 100 times higher than background concentrations measured upstream.

Because of ongoing concerns surrounding the status of the Nováky plant as a point source pollutant hotspot, Greenpeace International visited the site in February 2002. The purpose of the visit, conducted by representatives of Greenpeace Slovakia, Greenpeace Austria and the Greenpeace Research Laboratories (Greenpeace International) was to collect up to date samples of the waste streams associated with activities at the plant for scientific analysis. It was hoped that such samples would enable more detailed characterisation of the ongoing discharges of wastes from the plant and their potential for long-term damage to the Nitra River.

Materials and methods

i) Sample collection

A total of eight samples were collected in the vicinity of the Nováky plant on 17th February 2002.

Two separate wastewater discharges to the Nitra River were identified as associated with the plant:-

- discharge from the western boundary of the plant, *via* a part-covered channel running close to the railway line, which appeared to be the main process effluent stream from the plant (west discharge); and
- discharge *via* a circular-section concrete pipe located immediately to the north west of the plant, but on the opposite bank of the Nitra River, beneath a large sedimentation lagoon (north-west discharge).

Although it seems likely that the western discharge is that identified as "outfall II" in the Danube Pollution Reduction Programme Report, and that originating beneath the sedimentation lagoon is "outfall I", it was not possible to obtain conclusive confirmation of this. Hence they are referred to below as the western (or main) discharge and the north-western (or second) discharge.

Samples of the effluents being discharged at the time and of sediments which had accumulated inside the discharge channel/pipe were collected from both locations at the point of discharge to the Nitra River (samples CEE02004 & 5, CEE02007 & 10). In the case of the western discharge, the effluent channel was subsequently traced back to the point at which it exited beneath the plant boundary fence and a further sample of both effluent and sediment collected at that location (CEE02003 & 6).

Sample code	Sample type	Location
Main discharge		
CEE02003	Wastewater	From main discharge channel at western boundary of plant, at point at which channel exits beneath boundary fence
CEE02006	Sediment	As CEE02003
CEE02005	Wastewater	From main discharge channel at western boundary of plant, at point of discharge to Nitra River
CEE02004	Sediment	As CEE02005
Second discharge		
CEE02007	Wastewater	From second discharge pipe at north-western boundary of plant, beneath sedimentation lagoon, at point of discharge to Nitra River
CEE02010	Sediment	As CEE02007
Sedimentation lagoon		
CEE02011	Solid waste	From sedimentation lagoon located to the north-west of the plant and on the opposite bank of the Nitra River
Control site		
CEE02012	Sediment	From channel of Nitra River near confluence with Lehota River, upstream from both discharges from Nováky plant

Table 1: Summary of the samples collected in the vicinity of the Nováky Chemicals Plant in February 2002

A sample of solid waste (CEE02011) was collected from the sedimentation lagoon located to the north west of the plant boundary though on the opposite bank of the Nitra River, immediately above (and probably feeding in to) the second, smaller discharge.

In addition, a single (control) sample of sediment (CEE02012) was collected from the Nitra River channel immediately upstream from both discharges and the plant itself, close to the town of Nováky and immediately downstream from the confluence with the Lehota River.

Details of the samples collected are summarised in Table 1. Sampling locations are further indicated on the map at Figure 1.

All samples were collected and stored in pre-cleaned glass bottles that had been rinsed thoroughly with nitric acid and analytical grade pentane in order to remove all heavy metal and organic residues.

At each wastewater sampling location, a 1 litre sample was collected in a screw-cap bottle, as well as a separate 125 ml sample collected in an amber bottle with a ground-glass stopper. Sediment samples were collected in 100ml bottles. All samples were kept cool and returned to the Greenpeace Research Laboratories for analysis.

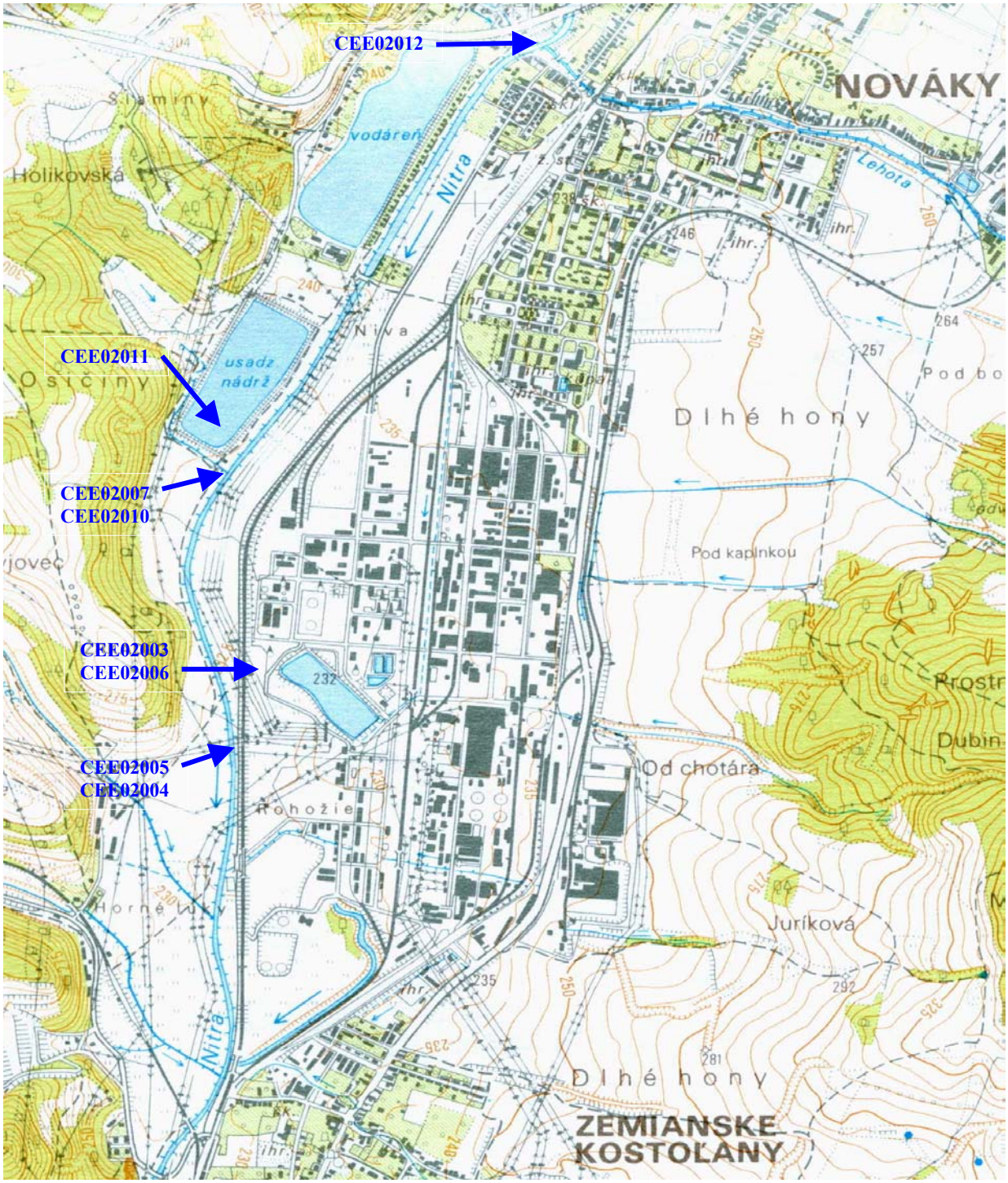

Figure 1: Locations of all sites sampled in the current study

Location	Main discharge (W)				Second discharge (NW)		Lagoon	Control
Sample Number	CEE02003	CEE02006	CEE02005	CEE02004	CEE02007	CEE02010	CEE02011	CEE02012
Sample Type	Wastewater	Sediment	Wastewater	Sediment	Wastewater	Sediment	Solid waste	Sediment
METALS								
Concentration	ug/l	mg/kg	ug/l	mg/kg	ug/l	mg/kg	mg/kg	mg/kg
Cadmium	<10	<1	<10	<1	<10	<1	<1	<1
Chromium	<20	27	<20	26	<20	3	4	18
Cobalt	<20	3	<20	7	<20	<2	<2	6
Copper	48	21	88	19	57	12	14	10
Lead	167	9	129	36	93	35	32	19
Manganese	111	171	127	222	<10	24	12	375
Mercury	112	131	28	106	2.6	27	197	2.5
Nickel	<20	12	<20	14	<20	4	<2	11
Zinc	28	64	24	94	<10	14	3	60
VOLATILE ORGANOHALOGEN COMPOUNDS								
Concentration	ug/l		ug/l		ug/l			
Chloroform	240	N/A	170	N/A	N/D	N/A	N/A	N/A
Vinyl chloride	1 900	N/A	2 550	N/A	400	N/A	N/A	N/A
Chloroethene	N/Q	N/A	N/Q	N/A	N/Q	N/A	N/A	N/A
1,1-Dichloroethene	*	N/A	*	N/A	N/D	N/A	N/A	N/A
cis-1,2-Dichloroethene	*	N/A	*	N/A	*	N/A	N/A	N/A
trans-1,2-Dichloroethene	*	N/A	*	N/A	N/D	N/A	N/A	N/A
Trichloroethene	27 970	N/A	21 540	N/A	820	N/A	N/A	N/A
Tetrachloroethene	14 280	N/A	11 500	N/A	850	N/A	N/A	N/A
1,2-Dichloroethane	*	N/A	*	N/A	*	N/A	N/A	N/A
Hexachloroethane	N/Q	N/A	*	N/A	N/D	N/A	N/A	N/A

*Table 2: Concentrations of heavy metals and volatile organochlorines in wastewater, sediment and solid waste samples, as well as an indication of other VOCs present. N/D – not detected, N/Q – identified by qualitative screen but not quantified, * - identified as present in trace quantities in selective ion mode (SIM) but below limits of quantification, N/A – not analysed.*

ii) Sample analysis

1 litre samples were used for quantitative analysis of heavy metals and for qualitative screening for organic contaminants. The separate 125ml samples were analysed quantitatively for volatile organic compounds (VOCs), specifically organochlorines. Sediment samples were analysed for heavy metals and screened for organic contaminants; VOCs were not determined for sediment samples. Detailed description of sample preparation and analytical procedures are provided in Appendix 1.

In addition to the analyses conducted within the Greenpeace Research Laboratories, two samples (sediment CEE02006, from the west discharge channel and solid waste CEE02011 from the sedimentation lagoon) were forwarded to an accredited laboratory in the UK for quantitative analysis of chlorinated dioxins and furans. Details of the analytical techniques employed can be provided on request.

Results and discussion

Concentrations of heavy metals in all samples are summarised in Table 2. For the three wastewater samples, quantitative data for those volatile organochlorine compounds identified

are also included in Table 2. Table 3 provides further information on the presence of organic contaminants in the samples, including an indication of the total number of compounds present

Location	Main discharge (W)				Second discharge (NW)		Lagoon	Control
Sample Number	CEE02003	CEE02006	CEE02005	CEE02004	CEE02007	CEE02010	CEE02011	CEE02012
Sample Type	Wastewater	Sediment	Wastewater	Sediment	Wastewater	Sediment	Solid waste	Sediment
No. of organic compounds isolated	98	95	92	64	57	34	63	51
No. identified to >90% match quality	55	63	44	31	28	11	33	18
No. organohalogens identified to >90%	36	44	31	14	14	2	16	1
OTHER (SEMI-VOLATILE) ORGANOHALOGEN COMPOUNDS								
Benzene, chloro-	1*	1*	1*	-	1*	-	-	-
Benzenes, dichloro-	1*	3	1*	2*	-	-	1*	1*
Benzenes, trichloro-	1*	1	2*	-	-	-	1*	-
Benzenes, tetrachloro-	3*	3*	2*	-	-	-	2*	-
Benzene, pentachloro-	1	1*	1*	-	-	-	1*	-
Benzene, hexachloro-	-	1*	-	-	-	-	1*	-
1,3-Butadiene, tetrachloro-	9	6	6	7	4	-	7	-
1,3-Butadiene, pentachloro-	4	3	3	2	1	-	2	-
1,3-Butadiene, hexachloro-	1	1	1	1	1*	-	1	-
Decane, 3-chloro-	1	-	-	-	-	-	-	-
7-Heptadecyne, 17-chloro-	-	-	1	-	-	-	-	-
Propane, 1,2-dichloro-	1	-	1	-	-	-	-	-
Propane, 1,1'-oxybis[3-chloro-	1	1	1	1	1	1	-	-
Propane, 2,2'-oxybis[1-chloro-	1	1	1	1	1	1	-	-
1-Propene, pentachloro-	1	-	-	-	-	-	-	-
Tetradecane, 1-chloro-	1	1	1	-	-	-	-	-

*Table 3: Summary of all other (semi-volatile) organochlorine compounds identified in wastewater, sediment and solid waste samples, plus an indication of the total number of organic compounds isolated from each sample and the number of those which could be identified. For the specific chemicals and groups listed, tabulated values indicate the number of individual isomers identified. * - compound/group identified by selective ion monitoring (SIM) only.*

and the proportions of these which were reliably identified (defined as identification to greater than 90% match quality reliability). Also shown is the number of those reliably identified compounds which were organohalogens (in this case, all organochlorines), as well as a detailed list of those specific organochlorine compounds or groups represented in each case.

i) Main discharge

Wastewater samples CEE02003 and 02005, collected at different locations along the same effluent channel, predictably yielded similar results. In both cases, concentrations of dissolved mercury (112 and 28 ug/l respectively) were particularly high, indicative of substantial releases of this toxic metal from industrial processes on site. The significant difference in concentration between the two samples may simply reflect the inherent chemical heterogeneity of the discharges. Such elevated mercury concentrations could reflect losses from a mercury cell chloralkali unit, which may be employed in the manufacture of chlorine as a feedstock for the various chlorinated products manufactured at the plant. The possibility of mercury arisings from other processes on site, including uses of mercury-based catalysts, cannot be discounted, however. For example, mercuric chloride is sometimes used in the gas-phase chlorination of acetylene (Stringer & Johnston 2001).

Background information on the environmental distribution and toxicity of mercury is presented in Appendix 2.

In addition to mercury, concentrations of lead in the wastewaters were also relatively high. Lead compounds are still widely used as stabilisers in rigid PVC products, including window profiles and other PVC building materials (Donelly 1999). The Nováky plant is engaged in the manufacture of such products, though again it is not possible to identify conclusively the specific source of the elevated lead concentrations in the wastewaters sampled. Concentrations of copper, commonly used as a catalyst in the production of ethylene dichloride (EDC) by oxychlorination, were slightly, though not substantially, elevated.

As may be expected, the sediment samples collected from the same locations as the two wastewater samples also showed evidence of substantial mercury accumulation (over 100 mg/kg at both sampling points). Given that the effluent channel originates from within the plant boundary and appears to be of artificial construction, it is likely that the sediments accumulating at the base of this channel comprise entirely of waste solids from industrial processes which have settled out over time, rather than natural river sediment which has become contaminated. This inference was further supported by the physical appearance of the sediments. Moreover, the highly turbid nature of the flowing effluent itself indicates high concentrations of suspended particulate matter which may settle out in the effluent channel or in the river channel following discharge.

Both wastewater samples also show evidence of heavy contamination with a wide range of organic compounds, especially organochlorines. Of almost 100 individual compounds which could be isolated in both cases, only around half could be identified to a high degree of reliability. The identity and, therefore, potential impacts of the other compounds will, of course, remain unknown.

The most prominent groups of organochlorine compounds present were the chlorinated ethenes (mono- to tetra- chloro-), chlorinated benzenes (mono- to penta- chloro-) and chlorinated butadienes (tetra-, penta- and hexa-). Of the more volatile organochlorines, trichloroethene (21-28 mg/l) and tetrachloroethene (11-14 mg/l) were by far the most abundant, although both vinyl chloride monomer (VCM, 1.9-2.5 mg/l) and chloroform (0.17-0.24 mg/l) were also present at substantial levels. Traces of 1,2-dichloroethane (ethylene dichloride or EDC, a key step in VCM manufacture *via* the ethylene chlorination route) were also present in these wastewaters.

Information on sources, environmental distribution and toxicity of many of these compounds and groups is provided in Appendix 2. Such contaminants are typical of waste streams from processes linked to PVC production (Johnston *et al.* 1996), an origin largely confirmed by the presence of vinyl chloride monomer itself. Moreover, chloroform, 1,2-dichloroethane and tetrachloroethene are among those chlorinated hydrocarbons reported to be detected regularly in the waters of the Nitra River at a sampling site downstream from the Nováky plant (DPRP 1999).

Another prominent component of the wastewaters and associated sediments, the chlorinated benzenes, are known precursors for the formation of chlorinated dioxins, a group of highly toxic and persistent organochlorines which are also characteristic by-products of steps in the PVC production process. The presence of dioxins in the sediment sample taken from the channel by the boundary fence (CEE02006) was confirmed by separate quantitative analysis, although the concentrations were relatively low (18.7 ppt as internationally accepted toxicity equivalents, I-TEQ) compared to those commonly found in the non-aqueous tarry wastes resulting from PVC production (often in the ug/kg [ppb, part per billion] or even mg/kg [ppm, part per million]

range, Stringer *et al.* 1995). Samples of those heavily chlorinated organic wastes were not available as part of the current study, and no information is available relating to their formation or handling on the Nováky site.

Despite relatively low dioxin concentrations in this sediment, the presence of substantial levels of other organochlorine compounds was confirmed by the organic screen analysis (Table 3). The range of contaminants present, principally the chlorinated benzenes and butadienes, was almost identical to that identified in the overlying wastewaters, as may be expected. In addition to providing a more integrated picture of contaminants released from the plant than perhaps provided by the effluent samples, these sediments may themselves act as substantial reservoirs of contaminants to the Nitra River over time if periodically suspended.

In addition to the compound groups identified above, two chlorinated propanes (1,1'-oxybis[3-chloropropane] and 2,2'-oxybis[1-chloropropane]) were also common to both wastewater and sediment samples. To the authors' knowledge, these compounds have not previously been reported as components of effluent streams from PVC production processes, although related compounds (1,1'-oxybis[1-chloroethane] and 1,1'-oxybis[2-chloroethane]) have been identified (Johnston *et al.* 1996).

Both wastewaters CEE02003 and 02005, and sediment CEE02006, also contained traces of chlorinated linear hydrocarbons (especially 1-chlorotetradecane). Although such contaminants could have arisen from a number of chlorination reaction pathways, the manufacture of chlorinated paraffins which, according to company literature, is conducted at the Nováky site, is one possible source.

ii) Second (north-west) discharge

The second discharge identified, thought to be carrying leachate or run-off from the sedimentation lagoon, was releasing only a small volume of wastewater at the time of sampling. Again, the precise origins of the wastes discharged *via* this route are not known, though it is possible that this is the outfall identified in the Danube Pollution Reduction Programme Report (DPRP 1999) as "outfall I". The pattern of contamination identified was, however, similar to that for the effluent and sediments associated with the main discharge, although concentrations were generally lower.

Wastewater sampled from this pipe (CEE02007) contained 2.6 ug/l mercury, and the sediment from inside the pipe (CEE02010), 27 mg/kg, both significant but lower than in the main discharge. Similarly, concentrations of VOCs were also lower; although tri- and tetrachloroethene were still the most abundant of those quantified, at 0.82 and 0.85 mg/l respectively these were an order of magnitude or more below those in the main discharge. Vinyl chloride was present at 0.4 mg/l.

In addition to the VOCs, both wastewater and sediment from this north-west discharge contained the two oxygenated chloropropane derivatives common to wastes from the main discharge, while the wastewater (CEE02007) also yielded several chlorinated butadienes. Together, these findings suggest a common source for at least a proportion of the wastewaters reaching the two discharge points.

Clearly both discharges identified are acting as point sources of hazardous chemical contaminants from the Nováky plant to the Nitra River. Nevertheless, the higher concentrations

of mercury and VOCs, coupled with the higher and seemingly more continuous flow of waste associated with the main (western) discharge, indicate that this is likely to be the more significant point source from the site.

iii) Solid waste from sedimentation lagoon

The pattern of high mercury and organochlorine contamination was repeated again in the sample of dewatered solid waste collected from the lagoon located to the north-west of the plant, on the opposite bank of the Nitra River, and from which the second discharge appeared to originate. At 197 mg/kg, this was the most heavily mercury contaminated waste sampled during the current investigation. Again a link to the production of chlorine and caustic through the use of mercury cell electrolysis seems likely, though other sources cannot be excluded. The range of organochlorines present, primarily chlorinated butadienes and chlorinated benzenes, is again typical for complex wastes from chlorine-based chemical manufacturing processes (Johnston *et al.* 1996, Stringer & Johnston 2001). The chlorinated propane derivatives common to all the other wastes sampled, however, could not be detected in this solid waste sample.

Residues of chlorinated dioxins and furans were detectable although, in common with the sediment from the main discharge channel, concentrations (15.8 ppt [part per trillion] I-TEQ) were lower than may have been expected for such a complex chlorinated waste. It is likely, therefore, that the more heavily chlorinated and dioxin-contaminated organic wastes generated on site are not being directed to this sedimentation lagoon.

Just as for the process origin of the wastes which are present in the lagoon, the final fate of this dewatered waste is currently unknown. Nevertheless, it is clear that while it is stored within an open lagoon adjacent to the Nitra River it is acting as a further point source of toxic and persistent contaminants to the atmosphere and the river itself *via* the adjacent discharge.

iv) Control sample

This sample of sediment from the Nitra River, collected upstream from the two identified discharges and the sedimentation lagoon, showed substantially different, and lower levels of, contamination than any of the wastes described above. At 2.5 mg/kg, mercury contamination still appears to be significant, though it is possible only to speculate on other likely upstream sources. There are a number of urban developments, and possibly other smaller industries, upstream from the Nováky site, which might contribute contaminants to the river system either directly to the Nitra River or to its tributaries. The presence of 1,4-dichlorobenzene, the only chlorinated compound identified in this upstream sample, could for example result from the widespread use of this compound as a sanitary disinfectant and deodorant (Ware 1988) and hence contamination *via* the sewers.

Although a fairly high number of other organic compounds were isolated from this sediment in total (51), the majority of those which could be identified were non-chlorinated long-chain or branched hydrocarbons. Although some may be biogenic in nature, run-off of hydrocarbons from urban areas and sewage discharges can also make substantial contributions.

v) Comparison of discharge concentrations with limits set in other regions

Despite the hazards associated with the majority of the organochlorine compounds identified as components of the wastewater in the current study, few regulatory limits apply to releases from PVC manufacture anywhere in the world. Within the European context, perhaps the most

relevant forum is the OSPAR Convention (for the protection of the marine environment of the North East Atlantic region) although, as Slovakia is not a Contracting Party, the following comparisons are for illustrative purposes only.

OSPAR Decision 98/4 (OSPAR 1998a) set limits for releases of chlorinated hydrocarbons and copper from plants manufacturing EDC and VCM, although as these are expressed in grammes per tonne of production/processing capacity and not on an effluent concentration basis, it is not possible to make a direct comparison. The only possible comparison with data from the current study is then the limits set on discharges to water of 1mg/l VCM for both suspension-PVC plants (OSPAR Decision 98/5, OSPAR 1998b) and emulsion-PVC plants (OSPAR Recommendation 2000/3, OSPAR 2000). Against these limits, levels in the main Nováky discharge of 1.9-2.55 mg/l VCM appear relatively high. It must be stressed, however, that this comparison does not imply an exceedence of any regulatory limits which may be applicable to Slovakia.

More detailed regulations apply in the US with regard to chlorinated hydrocarbons in wastewaters arising from the plastics industry under Title 40, part 414 (US GPO 2000). These limits are summarised in Table 4 for comparative purposes, along with the concentrations determined in the three wastewaters analysed in the current study.

Contaminant	US maximum daily concentration limits for discharges		Concentrations in wastewaters collected from the Nováky plant, 17th February 2002		
(All concentrations in ug/l)	with biological treatment	without biological treatment	CEE02003	CEE02005	CEE02007
Total Copper	3380	3380	48	88	57
Total Zinc	2610	2610	28	24	<10
Chloroform	46	325	240	170	N/D
Carbon Tetrachloride	38	380	N/D	N/D	N/D
Vinyl Chloride	268	172	1900	2550	400
1,1-Dichloroethene	25	60	*	*	*
trans-1,2-Dichloroethene	54	66	*	*	N/D
Trichloroethene	54	69	27 970	21 540	820
Tetrachloroethene	56	164	14 280	11 500	850
1,1-Dichloroethane	59	59	N/D	N/D	N/D
1,2-Dichloroethane	211	574	*	*	*

*Table 4: Comparison of concentrations of key contaminants in the three wastewater samples analysed in the current study with limits established for direct discharge point sources for the plastics industry under US Federal Law (Title 40, part 414). N/D – not detected, * - identified as present in trace quantities in selective ion mode (SIM) but below limits of quantification*

The particularly high levels of trichloroethene and tetrachloroethene in the main discharge from Nováky are instantly apparent, as are the elevated levels of VCM. Although, once again, this does not imply any contravention of regulatory limits (should any exist) applicable to this sector in Slovakia, it nevertheless serves as a useful basis for comparison given the paucity of other data.

It is important to stress that, given the very volatile nature of some of the more abundant organochlorines determined in the current study, emissions and losses to atmosphere are also likely to be substantial. Such releases may occur from storage, handling and processing of chlorinated chemicals on the site, as well as by volatilisation from effluent streams, treatment plants and settling lagoons. Emissions to atmosphere were not quantified in the current study.

Conclusions

The Nováky Chemical Plant is clearly acting as a point source of mercury and hazardous organochlorine chemicals to the surrounding environment, in particular the Nitra River. The complex mix of organic compounds present in the wastewater, dominated by chlorinated compounds, is typical of discharges from plants engaged in the manufacture of PVC and other chlorinated materials through the chlorination of hydrocarbons. Indeed, many of the hazardous compounds identified in the current study are inevitable by-products of the chlorine chemical industry.

It is not possible to determine from the results of the current study what the likely total discharge of mercury and organochlorines to the Nitra River is over time; indeed, this was not the purpose of the study. Nevertheless, the high concentrations of some key chlorinated compounds, especially the chlorinated ethylenes, coupled with the flow of effluent observed in the main channel at the time of sampling, indicate that the plant is a substantial source of these compounds to the Nitra. This confirms previously published data indicating the presence of these and other volatile organochlorines downstream from the Nováky plant.

Given that many of the chlorinated compounds identified in the wastewater are, in common with mercury, highly toxic to aquatic life, impacts on the physiology and ecology of freshwater organisms in the Nitra River downstream from the plant are likely (though they were not the subject of the current investigation). Moreover, given that some of the compounds identified, including the higher chlorinated benzenes and butadienes, are highly resistant to degradation, accumulation of these toxic chemicals through the food chain may also be a problem.

Legislation introduced in Europe and other regions to control emissions and discharges of hazardous chemicals from EDC, VCM and PVC production has aimed primarily at describing best practice for continued manufacture, setting discharge limits for only a very limited number of these chemicals. While there are undoubtedly further measures which could be taken to reduce losses to the environment of such chemicals from the Nováky plant, such approaches will never address fully the problem of complex waste streams containing toxic and persistent chemicals. The only real solution is to work towards the cessation of discharges, emissions and losses of such hazardous substances through progressive substitution of chlorine-chemistry and chlorinated products with non-chlorinated, non-hazardous alternatives.

References

Bátora, J., Schmidt, K., Kubik, J. & Jakubis, P. (1998) Angiosarcoma of the liver in workers professionally exposed to vinyl chloride (in Slovakia). *Central European Journal of Occupational and Environmental Medicine* 4(3): 252-264

Donelly, P.J. (1999) A regulatory and marketing update on stabilisers used in PVC. Proceedings of "PVC 99: from strength to strength" Conference, Brighton, UK, April 1999, Publ. Institute of Materials, UK: 150-159

DPRP (1999) Danube Pollution Reduction Programme, Transboundary Analysis – Final Report, June 1999, Prepared by Donald L. Graybill, GCP International Inc., USA: pp 123-125

Johnston, P.A., Stringer, R.L., Clayton, R. & Swindlehurst, R.L. (1996) Regulation of toxic chemicals in the North Sea: the need for an adequate control strategy. In: *Proceedings of the*

Scientific Symposium on the North Sea Quality Status Report 1993, 18-21 April 1994, Ebeltoft, Denmark. Publ: Danish Environmental Protection Agency, ISBN 87-7810-699-0, pp 269-274

NCZ (2000) Novácke chemické závody Annual Report, 2000

OSPAR (1998a) OSPAR Decision 98/4 on Emission and Discharge Limit Values for the Manufacture of Vinyl Chloride Monomer (VCM) including the Manufacture of 1,2-dichloroethane (EDC), OSPAR Commission for the Protection of the Marine Environment of the North East Atlantic, Sintra 1998. http://www.ospar.org/eng/dra/welcome.html

OSPAR (1998b) OSPAR Decision 98/5 on Emission and Discharge Limit Values for the Vinyl Chloride Sector, Applying to the Manufacture of Suspension-PVC (s-PVC) from Vinyl Chloride Monomer (VCM), OSPAR Commission for the Protection of the Marine Environment of the North East Atlantic, Sintra 1998. http://www.ospar.org/eng/dra/welcome.html

OSPAR (2000) OSPAR Recommendation 2000/3 on Emission and Discharge Limit Values for the Manufacture of Emulsion PVC (e-PVC) from Vinyl Chloride Monomer, OSPAR Commission for the Protection of the Marine Environment of the North East Atlantic, Sintra 1998. http://www.ospar.org/eng/dra/welcome.html

Stringer, R.L., Costner, P. & Johnston, P.A. (1995) PVC manufacture as a source of PCDD/Fs. *Organohalogen Compounds* 24: 119-123

Stringer, R.L. & Johnston, P.A. (2001) *Chlorine and the Environment: An Overview of the Chlorine Industry*. Publ. Kluwer Academic Publishers, Dordrecht, Netherlands. ISBN 0-7923-6797-9, 429pp

USGPO (2000) Title 40- Protection of the Environment. Chapter 1, Part 414- Organic chemicals, plastics and synthetic fibers. Publ: US Government Printing Office, http://www.access.gpo.gov/nara/cfr/waisidx_00/40cfr414_00.html

Ware, G.W. (Ed.)(1988). Ortho-, meta- and para- dichlorobenzene. *Rev. Environ. Contam.Toxicol.* 106: 51-56

APPENDIX 1 ANALYTICAL METHODOLOGY

A1.1 Organic analysis

Preparation of samples for organic screen analysis

All solvents were of High Purity Grade (PRAG or low haloform). Glassware used in extraction and clean up procedures was cleaned in detergent, rinsed with tap water and deionised water, dried in the oven overnight at 105^0C and rinsed three times with low haloform pentane.

Solid Samples

In preparation for analysis of extractable organic compounds, approximately 30g (wet weight) was weighed and transferred to a clean 100 ml glass bottle. Samples were spiked with deuterated naphthalene (an internal standard) at a concentration of 4.7 mg/kg. 15ml of pentane was added, followed by 5ml of acetone. All samples were then sonicated for 2 hours.

Extracts were decanted, filtered through a pre-cleaned hydrophobic phase separator filter and collected in reagent tubes. The samples were then acidified to pH 2 with 10% nitric acid. Following this, a second portion of 15ml of pentane was added, followed by 5ml of acetone and the extraction procedure repeated. Finally, both extracts obtained for each sample were combined and evaporated to a volume of approximately 3ml. 3ml of iso-propanol and 3ml of fresh prepared TBA-reagent (mixture of 3% tetrabutylammonium hydrogen sulfate and 20% sodium sulfite anhydrous in deionised water) were added to the concentrated extract and the mixture shaken for 1 minute. After shaking, 20ml of deionised water was added to reagent tube and the phases were allowed to separate. Finally, the organic layer was transferred with a Pasteur pipette into a pentane pre-washed Florisil column. The compounds were eluted with a 95:5 mixture of pentane: toluene and the eluent evaporated down to a volume of 2 ml under a stream of analytical grade nitrogen. 1-Bromonaphthalene was then added at a concentration of 10mg/l to provide an indication of GC/MS performance.

Aqueous Samples

Prior to the extraction, samples were spiked with deuterated naphthalene (an internal standard) at a concentration of 10mg/l. 20ml of pentane were added and the sample agitated for 2 hours on a bottle roller to maximise contact between solvent and sample.

After separation of the phases, the solvent extract was filtered through a hydrophobic phase separator filter and collected in a reagent tube. The aqueous sample was acidified to pH 2 with 10% nitric acid, a second portion of 20ml pentane was added and the extraction procedure repeated. Both extracts were combined and cleaned up as described above for solid samples.

Chromatographic Analysis

Compound	Ions to monitor
Benzene, 1,3-dichloro-	146, 148, 111, 75
Benzene, 1,4-dichloro-	146, 148, 111, 75
Benzene, 1,2-dichloro-	146, 148, 111, 75
Benzene, 1,3,5-trichloro-	180, 182, 145, 74

Phenol, 2,4-dichloro-	162, 164, 63, 98
Benzene, 1,2,4-trichloro-	180, 182, 145, 109
Benzene, 1,2,3-trichloro-	180, 182, 145, 109
Dichlorvos	109, 185, 79, 47
Benzene, 1,2,3,5-tetrachloro-	216, 214, 218, 179
Benzene, 1,2,4,5-tetrachloro-	216, 214, 218, 179
Benzene, 1,2,3,4-tetrachloro-	216, 214, 218, 179
Benzene, pentachloro-	250, 252, 248, 215
alpha-HCH	181, 183, 219, 217
Benzene, hexachloro-	284, 286, 282, 249
Simazine	200, 215, 202, 173
beta-HCH	181, 183, 219, 217
gamma-HCH	181, 183, 219, 217
delta-HCH	181, 183, 219, 217
o,p'-DDE	246, 248, 318, 176
p,p'-DDE	246, 318, 246, 316
o,p'-DDD	235, 237, 165, 199
p,p'-DDD	235, 237, 165, 199
o,p'-DDT	235, 237, 165, 199
p,p'-DDT	235, 237, 165, 199

List of compounds in the Standard I used for SIM analysis

Organic compounds were identified qualitatively using Gas Chromatography Mass Spectrometry (GC-MS). Instrumentation was a Hewlett Packard (HP) 5890 Series II gas chromatograph, interfaced with a HP Chem-Station data system and linked to a HP 5972 Mass Selective Detector operated in scan mode. The identification of compounds was carried out by computer matching against a HP Wiley 275 library of 275,000 mass spectra combined with expert interpretation. Extracts were also analysed using selective ion monitoring (SIM) method against two standard solutions. The compounds contained in Standard I and Standard II are listed below. All individual standards were obtained from Sigma Aldrich Co. Ltd., Supelco, UK.

Results are reported as either reliably or tentatively identified. Match qualities of 90% or greater against HP Wiley 275 library or identification confirmed against standard compounds (using retention times and mass-spectra obtained during calibration) are assumed to give reliable identifications. Tentative identification refers to qualities between 51% and 90% against HP Wiley 275 library only. Analytes yielding match qualities of 50% or less are assumed to be unidentified.

Compound	**Ions to monitor**
Phenol, 2-chloro-	128, 64, 92, 39
Phenol, 2-methyl-	108, 79, 90, 51
Phenol, 3-methyl- and 4-methyl-	108, 107, 79, 77
Phenol, 2,5-dichloro-	162, 164, 63, 99
Phenol, 2,3-dichloro-	162, 126, 63, 99
Phenol, 4-chloro-	128, 65, 130, 100
Phenol, 2,6-dichloro-	162, 164, 63, 98
Butadiene, hexachloro-	225, 190, 260, 118
Phenol, 2,3,5-trichloro-	196, 198, 160, 97

Phenol, 2,4,5-trichloro-	196, 198, 97, 132
Phenol, 3,5-dichloro-	162, 164, 99, 63
Phenol, 2,3,6-trichloro-	196, 198, 97, 132
Phenol, 3,4-dichloro-	162, 164, 99, 63
Atrazine	200, 215, 202, 173
Phenol, pentachloro-	266, 268, 264, 165
Chlordane I	373, 375, 272, 237
Chlordane II	373, 375, 272, 237
PCB-153	360, 362, 290, 218
PCB-138	360, 362, 290, 292
PCB-180	394, 396, 324, 252

List of compounds in the Standard II used for SIM analysis

Volatile Organic Compounds (VOCs) analysis

For volatile organic compound analysis, no sample preparation was required. The original sample was sub-sampled immediately after opening. Three portions of 10ml each were transferred into 20ml headspace vials and sealed with Teflon-lined vial caps. One sub-sample was used for the organic screen analysis to evaluate the whole range of volatile compounds in the sample. The second sub-sample was analysed using Selective Ion Monitoring (SIM) method to detect the VOCs listed in the Table below. The third sub-sample was used for quantification of the detected compounds with an external standard using SIM method. All standard compounds were obtained from Sigma-Aldrich Co. Ltd./Supelco UK.

Name of compound	Target ion	Qualifying ions
1,1,1-Trichloroethane	97	61, 26, 117
1,1-Dichloroethane	63	27, 83, 98
1,1-Dichloroethene	61	96, 26, 35
Carbon tetrachloride	117	35, 47, 82
Chlorobenzene	112	77, 51, 38
Chloroform	83	47, 35, 118
cis-1,2-Dichloroethene	61	96, 26, 35
1,2-Dichloroethane	62	27, 49, 98
Hexachlorobutadiene	225	260, 190, 118
m- & p-Xylene	91	106, 77, 51
o-Xylene	91	106, 77, 51
Tetrachloroethene	166	129, 94, 47
Toluene	91	39, 65, 51
trans-1,2-Dichloroethene	61	96, 26, 37
Trichloroethene	130	95, 60, 35
Vinyl chloride	27	62, 37, 47

List of volatile organic compounds and appropriate ions that were monitored during GC/MS analysis using SIM method.

A1.2. Heavy Metal Analysis

Preparation of samples for heavy metal analysis

All chemicals were of High Purity Aristar Grade. All glassware was cleaned in detergent, rinsed with tap water and deionised water, soaked in 10% nitric acid overnight, rinsed with deionised water and dried in an oven.

Solid Samples

Samples were air dried until weighing readings became constant (approx. 5 days). They were then crushed using a pestle and mortar until homogenous and sieved through a 2-mm mesh.

0.5 g of sample was weighed into a glass 100 ml boiling tube and to this 10 ml of deionised water was added, followed by 7.5 ml of concentrated hydrochloric acid and 2.5 ml of concentrated nitric acid. The samples were digested at room temperature overnight prior to being placed onto a Gerhardt Kjeldatherm digestion block (40 space) connected to a Gerhardt Turbosog scrubber unit (filled with 10% w/v sodium hydroxide). The samples were then refluxed at 130°C for four hours.

After cooling to ambient temperature, the digests were filtered into volumetric flasks, diluted with deionised water, made up to a volume of 50 ml and mixed. A Standard Reference Material, BCR-143 (trace elements in a sewage sludge amended soil), certified by the Commission of the European Communities, Brussels and a blank sample, were prepared with the batch of samples. All were prepared in 15% v/v hydrochloric acid and 5% v/v nitric acid.

Aqueous samples

On arrival, 100ml of sample was transferred to a clean glass bottle and acidified with nitric acid (10% v/v). 50 ml of this solution was subsequently transferred to a 100ml boiling tube, placed onto the Gerhardt Kjeldatherm digestion block and refluxed at 130°C for four hours. After cooling to ambient temperature, the digests were filtered into volumetric flasks, diluted with deionised water, made up to a volume of 50 ml and mixed.

Inductively Coupled Plasma Atomic Emission Spectrometry (ICP-AES)

Following preparation, samples were analysed by ICP-AES, using a Varian Liberty-100 Sequential Spectrometer. The following metals were quantified directly: manganese, chromium, zinc, copper, lead, nickel, cobalt and cadmium. A multi-element instrument calibration standard was prepared at a concentration of 10 mg/l, matrix matched to the samples (i.e. in 15% v/v hydrochloric acid and 5% v/v nitric acid for solid samples, and 10% v/v nitric acid for aqueous samples). The calibration was validated using a quality control standard (8 mg/l), prepared internally from different reagent stocks. Any sample exceeding the calibration range was diluted accordingly, in duplicate, and re-analysed.

Mercury (Hg) was determined using Cold Vapour Generation ICP-AES. Hg (II) was reduced to Hg (0) i.e. a vapour, following reduction of the samples with sodium borohydride (0.6% w/v), sodium hydroxide (0.5% w/v) and hydrochloric acid (10 molar). The vapour was carried in a stream of argon into the spectrometer. Two calibration standards were prepared, at 10 ug/l and 100 ug/l, matrix matched to the samples (i.e. in 15% v/v hydrochloric acid and 5% v/v nitric

acid for solid samples, and 10% v/v nitric acid for aqueous samples). The calibration was validated using a quality control standard (80 ug/l), prepared internally from different reagent stock. Any sample exceeding the calibration range was diluted accordingly, in duplicate, and re-analysed.

APPENDIX 2 TOXICOLOGICAL OUTLINES FOR KEY CONTAMINANTS IDENTIFIED

Mercury (Hg)

Mercury is a naturally occurring metal, which can exist in several forms. Metallic mercury is a shiny, silver-white, odorless liquid, which forms a colorless, odorless gas if heated. Mercury combines with other elements such as chlorine, sulfur, or oxygen to form inorganic mercury compounds or salts, which are usually white solids. Mercury also combines with carbon to make organic mercury compounds, the most common of which is methylmercury.

Mercury is a very rare metal, found in the earth's crust at concentrations frequently below 0.03 mg/kg (Alloway 1990). It is estimated that the total amount of mercury released to the atmosphere from natural sources is of the order of 2700-6000 tonnes/year, mainly from volcanic activity and biogenic processes. This compares with an estimated anthropogenic load of 3000 tonnes/year (WHO 1991). The major sources of air emissions of inorganic mercury (metallic mercury and inorganic mercury compounds) are from mining ore deposits, burning coal and waste and from manufacturing plants.

Uses of mercury are extensive, due to its unique properties of fluidity, its high surface tension and its ability to alloy with other metals. It is primarily used in the electrical industry in alkaline batteries, electric lamps and wiring and switching devices, such as thermostats and cathode tubes. It is also used in the chemical industry as a catalyst; for example to form compounds such as vinyl chloride (VCM) via the acetylene process (Matthews 1996), and as the cathode in the chlor-alkali electrolytic separation of brine to produce chlorine and sodium hydroxide (caustic soda). At present, there is an estimated 12 000 tonnes of pure mercury in use in mercury cells within the EU alone.

Metallic mercury is used in dental restorations, and in medical equipment, such as thermometers and manometers (ATSDR 1997).

Mercuric oxide and mercuric sulphide are used as pigments in paints; and gold mining operations utilise mercury to extract gold from ores through amalgamation. Until 30 years ago, mercury compounds were used extensively as pharmaceuticals and agrochemicals, e.g. as components of antiseptics, diuretics, skin lightening creams, laxatives, anti-syphilitic drugs, fungicides, bactericides, wood and felt preservatives. However, due to the high toxicity of mercury, most of these applications are banned in most parts of the World (ATSDR 1997).

Mercury is found at very low concentrations in many aquatic and terrestrial ecosystems (see Table 1). Due to the fact that mercury is the only metal that can exist as both a liquid and a vapour at ambient temperatures, its environmental behaviour differs from that of most other toxic elements (ATSDR 1997, WHO 1989). Mercury can exist in three valence states, Hg (0) metallic, Hg (I) mercurous and Hg (II) mercuric. In the atmosphere, elemental mercury is by far the most common form, and, as a vapour, it is responsible for the long-range, global cycling of mercury. In addition, though to a far lesser degree, mercury may be associated with particulates, which can be moved by dry or wet deposition (ATSDR 1997, WHO 1991).

Environmental Matrix	Concentration	Reference
Seawater (open ocean)	0.001-0.004 ug/l 0.02 ug/l	Bryan & Langston 1992, WHO 1989 ATSDR 1997
Freshwater	<0.005 ug/l	ATSDR 1997

Marine sediment	0.02-0.1mg/kg	WHO 1989
Freshwater sediment	0.2-0.35 mg/kg	Salomons & Forstner 1984
Soil	0.02-0.625 mg/kg	Alloway 1990, WHO 1989
Fish	<0.2 mg/kg	ATSDR 1997

Table 1. Background concentrations of mercury found in water, sediments, soil and fish.

In the aquatic environment, mercury is most commonly found in the mercuric (II) state. Once released, its fate is dominated by rapid adsorption to soluble and particulate organic material; followed by flocculation, precipitation and final accumulation in the bottom sediment. Because of the strength with which mercury is bound to sediment, exchange back to the water column is generally slight, although it can be accelerated in saline waters, and in the presence of high concentrations of sulphide (anoxic conditions) (ATSDR 1997, Bryan & Langston 1992). Dredging or re-suspension of bed materials may cause short-term release of mercury, although levels of dissolved metal quickly return to pre-disturbance values. Mercury accumulation from sediments may therefore be a dominant pathway for uptake in aquatic organisms and accounts for relatively high concentrations in deposit feeders, in both freshwater and marine systems (Bryan & Langston 1992).

Inorganic mercury can be converted to an organic form by micro-organisms indigenous to soils, fresh water and marine sediments. The most common form of organic mercury is methylmercury (MeHg), which is soluble, mobile and quick to enter the aquatic food chain. Increased levels of mercury in the environment can increase the amount of methylmercury that these bacteria produce. The selective retention of MeHg at each step in the food chain, relative to inorganic mercury, is related to its high lipid solubility, its long biological half-life, and the increased longevity of top predators (Bryan and Langston 1992). As a result, MeHg provides one of the rare examples of metal biomagnification in food chains (ATSDR 1997, WHO 1989). For example, concentrations in carnivorous fish at the top of freshwater and salt water food chains (e.g., pike, tuna, and swordfish) are biomagnified 10,000-100,000 times the concentrations found in ambient waters (ATSDR 197). The significance of this bioaccumulation is that it is generally the most important source of human, non-occupational mercury exposure (ATSDR 1997, WHO 1989).

Exposure to mercury generally occurs from breathing contaminated air, ingesting contaminated water and food and having dental and medical treatments.

Mercury is an extremely toxic, non-essential trace metal, having no biochemical or nutritional function. Biological mechanisms for its removal are poor, and, as mentioned above, mercury is the only metal known to biomagnify i.e. progressively accumulate though the food chain (WHO 1989).

Acute inhalation of high levels of mercury vapour may cause nausea, vomiting, diarrhoea, increases in blood pressure or heart rate, skin rashes, eye irritation, corrosive bronchitis and pneumonitis, and may be associated with central nervous system (CNS) effects such as tremor or increased excitability (ATSDR 1997, Goyer 1996). With chronic exposure, the major effects are on the CNS (tremor, spasms, loss of memory, increased excitability, severe depression, personality changes, even delirium and hallucination), although renal damage, associated with chronically exposed workers, has also been shown (Ratcliffe *et al*. 1996, Goyer 1996). These effects have also been reported in animal studies (ATSDR 1997).

Acute exposure to high levels of mercury salts, or chronic low-dose exposure, is directly toxic to the kidney (Zalups & Lash 1994). In addition, nausea and diarrhoea may result after swallowing large amounts of inorganic mercury salts, and some nervous system effects have also been recorded (ATSDR 1997, WHO 1989).

Exposure to MeHg has resulted in permanent damage to the CNS, kidneys, and the developing foetus. The levels of MeHg that result in these effects are not usually encountered by the general population, however they were encountered by the population of Minamata, in Japan, who were exposed to high levels of MeHg from eating contaminated fish and seafood collected from the Bay (ATSDR 1997). Symptoms such as brain damage, numbness of extremities, and paralysis, along with the loss of hearing, speech and sight were reported (D'Itri 1991). The problem of methylation of past and present inorganic mercury discharges continues, and the long retention time of mercury by sediments delays the elimination of contamination for many years (Harada 1997, Barbosa 1997, Akagi *et al.*. 1995, Bryan &Langston 1992, D'Itri 1991).

There are inadequate human cancer data available for all forms of mercury. Mercuric chloride has caused increases in several types of tumors in rats and mice, while methylmercury increased kidney tumors in male mice. The USEPA has determined that mercuric chloride and methyl mercury are possible human carcinogens (ATSDR 1997)

Studies on the aquatic toxicity of mercury are numerous, and again show that MeHg is more toxic than any of the inorganic forms. (WHO 1989)

Inorganic mercury is toxic to fish at low concentrations. The 96-h LC_{50}s vary between 33-400 ug/l for freshwater fish and are higher for salt-water fish; with organic mercury compounds being more toxic to both (Bryan & Langston 1992, WHO 1989).

Studies have reported a wide range of adverse reproductive effects in fish exposed to elevated levels, including prevention of oocyte development in the ovary and spermatogenesis in the testis of freshwater fish. Reductions in embryo survival and hatching success of *Fundulus heteroclitus* has also been reported, along with reductions in growth and an increase in deformities in trout (WHO 1989). Blindness and reduced respiratory rate have also been found in rainbow trout, bass and roach exposed to high levels of mercury (WHO 1989).

High incidences of abnormalities have also been observed in seabirds, abnormalities that seem to correlate with mercury residues in tissues. Even at sites apparently remote from contamination, elevated mercury concentrations have been determined in the liver and kidneys of fish-eating seabirds (Bryan and Langston 1992).

Council Directive 80/778/EEC relating to the quality of water intended for human consumption. A maximum permissible concentration of 1 ug/l is set (EEC 1980). The US Environment Protection Agency (USEPA) and Food and Drug Administration (FDA) set a maximum permissible level for drinking water of 2 ug/l (ATSDR 1997). The Food and Drug Administration (FDA) has set a maximum permissible level of 1 ppm of methylmercury in seafood (ATSDR 1997).

Relating to mercury emissions, the international body which controls the pollution of the North-east Atlantic has recommended that existing mercury cell chlor-alkali plants be phased out as soon as practicable, and that they should be phased out completely by 2010 (PARCOM 1990). This has been reinforced in the Sintra Statement at the 1998 Ministerial Meeting of the OSPAR

Commission (OSPAR 1998a) in which mercury has been selected for priority action, and included in Annex 2 of the OSPAR Strategy with regard to Hazardous Substances (OSPAR 1998b). This agreement states that environmental concentrations of naturally occurring hazardous substances should be reduced to near background levels within the next 25 years.

References

Akagi, H., Malm, O., Kinjo, Y., Harada, M., Branches, F.J.P, Pfeiffer, W.C. & Kato, H. (1995) Methylmercury pollution in the Amazon, Brazil. The Science of the Total environment 175: 85-95

Alloway, B.J. (1990) Heavy metals in soils. John Wiley and Sons, Inc. New York, ISBN 0470215984

ATSDR (1997) Toxicological profile for Mercury on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service

Barbosa, A.C. (1997) Mercury in Brazil: present or future risks? Journal of the Brazilian Association for the Advancement of Science 49,1/2: 111-116

Bryan, G.W. & Langston, W.J. (1992) Bioavailability, accumulation and effects of heavy metals in sediments with special reference to United Kingdom estuaries: a review. Environmental Pollution 76: 89-131

D'Itri, F.M. (1991) Mercury contamination: what we have learned since Minamata. Environmental Monitoring and Assessment 19: 165-182

EC (1999) Integrated Pollution Prevention and Control (IPPC) Draft best available techniques reference document on the chlor-akali industry. February 1999, 160pp

EEC (1980) Council Directive 80/778/EEC of 15 July 1980 relating to the quality of water intended for human consumption OJ L 30.8.1980, 229:11

Goyer, R.A. (1996) Toxic effects of metals. In Casarett & Doull's Toxicology. The Basic Science of Poisons, Fifth Edition, Klaassen, C.D. [Ed]. McGraw-Hill Health Professions Division, ISBN 0071054766

Harada, M. (1997) Neurotoxicity of methylmercury; Minamata and the Amazon. In Mineral and Metal Neurotoxicology. Yasui, M., Strong, M.J., Ota, K. and Verity, M.A.[Eds]. CRC Press Inc., ISBN 0849376645

Matthews, G. (1996) PVC: Production, properties and uses. Publ: The Institute of Materials, London

OSPAR (1998a) OSPAR Convention for the Protection of the Marine Environment of the North-East Atlantic. Sintra Statement, Ministerial Meeting of the OSPAR Commission, Sintra 22-23 July 1998

OSPAR (1998b) OSPAR Convention for the Protection of the Marine Environment of the North East Atlantic. OSPAR Strategy with regard to Hazardous Substances, Reference no. 1998-16, OSPAR 1998/14/1/Annex 34, Ministerial Meeting of the OSPAR Commission, Sintra 22-23 July 1998

PARCOM (1990) PARCOM decision 90/3 of 14 June 1990 on reducing atmospheric emissions from existing chlor-alkali plants

Ratcliffe, H.E., Swanson, G.M. & Fischer, L.J. (1996) Human exposure to mercury: a critical assessment of the evidence of adverse health effects. Journal of Toxicology and Environmental Health 49: 221-270

Salomons, W. & Forstner, U. (1984) Metals in the hydrocycle. Springer-Verlag, Berlin, Heidelberg, New York, Tokyo, ISBN 3540127550

World Health Organisation (1989) Mercury. Environmental Health Criteria 86. ISBN9241542861

World Health Organisation (1991) Inorganic mercury. Environment Health Criteria 118. ISBN9241571187

World Health Organisation (1993) Guidelines for drinking water quality. Volume 1: Recommendations. ISBN 9241544600

Zalups, R.K. & Lash, L.H. (1994) Advances in understanding the renal transport and toxicity of mercury. Journal of Toxicology and Environmental Health 42: 1-44

1,2-Dichloroethane (EDC)

Ethylene dichloride (EDC) is more properly called 1,2-dichloroethane. It is a pleasant-smelling, colourless, volatile liquid, which does not persist long in the environment but is both hazardous and toxic. It is highly flammable and may pose an explosion hazard. Violent reactions can take place with aluminium, nitric acid or ammonia. Its primary use is in the manufacture of vinyl chloride, which accounts for 96% of its consumption in the USA (Reed 1993).

1,2-Dichloroethane (EDC) is produced by chlorination or oxychlorination of ethene. It is one of the leading bulk chemical products and production nearly trebled in the decade to 1987. The

1988 EDC production capacity was about 31 million t y^{-1} (9 000 kt in North America, 10 000 kt in Western Europe and 3 000 kt in Japan) (Snedecor 1993). 96% of US production is used in the production of vinyl chloride monomer (VCM) used in the manufacture of PVC (Reed 1993). Other applications include solvent manufacture, ethyleneamine preparation and addition to fuel anti-knock mixtures (Snedecor 1993). EDC is regulated by the USEPA because of its contribution to the formation of tropospheric ozone (Sullivan 1997).

The production of EDC results in the generation of chlorinated toxic distillation residues called heavy ends. These are contaminated with dioxin (EA 1997, ICI 1994) and concentrations can reach part per million levels (Stringer *et al*. 1995).

Because of its volatility, the most usual route of exposure is via inhalation, though it can also be absorbed through the skin or gastro-intestinal tract (IPCS 1995). It is one of the more toxic chlorinated solvents and can be toxic at concentrations too low to be detected by smell (Snedecor 1993). Inhalation or ingestion of large amounts by humans can impact the central nervous system, liver, kidneys, lungs and cardiovascular system (IPCS 1995). It can irritate the eyes, nose and throat, upset the nervous and gastrointestinal systems, cause dizziness, nausea and vomiting, and may damage the liver, kidney and adrenal gland. It is distributed to all tissues of the body and can pass both the blood/brain barrier and the placenta. EDC is classified by the IARC in Group 2B (possibly carcinogenic to humans) and the US Department of Health and Human Services classifies it as reasonably anticipated to be a human carcinogen (USDHHS 2000).

References

EA (1997) Regulation of dioxin releases from the Runcorn operations of ICI and EVC. UK Environment Agency Information Report.

ICI (1994) Report to the Chief Inspector HMIP authorisation AK6039 Improvement Condition part 8, Table 8.1, item 2: Formation of dioxins in oxychlorination, significance for human health and monitoring proposals. ICI Chemicals and Polymers report NWJP/BMTD, 27th April 1994, 16pp

IPCS (1995) 1,2-Dichloroethane (second edition). International Programme on Chemical Safety Environmental Health Criteria 176, Publ: World Health Organisation, ISBN 92 4 157176 4, 148pp.

Reed D.J. (1993) Chlorocarbons, -hydrocarbons (survey). In: Kroschwitz, J.I. & Howe-Grant, M. (Eds). The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition. Publ. Wiley-Interscience, N.Y. Volume 5, pp 1017-1028

Snedecor, G (1993) Other chloroethanes. In: Kroschwitz, J.I. & Howe-Grant, M. (Eds), The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition. Publ: Wiley-Interscience, N.Y., Vol. 6, pp.11-36

Stringer, R.L., Costner, P. & Johnston, P.A. (1995) PVC manufacture as a source of PCDD/Fs. Organohalogen Compounds 24: 119-123

Sullivan, D.A. (1997) Solvents, industrial. In: Kroschwitz, J.I. & Howe-Grant, M. (Eds). The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition. Publ. Wiley-Interscience, N.Y. Vol. 22, pp 529-571

USDHHS (2000) 9th report on carcinogens. Publ: US Department of Health and Human Services, 334pp.

Vinyl chloride

Vinyl chloride (also known as vinyl chloride monomer or VCM) is a colourless, sweet smelling gas under normal conditions. Vinyl chloride manufacture accounts for 36% of global chlorine consumption (Chemical Week 1998). In the US in 1992, 98% of vinyl chloride was used to make PVC (ATSDR 1997). Approximately 95% of the VCM produced globally has an end use in polymer or copolymer applications, of which by far the largest is PVC production. Non-polymeric uses occur in the synthesis of a variety of industrial compounds and in the manufacture of vinylidene chloride and tri- and tetra-chloroethene. Total world capacity of

VCM in 1996 was approximately 25 500 kt y^{-1}, of which about 54% was concentrated in the United States and Western Europe (Cowfer & Gorensek 1997).

In the wider environment, VCM is not persistent. It is degraded in a few hours by the action of light and reactions with the atmospheric OH radical. If released to water, vinyl chloride volatilises rapidly to the air. However, in soil, vinyl chloride migrates rapidly to groundwater. Here it is far more persistent, with a maximum estimated degradation half-life approaching eight years (Howard *et al.* 1991).

Vinyl chloride is an extremely hazardous substance. Mixed with air, it can be explosive and it causes a wide variety of toxic effects in humans and animals. It has been known for over 20 years that it causes cancer in humans. It was classified as known to be a human carcinogen by the US government in 1980. The cancer most strongly associated with vinyl chloride is angiosarcoma of the liver in the occupationally exposed and, although the evidence is less strong, other studies have shown elevated levels of cancers of the brain and nervous system, lung and respiratory tract and the lymphatic/haemapoietic system. There are less well supported indications of cancers at still further sites (ATSDR 1997). Retrospective analysis of histories of individuals with angiosarcomas at different sites suggest that exposure to vinyl chloride, PVC and other polymeric materials may have been a factor (Rhomberg 1998).

The most usual route of exposure to vinyl chloride is through inhalation. Vinyl chloride appears to cause emphysema, dyspnea, pulmonary lesions and a number of other lung problems (ATSDR 1997). Exposure to high levels of vinyl chloride can cause Raynaud's phenomenon, where the blood circulation in workers' fingers is damaged so that they become white and painful in cold conditions. This illness is sometimes followed by resorption of the bones in the tips of the fingers or lesions on bones in other parts of the body (ATSDR 1997).

Workers have also been reported to die more frequently from cardiovascular and cerebrovascular disease (e.g. heart attacks and strokes). Vinyl chloride can also reduce the blood's ability to clot normally (ATSDR 1997).

Vinyl chloride is narcotic and inhalation can cause dizziness, headaches, drowsiness or unconsciousness, euphoria, memory loss, visual and/or hearing disturbances, sleep disturbance, nausea, irritability and nervousness. Damage to the nervous system manifests itself in peripheral neuropathy with tingling, pain or numbness in the fingers (ATSDR 1997). Also seen are toxic effects on the immune systems, livers, spleens, thyroid function, eyes and skin of workers. Anorexia (weight loss) has been reported and there are some indications that vinyl chloride can affect the reproductive systems of both men and women (ATSDR 1997).

To protect workers, many countries have set maximum limits for vinyl chloride in air. These range from 1ppm (2.6mg/m^3) in the USA to 7ppm (18.2 mg/m^3) in Finland, Germany and the UK. These limits are below odour thresholds (IPCS 1999).

The high toxicity of vinyl chloride has lead to it being banned for domestic use in both the USA and the European Community. The US ban dates from 1974 (USDHHS 2000). EC directive 76/769/EEC controls the sales and use of dangerous substances within the European Community. When this directive was brought in in 1976, it banned the use of vinyl chloride as an aerosol propellant on the basis of its hazards to human health (EEC 1976). Vinyl chloride was one of the first three substances controlled under this legislation, a measure of the hazard is represents.

VCM can be produced by the hydrochlorination of ethyne (acetylene), but currently over 90% of world production is carried out by the thermal cracking of 1,2-dichloroethane (EDC) (Cowfer & Gorensek 1997). It is then polymerised to PVC by the suspension, emulsion or mass process.

Substance	Emission to:	Limit value
VCM	air	80 g per tonne of s-PVC produced
VCM	water (after effluent stripper, before secondary treatment)	1 mg VCM per litre 5 g VCM per tonne s-PVC produced
COD	water (at outlet of effluent water treatment plant)	125 mg per litre for single plants 250 mg per litre for combined plants*
suspended solids	water (at outlet of effluent water treatment plant)	30 mg suspended solids per litre

*Limit values for suspension polymerisation PVC plants as set by the OSPAR Convention. *single plants manufacture only suspension-polymerisation PVC (s-PVC); combined plants are those where s-PVC is produced as part of an industrial site, where other chemical processes are being carried out.*

Emission limits for the ethylene dichloride/vinyl chloride production and PVC polymerisation using the suspension process are given in OSPAR decisions 98/4 and 98/5 respectively. Plants built after February 1999 would have to adhere to these limits immediately, but plants already in existence would have until the beginning of 2006 to meet the EDC/VCM limits and until the beginning of 2003 to meet the polymerisation plant limits. The European Commission has also put forward a proposed Decision which would incorporate these limits into EC law (EC 1999).

In the USA, federal legislation has been designed to control the emissions of vinyl chloride from plastics manufacturers. Title 40, Part 414 specifies a range of limits depending on the type of discharge and measurement period (USGPO 2000). These are summarised below.

Type of discharge	Maximum concentration for any one day	Maximum for any monthly average
Direct discharge point sources that use end-of-pipe biological treatment	268 ug/l	104 ug/l
Direct discharge point sources that do not use end-of-pipe biological treatment	172 ug/l	97 ug/l
Indirect source point discharges	172 ug/l	97 ug/l

References

ATSDR (1997) ATSDR's toxicological profiles on CD-ROM, CRC Press, Boca Raton, FL.

Chemical Week (1998) Polyvinyl chloride. Chemical Week 160(42): 36

Cowfer, J.A. & Gorensek, M.B. (1997) Vinyl chloride. In: Kroschwitz, J.I. & Howe-Grant, M. (Eds). The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition. Publ. Wiley-Interscience, N.Y. Volume 24, pp 851-882

EC (1999) Proposal for a Council Decision concerning the approval, on behalf of the Community, of OSPAR Decision 98/4 on emission and discharge limit values for the manufacture of vinyl chloride monomer and of OSPAR Decision 98/5 on emission and discharge limit values for the vinyl chloride sector (1999/C 158/04) COM(1999) 190 final – 99/0098 (CNS). OJ C 158, 4.6.1999: 19-32

EEC (1976) Council Directive 76/769/EEC of 27 July 1976 on the approximation of the laws, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations. OJ L 262, 27.9.76: 201-203

Howard, P.H., Boethling, R.S., Jarvis, W.F., Meylan, W.M. & Michalenko, E.M. (1991) Handbook of environmental degradation rates. Publ: Lewis Publishers, ISBN 0-87371-358-3, 725pp

IPCS (1999)Vinyl Chloride. Environmental Health Criteria 215. Publ: WHO, ISBN 92 4 157215 9, 356pp.

Rhomberg, W. (1998) Exposure to polymeric materials in vascular soft-tissue sarcomas. International Archives of Occupational and Environmental Health 71: 343-347

USDHHS (2000) 9th report on carcinogens. Publ: US Department of Health and Human Services, 334pp.

USGPO (2000) Title 40- Protection of the Environment. Chapter 1, Part 414- Organic chemicals, plastics and synthetic fibers. Publ: US Government Printing Office, http://www.access.gpo.gov/nara/cfr/waisidx_00/40cfr414_00.html

Chloroform

Chloroform is a heavy, colourless, non-flammable liquid. It has a characteristic pleasant, sweet odour and a sweetish burning taste (CEC 1986).

It has been extensively used in the past as an anaesthetic (Snyder & Andews 1996). Currently the largest use of chloroform is to make HCFC-22, an ozone-depleting refrigerant (Holbrook 1993). The Montreal Protocol, the international legislation which protects the ozone layer, has set targets for reducing the use of HCFC-22, but it will not be totally phased out until 2030 (UNEP 1997).

Chloroform is the most abundant of the trihalomethanes (THMs) which are generated as by-products during water disinfection using chlorine-containing compounds (Oxenford 1996, ATSDR 1997, Health Canada 1996). Additionally it can be formed in washing machines into which chlorinated bleach has been added (Shepherd & Corsi 1996), in the natural waters where chlorine-containing effluents have been discharged (Mills *et al.* 1998). Exposure to chloroform may occur when breathing contaminated air, drinking contaminated water or through skin contact (Weisel & Chen 1994; Weisel & Jo 1996). Water is possibly now the major source of environmental exposure to chloroform.

Chloroform has been specified by the International Agency for Research on Cancer in the Group 2B as possibly carcinogenic to humans (IARC 1998). Investigations on animals have shown that the main target organs for carcinogenicity from chloroform are liver, kidney and/or intestine (Dunnick & Melnik 1993, Snyder & Andews 1996, Chiu *et al.* 1996). A guideline value of 200ug/l was calculated to correspond to an excess lifetime cancer risk of 10^{-5} by the World Health Organisation (WHO 1993). There are four contaminants included in this group: chloroform, bromodichloromethane, dibromochloromethane and bromoform (Oxenford 1996).

It is not known whether chloroform causes reproductive effects or birth defects in people, but animal studies have shown that miscarriages occurred in rats and mice that breathed air containing 30–300 ppm chloroform during pregnancy and also in rats that ate chloroform during pregnancy. Offspring of rats and mice that breathed chloroform during pregnancy had birth defects. Abnormal sperm were found in mice that breathed air containing 400 ppm chloroform for a few days (ATSDR 1997).

The levels of chloroform found in treated drinking water depends upon water treatment practice, age of the water, water temperature (Health Canada 1996) and can vary in the range from less than 1ug/l to 200ug/l (Wallace 1997, Health Canada 1996). Levels less than 10ug/l

were found in US rural ground water (Wallace 1997). A mean value of 84ug/l was reported for surface waters (if detected) in the same survey.

Chloroform evaporates easily into the air. Most of the chloroform in air breaks down eventually, but it is a slow process. The breakdown products in air include phosgene and hydrogen chloride, which are both toxic (ATSDR 1997). It is poorly absorbed to soil and can travel through soil to groundwater where it can persist for years. Chloroform dissolves easily in water and some of it may break down to other chemicals (ATSDR 1997).

The presence of chloroform (as an organohalogen compound) in groundwater is controlled by European Community Environmental Legislation. Article 3 of EC Council Directive 80/68/EEC of 17 December 1979 on the protection of groundwater against pollution caused by certain dangerous substances (EEC 1979) and amended later (EEC 1991) says that Member States shall take necessary steps to prevent the introduction into groundwater of substances in List I and organohalogen compounds are among the groups of the compounds listed there.

The quality objective of 12ug/l for the aquatic environment (including inland surface waters, estuary waters, internal coastal waters other than estuary waters and territorial waters) is set for chloroform by the EC Council Directive 86/280/EEC (EEC 1986) and amended in 1988 (EEC 1988).

The EC Council Directive 76/769/EEC (EEC 1976) which was last amended in 1996 (EEC 1996) restricts marketing and use of chloroform. Chloroform may not be used in concentrations equal to or greater than 0.1% by weight in substances and preparation placed on the market for sale to the general public and/or in diffusive applications such as in surface cleaning and cleaning of fabrics.

In the USA, federal legislation has been designed to control the emissions from plastics manufacturers. The Environmental Protection Agency Code of Federal Regulations, Title 40, Part 414 specifies a range of limits depending on the type of discharge and measurement period (USGPO 2000). These are summarised below.

Type of discharge	Maximum concent-ration for any one day	Maximum for any monthly average
Direct discharge point sources that use end-of-pipe biological treatment	46 ug/l	21 ug/l
Direct discharge point sources that do not use end-of-pipe biological treatment	325 ug/l	111 ug/l
Indirect source point discharges	325 ug/l	111 ug/l

References

ATSDR (1997) ATSDR's Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. CRC Publishers

CEC (1986) Chloroform. In: Organochlorine solvents: health risk to workers. Publ: Commission of the European Communities, ISBN 0-85186-078-8: 17-53

Chiu, N., Orme-Zavaleta, J., Chiu, A., Chen, C., DeAngelo, A., Brattin, W. & Blancato, J. (1996) Characterization of cancer risk associated with exposure to chloroform. Environmental Carcinogenesis & Ecotoxicology Reviews. Journal of Environmental Science and Health C14(2): 81-104

Dunnick, J.K & Melnik, R.L. (1993) Assessment of the carcinogenic potential of chlorinated water: experimental studies of chlorine, chloramine and trihalomethanes. Journal of the National Cancer Institute 85(10): 818-823

EEC (1976) Council Directive 76/769/EEC of 27 July 1976 on the approximation of laws, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations. OJ L 262: 201-203
EEC (1979) Council Directive 80/68/EEC of 17 December 1979 on the protection of groundwater against pollution caused by certain dangerous substances. OJ L 020: 43-48
EEC (1986) Council Directive 86/280/EEC of 12 June 1986 on limit values and quality objectives for discharges of certain dangerous substances included in List I of the Annex to Directive 76/464/EEC. OJ L 181: 16-27
EEC (1988) Council Directive 88/347/EEC of 16 June 1988 on limit values and quality objectives for discharges of certain dangerous substances included in List I of the Annex to Directive 76/464/EEC. OJ L 158: 35-41
EEC (1991) Council Directive 91/692/EEC of 23 December 1991 standardizing and rationalizing reports on the implementation of certain Directives relating to the environment. OJ L 377: 48-54
EEC (1996) Commission Directive 96/55/EEC of 4 September 1996 adapting to technical progress for the 2nd time Annex I to Council directive 76/769/EEC of 27 July 1976 on the approximation of lows, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations (chlorinated solvents). OJ L 231: 20-21
Health Canada (1996) A one-year survey of halogenated disinfection by-products in the distribution system of treatment plant using three different disinfection processes. Published by authority of the Minister of Health, Canada. ISBN 0-662-25172-5, 59p.
Holbrook, M.T. (1993) Chloroform. IN: Kroschwitz, J.I. & Howe-Grant, (Eds). The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition .Publ. Wiley-Interscience, N.Y. Volume 5, pp 1051-1062
IARC (1998) Overall evaluation of carcinogenicity: an updating of IARC Monographs Volumes 1 to 42. Chloroform. Supplement 7, p.152
Mills, W.B., Lew, C.S. & Loh, J.Y. (1998) Predictions of potential human health and ecological risks from power plant discharges of total residual chlorine and chloroform into rivers. Environmental Science and Technology 32(14): 2162-2171
Oxenford, J.L. (1996) Disinfection by-products: current practices and future directions. In: Disinfection by-products in water treatment: the chemistry of their formation and control. Minear, R.A. & Amy G.A. [Ed] CRC Press, Inc. ISBN 1-56670-136-8: 3-16
Shepherd, J.L. & Corsi, R.L. (1996) Chloroform in indoor air and wastewater: the role of residential washing machines. Journal of Air and Waste Management Association 46: 631-642
Snyder, R. & Andews, L.S. (1996) Toxic effects of solvents and vapours. In: Casarett and Doull's Toxicology: The Basic Science of Poisons. Fifth Edition. Klaasen, C.D, Amdur, M.O. & Doull, J. [Ed]. Mc-Graw-Hill, ISBN 0-07-105476-6, pp.737-772
USGPO (2000) Title 40- Protection of the Environment. Chapter 1, Part 414- Organic chemicals, plastics and synthetic fibers. Publ: US Government Printing Office, http://www.access.gpo.gov/nara/cfr/waisidx_00/40cfr414_00.html
UNEP (1997) The 1987 Montreal Protocol on Substances that Deplete the Ozone Layer, as adjusted and amended by the second Meeting of the Parties (London, 27-29 June, 1990) and by the fourth Meeting of the parties (Copenhagen, 23-25 November 1992) and further adjusted by the seventh Meeting of the Parties (Vienna, 5-7 December 1995) and further adjusted and amended by the ninth Meeting of the Parties (Montreal, 15-17 September 1997)
Wallace, L.A. (1997) Human exposure and body burden for chloroform and other trihalomethanes. Critical Reviews in Environmental Science and Technology 27(2): 113-194
Weisel, C.P. & Chen, W.J. (1994) Exposure to chlorination by-products from hot water uses. Risk Analysis 14(1): 101-107
Weisel, C.P. & Jo W-K. (1996) Ingestion, inhalation and dermal exposures to chloroform and trichloroethene from tap water. Environmental Health Perspectives 104(1): 48-51
WHO (1993) Guidelines for drinking-water quality. Vol.1: Recommendations. Second Edition, ISBN 92 4 154460 0, 188p.

Hexachloro-1,3-butadiene

Hexachloro-1,3-butadiene (HCBD) is a colourless liquid with a turpentine-like odour. This compound is not found naturally in the environment. Hexachlorobutadiene was first prepared in 1877 by the chlorination of hexyl oxide (ATSDR 1997). HCBD either commercially manufactured or is known to be a by-product of the manufacture of chlorinated hydrocarbons such as tetrachloroethene, trichloroethene, and carbon tetrachloride (ATSDR 1997, US EPA,

1986, Johnston *et al.* 1994, Botta *et al.* 1996). It is present in relatively small quantities (up to 5% and sometimes over hexachlorobutadiene in the chlorolysis process of 1,2-dichloroethane for the production of carbon tetrachloride and tetrachloroethene), but, because of the huge production of volatile chlorinated solvents, the amounts of hexachlorobutadiene from the different processes are relevant (Botta *et al.* 1996). It is also reported as a contaminant in technical formulations of pentachlorophenol, used widely as a wood preservative (Goodrichmahoney *et al.* 1993).

Hexachlorobutadiene is used as a chemical intermediate in the manufacture of rubber compounds (ATSDR 1997). Lesser quantities of hexachlorobutadiene are used as a solvent, a fluid for gyroscopes, a heat transfer liquid, hydraulic fluid, and as a chemical intermediate in the production of chlorofluorocarbons and lubricants. Small quantities are also used as a laboratory reagent. In the international market, Russia is reported to be one of the major users of hexachlorobutadiene, where it is used as a fumigant on grape crops (ATSDR 1997).

Hexachlorobutadiene is a wide spread environmental contaminant. It can exist in the atmosphere as a vapour or adsorbed to airborne particulate matter. HCBD has been found in wastewater from the chlorine industry, leachate from landfills and hazardous waste sites and also in air, soils, surface water and sediments (ATSDR 1997, Santillo & Labounskaia 1997a&b, Choudhary 1995). It has also been detected in fly ash from the incineration of HCBD-containing hazardous wastes (Choudhary 1995).

Hexachlorobutadiene is toxic compound. Acute toxic effects may include the death of animals, birds, or fish, and death or low growth rate in plants. Acute effects are seen two to four days after animals or plants come in contact with it (US EPA 1986, Choudhary 1995). Chronic toxic effects may include shortened lifespan, reproductive problems, lower fertility, and changes in appearance or behaviour. Hexachlorobutadiene has high acute and chronic toxicity to aquatic life (US EPA 1986).

The kidney was found to be an important target organ for HCBD (Jonker *et al.* 1996, Choudhary 1995). If ingested, HCBD concentrates in the kidney, interferes with fundamental processes of cell respiration and can, as a result of conjugation with other compounds in the body, react with DNA resulting in cell death or the development of tumours (Choudhary 1995, ATSDR 1997). Short and longer-term exposure to very low doses via food induced kidney and liver damage in laboratory animals, with juveniles more at risk than adults. It was shown that human exposures to HCBD were associated with highly significant increases in a number of individual and summed bile acid measures in the study of the possible hepatic effects of different chlorinated compounds including HCBD (Driscoll *et al.* 1992)

The International Agency for Research on Cancer concluded that there was limited evidence of HCBD carcinogenicity in rats and classified HCBD as compound not classifiable as to human carcinogenicity (Group 3) (IARC 1999).

References

ATSDR (1997) ARSDR's Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. CRC Publishers

Botta, D. Dancelli, E. & Mantica, E. (1996) A case history of contamination by polychloro-1,3-butadiene. Environmental Science and Technology 30(2): 453-462

Choudhary, G. (1995) Human health perspecives on environmental exposure to hexachlorobutadiene: a review. Environ. Carcino. & Ecotox. Revs. C13(2): 179-203

Driscoll, T.R., Hamdam, H.H., Wang, G., Wright, P.F.A. & Stacey, N.H. (1992) Concentrations of individual serum or plasma bile acids in workers exposed to chlorinated aliphatic hydrocarbons. British Journal of Industrial Medicine 49: 700-705

Goodrichmahoney, J.W., I.P. Murarka, L.J. Hocombe & M.E. Horn (1993). Pentachlorophenol-treated wood poles and crossarms - toxicity characteristic leaching procedure (tclp) results. Environment International 19(6): 535-542.

IARC (1999). 1,1,2,3,4,4-Hexachlorobutadiene. IARC Monographs. International Agency for Research on Cancer. Geneva. Vol. 73, p. 277

Johnston, P.A., R.L. Stringer, R. Clayton & R.J. Swindlehurst (1994). Regulation of toxic chemicals in the North Sea: The need for an adequate control strategy. North Sea Monitor June 1994: 9-16.

Jonker, D., Woutersen, R.A. & Feron, V.J. (1996) Toxicity of mixutes of nephrotoxicants with similar or dissimilar mode of action. Food and Chemical Toxicology 34: 1075-1082

Santillo, D. & Labounskaia, I. (1997a) Petkim: Organic Analytical report 1: Analysis of marine sediments and seawater from locations adjacent to the main wastewater discharges of the Petkim chemical complex, Aliaga, Turkey. Greenpeace Research Laboratories Technical note 08/97

Santillo, D. & Labounskaia, I. (1997b) Petkim: Organic analytical report 2: Analysis of wastes generated by integrated chemical production processes, including the manufacture of VCM and chlorinated organic solvents, at the Petkim chemical complex, Aliaga, Turkey. Greenpeace Research Laboratories Technical Note 09/97

US EPA (1986) Factsheets for regulated chemicals: Hexachlorobutadiene. Office of Environmental Health Hazard Assessment.

Trichloroethene

Trichloroethene is a clear, colourless, heavy liquid with a pleasant, sweetish, chloroform-like odour, and a sweet burning taste. It easily evaporates at room temperature. Trichloroethene is non-flammable but it can decompose at high temperature in the air producing hydrochloric acid, phosgene and other compounds (CEC 1986). Other names for trichloroethene are trichloroethylene and ethylene trichloride.

Trichloroethene is a chlorinated solvent which was produced since the 1920s in many countries by chlorination of ethylene or acetylene (IARC 1995). Another method of producing trichloroethene is by direct chlorination of ethylene dichloride to form trichloroethene and tetrachloroethene (ATSDR 1997). Oxychlorination of chlorinated wastes from PVC manufacturing (EDC tars) can be used to make chlorinated solvents including trichloroethene, but this method also results in the generation of large quantities of dioxins (EA 1997, ICI 1994). It has been used in vapour degreasing in the 1920s, later it was introduced for use in dry cleaning but its use in this industry has declined sharply since the 1950s. In the 1990s, 80-90% of trichloroethene worldwide was used for degreasing metals (IARC 1995). Use for all applications in Western Europe, Japan and the United States in 1990 was about 225 thousand tonnes. Other uses include processes requiring strong solvent action to dissolve rubbers and resins and in the manufacture of paints, lacquers and adhesives and oils from animal and vegetable matter. The textile industry also uses trichloroethene as a solvent in waterless dying and finishing operations (ASTDR 1997). It is also used as a chain terminator in the production of polyvinyl chloride, has also been used in fire extinguishing and fire retarding applications (CEC 1986).

Trichloroethene is a photochemically reactive compound and it can decompose in the presence of free radicals. Stabilisers such as epoxides (including the carcinogen epichlorohydrin) or combinations of epoxides, esters and amines are added to commercial trichloroethene to prevent it becoming acidic towards equipment and degreased materials (CEC 1986).

Trichloroethene is not thought to occur naturally in the environment. However, it is present in most underground water sources and many surface waters as a result of the manufacture, use, and disposal of the chemical (Hughes *et al.* 1994, WHO 1993, ATSDR 1997).

Trichloroethene was found at different levels in drinking water supplies: in Galicia (Spain) in a range of concentrations between 1 and 11.6ug/l (Freiria-Gandara *et al.* 1992), in drinking water samples from Zagreb, Croatia, betwen 0.69 to 35.90 ug/l (ATSDR 1997), and up to 212 ug/l in the drinking water from two villages in Finland (Vartiainen *et al* 1993). It was also detected in the breath of people after inhalation and dermal exposure to tap water contaminated with trichloroethene (Weisel & Jo 1996). The World Health Organisation guideline value for trichloroethene in drinking water is 70ug/l, assuming that this route provides 10% of exposure (WHO 1993). The maximum contaminant level (MCL) for trichloroethene in drinking water set by US Environmental Protection Agency is 5 ug/l (US EPA 1999). Some surface waters have been found to contain more than 400ug/l of this contaminant (CEC 1986).

Trichloroethene dissolves slightly in water. Under anaerobic conditions (for example in ground water) trichloroethene may degrade to more toxic compounds including vinyl chloride (Klier *et al.* 1999, Su & Puls 1999, WHO 1993). Trichloroethene itself could be formed as a degradation product of another chlorinated volatile compound – 1,1,2,2-tetrachloroethane (Loran & Olsen 1999).

Trichloroethene may absorb to particles in water, which will cause it to eventually settle to the bottom sediment (ATSDR 1997). Soil contamination by trichloroethene has been reported with concentration ranging from below 1mg/kg to approximately 1500mg/kg (Ho *et al.* 1999). Trichloroethene evaporates less easily from the soil than from water and may remain in soil for a long time.

Among the most heavily trichloroethene-exposed people are those working in the degreasing of metals, who are exposed by inhalation. Breathing large amounts of trichloroethene may cause impaired heart function, coma, and death. Breathing it for long periods may cause nerve, lung, kidney and liver damage. Inhaling small amounts for short periods of time may cause headaches, lung irritation, dizziness, poor co-ordination, and difficulty concentrating. Drinking large amounts of trichloroethene may cause nausea, liver and kidney damage, convulsions, impaired heart function, coma, or death. Drinking small amounts of trichloroethene for long periods may cause liver and kidney damage, nervous system effects, impaired immune system function and impaired foetal development in pregnant women, although the extent of some of these effects is not yet clear. Skin contact with trichloroethene for short periods may cause skin rashes (ATSDR 1997).

Trichloroethene has been classified by International Agency for Research on Cancer in Group 2A (probably carcinogenic to humans) (IARC 1995) and the US authorities reasonably anticipate it to be a human carcinogen (USDHHS 2000). The most important observations are the elevated risk for cancer of the liver and biliary tract and the modestly elevated risk for non-Hodgkin's lymphoma. Trichloroethene-contaminated groundwater may marginally increase a risk for non-Hodgkin's lymphoma. It has been shown to induce lung and liver tumours in various strains of mice (Fisher & Allen 1993, WHO 1993). It is also a weakly active mutagen in bacteria and yeast (WHO 1993).

Trichloroethene is in the first list of priority substances of the Commission Regulation (EC) No 1179/94 (EC1994) which is a part of the Council Regulation (EEC) No 793/93 on the evaluation and control of the risks of existing substances (EEC 1993).

Discharges of trichloroethene during several industrial processes (including production of tetrachloroethene and trichloroethene) and usage of trichloroethene for degreasing of metals are controlled by the European Community Legislation and special provisions are set relating to trichloroethene in the Council Directive 90/415/EEC (EEC 1990). The quality objective of 10ug/l for the aquatic environment (including inland surface waters, estuary waters, internal coastal waters other than estuary waters, and territorial waters) is set for trichloroethene in the same Directive.

Article 5(6) of the recent document (EC 1999) concerning limitation of emissions of volatile organic compounds due to the use of organic solvents in certain activities and installations says that substances or preparations which, because of their content of volatile organic compounds classified as carcinogens, mutagens, or toxic to reproduction, shall be replaced as far as possible by less harmful substances or preparations within the shortest possible time.

References

ATSDR (1997) ATSDR's Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. CRC Publishers

CEC (1986) Trichloroethylene. In: Organochlorine solvents: health risk to workers. Publ: Commission of the European Communities. ISBN 0-85186-078-8: 93-130

EA (1997) Regulation of dioxin releases from the Runcorn operations of ICI and EVC. Information Report. Environmental Agency Licence GD03177G0003. Pub. By Crown. 36pp.

EC (1994) Commission Regulation (EC) No 1179/94 of 25 May 1994 concerning the first list of priority substances as foreseen under Council Regulation (EEC) No 793/93. OJ L 131: 3-4

EC (1999) Council Directive 1999/13/EC of 11 March 1999 on the limitation of emission of volatile organic compounds due to the use of organic solvents in certain activities and installations. OJ L 85: 1-22

EEC (1990) Council Directive of 27 July 1990 amending Annex II to Directive 86/280/EEC on limit values and quality objectives for discharges of certain dangerous substances included in list I of the Annex to Directive 76/464/EEC. OJ L 219:49-57

EEC (1991) Council Directive 91/692/EEC of 23 December 1991 standardizing and rationalizing reports on the implementation of certain Directives relating to the environment. OJ L 377: 48-54

EEC (1993) Council Regulation (EEC) No 793/93 of 23 March 1993 on the evaluation and control of the risks of existing substances. OJ L 84: 1-75

Fisher, J.W. & Allen, B.C. (1993) Evaluating the risk of liver cancer in humans exposed to trichloroethylene using physiological models. Risk Analysis 13(1): 87-95

Freiria-Gandara, M.J., Lorenzo-Ferreira, R.A., Alvarez-Devesa, A. & Bermejo, F. (1992) Occurance of halogenated hydrocarbons in the water supply of different cities of Galicia (Spain). Environmental Technology 13: 437-447

Ho, S.V., Athmer, C., Sheridan, P.W., Hughes, B.M., Orth, R., McKenzie, D., Brodsky, P.H., Shapiro, A.M., Sivavec, T.M., Salvo, J., Schultz, D., Landis, R., Griffith, R. & Shoemaker, S. (1999) The Lasagna technology for *in situ* soil remediation. 2. Large field test. Environmental Science and Technology 33(7): 1092-1099

Hughes, K., Meek, M.E. & Windle, W. (1994) Trichloroethylene: Evaluation of risks to health from environmental exposure in Canada. Environmental Carcinogenesis and Ecotoxicology Reviews. Journal of Environmental Science and Health 12(2): 527-543

IARC (1995) Trichloroethylene. IARC Monographs Vol.63, p.75

ICI (1994) Report of the Chief Inspector HMIP authorisation AK6039 Improvement Condition part 8, table 8.1, item 2: Formation of dioxines in oxychlorination, significance for human health and monitoring proposals. ICI Chemicals and Polymers report NWJP/BMTD, 27th April, 1994, 16pp.

Klier, N.J., West, R.J. & Donberg, P.A. (1999) Aerobic biodegradation of dichloroethylenes in surface and subsurface soils. Chemosphere 38(5): 1175-1188

Loran, M.M. & Olsen, L. (1999) Degradation of 1,1,2,2-tetrachloroethane in a freshwater tidal wetland: field and laboratory evidence. Environmental Science and Technology 33(2): 227-234

Su, C. & Puls, R.W. (1999) Kinetics of trichloroethene reduction by zerovalent iron and tin: pretreatment effect, apparent activation energy, and intermediate products. Environmental Science and Technology 33(1): 163-168
USDHHS (2000) 9th report on carcinogens. Publ: US Department of Health and Human Services, 334pp.
US EPA (1999) Current drinking water standards. National primary and secondary drinking water regulations. Office of Ground Water and Drinking Water. US Environmental Protection Agency
Vartiainen, T., Pukkala, E., Rienoja, T., Strandman, T., Kaksonen, K. (1993) Population exposure to trichloroethene and tetrachloroethene and cancer risk - 2 cases of drinking-water pollution. Chemosphere 27(7): 1171-1181
Weisel, C.P. & Jo, W-K (1996) Ingestin, inhalation, and dermal exposures to chloroform and trichloroethene from tap water. Environmental Health Perspectives 104(1): 48-51
WHO (1993) Guidelines for drinking-water quality. Vol.1: Recommendations. Second Edition, ISBN 92 4 154460 0, 188p.

Tetrachloroethene

Tetrachloroethene is a clear liquid, which is heavier than water, with a sweet chloroform-like odour. Tetrachloroethene does not burn but it can produce poisonous gases in fire including hydrogen chloride and phosgene (US EPA 1989). Other names for tetrachloroethene include perchloroethylene and tetrachloroethylene.

Tetrachloroethene was first prepared in 1821 by Faraday by thermal decomposition of hexachloroethane (Hickman 1993). Tetrachloroethene is one of the most important chlorinated solvents worldwide and it has been produced commercially since the early 1900s. About 513 thousand tonnes were used in all applications in Western Europe, Japan and the United States in 1990 (IARC 1995). Tetrachloroethene is typically produced as a co-product with either trichloroethene or carbon tetrachloride from hydrocarbons, partially chlorinated hydrocarbons, and chlorine (Hickman 1993). Oxychlorination of chlorinated wastes from PVC manufacturing (EDC tars) can be used to make chlorinated solvents including tetrachloroethene, but this method also results in the generation of large quantities of dioxins (EA 1997, ICI 1994). Most of the tetrachloroethene produced was used for the dry cleaning garments and smaller amounts were used for degreasing and in the production of chlorofluorocarbons (CFCs) (IARC 1995). However, this latter application will have been reduced since the Montreal Protocol banned CFC-11 and CFC-12 over most of the world (UNEP 1997). Tetrachloroethene was used in the textile industry for processing, finishing and sizing (US EPA 1998). Other uses include: insulating/cooling fluid in electric transformers; in typewriter correction fluids, as veterinary medication against worms, and it was once used as a grain fumigant (US EPA 1998).

Tetrachloroethene is well known environmental contaminant. It has been detected in air, lakes, rainwater, seawater, rivers, soil, food and human tissues (ATSDR 1997, Bauer 1990, CEC 1986). It has also been found in drinking water at concentration in the range from 10 ug/l to 180 ug/l (Bauer 1990, Freiria-Gandara *et al.* 1992, Vartiainen *et al.* 1993, CEC 1986). Contamination of well water with the concentration of 375ug/l was recorded at a waste disposal site due to tetrachloroethene leaching through soil (CEC 1986). The World Health Organisation guideline value for tetrachloroethene in drinking water is 40ug/l assuming that 10% of exposure comes from this source (WHO 1993). The maximum contaminant level (MCL) for tetrachloroethene in drinking water set by US Environmental Protection Agency is 5 ug/L (US EPA 1999). Tetrachloroethene has also been detected in the effluents from industrial plants and refineries and in sewage treatment plant effluents before and after chlorination (Santillo *et al.* 1997, US EPA 1989, CEC 1986).

The majority of the produced tetrachloroethene (80-85%) is lost in the atmosphere as a result of evaporation during production, storage and use (US EPA 1994, CEC 1986) and only 1% is released to water. Releases of tetrachloroethene to the environment are primarily from alkali and chlorine industries (US EPA 1998). In 1992, more than 12.3 million pounds (5584.2 tonnes) were released to the atmosphere, 10 thousand pounds (4.54 tonnes) to surface water, 13 thousand pounds (5.9 tonnes) to underground injection sites and 9 thousand pounds (4.07 tonnes) to land from U.S. facilities (US EPA 1994). Once released into the environment tetrachloroethene can undergo transformation. The degradation of tetrachloroethene through biotic mechanism includes the formation of lesser chlorinated compounds including trichloroethene, cis- and trans-1,2-dichloroethene, and vinyl chloride (Klier *et al.* 1999). In the air a photochemical degradation occurs with trichloroacethyl chloride as a major degradation product and phosgene a lesser one (CEC 1986).

The major route of human exposure to tetrachloroethene is from inhalation of contaminated urban air, especially near point sources such as dry cleaners, drinking contaminated water from contaminated aquifers (US EPA 1998), drinking water distributed in pipelines with vinyl liners (Webler & Brown 1993), and inhalation of contaminated occupational atmospheres in metal degreasing and dry cleaning industries (US EPA 1998).

Exposure to very high concentrations of tetrachloroethene can cause dizziness, headaches, sleepiness, confusion, nausea, difficulty in speaking and walking, and unconsciousness (ATSDR 1997). Prolonged and frequently repeated dermal exposure can cause irritation, dryness, and dermatitis due to defatting (US EPA 1994). Tetrachloroethene is classified as Group 2A carcinogen (probably carcinogenic to humans) by the International Agency for Research on Cancer (IARC 1995) and reasonably anticipated to be a human carcinogen by the US Department of Health and Human Services (USDHHS 2000). This compound induces leukemia in rats and increases risk for oesophageal cancer, non-Hodgkin's lymphoma and cervical cancer (IARC 1995). Tetrachloroethene has been shown to cause liver tumours in mice and kidney tumours in male rats (ASTDR 1997). Exposure to tetrachloroethene-contaminated drinking water was associated with an increased risk of leukemia and bladder cancer. The risk was dose related (Aschengrau *et al.* 1993).

Specific provisions are set by the European Community Legislation relating to tetrachloroethene in the Council Directive 90/415/EEC (EEC 1990) which controls discharges of tetrachloroethene during several industrial processes (including production of tetrachloroethene, trichloroethene, carbon tetrachloride and chlorofluorocarbons) and usage of tetrachloroethene for degreasing of metals. The quality objective of 10ug/l for the aquatic environment (including inland surface waters, estuary waters, internal coastal waters other than estuary waters, and territorial waters) is set for tetrachloroethene in the same Directive.

Article 5(6) of the recent document (EC 1999) concerning limitation of emissions of volatile organic compounds due to the use of organic solvents in certain activities and installations says that substances or preparations which, because of their content of volatile organic compounds classified as carcinogens, mutagens, or toxic to reproduction, shall be replaced as far as possible by less harmful substances or preparations within the shortest possible time.

Tetrachloroethene is in the first list of priority substances of the Commission Regulation (EC) No 1179/94 (EC1994) which is a part of the Council Regulation (EEC) No 793/93 on the evaluation and control of the risks of existing substances (EEC 1993).

References

Aschengrau, A., Ozonoff, D., Paulu, C., Coogan, P., Vezina, R., Heeren, T. & Zhang, Y. (1993) Cancer risk and tetrachloroethylene-contaminated drinking water in Massachusetts. Archives of Environmental Health 48(5): 284-292

ATSDR (1997) ATSDR's Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. CRC Publishers

Bauer, U. (1990) Occurrence of tetrachloroethylene in the FRG. In: Organohalogen compounds. Vol.2: Dioxin'90 – EPRI-Seminar. Hutzinger, O. & Fiedler, H. [Ed] published by Ecoinforma Press, Bayreuth, FRG. pp385-388

CEC (1986) Perchloroethylene. In: Organochlorine solvents: health risk to workers. Publ: Commission of the European Communities, ISBN 0-85186-078-8: 191-224

EA (1997) Regulation of dioxin releases from the Runcorn operations of ICI and EVC. Information Report. Environmental Agency Licence GD03177G0003. Pub. By Crown. 36pp.

EC (1994) Commission Regulation (EC) No 1179/94 of 25 May 1994 concerning the first list of priority substances as foreseen under Council Regulation (EEC) No 793/93. OJ L 131: 3-4

EC (1999) Council Directive 1999/13/EC of 11 March 1999 on the limitation of emission of volatile organic comp[ouinds due to the use of organic solvents in certain activities and installations. OJ L 85: 1-22

EEC (1990) Council Directive of 27 July 1990 amending Annex II to Directive 86/280/EEC on limit values and quality objectives for discharges of certain dangerous substances included in list I of the Annex to Directive 76/464/EEC. OJ L 219: 49-57

EEC (1993) Council Regulation (EEC) No 793/93 of 23 March 1993 on the evaluation and control of the risks of existing substances. OJ L 84: 1-75

Freiria-Gandara, M.J., Lorenzo-Ferreira, R.A., Alvarez-Devesa, A. & Bermejo, F. (1992) Occurrence of halogenated hydrocarbons in the water supply of different cities of Galicia (Spain). Environmental Technology, 13: 437-447

Hickman, J.C. (1993) Tetrachloroehylene. Chlorocarbons, -hydrocarbons ($CHCl=CCl_2$). In: Kirk-Othmer Encyclopedia of Chemical Technology. [Eds] Jacqueline I. Kroschwits; Mary Howe-Grant. Fourth edition, Vol. 6. John Wiley & Sons, Inc. pp.50-59

IARC (1995) Tetrachloroethylene. IARC Monographs Vol.63, p.159

ICI (1994) Report of the Chief Inspector HMIP authorisation AK6039 Improvement Condition part 8, table 8.1, item 2: Formation of dioxines in oxychlorination, significance for human health and monitoring proposals. ICI Chemicals and Polymers report NWJP/BMTD, 27th April, 1994, 16pp.

Klier, N.J., West, R.J. & Donberg, P.A. (1999) Aerobic biodegradation of dichloroethylenes in surface and subsurface soils. Chemosphere 38(5): 1175-1188

Santillo, D., Labounskaia, I., Stringer, R.L. & Johnston, P. A. (1997) Report on the analysis of industrial wastewaters from the Frutarom VCM/PVC plant near Haifa, Israel and adjacent shoreline sediments for organic contaminants. Greenpeace Research Laboratories Technical Note 03/97

UNEP (1997) The 1987 Montreal Protocol on Substances that Deplete the Ozone Layer, as adjusted and amended by the second Meeting of the Parties (London, 27-29 June, 1990), and by the fourth Meeting of the parties (Copenhagen, 23-25 November 1992), and further adjusted by the seventh Meeting of the Parties (Vienna, 5-7 December 1995), and further adjusted and amended by the ninth Meeting of the Parties (Montreal, 15-17 September 1997)

USDHHS (2000) 9th report on carcinogens. Publ: US Department of Health and Human Services, 334pp.

US EPA (1989) Tetrachloroethylene. US Environmental Protection Agency factsheets for regulated chemicals. Aquire Database, ERL-Duluth, US Environmental Protection Agency

US EPA (1994) Chemical summary for perchloroethylene. Office of Pollution Prevention and Toxics. US Environmental Agency

US EPA (1998) Technical factsheet on: Tetrachloroethylene. National primary drinking water regulations. Drinking water and health. Office of Ground Water and Drinking Water, US Environmental Protection Agency

US EPA (1999) Current drinking water standards. National primary and secondary drinking water regulations. Office of Ground Water and Drinking Water. US Environmental Protection Agency

Vartiainen, T., Pukkala, E., Rienoja, T., Strandman, T., Kaksonen, K. (1993) Population exposure to trichloroethene and tetrachloroethene and cancer risk - 2 cases of drinking-water pollution. Chemosphere 27(7): 1171-1181

Webler, T. & Brown, H.S. (1993) Exposure to tetrachloroethylene via contaminated drinking pipes in Massachusetts: a predictive model. Archives of Environmental Health 48(5): 293-297

WHO (1993) Guidelines for drinking-water quality. Vol.1: Recommendations. Second Edition, ISBN 92 4 154460 0, 188p.

Chlorinated benzenes

The production of chlorinated benzenes is a multiple product operation achieved by direct chlorination of benzene in the liquid phase using a ferric chloride catalyst. Only limited control can be exerted over the final product mix. The distillation train used for separating the mixture has a limited resolving power and the distillates are always mixtures of close boiling isomers which can be further separated by crystallisation (see eg Bryant 1993). Distillation also gives rise to chlorinated tars.

12 chlorinated benzenes are possible, with substitution patterns as follows:
1 chlorine monochlorobenzene,
2 chlorines 1,2-di-, 1,3-di- and 1,4-dichlorobenzenes
3 chlorines 1,2,3-tri-, 1,2,4-tri- and 1,3,5-trichlorobenzenes
4 chlorines 1,2,3,4-tetra-, 1,2,3,5,-tetra- and 1,2,4,5-tetrachlorobenzenes
5 chlorines pentachlorobenzene
6 chlorines hexachlorobenzene.

Both technological changes and environmental concerns have severely affected the production of chlorobenzenes; today only monochlorobenzene and 1,2- and 1,4-dichlorobenzenes are manufactured in large quantities. These are often produced together, with the economically optimised reaction yielding approximately 85% monochlorobenzene, 10% 1,4-dichlorobenzene and 5% 1,2-dichlorobenzene. Monochlorobenzene yield can be increased to 90% by careful monitoring of the reaction mix density and recycling of unreacted benzene, but total elimination of dichlorobenzene formation is not economical. Should the primary interest be in the para-isomer, yield may be increased by use of a selective catalyst, or the mix can be further chlorinated to produce a mixture of 1,4-dichlorobenzene and 1,2,4-trichlorobenzene. These two products can easily be separated by distillation (Bryant 1993, CEC 1986).

Mono- and di-chlorobenzenes

Chlorobenzene, 1,2-dichlorobenzene and 1,3-dichlorobenzene are colourless liquids; 1,4-dichlorobenzene forms colourless crystals at room temperature (Ware 1988a & b).

One of the earliest uses of chlorobenzene was as an intermediate for the explosive picric acid during the first World War (CEC 1986). It is used as a solvent and as an intermediate in chemical synthesis. In the US in the 1980s, the predominant use was for the production of ortho- and para-chlorobenzenes. Theses are used as intermediates for rubber chemicals, antioxidants, dyes and pigments, pharmaceuticals and agricultural chemicals. The fungicide benomyl, and carbofuran and the parathion group of insecticides are all derived from chlorobenzene. One previously important use was in the manufacture of DDT. Chlorobenzene production has fallen due to the development of other routes to aniline and phenol and the restriction of DDT use. By various routes, chlorobenzene is also used for the manufacture of specialty silicones, Grignard reagents and catalysts (Bryant 1993). Release to the environment is expected to derive from its use as a solvent, either through fugitive emissions or volatilisation from pesticides for which it is used as a carrier. Thus, inhalation is thought to be a major route of exposure for humans since it is rarely if ever found in food. It bioaccumulates in algae, fish and aquatic invertebrates. Mammalian metabolites are reported to be p-chlorophenol, p-chlorocatechol and p-chlorophenyl mercapturic acid. Human exposure causes CNS depression and respiratory tract irritation and animal studies have reported liver necrosis, renal toxicity and effects on the pancreas, blood and lymph and adrenal glands (Ware 1988a, Meek *et al.* 1994a).

Canada has derived a TDI of 8.1ug/kg body weight/day; estimated exposures (0.05-0.14ug/kg/day) are considerably lower than this (Meek *et al.* 1994a).

Ware (1988b) reports human symptoms after exposure to DCBs, but does not distinguish between isomers. Effects reported are anaemia, skin lesions, vomiting, headaches, eye and respiratory tract irritation, anorexia, weight loss, yellow atrophy of the liver, blood dyscrasias, porphyria, and chromosomal breaks in blood samples. Animal experiments recorded liver and kidney damage to be the most frequent effects, though high doses caused CNS perturbation and death through respiratory depression. The dichlorobenzenes are bioaccumulative in algae, aquatic invertebrates and fish (Ware 1988b). All three have also been reportedly found in blood (Ware 1988b).

1,2-Dichlorobenzene is produced unavoidably in the production of monochlorobenzene, but it is also possible to maximise dichlorobenzene production to 98% of the reaction mixture using suitable catalysts or alternative production methods leading to specific isomers. It is used mainly in the production of dyes and pesticides after conversion to 1,2-dichloro-4-nitrobenzene or dichloroaniline. Other uses include the solvent phase in the production of toluene di-isocyantes, production of deodorants and disinfectants and on a small scale as a heat transfer fluid. According to Meek *et al.* (1994b), the largest use is in degreasing for the metal and automotive industries.

Exposed laboratory animals exhibited hepatic, renal and haematological effects as well as lymphoid depletion of the thymus and spleen and multifocal mineralisation of both muscular and heart muscles (Ware 1988b, Meek *et al.* 1994b). Developmental toxicity was only observed at concentrations, which were overtly toxic to the mother. Human toxicity data are sparse, but chromosomal aberrations, anaemia and leukemia have been reported (Meek *et al.* 1994b). Mammals metabolise 1,2-dichlorobenzene to phenols and catechols, most of which are excreted after conjugation with glucoronic or sulphuric acids. Mercapturic acids may also be produced. The primary metabolites in humans are conjugated phenols (Ware 1988b). 1,2-Dichlorobenzene is found in air, food, breast milk and drinking water (Meek *et al.* 1994b). It is also toxic to higher plants, inducing abnormal mitosis (cell division) in onions (Ware 1988b).

1,3-Dichlorobenzene is growing in importance as a starting product in the manufacture of dyes, pesticides and pharmaceuticals. However, this has not yet reached commercial importance. There are some other small, specialised uses, but larger markets have not been developed, mainly because 1,3-dichlorobenzene only occurs as a minor constituent (approx 1%) of the technical dichlorobenzene reaction mix, and to produce it by other routes is expensive (Bryant 1993). Mammalian (and human) metabolism is as for 1,2-dichorobenzene above, but generally little is known about this 1,3-dichlorobenzene in comparison to the more commercially important dichlorobenzenes.

1,4-Dichlorobenzene (p-dichlorobenzene) is used largely in the production of deodorant blocks and room deodorants. It is also used as a moth control agent, as an insecticide and an intermediate for production of insecticides and dyes. An emerging market is in the manufacture of poly(phenylene sulphide) resin (PPS), and minor uses are as a germicide, fungicide and extreme pressure lubricant (Bryant 1993, CEC 1986). 1,4-dichlorobenzene is not spontaneously combustible and does not assist fire, but it is flammable nevertheless. It may be absorbed both through the inhalation of vapours, through the skin and though consumption of contaminated food. Human symptoms include damage to the liver, kidneys and lungs. Accidental poisoning

of children, presumably who have eaten moth repellent was widespread in the 1970s (CEC 1986). Once absorbed, 1,4-dichlorobenzene is stored in the adipose tissue, and has been detected in human samples (CEC 1986, Ware 1988b). The metabolism of 1,4-dichlorobenzene by mammals varies from that of the other two isomers in that mercapturic acids are not formed. 1,4-dichlorobenzene causes abnormal mitosis in higher plants. 1,4-Dichlorobenzene has been reported in human adipose tissue, as well as in blood (Ware 1988b).

Trichlorobenzenes

1,2,3- and 1,2,4-trichlorobenzene have been produced from the dehydrohalogenation of the unwanted isomers of the production of the pesticide hexachlorocyclohexane (HCH). This is of limited application.

Environmental regulations have curbed the use and discharge of trichlorobenzenes to the environment, at least in Europe and the USA (Harper *et al.* 1992, Bryant 1993). Not surprisingly, therefore, little research appears to have been carried out in comparison with some other chlorobenzenes.

The general human population would probably receive their greatest exposure to trichlorobenzenes through inhalation. The toxicity of all three appear similar; they damage the liver, kidney and thyroid. There is some indication of slight fetotoxicity at high doses. There is little evidence of mutagenicity and too few data are available for the trichlorobenzenes to given a carcinogenicity classification (Giddings *et al.* 1994a). All three isomers are toxic to phytoplankton (Sicko-Goad *et al.* 1989a-d, Sicko-Goad & Andresen 1993a & b).

1,2,3-trichlorobenzene has been detected in air, drinking water, food and breast milk (Giddings *et al.* 1994a) as well as industrially polluted surface waters (Harper *et al.* 1992), though it was not found in human adipose tissue from Canada (Hermanson *et al.* 1997). Little is known about its toxicity other than its ability to damage the liver, kidney and thyroid (Giddings *et al.* 1994a).

More information is available about 1,2,4-trichlorobenzene. According to Giddings *et al.* (1994a), only 1,2,4-trichlorobenzene has industrial application in Canada. It is imported for solvent and intermediate use. Environmental releases come from industrial discharges and from spillage of dielectric fluids. As mentioned above, it is toxic to the liver, thyroid and kidney. Liver and kidney weights and porphyrin excretion increase. In some studies, more severe liver damage has occurred, including necrotic and non-necrotic degeneration. 1,2,4-trichlorobenzene may be found in all environmental media, though there is insufficient analytical data to tell how widespread contamination is and it was not found in human adipose tissue from Canada (Hermanson *et al.* 1997).

Giddings *et al.* (1994a) report 1,3,5-trichlorobenzene air, drinking water, food, breast milk, though it was not found in human adipose tissue from Canada (Hermanson *et al.* 1997). It can be found in association with industrial operations (Harper *et al.* 1992) including PVC industry (Johnston *et al.* 1993).

Tetrachlorobenzenes

Giddings *et al.* (1994b) reviewed toxicity and exposure data for the tetrachlorobenzenes. They are no longer used or produced in Canada and releases come only from dielectric fluid spills and long-range transport. 1,2,4,5-Tetrachlorobenzene used to be used in the production of

2,4,5-trichlorophenol on a large scale, but this use has now been largely discontinued. There are not expected to be large differences between the behaviour of the isomers. Uptake of 1,2,4,5-tetrachlorobenzene was studied in rainbow trout. It is not volatile enough to evaporate from water easily, and is accumulated by the fish, through its gills. Bioaccumulation depended upon the rate of activity and oxygen uptake of the fish, and only the low water solubility prevented significant toxicity occurring (Brauner *et al.* 1994).

The greatest exposure of the general population is probably through food. All isomers were found to affect the liver, kidney, thyroid and lungs, with 1,2,4,5-tetrachlorobenzene being the most toxic. Not enough information was available to classify tetrachlorobenzenes as to carcinogenicity.

In addition to the effects noted above, 1,2,4,5-tetrachlorobenzene has also caused changes in the spleen, thymus, lymph nodes and haematological parameters in animals (Giddings *et al.* 1994b). An increase in chromosomal aberrations was seen in workers exposed to 1,2,4,5-tetrachlorphenol at a pesticide manufacturing complex (Giddings *et al.* 1994b).

In rats, 1,2,3,4- and 1,2,3,5-tetrachlorobenzene caused reduction in the number of live offspring at concentrations too low to adversely affect the mother (Giddings *et al.* 1994b).

All isomers have been detected in ambient air, drinking water and food and 1,2,3,4- and 1,2,3,5-tetrachlorobenzene have been identified in breast milk (Giddings *et al.* 1994b), though none of the isomers were detected in Canadian human adipose tissue (Hermanson *et al.* 1997).

Pentachlorobenzene

Giddings *et al.* (1994c) found that though no longer manufactured or used in Canada, pentachlorobenzene could still enter the environment through spillage of dielectric fluids or atmospheric transport. Animal studies demonstrate weight loss and effects on the liver, thymus, kidney, adrenal glands and digestive tract. Anaemia and malformation of sperm also occurred. There is some indication of fetotoxicity and developmental toxicity. The thyroid was impacted, with thyroid hormone (free and total thyroxin) concentrations reduced. Pentachlorobenzene cannot be assigned a carcinogenicity classification because of lack of data. Pentachlorbenzene accumulates in, and is toxic to algae (Sicko-Goad *et al.* 1989d).

Pentachlorobenzene has been detected in air, drinking water, food and breast milk (Giddings *et al.* 1994b), though according to Hermanson *et al.* (1997) it was found in less than 15% of human adipose samples collected in Ontario, Canada.

Hexachlorobenzene

Hexachlorobenzene (HCB) is a manufactured chemical, which was used as a wood preservative, as a fungicide for treating seeds and as an intermediate in organic syntheses (Budavari *et al.* 1989). Additionally, hexachlorobenzene may be formed as an unwanted by-product in the synthesis of other organochlorine compounds and in high-temperature processes (Newhook & Meek 1994, Sala *et al.* 1999). The UNECE (1998) lists HCB alongside PCDD/Fs and PAHs as being the most important POPs emitted from stationary sources. HCB emissions from waste incineration, metallurgical industries and burning of chlorinated fuels are highlighted (UNECE 1998)(Annex V).

HCB is toxic to aquatic life, land plants, land animals, and humans. It is listed by the IARC as a Group 2B carcinogen, i.e. possible carcinogen to humans and also appears to be a tumour promoter. Hexachlorobenzene may damage the developing foetus, liver, immune system, thyroid and kidneys and CNS. The liver and nervous system are the most sensitive to its effects. Porphyria is a common symptom of HCB toxicity. High or repeated exposure may damage the nervous system and can cause irritability, difficulty with walking and co-ordination, muscle weakness, tremor and/or a feeling of pins and needles on the skin. Repeated exposure, especially when skin effects occur, can lead to permanent skin changes, such as changes in pigmentation, tight, thickened skin, easy wrinkling, skin scarring, fragile skin, and increased hair growth, especially on the face and forearms (ATSDR 1997, Newhook & Meek 1994, van Birgelen 1998). Recent research (van Birgelen 1988) suggests that HCB has dioxin-like toxicity and that, based on a preliminary toxic equivalence factor (TEF) of 0.0001, HCB could contribute significantly to the dioxin-type toxicity of human milk based on PCB/PCDD/PCDF toxicity equivalents. In many countries, this could mean an increase of 10% - 60%, but in countries with high HCB exposure levels, the effects could be even greater. In Spain and the Czech Republic, inclusion of HCB in total breastmilk TEQ estimates could lead to totals 6 times higher than based only on PCBs and PCDFs. Slovakia and India also have very high HCB levels; other countries (eg Austria) had high levels in previous decades. It has been suggested that more epidemiological studies should be undertaken, especially in the most highly contaminated countries.

With the exception of occupational settings, almost all human exposure occurs via food. The greatest body of information on HCB toxicity to humans derives from an incident in Turkey between 1955 and 1959, when HCB-treated grain was made into bread. More than 600 people experienced porphyria cutanea tarda. Children of exposed women had skin lesions and 95% of them died at less than one year old. In the long term (20-30 years), some people continued to have abnormal porphyrin biochemistry and neurological, orthopaedic and dermatological symptoms persisted. Hexachlorobenzene is also thought to have caused porphyria cutanea tarda in populations exposed industrially and through food (Newhook & Meek 1994). High concentrations of HCB were found in the air around a chlor-alkali and organochlorine manufacturing plant at Flix in Spain and in blood of workers and local residents (Sala *et al.* 1999, Grimalt *et al.* 1994). One study found a significant elevation in incidence of cancer of the thyroid, soft tissues and at unspecified sites in the men of the community (Grimalt *et al.* 1994) and the authors of one study stated that HCB exposure was associated with specific health effects in the most highly exposed subjects (Sala *et al.* 1999).

Once introduced into environment, HCB strongly adsorbs to soil materials and almost no desorption take place (Bahnick & Doucette 1988). It is bioaccumulative and biomagnifies. It can be measured in ambient air, drinking water, soil, food and breast milk (Newhook and Meek 1994).

HCB is one of twelve priority POPs intended for global action by the UN Environment Programme (UNEP) Governing Council. It is intended that HCB will be phased out worldwide under the POPs Convention (UNEP 1995, 1997), which was signed in Stockholm in May 2001 (UNEP 2001). Furthermore, HCB is included on Annex I of the UNECE POPs Protocol under the Convention on Long-Range Transboundary Air Pollution (LRTAP)(UNECE 1998).

Within the EC, discharges of HCB are controlled as stipulated by EC Council Directive 86/280/EEC, which amends Directive 76/464/EEC, regarding pollution caused by certain dangerous substances discharged into the aquatic environment (EC 1986, 1976).

HCB is also included in the list of priority hazardous substances agreed by the Third and Fourth North Sea Conferences (MINDEC 1990 & 1995), where continuous reduction of all hazardous substances was agreed with the ultimate aim of reducing environmental concentrations of hazardous substances to near background levels (synthetic substances to zero) within the next 25 years. The 1998 Ministerial Meeting of the OSPAR Commission (OSPAR 1998a) further reinforced these objectives. HCB is included on the OSPAR 1998 List of Candidate Substances, Annex 3 of the OSPAR Strategy with regard to Hazardous Substances (OSPAR 1998b). In addition, HCB is regulated under the 1995 Barcelona Convention, the Rotterdam (PIC) Convention and the International Joint Comission on the Great Lakes (IJC) has called for all uses to be eliminated.

References

ASTDR (1997) ATSDR's toxicological profiles on CD-ROM. U.S. Department of Health and Human Services, Public Health Service, CRC Press Inc

Bahnick, D.A. & Doucette, W.J. (1988) Use of molecular connectivity indices to estimate soil sorption coefficients for organic chemicals. Chemosphere 17: 1703-1715 .

Brauner, C.J., Randall, D.J., Neuman, J.F. & Thurston, R.V. (1994) The effect of exposure to 1,2,4,5-tetrachlorobenzene and the relationship between toxicant and oxygen uptake in rainbow trout (*Oncorhynchus mykiss)* during exercise. Environ. Toxic. Chem. 13(11): 1813-1820

Bryant, J.G. (1993) Chlorinated benzenes. IN: Kroschwitz, J.I. & Howe-Grant, (Eds). The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition .Publ. Wiley-Interscience, N.Y. Volume 6: 87-100

Budavari, S.M., O'Neil, J., Smith A. & Heckleman P.E. [Eds] (1989) The Merck index: an encyclopaedia of chemicals, drugs and biologicals. 11th Edn, Merck and Co, Inc., New Jersey, USA

CEC (1986) p-dichlorobenzene. IN: Organo-chlorine solvents: Health risks to workers. Publ: Commission of the European Communities, ISBN 0-85186-078-8, pp1-16

EEC (1976) Council Directive 76/464/EEC of 4 May 1976 on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community. OJ L 129: 23-29 as amended.

EEC (1986) Council Directive 86/280/EEC of 12 June 1986 on limit values and quality objectives for discharges of certain dangerous substances included in List 1 of the Annex to Directive 76/464/EEC.

Giddings, M., Meek, M.E. & Gomes, R. (1994a) Trichlorobenzenes: Evaluation of risks to health from environment exposure in Canada. Environ. Carcin. Ecotox. Revs. C12(2): 517-525

Giddings, M., Meek, M.E. & Gomes, R. (1994b) Tetrachlorobenzenes: Evaluation of risks to health from environment exposure in Canada. Environ. Carcin. Ecotox. Revs. C12(2): 473-481

Giddings, M., Meek, M.E. & Gomes, R. (1994c) Pentachlorobenzene: Evaluation of risks to health from environment exposure in Canada. Environ. Carcin. Ecotox. Revs. C12(2): 435-441

Grimalt, J.O., Sunyer, J., Moreno, V., Amaral, O.C., Sala, M., Rosell, A., Anto, J.M. & Albaiges, J. (1994) Risk excess of soft-tissue sarcoma and thyroid cancer in a community exposed to airborne organochlorinated compounds with a high hexachlorobenzene content. International Journal of Cancer 56: 200-203

Harper, D.J., Ridgeway, I.M. & Leatherland, T.M. (1992) Concentrations of hexachlorobenzene, trichlorobenzenes and chloroform in the waters of the Forth estuary, Scotland. Mar. Poll. Bull. 24(5): 244-24

Hermanson, M.H., Monosmith, C.L. & Donnelly-Kelleher, M.T. (1997) Seasonal and spatial trends of certain chlorobenzene isomers in the Michigan atmosphere. Atmos. Environ. 31(4): 567-573

Johnston, P.A., Stringer, R.L., Clayton, R. & Swindlehurst, R.L. (1993) Regulation of toxic chemicals in the North Sea: the need for an adequate control strategy. Proceedings of the Scientific Symposium on the North Sea Quality Status Report 1993, 18-21 April 1994, Ebeltoft, Denmark: 269-274

Meek, M.E., Giddings, M. & Gomes, R. (1994a) Monochlorobenzene: evaluation of risks to health from environmental exposure in Canada. Environ. Carcin. Ecotox. Revs. 12(2): 409-415

Meek, M.E., Giddings, M. & Gomes, R. (1994b) 1,2-Dichlorobenzene: evaluation of risks to health from environmental exposure in Canada. Environ. Carcin. Ecotox. Revs. 12(2): 269-275

MINDEC (1990) Final Declaration of the Third Ministerial Conference on the Protection of the North Sea. Ministry of Transport Conference on the Protection of the North Sea. Ministry of Transport and Public Works, The Haugue.

MINDEC (1995) Ministerial Declaration of the Fourth International Conference on the Protection of the North Sea. 8-9 June 1995, Esjberg, Denmark.

Newhook, R. & Meek, M.E. (1994) Hexachlorobenzene: Evaluation of risks to health from Environmental Exposure in Canada. Environ. Carcin. Ecotox. Revs. C12(2): 345-360

OSPAR (1998a) OSPAR Convention for the Protection of the Marine Environment of the North-East Atlantic. Sintra Statement, Ministerial Meeting of the OSPAR Commission, Sintra 22-23 July 1998.

OSPAR (1998b) OSPAR Convention for the Protection of the Marine Environment of the North East Atlantic. OSPAR Strategy with regard to Hazardous Substances, Reference no. 1998-16, OSPAR 1998/14/1/Annex 34, Ministerial Meeting of the OSPAR Commission, Sintra b22-23 July 1998.

Sala, M., Sunyer, O., Otero, R., Santiago-Silva, M., Ozalla, D., Herrero, C., To-Figueras, J., Kogevinas, M., Anto, J., Camps, C. & Grimalt, J. (1999) Health effects of chronic high exposure to hexachlorobenzene in a general population sample. Arch. Environ. Health 54(2): 102-109

Sicko-Goad, L. & Andersen, N.A. (1993a) Effect of lipid composition on the toxicity of trichlorobenzene isomers to diatoms. I. Short-term effects of 1,3,5-trichlorobenzene. Arch. Environ. Contam. Toxicol. 24: 236-242

Sicko-Goad, L. & Andersen, N.A. (1993b) Effect of lipid composition on the toxicity of trichlorobenzene isomers to diatoms. II. Long-term effects of 1,2,3-trichlorobenzene. Arch. Environ. Contam. Toxicol. 24: 243-248

Sicko-Goad, L., Evans, M.S., Lazinsky, D., Hall, J. & Simmons, M.S. (1989d) Effect of chlorinated benzenes on diatom fatty acid composition and quantitative morphology. IV. Pentachlorobenzene and comparison with trichlorobenzene isomers. Arch. Environ. Contam. Toxicol. 18: 656-668

Sicko-Goad, L., Hall, J., Lazinsky, D. & Simmons, M.S. (1989b) Effect of chlorinated benzenes on diatom fatty acid composition and quantitative morphology. II. 1,3,5-Trichlorobenzene. Arch. Environ. Contam. Toxicol. 18: 638-646

Sicko-Goad, L., Hall, J., Lazinsky, D. & Simmons, M.S. (1989c) Effect of chlorinated benzenes on diatom fatty acid composition and quantitative morphology. III. 1,2,3-Trichlorobenzene. Arch. Environ. Contam. Toxicol. 18: 647-655

Sicko-Goad, L., Lazinsky, D., Hall, J. & Simmons, M.S. (1989a) Effect of chlorinated benzenes on diatom fatty acid composition and quantitative morphology. I. 1,2,4-Trichlorobenzene. Arch. Environ. Contam. Toxicol. 18: 629-637

UNECE (1998) Protocol to the 1979 Convention on long-range transboundary air pollution (LRTAP) on persistent organic pollutants (Aarhus POPs Protocol). Publ UNECE, 61pp.

UNEP (1995) Decision 18/32 of the UNEP Governing Council: Persistent Organic Pollutants. UNEP Governing Council, 25th May 1995.

UNEP (1997) Decisions adopted by the Governing Council at its nineteenth session: 13c. International action to protect human health and the environment through measures which will reduce and/or eliminate emissions and discharges of persistent organic pollutatns, including the development of an international legally binding instrument. UNEP Governing Council, 7th February 1997.

UNEP (2001) Final Act of the Conference of Plenipotentiaries on the Stockholm Convention on Persistent Organic Pollutants, UNEP/POPS/CONF/4, 5 June 2001, United Nations Environment Programme, 44 pp.

US EPA (1989). EPA Factsheets for Regulated Chemicals. Hexachlorobenzene. Washington, DC.

Van Birgelen, A.P.J.M. (1998) Hexachlorobenzene as a possible major contributor to the dioxin activity of human milk. Environ. Health Persp. 106(11): 683-688

Ware, G.W. (Ed.)(1988a) Chlorobenzene. Rev. Environ. Contam.Toxicol. 106: 37-49

Ware, G.W. (Ed.)(1988b) Ortho-, meta- and para- dichlorobenzene. Rev. Environ. Contam.Toxicol. 106: 51-56