

6. Dioxin: Seveso, Vietnam and everyday exposure

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1. Chemistry / Nomenclature:

Dioxin is not a molecule, but a structural element of many molecules (see Fig. 1). The most simple dioxin in chemical perspective is 1,4-dioxan a relatively harmless solvent with 6 hydrogen atoms attached to this six-sided ring with the characteristic oxygen atoms.

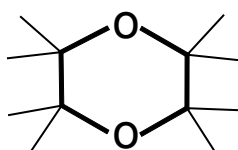


Fig. 1: Basic structure of all dioxins (*para*-dioxins, oxygen atoms are diametrical).

However there are certain members of this chemical family which are known as some of the most toxic, 'man-made' substances. This specific subgroup are polychlorinated dibenzo-*p*-dioxins (PCDDs). The basic structure is shown in Fig. 2, where up to 8 chlorine atoms can be attached to the outer rings.

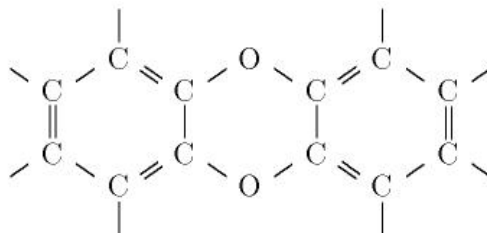


Fig. 2: Structure of dibenzo-*p*-dioxin.

The number and position of the chlorine atoms determine the toxicity of the PCDD and the most toxic among them is 2,3,7,8 tetrachloro dibenzo-*p*-dioxin (TCDD), which is shown in Fig. 3. This molecule is typically meant when the media refer to 'dioxin' and we will also use this unscientific nomenclature.

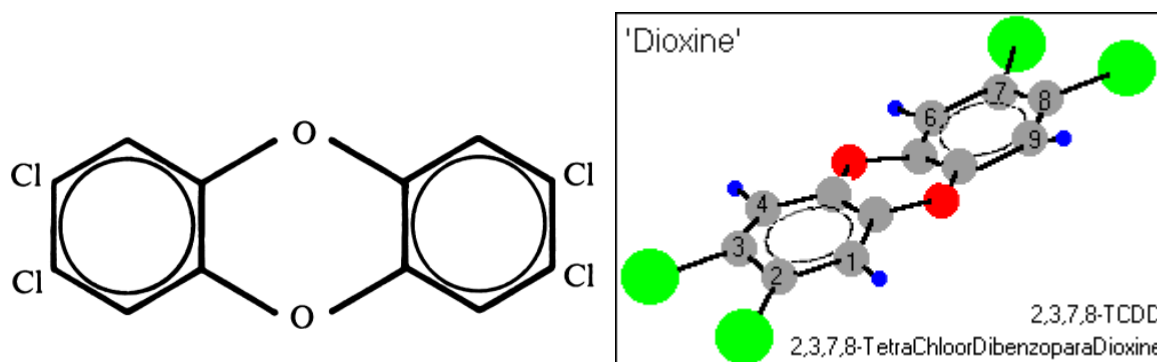


Fig. 3: Structure of is 2,3,7,8 tetrachloro dibenzo-*p*-dioxin (TCDD), the Seveso poison.

Furans are similar to dioxins (see Fig. 4). They have molecules in which chlorine atoms can be inserted at positions 1-4 and 6-9. Because of the asymmetrical nature of the parent molecule, there are 135 possible furan isomers.

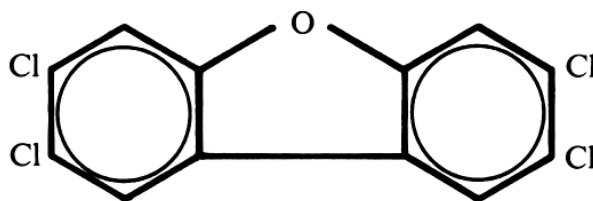


Fig. 4: Structure of 2,3,7,8 tetrachloro-dibenzofuran.

Dioxin then is a collective name for 75 different poly-chlorinated dibenzo-p-dioxins and 135 different poly-chlorinated-dibenzo furans. These compounds can be extremely toxic substances to some animal species. The most toxic is 2,3,7,8 tetrachlorodibenzo-p-dioxin (2,3,7,8-TDD), in which chlorine atoms are present at positions 2, 3, 7 and 8 in the dioxin molecule. This dioxin is the most studied one because of its extreme toxicity towards some laboratory animals, but there is very considerable doubt about any possible hazards it poses to man.

No human is known to have died from dioxin poisoning.

2. Properties of TCDD:

Pure 2,3,7,8-TCDD was synthesized in 1968.

Colorless to white crystals

thermal:

Melting point: 295 °C

Decomposes at 500 °C

Solubility at 25 °C

Water : <1 mg/ml

Benzene: 0.57 mg/ml

Chlorobenzene: 0.72 mg/ml

Lard oil: 0.04 mg/ml

Hexane: 0.28 mg/ml

Dioxins are slow to evaporate (i.e. they have a low vapour pressure), do not dissolve readily in water, but are **lipophilic** (or fat-loving) that is, soluble in fatty substances and in organic matter with fat-like properties. Dioxins **do not react** easily with other chemicals.

These characteristics explain why dioxins are usually found adhered to or dissolved **in fatty tissue** where they can **(bio)accumulate**. In lakes and rivers, dioxins are often detected bound to **sediment** or other organic substances.

3. Creation:

Concentrations:

Amounts of dioxins are stated in **equivalents of TCDD or TEQ** (toxic equivalent), a measure which weights together the seventeen most poisonous dioxins and dibenzofurans (see part 4).

$$1 \text{ ng} = 10^{-9} \text{ g}$$

$$1 \text{ pg} = 10^{-12} \text{ g}$$

Nm³ - refers to the normalised gas volumes (cubic meters)

Loch Ness is Britain's largest loch. It's volume is 7,000,000,000 m³. A sugar lump weight 3 grams. Hence a quarter of a sugar lump dissolved in Loch Ness is the equivalent of 0.1 ng/m³ in the chimney gases of an incinerator.

Dioxins are **not manufactured** as commercial products or ingredients. They occur as **unintended by-products** of incomplete combustion (low temperature), photochemical and certain chemical processes. These combustion processes include: municipal, hospital and hazardous waste incinerators; motor vehicles; wood burning, smoking; forest fires (e.g. ignited naturally by lightning strikes); volcanoes; compost heaps, etc. The metal industry is another source of dioxins, which appear during melting and refining operations or scrap recovery. A few chemical manufacturing processes also emit trace quantities of dioxins.

As a result of the multiplicity of emission sources (either natural or anthropogenic), scientists have **detected dioxins virtually everywhere**, even in samples from the pre-industrial era. However, many of the **primary sources of dioxins in the environment remain unknown**.

3.1 Dioxin from natural processes

Wood fires are one possible source of dioxins in nature. Estimates are varying from a few grams a year to several kg. Indeed, dioxin is found in the bark of thousands of years old redwoods and in human bodies of 6,000 years old. If this is a big source of dioxins in nature is not clear. Also after a recent dioxin scare in chicken meat in the US, the origin of the dioxins was found in millions of years old clay layers, not influenced by any man-made source.

Also interesting are **complete biological routes** which makes dioxins. A lot of wood rotting fungi and about half of all mushrooms use chlorinating and oxidising compounds to break down lignin, the glue which makes part of the strength of all plants. They need that to reach the cellulose, which they use as their source of energy. These organisms are very effective in transforming inorganic chlorine from salt into all kinds of chlorinated organics, mainly methylchloride, but also chlorinated phenols or alike compounds.

The remainder of this normal biological recycling of wood, is an amount of chlorinated humic acids and chlorinated lignin in water, chlorinated humus and chlorinated phenols in soil, the latter about **seven times higher than allowed by legislation** in The Netherlands! From chlorinated phenols to dioxins is only a small step, you only need hydrogen peroxide, which is produced by a lot of micro-organisms. This results in the fact that, except for 'hot spots', near old incinerators and other point sources from the past, the **highest amount of dioxins in soil is found in woods, not in industrial or heavy traffic areas**.

While a bad incinerator emits nearly as much dioxins as what was going into the incinerator (but concentrates that in the vicinity) and a good one reduces the amount with 99.9% or more, the biological destruction of municipal sludge and the biological composting of natural organic material *triples* the amount of dioxins! Most probably this is the result of the same biological mechanism, which oxidises everywhere present natural chlorophenols.

Tab. 1: Dioxin found in soil at different places. All figures as ng I-TEQ/kg dry material [2].

Dioxin found in soil at different places:	
Place	average
Municipal biological sludge:	62
Soil in woods:	26
Soil in industrial areas:	17
Bio-compost:	14
Soil from roadsides:	8
Soil from fields:	3.5

3.2 Anthropogenic Sources:

Toxic amounts are only **produced unintentionally** by man and besides waste combustion and degeneration of PVC the primary source of PCDDs is/was that of a by-product in the industrial fabrication of **chlorinated phenols** (Fig. 5), which are still used as herbicides, fungicides and bactericides (world production 1980: 150,000 t), although their use has been dramatically restricted since the 80ies. (You can still buy some as weed killers for gardening, but vast use in industry is very limited these days)

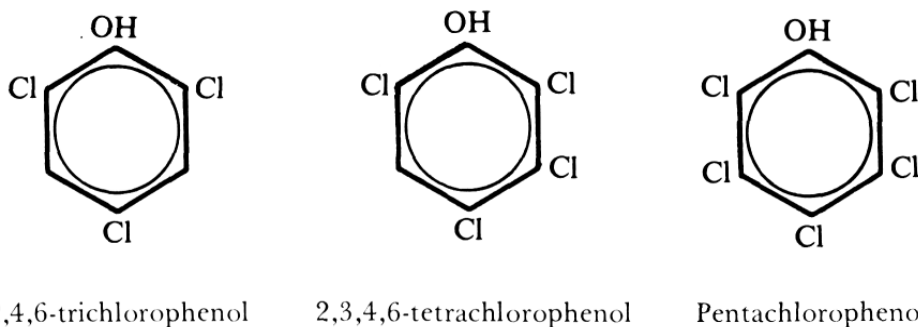


Fig. 5: Some chlorinated phenols. They are a stage in the reactions for synthesising highly potent herbicides etc. [1].

The occurrence of PCDDs during the production of these chlorinated phenols strongly depend on the production process (especially the temperature). If the temperature rises the production of the more stable dioxins and furans is preferred (see Fig. 6).

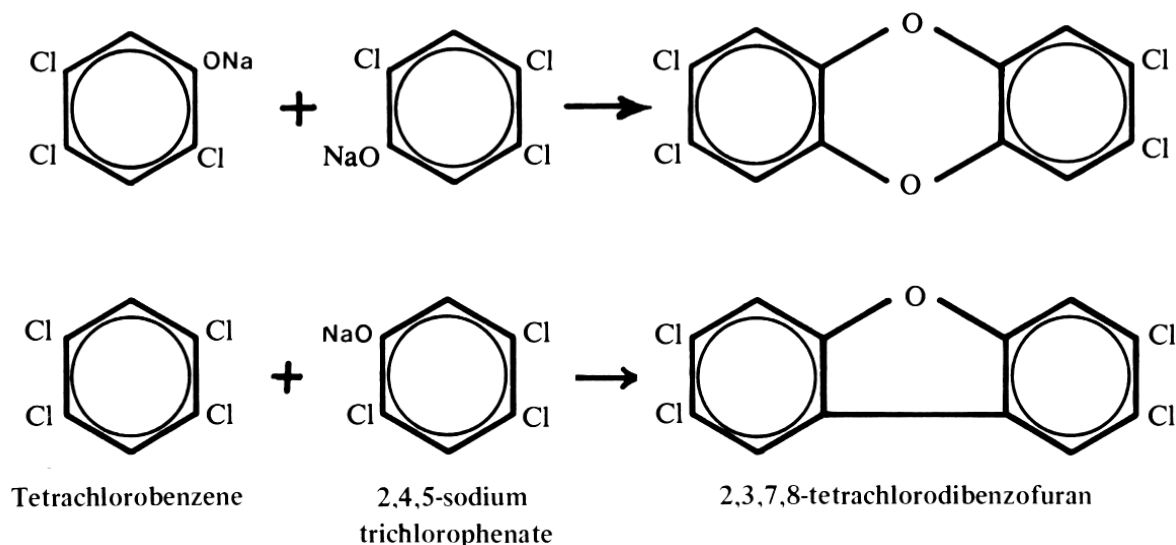


Fig. 6: Top: Condensation of 2 molecules of sodium trichlorophenolate to TCDD. Bottom: Condensation of tetrachlorobenzene and sodium trichlorophenolate to 2,3,7,8-tetrachlorodibenzofuran.

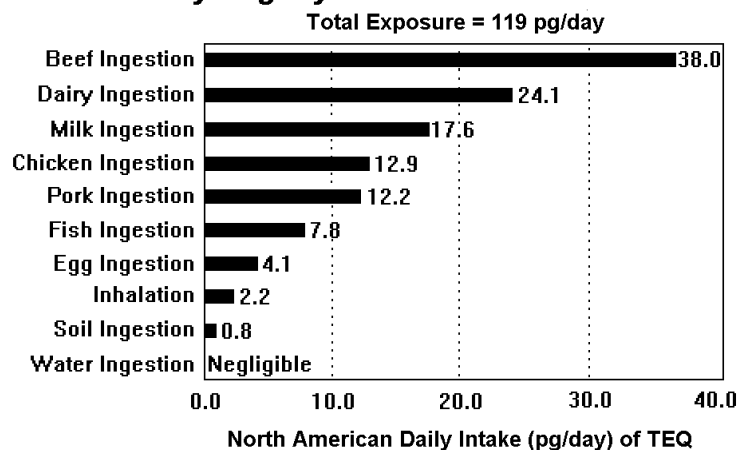
It is also formed during various combustion processes. Incineration of chemical wastes, including chlorophenols, chlorinated benzenes and biphenyl ethers, may result in the presence of TCDD in flue gases, fly ash and soot particles.

3.3 Human exposure

Most human exposure to dioxins stems from what are called '**secondary**' exposure pathways, that is, through the **food chain** (e.g. milk and dairy products, meat and fish containing small amounts of dioxins, see Fig. 7). These secondary exposures sum up to about 95% of average human exposure (according to EPA = U.S. Environmental Protection Agency)

Based on numerous published studies on human exposure to dioxins, virtually everyone in the world has some small amount of dioxins in their body. Human fat concentrations of 2, 3, 7, 8 TCDD throughout the world have been reported in the range of 5 to 15 parts per trillion. This is equivalent to approximately 0.00000015 grams of dioxins in an individual weighing 70kg. Exposure to dioxins originating from human activities is declining. This is due in part to improved incineration technology.

Tobacco smoke and other products finished by **roasting or smoking** (coffee, BBQ, smoked ham, fish, etc.) might have increased levels of dioxins due to low temperature combustion involved.

This is where you get your dioxin from:

Is this a good case for vegetarianism or what?

[A TEQ is a dioxin Toxic Equivalent]

Fig. 7: The secondary exposure.

Table 2: Sources of dioxin in the UK. Measured and estimated in 1995 to air: All figures expressed in gram I-TEQ per year [3].

Process	1995		future max
	min	max	
MSW combustion:	460	580	15
Clinical waste combustion:	18	88	5
Industrial coal combustion:	5	67	67
Sinter plants:	29	54	47
Traffic:	1	45	45
Iron and steel:	3	41	14
Non-ferrous metals:	5	35	10
Crematoria:	1	35	35
Domestic coal combustion:	20	34	34
Domestic wood combustion:	2	18	18
Natural fires:	0.4	12	12
Cement manufacturing:	0.2	11	11
Straw combustion:	3.4	10	10
Chemical waste combustion:	1.5	8.7	0.3
Sewage sludge combustion:	0.7	6	0.9
Landfill gas combustion:	1.6	5.5	5.5
Industrial wood combustion:	1.4	2.9	2.9
Waste oil combustion:	0.8	2.4	2.4
Lime manufacture:	0.04	2.2	2.2
Coke production:		2	2
Tyres combustion:		1.7	1.7
Asphalt mixing:	0.047	1.6	1.6
PCP in timber processes:		0.8	0.8
Pesticide production:	0.1	0.3	0.3
Ceramic production:	0.02	0.06	0.06
Halogenated chemicals:		0.02	0.02
Glass manufacturing:	0.005	0.01	0.01
Carbon regeneration:		0.006	0.006
Total:	560	1100	350

3.4. World wide dioxin emission inventory:

The United Nations Environmental Program (UNEP) has made an inventory of official inventories of sources of dioxins/furans. Although only from industrial countries, it gives a good impression of what world wide is known of emitted quantities. Also some trends can be seen: in most countries the **amounts of dioxins are falling rapidly**, mainly because of stringent rules for incinerator emissions. There still is a large gap between the known emissions and what can be estimated as total emissions, based on measured world wide deposits.

The **total deposit** of PCDD/PCDF to land was calculated to be 220 ± 30 kg I-TEQ/year. A rough estimate of known sources in **industrial** countries, recalculated for **global emissions** comes to 50 ± 10 kg I-TEQ/year. That is less than one quarter of the estimated deposits, which leads to the impression that the **emissions of biomass combustion, especially forest fires, are highly underestimated**.

However from sediment analysis in lakes and rivers it can be seen, that at least in dense populated areas the human factor causes local (toxic?) accumulation (Fig. 8).

Table 3: World wide sources of dioxin estimated in 1990 [4].

Process	Emission Factor sum $\mu\text{g}/\text{kg}$	Production Mton/yr	Total Emission sum kg/yr
Waste incineration	13	87	1,130
Cement kilns (+hazwaste)	2.6	260	680
Biomass combustion	0.04	8700	350
Ferrous metal production	0.5	700	350
Cement kilns (no hazwaste)	0.2	1600	320
Medical waste incineration	22	4	84
Sec. copper smelting	39	2	78
Leaded fuel combustion	2800	3800	11
Unleaded fuel combustion	320	3800	1
			3000
			50

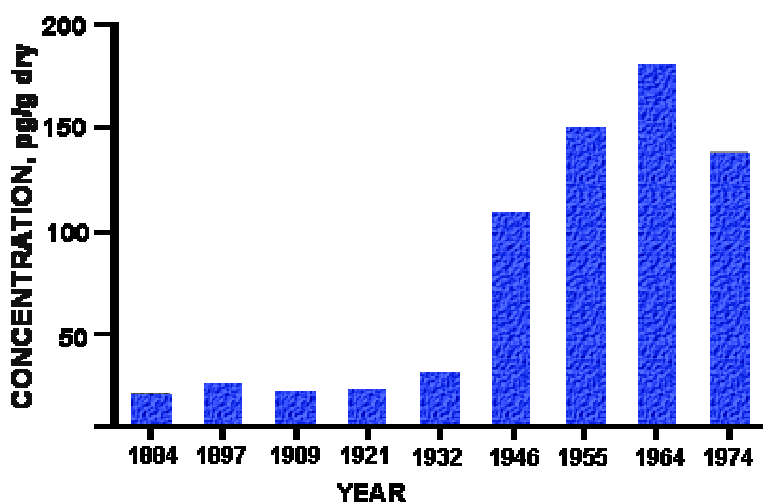


Fig. 8: Concentrations of Total Dioxins in Bottom Sediment Layers Corresponding to Year of Deposition Beaver Lake, Olympic Peninsula, Washington

4. Toxicity:

One international standard measures the toxicity of substances by **LD₅₀**, that is the dosage at which 50% of the samples (lab animals) die. Table 4 gives an overview for PCDDs [1]. It clearly shows that mice are much less affected than guinea pigs. There are models to scale for man, but the accuracy is doubtful!

Table 4: Estimated single oral LD₅₀ of PCDDs for guinea pigs and mice [1].

Chlorination	Guinea pigs		Mice	
	μg/kg	μmol/kg	μg/kg	μmol/kg
2,8	> 300,000	> 1,180	—	—
2,3,7	29,444	120.41	> 3,000	> 10
2,3,7,8	2	0.006	283.7	0.88
1,2,3,7,8	3.1	0.009	337.5	0.94
1,2,4,7,8	1,125	3.15	> 5,000	> 14
1,2,3,4,7,8	72.5	0.185	825	2.11
1,2,3,6,7,8	70–100	0.178–0.255	1,250	3.19
1,2,3,7,8,9	60–100	0.153–0.255	> 1,440	> 3.67
1,2,3,4,6,7,8	> 600	> 1,400	—	—
1-NO ₂ -3,7,8	> 30,000	> 90	—	—
1-NH ₂ -3,7,8	> 30,000	> 99	—	—
1-NO ₂ -2,3,7,8	47.5	0.129	> 2,000	> 5.4
1-NH ₂ -2,3,7,8	194.2	0.576	> 4,800	> 14.2

Because of the fact that in contaminated samples typically a vast of PCDDs is present, an average toxicity is defined, where the concentration of each component is scaled by its toxicity, defining the so-called I-TEQ!

Definition of I-TEQ (= International toxicity equivalents):

From the total 210 different (poly)chlorinated dioxins and -furans, called congeners, only seventeen are (very) toxic. Between these seventeen, there are differences up to a thousand times in toxicity. To make any comparison possible, the amounts of the seventeen toxic chlorinated dioxins/furans are multiplied with a toxicity factor (I-TEF) and added together. The toxicity factor compares the toxicity of a congener with the most toxic dioxin, which escaped in Seveso.

What are the symptoms of being exposed to a larger quantity of TCDD?

Symptoms of exposure to this compound may include eye irritation, allergic dermatitis, wasting, hepatic necrosis, thymic atrophy, hemorrhage, lymphoid depletion and chloracne. It may cause hypercholesterolemia and psychiatric disturbances. It may also cause hyperpigmentation, liver damage, Hodgkin's lymphoma, raised serum hepatic enzyme levels, disorders of fat metabolism, disorders of carbohydrate metabolism, cardiovascular disorders, urinary tract disorders, respiratory disorders, pancreatic disorders, poly-neuropathies, lower extremity weakness, sensorial impairments and neurasthemic or depressive syndromes.... (this list goes on for **2 more pages!**)

(...and still going on → Seveso people and Vietnam veterans)

What have studies shown about the effect of dioxin on the immune system?

One of the primary toxic effects of dioxin in laboratory animals is immune suppression, which results in decreased resistance to infectious agents and some cancers. The mechanisms and relationship between altered host resistance and immune dysfunction is complex, poorly defined, but extremely important to understanding health effects.

Some effects -such as degradation of the human immune system- seem to occur at dioxin levels that the average inhabitant of industrialised nations is already carrying around in his or her body.

What have studies shown about dioxin and its role in causing birth defects?

Dioxin's causation of birth defects (teratogenicity) has not been established in humans but studies in mice have shown that dioxin and similar chemicals produce cleft palate and hydronephrosis, a congenital obstruction of the ureter.

What is known about the developmental effects of dioxin?

A previous study in rats showed severe hormonal imbalance caused by low doses of dioxin, and grantees are studying effects of this imbalance on the development and function of endocrine organs such as the adrenal cortex, thyroid and pancreas.

What is known about the reproductive effects of dioxin?

Most sensitive of all are new-born infants and foetuses exposed while in the womb. In mammals, postnatal functional alterations involving learning behaviour and the developing reproductive system appear to be the developmental events most sensitive to perinatal (shortly after birth) dioxin exposure. The developing immune system may also be highly sensitive. In other words, dioxin exposure of mammals (including humans) shortly before or shortly after birth are most likely to impair intellectual development and the immune system.

Dioxin adversely affects the male reproductive system by causing major androgenic deficiencies, due to decreased testosterone synthesis.

Does dioxin cause cancer?

The mechanism by which dioxins can cause a toxic effect has been studied in numerous animal experiments. TCDD has been shown to be carcinogenic for animals. A possible weak increased frequency of cancer may be found in heavily-exposed workers. Although questionable, IARC (International Agency for Research on Cancer) decided in 1997 to classify TCDD as carcinogenic to human. It is unlikely that dioxins increase cancer incidence in people at low exposure levels such as those found in the environment.

Even at extreme exposure levels, those to which some Seveso residents were exposed, the only clear effect is **chloracne** - a serious but reversible skin condition.

The length of time required for half a quantity of drug, or other substance residing in a living organism to be metabolized, or eliminated by normal biological processes. It is also called biological half-life. The **half-life** of dioxin is 8.7 years in humans (as determined in the Ranch Hand (follow-up) studies, see 6).



5. Seveso: July 10, 1976

An industrial accident during the **manufacture of 2,4,5-trichlorophenol** in Seveso, Italy on July 10, 1976 caused the release of estimated **1 - 5 kg of TCDD** into the environment. Concentrations as high as **51.3 ppm TCDD** were found in some samples [5].

The company ICMESA causing the accident was located in Seveso (ca. 30 km north of Milan, Italy) and a subsidiary of the Swiss chemical company Givaudan, which was a subsidiary of the giant pharmaceutical corporation **F. Hoffmann-La Roche** (Basle, Switzerland).

A diary of the disaster (for completeness see [6])

- 1969–1970 ICMESA starts trichlorophenol (TCP) production. Givaudan needs high-grade TCP for the production of hexachlorophene, a disinfectant used in medicinal soaps.
- 1970 to July 1976 Rising TCP production, all of which is delivered to Givaudan.

Friday, 9 July 1976

- 14:30 Seveso/ICMESA. Dr Paolo Paoletti, director of production at ICMESA, discusses the production programme with the various foremen, including the one responsible for Building B. As usual in Building B, trichlorophenol (TCP) was to be produced. Trichlorophenol is an intermediate used in the production of the disinfectant hexachlorophene.
- 16:00 The TCP reaction vessel is filled with the various starting materials.

Saturday, 10 July 1976

- 02:30 According to the temperature diagram the reaction is completed.
- 04:45 The foreman in charge gives the order to interrupt a distillation which is not completed. The heating is turned off and the vessel contents mixed for a further 15 minutes. The last measured temperature is 158°C. (water cooling is not turned on, because the reaction is believed to be finished)
- 06:00 The night shift is over. The workers leave the factory, and only the cleaning and maintenance crew remains behind.
- 12:37 The rupture disk in the safety valve bursts as a result of excessive pressure, caused by an exothermic reaction in the TCP vessel. A chemical mixture in the form of an aerosol cloud escapes into the air in a south-easterly direction. It is later learned that the mixture falls mainly on the communes of Seveso, Meda, Cesano Maderno and Desio.
- 13:00 A foreman present at the plant telephones Dr Clemente Barni, the deputy head of production, who is on standby call that day.
- 13:10 Dr Barni arrives at the ICMESA plant.
- 13:45 A foreman turns on the cooling system, thereby stopping the escape of the mixture.
- 14:30 Dr Barni inspects the area immediately surrounding the factory but notices nothing out of the ordinary.
- 17:45 Dr Barni inspects the immediate surroundings of the factory and warns the inhabitants not to eat any local vegetables or fruit.
- 19:00 The factory porter is instructed by Dr Barni to reach the ufficiale sanitario, the local health officer for Seveso and Meda, Professor Giuseppe Ghetti. He is absent, and it is not possible to find out who his deputy is.
- 20:30 Dr Barni telephones the carabinieri of Meda and reports the incident. He inquires whether damage has been reported and requests that any such reports should be transmitted to ICMESA.

next days:

- 11.7.76: Dr Paoletti and Dr Barni inspect the surroundings for signs of damage to plants. It is decided that first the ufficiale sanitario or his deputy have to be informed; then visit the mayor of Meda and the mayor of Seveso; they then would again contact the carabinieri and then collect samples for analysis from the vessel and in the surrounding area. Dr Barni, together with a carabinieri, go around the area of Meda and warn the inhabitants. It is not possible to do the same in Seveso as the mayor cannot find any official.
- 12.7.76 ICMESA. Dr Barni closes off Building B. First samples arrive in Switzerland.
- 13.7.76 Death of smaller animals (rodents) is reported.
- 14.7.76 The first signs of skin inflammation (chloracne, see Fig. 9) occur in children. Traces of TCDD were found in the samples. The precise quantity of dioxin that escaped could not be determined after the accident. Estimates by experts vary from a few hundred grams to a few kilograms at most.
- 15.7.76 Examination of factory workers and inhabitants (2 cases of chloracne reported with local children, which were hospitalised). Population of affected villages was asked not to eat or touch fruit or vegetables. Warning signs are to be put up and the danger zone fenced off.
- 16.7.76 Between 12 and 16 children have been hospitalized. The evacuation recommended by ICMESA is postponed. (in total: The accident caused 447 cases of acute chemical burns and 193 cases of chloracne)
- 17.7.76 The management orders the factory to be closed as a precautionary measure. It was announced that in the accident TCDD was released.
- 20.7.76 Chemists confirm that TCDD was found in the samples.
- 21.7.76 Manager of factory is arrested. Italian government is (officially) informed. Other companies, who experienced dioxin accidents [Coalite (UK), BASF (D), Phillips (NL), Dow (USA)] are asked for assistance. Evacuation is recommended.
- 26.7.76 To start with, 208 people from 37 houses leave the contaminated area. Approximately 3 300 animals (mostly poultry and rabbits) are slaughtered. By 1978 some 77 000 animals will have been slaughtered.
- 30.7.76 Zone plan. The contaminated area is divided into three zones. Zone A has the highest degree of contamination (more than 50 micrograms per square metre). The contamination is less severe in Zone B (5-50 micrograms per square metre), while in Zone R no TCDD or only slight traces (less than 5 micrograms per square metre) are found. Zone A is further divided into 7 subzones according to the severity of contamination. The number of people affected in Zone A is 736 (they are all evacuated), in Zone B, about 4 700 and in Zone R, about 31 800.



Fig. 9: Seveso child with chloracne.

next months:

- July 76 1600 people of all age groups are examined at the outpatient clinic in Seveso. 447 of them are suffering from acute skin lesions, presumably caused by the escape of chemicals. The patients recover within two to three weeks.
- 2.8.76 New analyses make it imperative to evacuate a further 500 people from 90 houses in Zone A. A total of 736 persons have now been evacuated. The health authorities decide that children and pregnant women should leave Zone B during the day to minimise the danger of contact with TCDD. The regional health authorities permit pregnant women in the contaminated zones to decide freely whether they wish to continue their pregnancy to the full term (some women decide for abortion).
- 11.8.76 The Roche group commits itself to covering the costs of the damage.

24.8.76 Givaudan submits an initial decontamination plan worked out by the British engineering firm Cremer & Warner.

the years after:

May 77 Decontamination starts (Fig. 10): The insides of the buildings are cleaned with equipment for sucking up dust and water and the outsides with a special soap solution. The contaminated waste water is collected in special containers. In the gardens plants are removed and the surface soil cleared away. The success of the decontamination work is assessed.



Fig. 10: Decontamination of zone A.

17.6.77 A programme of systematic epidemiological health monitoring of 220,000 people is launched.

10.7.77 First anniversary of the accident. In the meantime 7 000 soil analyses have been made. A high, 4 000 metre long fence now surrounds subzones A1-A5, while subzones A6 and A7 are sealed off by a smaller fence. The soil of subzones A6 and A7 is decontaminated. 112 houses, including gardens and yards, in subzones A6 and A7 are decontaminated.

Oct. 77 The first evacuated families return to their decontaminated houses. By year end a total of 511 persons can reoccupy their homes. Those houses in Zone A which are heavily contaminated are to be demolished and new houses built in another residential area of Seveso.

Nov. 77 Decontamination in the less affected zones (B-R) starts.

Spring 78 The fields are ploughed and feed crops planted. The harvest is to be analysed and then destroyed. The dioxin content of the soil is also to be monitored.

Jan. 80 The important feeder road from Meda to the Milan-Como motorway is decontaminated and opened to traffic. (after only 4 years!)

Feb. 80 A special disposal site is opened and contaminated material stored (Fig. 11)



Fig. 11: Waste deposit in Zone A.

- Oct. 80 One third of Zone R, or about 417 hectares, is released for agricultural use and horticulture.
- Nov. 81 With a few exceptions, all parts of Zone R are approved for agricultural use.
- Spring 82 Before the reaction vessel in Building B can be emptied, the residues must be disposed off. No viable solution can be found in Italy or in Switzerland. The negotiations subsequently conducted in various European countries are not successful. No one wants to dispose of the waste from Seveso, even though special waste disposal sites and high temperature incinerators are available. In the spring of 1982 the Italian authorities recommend the firm Mannesmann Italiana, which had worked for the Italian Republic in the past. Mannesmann Italiana then submits a disposal plan, in which the company undertakes to ensure transport and disposal of all waste containing dioxin from the reaction vessel in a suitable and authorised site and to obtain all necessary Italian and foreign permits. On account of the origin of the residue, Mannesmann Italiana makes it a condition that the disposal site should be kept secret from Givaudan.
- July 82 Emptying the reaction vessel. When the official permits have been obtained, volunteers from Roche Basel begin the extensive task of emptying the vessel in a carefully planned operation conducted under strict safety precautions. By the evening of 9 September, 41 barrels are ready for transport.
- 10.9.82 The contaminated residue from the reaction vessel, packed in 41 barrels, leave the ICMESA premises. The truck has an official escort as far as the Italian border at Ventimiglia by the Italian authorities.
- 23.2.83 Search for the barrels. The French Swiss television programme 'A bon entendeur' reconstructs on film the journey of the 41 barrels to St-Quentin in France. After that, all trace of them is lost. Various hypotheses on the whereabouts of the barrels are put forward.
- 30.3.83 St-Quentin. Bernard Paringaux, head of the disposal company Spelidec in Marseille and the second subcontractor of Mannesmann Italiana, who had arranged the transport of the barrels, is arrested in St-Quentin. He refuses to make any statement on the whereabouts of the barrels.
- 19.5.83 Finally the 41 barrels are found in an unused abattoir in Anguilcourt-le-Sart, a village in northern France. The barrels are transferred the very same evening to a military base near Sissonne.
- 24.9.83 The Criminal Court of Monza sentences five former employees of ICMESA and Givaudan to periods of imprisonment ranging from 2 1/2 to 5 years. The five accused all appeal against the conviction, claiming that the accident could not have been foreseen.
- Feb. 84 In its final report, the International Steering Committee points out that, with the exception of chloracne (a total of 193 patients), no ill effects can be attributed to TCDD.
- Apr. 84 Decontamination of the most heavily contaminated area, Zone A, is completed.
- 19.3.85 A second incineration test (at a high temp. incinerator at CIBA), involving 360 kilograms, is completed successfully.
- 17.6.85 Main incineration. As the results of the second test are also positive, the incineration of the main batch can be carried out as planned. Ongoing analysis of flue gases ensures that no TCDD escapes into the atmosphere during incineration.

Costs:

Disbursements for the reparation of the damage caused by the Seveso chemical accident now totals over 300 million Swiss francs. Still pending are 24 civil lawsuits in the courts of Monza and Milan and the Supreme Court in Rome. The suits are claims for damage or amends by private citizens, the basis or amount of which is contested by Givaudan and ICMESA.

Longer term study: [7]

Although the follow-up period is only 15 years (and some cancers take 20 or more years to appear following exposure), the results are interesting.

When all types of cancers were grouped into one category, no statistically significant excess of cancer was observed.

6. Defoliants in the Vietnam War: *Operation Ranch Hand & Agent Orange*

Agent Orange was the code name for a herbicide developed for the military, primarily for use in tropical climates. Serious testing for military applications did not begin until the early 1960's.

The purpose of the product was to deny an enemy cover and concealment in dense terrain by **defoliating trees** and shrubbery where the enemy could hide. The product "Agent Orange" (a code name for the orange band that was used to mark the drums it was stored in, was principally effective against broad-leaf foliage, such as the dense jungle-like terrain found in Southeast Asia.



The product was tested in Vietnam (also Cambodia and Laos) in the early 1960's, and brought into ever widening use during the height of the war (Operation Ranch Hand 1967-68), though it's use was diminished and eventually discontinued in 1971.

Agent Orange was essentially a 50-50 mix of 2,4,D and 2,4,5,T. The combined product was mixed with kerosene or diesel fuel and dispersed by aircraft, vehicle, and hand spraying. An estimated **19 million gallons** of Agent Orange were used in South Vietnam during the war.

The earliest health concerns about Agent Orange were about the product's contamination with TCDD.

Table: 5: Defoliants used in Vietnam:

Description	TCDD (Dioxin)Amounts	used
Agent Orange	1.77 to 40 ppm	1965 - 1970.
Agent Blue (Purple)	32.8 to 45 ppm	1962 - 1964
Agent Red (Pink)	65.6 ppm	1962-1964
Agent White (Green)	65.6 ppm	1962-1964
2,4,5-T (Current)	0.1 ppm or less	

Facts:

- In the early years of WWII, a grant was provided by the National Research Council to develop a chemical to destroy rice crops in Japan (the major food source of the Japanese). 2,4-D and 2,4,5-T (Agent Orange) was the result. A discussion between President Roosevelt and White House Chief of Staff, Admiral William D. Leahy determined that this heinous chemical should not be used. Agent Orange was not used during WWII.
- In 1961, President Kennedy signed two orders allowing Agent Orange to be used in Vietnam. One order to destroy crops, and another order to defoliate the jungle. [Note: These orders were signed prior to major U.S. intervention.]
- Agent Orange and other herbicides were used extensively through 1970 (and thereafter until the end of the Vietnam War).
- The U.S. Air Force military operation *Ranch Hand* began spraying Agent Orange on areas of South Vietnam in August 1965 (though it was used prior to this date).
- The US Air Force Herb Tapes accounted for just over **8 million gallons** (other sources say **18 million gallons**) of the herbicides used in South Vietnam.
- Agent Orange caused Vietnamese farmers to lose about 70% of their crops. (One of the goals for using herbicides was to deprive the enemy of food.)

- 6250+ square miles of South Vietnam can not be farmed because of defoliation (see Fig. 12) . This is still true almost 30 years later.
- 1984: Vietnam Veterans and their families filed a class action suit against seven chemical companies (Dow Chemical, Monsanto, Uniroyal, Hercules, Diamond Shamrock, Thompson Chemical, and T.H. Agriculture). It was settled out of court in May 1984 for victims and families of those exposed to herbicides for \$180,000,000 (the lawyers got a staggering 100 million dollars) [8]

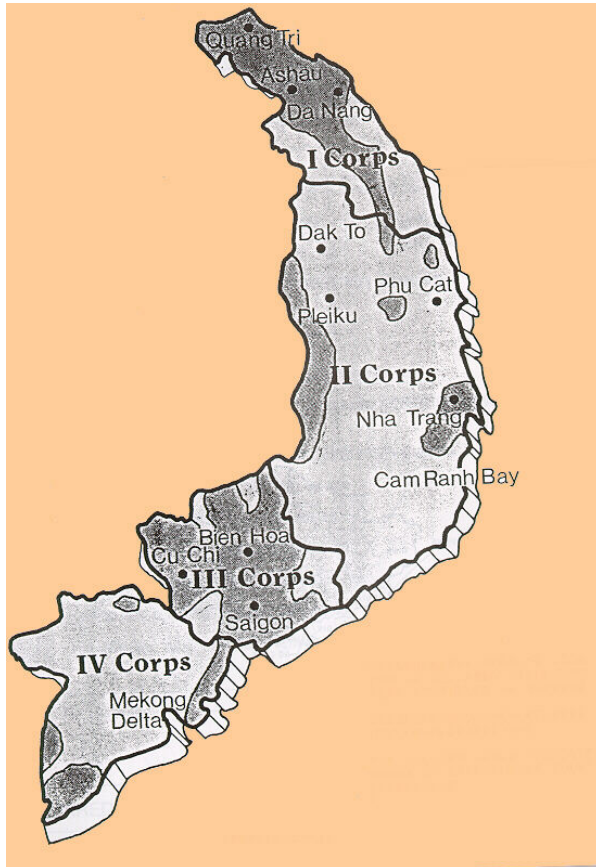


Fig. 12: This map is a representation of herbicide spray missions in Vietnam. The dark areas represent concentrated spraying areas. This map only represents fixed-wing aircraft spraying, and does not include helicopter spraying of perimeters, or other spray methods.

The III Corps area received the heaviest concentrations of spraying, followed by I Corps, II Corps and IV Corps.

The Department of U.S. Veterans Affairs offers service-connected compensation for the following diseases believed to be associated with Agent Orange exposure.

- Chloracne
- Non-Hodgkin's Lymphoma
- Soft tissue sarcoma
- Hodgkin's Disease
- Porphyria Cutanea Tarda (PCT)
- Multiple Myeloma
- Respiratory cancers (including cancers of the lung, larynx, trachea and bronchus)
- Prostate cancer
- Peripheral neuropathy (acute or subacute)
- Spina bifida (in the Children of Vietnam veterans)



7. PVC:

What's So Dangerous About PVC?

PVC (polyvinyl-chloride) is produced from imported, non-renewable fossil fuels (**what other polymer does not?**) and lethal chlorine gas. Production involves transport of dangerous explosive materials and the creation of toxic waste. Additives like heavy metals or plasticisers needed for various applications of PVC can be carcinogenic or otherwise harmful, particularly when plasticisers evaporate or leach into food (**same is true for other plastics**). In the production or incineration of PVC (**what temperature?**), chlorine reacts with other chemicals to form unwanted by-products like dioxin, PCBs, hexachlorobenzene, acidic gases, and others. PVC cannot be effectively recycled because of its different chemical additives. If landfilled, it eventually releases its additives, threatening ground water (**Dioxin isn't solvable in water**). Fires and incinerators cause the release of PVC's toxic chemicals such as dioxin (**again temp.!**).

8. Consequences:

European Directive: Seveso II Directive [96/082/EEC]

This Directive is aimed at the prevention of major accidents which involve dangerous substances, and the limitation of their consequences for man and the environment, with a view to ensuring high levels of protection throughout the Community in a consistent and effective manner. (for details see [9])

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