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**TRICHLOROACETIC ACID**  
**CAS N°: 76-03-9**

## SIDS INITIAL ASSESSMENT PROFILE

CAS Nr.	76-03-9
Chemical Name	Trichloroacetic acid
Structural formula	CCl <sub>3</sub> -COOH
<b><u>CONCLUSIONS AND RECOMMENDATIONS</u></b>	
<p>Environment: A potential risk to the aquatic compartment is identified due to high toxicity to algae and local exposure from use as auxiliary in textile dyes, waste water from electroplating facilities, textile washing and pulp mills. A potential risk to the terrestrial environment is identified due to high toxicity to plants and global exposure from the decomposition of C2-chlorocarbons.</p> <p>Human health: The chemical is reprotoxic, corrosive and an eye irritant but adequate protection measures are currently being applied. Trichloroacetic acid is currently considered of low potential risk and low priority for further work.</p>	
<b><u>SHORT SUMMARY WHICH SUPPORTS THE REASONS FOR THE CONCLUSIONS AND RECOMMENDATIONS</u></b>	
<p>The production volume of trichloroacetic acid (TCA) is ca. 1000 t/a in Germany. TCA is mostly used in the production of TCA Na-salt used as a herbicide. TCA is also used as an auxiliary in textile dyeing processes.</p> <p>TCA is stable in neutral solution and is classified as "non biodegradable" with a "low bioaccumulation potential" for fish and a "high bioaccumulation potential" for terrestrial plants. The most sensitive environmental species to TCA is the alga <i>Chlorella pyrenoidosa</i> (14d-NOEC = 0.01 mg/l) and pine (60d-EC10 = 0.12 mg/kg).</p> <p>The acute oral, dermal and inhalation toxicity is low. This chemical is corrosive and strongly irritant to the eyes. The NOEL in a 90-day study in dogs - the most sensitive species tested - was determined as 500 ppm (approx. 30 mg/kg bw/day). The NOEL for repeated dose toxicity in a 4-month feeding study with rats was 4000 ppm (365 mg/kg bw/day), the NOEL in a 2-year feeding study in rats was 1600 ppm (80 mg/kg bw/day).</p> <p>An inconsistent picture was found in tests on genotoxic action. Point-mutation tests were predominantly negative. In-vivo tests of chromosome mutations were mostly positive, but effects only appeared after high loading of the animals. The SCE test in mice was negative. The results of a micronucleus test in mice are apparently not reproducible. The end point of the sperm anomaly test is not necessarily due to genetic damage. The validity of the positive test results described for the clastogenic effects in mice suffers from the partly insufficient experimental procedure.</p> <p>Drinking water studies in male and female mice to 52 or 61 weeks gave an increased incidence of tumours in the livers of the male mice only. A 2-year feeding study with rats and a drinking water study over 100 - 104 weeks in rats showed no evidence of carcinogenicity.</p> <p>Reproduction toxicology investigations in rats showed maternal and embryonic toxicity from 330 mg/kg bodyweight and from 800 mg/kg also embryo-lethality. In all dose-groups there was a dose-</p>	

dependent increase in visceral anomalies, particularly in the cardiovascular system. The mean frequency of soft tissue malformations ranged from 9% at the low dose (330 mg/kg) to 97% at the high dose (1800 mg/kg/day). A NOAEL could not be established. Based on these findings TCA was considered to be developmentally toxic in the pregnant rat at doses of 330 mg/kg and above.

The aquatic local PEC probably due to its use in textile finishing industry was estimated to be 7 - 27 µg/l. The PEC in natural soil due to atmospheric oxidation of C2-chlorocarbons is 8 - 150 µg/kg.

In conclusion, TCA represents a risk to both the hydrosphere as well as the soil compartment.

Considering the low exposure potential to humans, available toxicity data support a low risk to human health. The tumorigenic action in male mouse liver corresponds to the type, which leads to liver tumours preferentially in male mice via peroxisome proliferation, hepatotoxicity and liver cell proliferation.

**IF FURTHER WORK IS RECOMMENDED, SUMMARISE ITS NATURE**

There is a need for consideration of risk management measures, concerning the environment and these should be addressed by the Risk Management Advisory Group.

Measurements in forest soils should be performed to actualise the soil pollution.

## FULL SIDS SUMMARY

CAS-NO.: 76-03-9		PROTOCOL	RESULTS
PHYSICAL CHEMICAL			
2.1	Melting-Point	NA	56 °C
2.2	Boiling-Point	DIN 53171	197 °C (at 101.3 kPa)
2.3	Density	NA	ca. 900 kg/m <sup>3</sup>
2.4	Vapour Pressure	NA	0.12 kPa at 50°C
2.5	Partition Coefficient (Log Pow)	exp. calc.	-0.27 1.33
2.6 A	Water solubility	NA	miscible at 20°C
B	pH	/	at °C
	pKa	NA	0.7
2.12	Oxidation : Reduction potential	/	mV
ENVIRONMENTAL FATE / BIODEGRADATION			
3.1.1	Photodegradation	calc. (Atkinson)	In air T <sub>1/2</sub> = 446 days
3.1.2	Stability in water	NA	ca 1% hydrolysis in 4 - 6 weeks
3.2	Monitoring data		In air = / mg/m <sup>3</sup> In surface water = 0.09 - 370 µg/l In soil / sediment = 0.02 - 0.4 µg/g In biota = ND - 0.1 µg/g
	Transport and Distribution	calculated (fugacity level 1 type)	In air / % In water / % In sediment / % In soil / % In biota / %
3.5	Biodegradation	OECD 302 B OECD 302 A OECD confirmatory test	10% after 27 d 0 - 46% after 28 d max. 10%

ECOTOXICOLOGY (lowest effect data only)				
4.1	acute/prolonged toxicity to fish	Pimephales promelas	NA	LC <sub>50</sub> (96 hr) = 2000mg/l
4.2	acute/prolonged toxicity to aquatic invertebrates ( daphnia )	Daphnia magna	NA	EC <sub>50</sub> (48 hr) = 2000mg/l
		Somatochlora cingulata	NA	LOEC (8h) = 0.01 mg/l
4.3	toxicity to aquatic plants e. g. algae	Chlorella pyrenoidosa	NA	EC <sub>50</sub> (14 d) = 0.3 mg/l NOEC (14d) = 0.01 mg/l
4.4	toxicity to microorganisms	Spirulina platensis	NA	EC <sub>50</sub> (14 d) = 5 mg/l
4.5.1	chronic toxicity to fish	Cyprinus carpio	NA	LOEC (63d) = 7 mg/l
4.6.1	toxicity to soil dwelling organisms	Eisenia foetida	87/302/EEC	LC <sub>50</sub> (14 d) = 1140mg/kg dw
		Allobophora rosea	NA	LC <sub>100</sub> (90d) = 20mg/kg dw
4.6.2	toxicity to terrestrial plants	Avena sativa	BBA (1984)	EC <sub>50</sub> (14 d) = <1 mg/kg dw
		pine & spruce	NA	EC <sub>10</sub> (60d) = 0.14mg/kg dw
TOXICOLOGY				
5.1.1	acute oral toxicity	dog rat mouse	Hoechst internal	LD <sub>50</sub> = 1590 - 2000mg/kg LD <sub>50</sub> = 3310 - 6900mg/kg LD <sub>50</sub> = 4970 mg/kg
5.1.2	acute inhalation toxicity	rat, rabbit, cat, guinea pig	Hoechst internal	LC <sub>50</sub> (4h) > 4800 ppm
5.1.3	acute dermal toxicity	rat	Hoechst internal	LD <sub>50</sub> > 2000 mg/kg
5.4	repeated dose toxicity	rat dog	Hoechst internal	NOEL(2 years) = 80 mg/kg NOEL(90d) = 30 mg/kg
5.5	genetic toxicity in vitro bacterial test (gen mutation)	several	NA	- (with metabolic activation) - (without metabolic activation)
5.6	genetic toxicity in vivo	mouse mouse mouse	cytogenetic micronucleus SCE	+ and - + and - -
5.8	toxicity to reproduction	/	/	
5.9	developmental toxicity / teratogenicity	rat	NA	NOEL >= 330 mg/kg (general toxicity) NOEL >= 330 mg/Kg (pregnancy/litter) NOEL >= 330 mg/Kg (foetal data)
5.11	experience with human exposure			

## SIDS Initial Assessment Report

### 1. Identity

Name:	Trichloroacetic acid
CAS Nr.:	76-03-9
Empirical Formula:	C <sub>2</sub> HCl <sub>3</sub> O <sub>2</sub>
Structural Formula:	CCl <sub>3</sub> -COOH
Purity of industrial product:	98%
Major impurities	Dichloroacetic acid, sulfuric acid, water

### 2. Exposure

#### 2.1 General discussion

In 1993, ca. 1000 t were produced in Germany (6). There is no information available on production volumes in other countries.

In Germany and in the USA, trichloroacetic acid is industrially produced by chlorination of acetic acid (5) (6).

The use pattern in Germany is the following (6):

670 t	export
250 t	processing to sodium trichloroacetate (NaTCA)
50 t	processing to trichloroacetic acid-ethylester
30 t	sale in Germany (mainly to producers of pharmaceuticals)

Of the produced NaTCA, ca. 110 t/a are sold in Germany and the rest is exported.

For production and processing, discharges occur at the German site into waste water in amounts of ca. 1 kg/a and about 17 kg/a via exhaust air (6).

At a second German site, where ca. 100 t of NaTCA are used in the formulation of textile finishing products, 2 t/a are released to a waste water treatment plant (6).

The main application for NaTCA is its use as a selective herbicide and in formulations with 2,4-D and 2,4,5-T preparations as a total herbicide. In Germany and in Switzerland, the sale and import of NaTCA as a herbicide is prohibited since 1989. In the USA, NaTCA is still used as a herbicide.

Several other uses are reported in literature (1). TCA is employed as an etching or pickling agent in the surface treatment of metals, as an albumin precipitating agent in medicine, as an auxiliary in

textile finishing, and as an additive to improve high-pressure properties in mineral lubricating oils. Because it is strongly corrosive, TCA is used to remove warts and hard skin and to treat various skin afflictions. Particularly the esters of TCA are important starting materials in organic syntheses.

For Germany, the only environmentally exposure-relevant use of TCA, which could be identified, is its use as an auxiliary in textile finishing (ca. 110 t/a (6)). According to the German producer, TCA is included in dyes as an alkali-liberating auxiliary for fixing reactive dyes on fibres for printing processes. In the usual formulations, the relative ratio to the actual dye is 1:1. Its average concentration in the formulations is 40 g/kg. Ca. 2.9 g TCA are necessary to completely dye 100 g textile.

According to a US-importer, TCA is also used as a glue precipitant, and as an etching agent in the manufacture of photographic films (5).

Furthermore, TCA is formed as a by-product:

- from chlorination of drinking water and swimming pools;
- in chemical cleaning units using tetrachloroethylene;
- in electroplating facilities treating cyanide containing waste water with NaOCl;
- in textile washing facilities using NaOCl as bleaching or disinfection agent;
- during bleaching of paper in paper mills;
- during the atmospheric oxidation of C2-chlorocarbons. This subject is elaborated more detailed in Appendix I.

Some authors set up the hypothesis that TCA could be formed in soils by enzymatic chlorination of naturally occurring compounds. In an *in vitro* laboratory test, TCA formation was simulated incubating different natural occurring short chained aliphatic acids with the chlorinating enzyme chloroperoxidase in the presence of sodium chloride and hydrogenperoxide. Trichloromethane and chlorinated acetones were identified as main products, while TCA was formed in low yields (8% with acetic acid, less than 1% with malic acid, lactic acid, fumaric acid, malonic acid, citric acid and acetonedicarboxylic acid ). As a high peroxide concentration (3 g/l) was used, it seems to be questionable that under natural conditions TCA is formed in any significant amount (19).

## 2.2 Environmental exposure

### 2.2.1 General/Environmental fate

TCA is miscible with water. The pure solid compound has a vapour pressure of 120 Pa at 50 °C. In aqueous solution, TCA dissociates ( $pK_a = 0.7$ ) and volatilization from water is not to be expected to a high degree. The measured log  $P_{ow}$  amounts to - 0.27 (probably dissociated form). The log  $P_{ow}$  of the undissociated TCA was calculated to be 1.33. Bioaccumulation factors of 0.4 - 1.0 and <1.7 have been determined experimentally (OECD GL 305C). Bioaccumulation in fish is therefore considered to be low.

In plants, a rapid uptake and distribution of TCA occurs. 22 days old seedlings of oat and wheat were exposed to a solution containing 0.03 mg/l  $^{14}C$ -TCA for 42.5 h. After exposure the seedlings were transferred into pots containing clay laom soil and were grown to appropriate stages of maturity. Uptake and distribution of  $^{14}C$  was examined with time for 16 weeks. A rapid translocation of  $^{14}C$ -TCA from roots to shoots was observed especially for wheat. In the roots of both

plants a substantial reduction of  $^{14}\text{C}$  was found 3 weeks after absorption. However, elimination from shoots was very slow and incomplete (50% after 16 weeks).

With barley and oat as test species bioaccumulation of  $^{14}\text{C}$ -TCA in plants was examined. The plants were grown in a closed aerated system in sandy agricultural soil treated with TCA (1 mg/kg dw) and were thus able to take up the chemical both by the roots from the soil and by the leaves from the air where the TCA has been volatilized. Also non-contaminated soil was placed in the closed system and protected against contamination by the air by a special close cover. In this soil plants were grown, the roots being in non-contaminated soil and the leaves in contaminated air. High bioconcentration factors (330 - 970) were determined 7 days after application. Uptake was both by roots and by leaves and transport within the plants was in both directions. The  $^{14}\text{C}$  residues in the plants grown in treated soil contained only small portions of the parent compound. For barley, only 8.5 % of total  $^{14}\text{C}$  present in shoots was TCA, and only 10.6 % in roots. For oats the corresponding figures were 8.7 % in shoots and 9.9 % in roots. The plants grown in untreated soil contained only unknown polar metabolites.

The uptake of TCA in the needles of two year old potted Scots pine (*Pinus sylvestris*) seedlings was studied (18). Two exposure set-ups, the root route and the atmospheric route simulating wet deposition of fog were used. Seedlings were exposed via roots by applying 5 ml of a TCA solution containing 0.02 mg/l respectively 1 mg/l into each pot, giving doses of 0.1  $\mu\text{g}$  and 5  $\mu\text{g}$  TCA respectively. The wet deposition treatments to the foliage were done inside teflon chambers which were placed over the seedlings during the treatment. 10 ml of a TCA solution (1 mg/l and 50 mg/l) were fumigated with bottled synthetic air by nebulizers. The aqueous TCA fog was distributed on all surfaces in the chamber, thus the exact TCA dose obtained by the needles is not known. The pots of the seedlings exposed to TCA by wet deposition were covered with a piece of plastic to prevent contamination of the soil. In both experiments seedlings were exposed 3 times a week for 9 weeks. TCA was effectively incorporated into the needles via roots. TCA concentrations were 700 - 800  $\mu\text{g}/\text{kg}$  and 40 - 60  $\mu\text{g}/\text{kg}$  at the end of the experiment in the second year needles (C+1) treated with 5  $\mu\text{g}$  and 0.1  $\mu\text{g}$  TCA respectively. The levels in the current year needles (C) were 2-3 times lower. About 80 % of the TCA applied as wet deposition remained on the surface of the needles and could be removed by rinsing with water. The TCA levels inside the C+1 needles at the end of the exposure were about 270 - 320  $\mu\text{g}/\text{kg}$  and 60 - 80  $\mu\text{g}/\text{kg}$  in the needle treated with high and low doses of TCA respectively. The C-needles exhibited about 1.5-4 times lower concentrations than the older C+1 needles.

Bioaccumulation of TCA in plants is therefore considered to be high.

Due to the ionic nature of TCA, the leaching behaviour in soil can not be described with log  $P_{\text{ow}}$  and water solubility only. Binding to the mineral parts of soil is possible, although not quantifiable. As found in a laboratory test, there is strong indication that TCA is physically or chemically bound on humic acids. TCA was added to aqueous solutions of humic acids in different concentrations, and the recovery rate for TCA analysis was determined. With the lowest tested humic acid concentration of 3 mg/l, only about 10% of the TCA could be extracted, and the recovery rate deteriorated with increasing humic acid concentrations (19).

Tests on ready biodegradability of TCA in the aquatic medium are not available. Nevertheless several test results on inherent biodegradability are available. In two Zahn-Wellens-Tests with industrial activated sludge, TCA proved to be non biodegradable (ca. 5 - 10 % DOC removal after 27 days). In a modified MITI-II-Test (with 100 mg/l instead of 30 mg/l test substance; 100 mg/l inoculum) biodegradation rates of 0 - 46% in 28 days were recorded.

On the other hand, in a modified OECD confirmatory test, no significant biodegradation was observed; even after increasing the retention time up to 48 hours, only a maximum of 10 % biodegradation with regard to Cl<sup>-</sup>-liberation could be achieved.

In aerated lagoons receiving the effluents from mills producing pulp from wood the removal was 22% in one treatment plant (age 3 years; hydraulic retention time 2.5 days) and 99 % in a second (age 10 years; hydraulic retention time 5 days).

Despite the many negative biodegradation results in laboratory tests, a significant elimination of TCA in waste water treatment plants can be expected, based on monitoring data.

Indeed, the elimination of TCA was monitored in 5 domestic waste water treatment plants (wwtps) in Switzerland ranging from 24 to 90 %. An average removal rate of 66% was recorded (average influent concentration: 1.4 µg/l; effluent conc.: 0.43 µg/l). The elimination variation in the different wwtp cannot be conclusively explained as data on operating conditions of the wwtps are available only for 2 plants. A possible explanation would be a difference in hydraulic retention times, being 19 hours in one (87% elimination) and 12.5 hours in the second (47% elimination). The average hydraulic retention time in domestic wwtps is estimated to be about 7 - 14 hours though. Therefore, for a realistic worst case estimation, it would be appropriate to assume a wwtp elimination of 24%. On the other hand, for a complete exposure assessment, the sequential use of worst case situations, which would lead to unrealistic worst case situations have to be avoided. As other worst case assumption are already included in most of the exposure scenarios (e.g. a dilution factor in surface waters of 10), the average elimination rate of 66% can nevertheless be used in the following exposure estimation.

Biodegradation of TCA in soil was also investigated, but no standardized investigations or measurements comparable with standard procedures are available.

In suspensions of different soils the dt<sub>50</sub>-values of TCA biodegradation (with respect to Cl<sup>-</sup>-liberation) amounted to 10 to 30 days with probes of the upper 5 cm of soil profiles and 16 to 68 days for deeper soil levels, depending on the origin of soil samples. The high numbers of microorganisms in soil probes that were able to grow with TCA as sole source of energy indicate that there might be a preadaptation of the soil microorganisms to TCA.

In a laboratory study, after 5 month, biodegradation reached 29% in a fine sand and 81.7% in a humus soil estimated from remaining radioactivity in soil. The high numbers of microorganisms in soil probes that were able to grow with TCA as sole source of energy indicate also here that there might be a preadaptation of the soil microorganisms to TCA.

In two field studies, it could be demonstrated that TCA is biologically degraded from soil microorganisms. In one study dt<sub>50</sub> values varied between 18.1 and 25.5 days depending on soil types and experimental conditions. The initial chemical burden of soils could not be estimated from literature in both studies. It is demonstrated that replicated treatment of soil with TCA reduces the lag-period and dt<sub>50</sub>-values significantly.

As a conclusion, TCA is biologically degraded from adapted soil organisms. Biodegradability of TCA from non-adapted soil organisms can not be finally assessed from available data.

For the assessment, only a very slow biodegradation rate can be assumed. Based on the above results, at best a biodegradation half-life of 150 days can be assumed. This half-life is derived from the 5 month study measuring remaining radioactivity in soil.

Zakordonec et al. (1987) found dichloroacetic acid and monochloroacetic acid as metabolites of NaTCA degradation in forest soil.

### 2.2.2 Monitoring data

#### atmosphere

Only one investigation on the occurrence of TCA in the atmosphere is available. In 1992, it was detected in concentrations of 0.4 to 3 ng/m<sup>3</sup> in unfiltered air sampled in a German rural area (15).

#### hydrosphere

The average concentration in the lake "Greifensee" in Switzerland was measured to be 0.09 µg/l. The 90-percentile value was 0.12 µg/l (50 samples over a depth of 0 - 30 m from 6.12.92 - 4.10.93). The concentration in other Swiss lakes was below 0.1 µg/l.

The concentration in several Swiss rivers varied from 0 - 0.34 µg/l (average 0.14 µg/l). The 90-percentile was 0.33 µg/l (30 samples from 7 rivers).

The TCA-concentration in the Austrian river "Pöls" downstream of a pulp mill varied between 148 and 558 µg/l (10 samples; average: 370 µg/l). In the river "Mur", downstream of another pulp mill the concentrations were < 3 - 104 µg/l (6 samples; average 56 µg/l).

#### rain water

The measured TCA-concentrations in rain water in 1989 in Germany (Hessen) are 0.2 - 6.5 µg/l (94 samples from 8 locations). The average concentration was 1.7 µg/l. Further rain samples were taken over a year in 1990/91 in Berlin with concentrations of 0.1 - 20 µg/l. The yearly average was 2.1 µg/l in wooded areas and 0.9 µg/l elsewhere.

In 1991/92, TCA was measured in the rain water in a distance of about 30 km from Siegen (Germany), where metal industry is settled. The concentrations were < 0.1 µg/l in December and 2 µg/l in June with an annual average of 0.57 µg/l. At a site in the vicinity, 0.1 - 0.21 µg/l were detected, without seasonal dependence (19).

The average concentration in rain water was determined as 0.3 µg/l in Switzerland in 1993 (44 samples at 2 locations). The 90-percentile value was 0.62 µg/l (11).

In the summer of 1993, TCA concentrations between 0.044 and 0.710 µg/l were measured in the city of Zürich. The mean TCA concentrations decreased from 0.3 to 0.13 µg/l in the period 1993-1995. Simultaneously the concentrations of some chlorohydrocarbons decreased at a sampling point in the vicinity indicating that they could be the precursors. In Alpthal (a rural area in Switzerland) similar TCA concentrations than in Zürich were measured. The deposition by canopy runoff from the trees was larger than by rain in the open field. This is explained by the authors by dry deposition onto trees with subsequent rinsing (14).

During a monitoring program in Germany in 1993/94, TCA was measured in the rain water both in open fields and forested areas at 9 rural sites. The maximum concentrations (2-months-mixed samples) were in the range from 0.37 - 2.2 µg/l with mean values (averaged over 15 months) of 0.2 - 0.85 µg/l in forested areas. In open fields (the sampling sites were in the near vicinity of the forest sites), the maximum values were 0.33 - 0.47 µg/l and the mean values 0.17 - 0.28 µg/l (20).

The rain water monitoring data reveal that the TCA concentrations in urban and industrialized regions are substantially higher than in rural regions. Several investigations show a seasonal dependence, the concentrations in summer are higher than in winter. These results indicate that the main source of the atmospheric TCA is the photochemical degradation of tetrachloroethylene (cf Appendix I). TCA is not only deposited by rain water, but by dry deposition e.g. on leaves and needles from which it is rinsed during rainfalls; this leads to an advanced pollution of forest soils.

#### waste water

The average effluent concentration from 5 domestic wwtps in Switzerland was 0.43 µg/l. The effluent concentration in the wwtp of a paper mill in Switzerland was measured at 104 µg/l (one 24h-sample). The untreated waste water of 4 electroplating facilities, treating their waste water with NaOCl contained 0.1 - 87 µg/l TCA (6 samples; average: 32 µg/l). The untreated waste water of 4 textile washing facilities, using NaOCl for bleaching, contained 2,3 - 205 µg/l TCA (7 samples; average: 97 µg/l).

#### swimming pools

The water in 34 swimming pools in Switzerland were analyzed. The average TCA concentration was 64 µg/l.

#### soil compartment and biota

In 1986, trichloroacetic acid was measured in fir and spruce needles of forest trees (Black Forest, Northern Alps, Germany) in concentrations up to 0.1 mg/kg fresh needle weight. The TCA concentration greatly depended on the needle age, time and place of the sampling as well as on the meteorological conditions. The TCA concentrations were relatively high (up to 300 µg/kg needles) after dry- and sunny periods, while it dropped to 1/10 or less after rainy periods. The youngest needles on one spruce branch contained 30 µg/kg, and older needles contained 80 µg/kg. In soil samples from the same forests (20 cm depth) TCA was also detected at concentrations of 0.02 - 0.4 mg/kg soil in 1988 (not clear whether wet weight or dry weight). The high concentrations can be explained by the characteristics of the sampling sites: on the mountains a high photolytical activity is combined with high rainfall levels.

Further pine needles in the area of Berlin were sampled over 2 years in 1990/91. The concentrations were in the range of 0.7 to 175 µg/kg with monthly average concentrations of 10 - 55 µg/kg fresh weight.

In another monitoring programme in Germany, soil leachate (passing through a 10 cm layer of natural soil) was sampled at 4 locations. The concentrations (mean values for each location) were in the range of 0.14 to 2.80 µg/l (overall average 0.7 µg/l). Compared to the above reported concentrations in rain water (average 1.7 µg/l), which were measured during the same time at the same locations, the concentration is diminished during the passage of the rain water through the soil layer. The cause can be that TCA is degraded in soils by microorganisms or adsorbs onto soil solids (cf. 2.2.1).

In 1991/92, TCA was measured in the soil of 4 rural sites in the Netherlands. The concentrations were 1.0 - 2.7 µg/kg at a peat-moor, 2.6 - 4.6 µg/kg at a peat-bog, 0.2 - 0.9 µg/kg in a forest with beech trees and 0.2 - 1.3 µg/kg in a douglas forest. In douglas needles at the last site, 23 µg/kg were detected (21).

### 2.2.3 Exposure estimation / PEC - calculations

In the following, the releases leading to high local concentrations are modeled.

#### 2.2.3.1 hydrosphere

##### - local concentration due to formulation of textile dyes

At one German site, 2 t NaTCA/a are released to a wwtp during formulation of textile dyes (processing of 100 t NaTCA/a). With an assumed elimination rate in the wwtp of 66%, 0.7 t/a are released into surface waters. The site being located on the upper Rhine, a concentration at low flow (ca. 550 m<sup>3</sup>/s) is estimated at  $PEC_{local} = 0.04 \mu\text{g/l}$ . No data are available regarding other chemical producers performing the same operations. For a more generic approach, a site independent exposure scenario for intermediates is proposed in the the EU-Technical Guidance Documents (2), using a flow of 60 m<sup>3</sup>/s of the receiving river. Assuming the same processing volumes and release rates for other chemical producers, a  $PEC_{local}$  of 0.37 µg/l can be estimated.

##### - local concentration due to the use as auxiliary in textile dyes

According to the TGD, the colouring capacities of dye-houses vary within a wide range. An average weight of processed goods of 3000 kg/day is assumed implying a daily use of 86 kg TCA for dyeing the whole cloth. During the steaming process for fixation, TCA is decomposed under formation of sodium bicarbonate, soda and chloroform (3). In a laboratory simulation test, performed by the producer, TCA decomposed to 96.5%. With this decomposition rate, a daily release rate of 3 kg is estimated.

The default volumes of water used during wet process of textile material (including pretreatment like scouring, desizing, mercerizing, bleaching etc.) is ca. 250 m<sup>3</sup>/1000 kg textile (2). For those textile processing units, which do not perform all the pretreatment processes, lower waste water volumes would occur and subsequent higher concentrations of TCA.

With the above indicated waste water volumes, and the decomposition rate of 96.5 % a concentration in the raw waste water of 4 mg/l is calculated.

Most textile processing sites are rejecting their waste water into domestic waste water treatment plants (ca. 93 % in Germany ). Assuming a dilution factor of 5 upon entering the wwtp, an influent concentration of ca 0.8 mg/l is calculated.

Elimination during biological treatment is assumed to be 66 % (see above). With an elimination rate of 66% in wwtps and assuming a further dilution factor of 10 upon entering the surface water, a  $PEC_{local}$  of ca. 27 µg/l is calculated. Distribution processes immediately upon entering the surface water are not to be expected (see above).

For estimating the successive dilution steps, other data are available: in a recent survey of textile processing units by ETAD, the 20-percentile of the water flow of 25 waste water treatment plants receiving waste water from textile processing units is 5000 m<sup>3</sup>/d. The 20-percentile of the water flow of 18 rivers receiving waste water from these wwtps is 45000 m<sup>3</sup>/d.

With an release of 3 kg/d TCA, an elimination in wwtps of 66% and dilution in a total of 5000 + 45000 m<sup>3</sup>/d, the resulting PEC<sub>local</sub> is 20 µg/l.

The above scenario assumes that the whole cloth is dyed. On the other hand, print colour is usually applied only to specific areas by screen printing systems to achieve the planned design. For comparison, the PEC will be calculated for an average use of 30 kg dye per day (2), i.e. 30 kg TCA. With a decomposition rate of 96.5%, 1.05 kg TCA are rejected per day into the waste water. Eliminated in wwtps at a rate of 66% and diluted in 50000 m<sup>3</sup>/d, the resulting PEC<sub>local</sub> is 7 µg/l.

In conclusion, the expected PEC<sub>local</sub> in surface waters will be in the range of 7 to 27 µg/l.

- local concentration due to the treatment of cyanide containing waste water from electroplating facilities

The above described measurements in untreated waste water, reporting concentrations averaging 32 µg/l can be used for a PEC estimation. Assuming an elimination of 66% in wwtps and a dilution factor of 10 in receiving surface waters, a PEC<sub>local</sub> of 1 µg/l is estimated.

- local concentration due to textile washing with NaOCl bleach

The above described measurements in untreated waste water, reporting concentrations averaging 97 µg/l can be used for a PEC estimation. Assuming an elimination of 66% in wwtps and a dilution factor of 10 in receiving surface waters, a PEC<sub>local</sub> of 3.3 µg/l is estimated.

- local concentration due to pulp mill effluents

Monitoring data in surface water are available for this pathway. Due to the low number of samples (6 - 10), only the average concentrations can be used. A PEC<sub>local</sub> of 56 - 370 µg/l can be expected.

- regional concentration

The measured concentrations in the Swiss lake "Greifensee" can be considered as representative for a background concentration, i.e. PEC<sub>regional</sub> = 0.12 µg/l. For a correct exposure assessment, this background concentration needs to be added to the concentration due to point emissions, unless the latter are based on measurements in the receiving surface water.

In the following table, an overview of the different PECs are presented:

Scenario for PEC <sub>local,water</sub>	Concentration [µg/l]
formulation of textile dyes (generic)	0.37
formulation of textile dyes (site)	0.04
use as auxiliary in textile dyes	7 - 27
waste water from electroplating facilities	1
textile washing	3.3

pulp mills	56 - 370
<b>Scenario for PEC<sub>regional,water</sub></b>	<b>Concentration [<math>\mu\text{g/l}</math>]</b>
representative monitoring data from lake "Greifensee" (90-percentile)	0.12

### **2.2.3.2 atmosphere**

There are no direct emissions of TCA into the atmosphere during its production or use. However, the substance is formed due to the photooxidation of tetrachloroethene and 1,1,1-trichloroethane. The relative importance of both precursors to the environmental TCA is elaborated in Appendix I. The result is that tetrachloroethylene is the main source of TCA formed in the atmosphere.

Due to its low volatility, TCA is removed from the atmosphere by dry and wet deposition, which leads to an ubiquitous pollution of soils. Air-born TCA was detected in fir and spruce needles (cf. 2.2.2).

### **2.2.3.3 soil**

As adsorption onto sludge is not to be expected to a significant extent (log  $P_{ow}$  max. 1.33; no adsorption was observed in the Zahn-Wellens-Tests), agricultural soils are not polluted by this route.

The main release to soil, apart from its use as a herbicide, is expected through atmospheric deposition. The question whether TCA is formed in soil by a natural process (cf. 2.1) is not decided finally.

A realistic TCA concentration in soil can presently not be modelled as fundamental parameters like distribution constant and degradation rate are not known exactly. Thus, the assessment is performed on the basis of monitoring data.

The measured data in soil were 20 - 400  $\mu\text{g/kg}$  (assumed these are based on wet soil => ca. 23 - 450  $\mu\text{g/kg dw}$ ). As the tetrachloroethylene concentrations in German rural areas decreased by a factor of about 3 since 1986 (cf. Appendix III, Schauinsland), it can be assumed, as an approximation, that the TCA concentration also decreased by this factor. Thus, 8 - 150  $\mu\text{g/kg dw}$  can be assumed as the present concentration range being chosen for the PEC<sub>soil</sub> in this assessment.

Di- and monochloroacetic acid were found to be metabolites of TCA in soils (16). Several authors state that these compounds are more toxic than TCA, so the real risk which is only based on TCA is underestimated.

## **2.3 Consumer exposure**

In the USA, TCA is found as a disinfection by-product in drinking water. The quarterly median concentrations ranged from 4.0 to 6.0  $\mu\text{g/l}$  (35 water utilities). These concentrations being median values, the actual concentrations will be higher in 50% of all cases.

The extent to which TCA is used in consumer products is unknown.

## **2.4 Occupational exposure**

TCA is manufactured as powder and commonly used as intermediate immediately after manufacture. TCA, if not used immediately in a closed-loop production line, is bagged or drummed for shipment. The corrosive properties prompt workers to dramatically limit the potential exposure to TCA.

TCA is received by processing companies and users in drums/bags. Given TCA's corrosive properties, workers are likely to avoid contact.

Several occupational exposure limits have been developed for TCA. They ranged from 1 mg/m<sup>3</sup> to 7 mg/m<sup>3</sup>. No inhalation monitoring data was uncovered. Based on the TLV of 7 mg/m, inhalation doses for workers of 7 mg/day to 70 mg/day have been estimated. Considering the low dusting and the corrosive properties of TCA the exposure estimate was regarded as highly conservative.

### **3. Toxicity**

#### **3.1 Human Toxicity**

##### **a. Acute Toxicity**

Animal data:

After single oral (rat, mouse), dermal (rabbit), and inhalational (rat, guinea pig, rabbit, cat) administration in the form of the sodium salt, trichloroacetic acid can be designated as nontoxic.

TCA is corrosive to rabbit skin and strongly irritant to the eyes, but it does not provoke sensitization.

The sodium salt has a significantly lower local irritancy than the acid and is used in high concentrations (50 % solution) as a cauterizing agent.

Human experience:

The use of TCA-sodium salt as herbicide caused acute irritations of mucous membranes and respiratory difficulties when applied as an aerosol.

Medical reports of acute exposure effects showed mild to moderate dermal and ocular irritation. TCA is not readily absorbed through the skin.

Conclusion: non toxic after single application

Recommendation: no need for follow-up test

Priority setting: low priority or concern

##### **b. Repeated Dose Toxicity**

Animal data:

TCA causes an increase in mitosis in mouse hepatocytes after only a short exposure, and an increased incorporation rate of <sup>3</sup>H-Thymidine in mouse liver.

After repeated administration in the feed over 4 months in rats (sodium trichloroacetate was tested), 10,000 ppm (911 mg/kg bodyweight) led to an approximate 10 % reduction in bodyweight

development. No further findings of toxicological relevance were discovered. The NOEL was determined as 4000 ppm (365 mg/kg bodyweight/day).

In a subchronic feeding study, Na-TCA was administered daily over a 90-days period in the diet (moist feed) to five groups each consisting of 3 male and 3 female beagle dogs in test concentrations of 0, 500, 2000, 4000 and 8000 ppm. Toxicological examinations were carried out on behaviour, general health, body weights, food consumption, haematology, urinalyses and histopathological examination of the organs.

In the two highest dose groups, 5 males had to be killed in moribund state and one female died intercurrently of pleuritis. From 2000 ppm upwards body weight decreases occurred accompanied by impairment of general health, necroses of the gums and oral mucosa, anaemia (diminution of red cells, haemoglobin and haematocrit) and changes in the leucocyte count (leucocytosis and lymphocytopenia). From 2000 ppm upwards, the histopathological examinations showed hepatic and myocardial changes. Oligospermia and azoospermia were seen in 2 animals dosed with 2000 ppm and in all animals in the 4000 and 8000 ppm groups. From 4000 ppm upwards atrophy of skeletal muscles occurred. Urinalysis of the males from the 4000 ppm and 8000 ppm groups killed during the study revealed positive protein and bilirubin reactions.

Based on these findings the "no effect level" in this study was 500 ppm, equivalent to approx. 30 mg Na-TCA/kg body weight/day.

In a 2-year study in rats, 250, 630, 1600 or 10,000 ppm sodium trichloroacetate was administered with the daily feed. A significantly reduced bodyweight development was caused by 10,000 ppm. All other parameters investigated, such as the feed consumption, substance uptake, blood picture, serum enzymes, urine findings, and organ weights were in the normal range. Histopathological examinations showed that feeding with sodium trichloroacetate over 2 years in rats did not lead to any morphologically discernible organ damage. In particular, no evidence was found of a carcinogenic action. The NOEL was determined as 1600 ppm (approx. 80 mg/kg bodyweight/day).

Human experience: no data available

### **c. Carcinogenicity**

Animal data:

Drinking water studies of the carcinogenicity of trichloroacetic acid in male and female mice for 52 weeks (1000 or 2000 mg TCA/l) or 61 weeks (5000 mg TCA/l only in male mice) gave a significantly increased incidence of tumours in the livers of the male mice. Females showed no significant differences from the controls.

In a 2-year feeding study, TCA was administered to male and female rats in concentrations of 250, 630, 1600 or 10,000 mg/kg feed. There was no evidence of substance-specific neoplastic alterations in any of the groups.

A drinking water study over 100 - 104 weeks in rats with concentrations of 0, 50, 500 or 5000 mg TCA/l showed liver toxicity but no evidence of carcinogenicity.

Mechanistic investigations showed that TCA induces peroxisome proliferation in rat and mouse hepatocyte cultures, but not in human liver-cell cultures.

It is known that the male B6C3F1 mouse in particular is unusually sensitive to the development of liver tumours.

Because TCA causes liver tumours only in the male B6C3F1 mouse after the administration of very high concentrations, and the peroxisome proliferation detected represents an extensively rodent-specific process, which has a subordinate meaning in humans, a tumour-initiating action is unlikely for TCA.

Human experience: no data available.

Conclusion: While in male B6C3F1 mouse TCA is hepatocarcinogenic, there is no evidence that humans are at significant risk for hepatocellular toxicity.

Recommendation: No need for follow-up test. NTP has not conducted short-term toxicology or long-term toxicology and carcinogenesis bioassays on TCA.

Priority setting: low priority or concern

#### **d. Reproductive Toxicity**

Animal data:

Reproduction toxicology investigations after treatment of pregnant rats from 6th to the 15th day of gestation showed maternal and embryonic toxicity from 330 mg/kg bodyweight, and from 800 mg/kg also embryo-lethality. In all dose groups there was a dose-dependent increase in visceral anomalies, particularly in the cardiovascular system. Maternal spleen and kidney weights increased in a dose related manner. The mean frequency of soft tissue malformations especially in the cardiovascular system, ranged from 9% at the low dose (330 mg/kg/day) to 97% at the high dose (1800 mg/kg/day).

Human experience: no data available.

Conclusion: The authors of the developmental toxicity study considered TCA to be a developmental toxin in the rat at doses  $\geq$  330 mg/kg bodyweight.

Recommendation: no need for follow-up test

Priority setting: Low priority or concern considering the low exposure potential

#### **e. Genetic Toxicity**

Experimental data:

Trichloroacetic acid has been tested for genotoxicity in a series of Ames tests, in the Rec assay using *Bacillus subtilis*, and on the basis of point mutations in *Aspergillus nidulans* in vitro. In all these studies trichloroacetic acid gave negative results, except in one, in which dimethyl sulfoxide (DMSO) was used as a solubilizer. In the authors' opinion TCA reacts with DMSO to give a mutagenic intermediate which subsequently decomposes, because after an hour the mixture is no longer mutagenic.

Free as well as neutralized TCA were incubated with human lymphocytes (500, 2000 and 3500  $\mu\text{g/ml}$ ) and examined for induction of chromosome aberrations. The free acid acted clastogenic with and without S9-mix, however only in concentrations at which the pH value was significantly

reduced. The neutralized TCA did not cause a significant increase in chromosome damage (Styles et al., 1992).

TCA did not induce any DNA-strand breaks in hepatocyte cultures of B6C3F1-mice and Fisher-344 rats after a 4-hour incubation with up to 10 mM. No DNA-strand breaks were determined also after a 2-hour incubation with human CCRF-CEF cells (same concentration, Chang et al., 1992).

TCA was examined on the mouse for induction of chromosome aberrations, micronuclei and sperm-head anomalies. Three adults Swiss mice each were used per end-point; per animal, 100 metaphases were examined for chromosome aberrations, 1000 poly- and normochromatic erythrocytes for micronuclei and 500 sperms for anomalies. The study on chromosome aberration took place under various exposure conditions: the preparation of the bone marrow occurred 24 hours after the last application following intraperitoneal injection of 125, 250 or 500 mg TCA/kg bw or 100 mg TCA/kg bw and day for 5 days or after oral administration of 500 mg/kg bw. After intraperitoneal injection of 500 mg/kg bw, the bone marrow preparation took place after 6, 24 and 48 hours. An increase in chromosome aberrations in the range of 3-6 % compared to the controls (0.16-0.2 %), could be observed under all test conditions. Hereby, the single administration of the substance was more effective than the fractionated application. Moreover, the results depended on the time of the bone marrow preparation (strongest effects after 24 hours) and on the application route (effects after intraperitoneal application were stronger than after oral application; Bhunya and Behera, 1987).

In the micronucleus test, mice were intraperitoneally treated twice within 24-hour intervals with 125, 250 or 500 mg TCA/kg bw. The preparation of the bone marrow was performed 6 hours after the last application. There was an increase in the micronuclei in poly- and normochromatic erythrocytes. Nevertheless, the findings were not dosage-dependent, because the weakest effects were shown at a dosage of 250 mg/kg bw (Bhunya and Behera, 1987)

In vivo, a negative result was obtained in a well conducted and well reported bone marrow micronucleus study in mice (Mackay et al. 1995). In contrast, statistically significant (but non-dose-related) increases in the frequency of cells containing micronuclei were observed in the Bhunya and Behera-study, 1987. The study by Mackay et al. was conducted to GLP and QA standards in an attempt to confirm the study by Bhunya and Behera. It employed the same study design, but with the addition of an extra sampling time at 24 hours. Mackay et al. proposed „the positive results previously observed with TCA may have been due to a non-genotoxic mechanism, possible caused by physicochemically induced stress, resulting from intraperitoneal pH changes“. In addition, Bhunya and Behera reported a positive test for chromosome aberration in mouse bone marrow. It is possible that these results may have also arisen via a non-genotoxic mechanism attributable to pH changes as proposed by Mackay et al.

For studying sperm-head anomalies, the animals were treated for 5 days intraperitoneally with 125, 250 or 500 mg TCA/kg bw and day. The preparations were produced 35 days after the last application. A dosage-dependent increase in sperm-head anomalies of 4.4-7.6 % was found compared to the controls (2.06 %; Bhunya and Behera, 1987).

None of the cited studies carried along positive controls in order to define the sensibility of the test systems.

Two other micronuclei tests produced negative results (DeAngelo et al., 1989; Styles et al., 1992). In one test, the mice were intraperitoneally administered neutralized TCA twice within 24 hours (25 %, 50 % and 80 % of the mean lethal dosage, corresponding up to 1300 mg/kg bw). On the

whole, no biologically significant increase in micronuclei was measured in the polychromatic erythrocytes.

One sister-chromatide-exchange (SCE) test on the mouse was likewise negative. Nonetheless, data are missing on the dosage and type of application (DeAngelo et al., 1989).

Three male Sprague-Dawley rats and B6C3F<sub>1</sub>-mice each received different oral doses of TCA. After 4 hours, the animals were killed and the induction of single-strand breaks on the liver-DNA was measured by alkaline elution. A dosage-dependent increase in strand breaks could be detected as of dosages of 0.6 mmol/kg bw (about 98 mg/kg bw) for the rat and as of 0.006 mmol/kg bw (about 0.98 mg/kg bw) for the mouse. Hepatotoxic effects were not found at these dosages by measuring the content of aspartate-aminotransferase (AST) and alanine-aminotransferase (ALT). In addition, there was no indication of peroxisome proliferation (Nelson and Bull, 1988). Also after the single oral administration of 500 mg TCA/kg bw, there was an increase of single-strand breaks in the liver-DNA for B6C3F<sub>1</sub>-mice without signs of peroxisome proliferation. In contrast, the 10-day treatment with 500 mg TCA/kg bw and day caused peroxisome proliferation and a mild hypertrophy of the liver, while no strand breaks were detected in the liver-DNA (Nelson et al., 1989).

TCA induced DNA-strand breaks neither in mice (cells from the liver, spleen, intestine and stomach) nor in rats (liver cells) after oral administration respectively of 10 mmol/kg bw (about 1600 mg/kg bw; mouse) and 5 mmol/kg bw (about 800 mg/kg bw; rat; Chang et al., 1989, 1992).

The results of Chang et al. are supported by other studies in which male B6C3F<sub>1</sub>-mice were administered once, twice or thrice daily oral doses of 500 mg TCA/kg bw as a free acid or a neutralized solution and then were killed 1 hour or 24 hours after the last application. No significant occurrence of DNA-single-strand breaks was measured in the liver cells. Within the first 4 hours after application, there was also no indication found of a binding onto DNA or other macromolecules. However, a low but significant increase in hyperplasias and peroxisome proliferation was detected in the liver (Styles et al., 1991).

TCA was administered in drinking water to male B6C3F<sub>1</sub>-mice for up to 14 days in dosages of 0, 300, 1000 or 2000 mg/l and day (ca. 75, 250 or 500 mg/kg bw). After 2, 5 or 14 treatment days, the animals were intraperitoneally treated with [<sup>3</sup>H]thymidine and killed two hours later. A dosage-dependent and, in the highest dosage, significant increase of the incorporation of [<sup>3</sup>H]thymidine could be observed in the livers of the animals that were treated for 5 and 14 days, respectively. Because the cell division rate did not simultaneously increase, the authors contend that the observed effects could indicate an increased DNA-repair synthesis rate (Sanchez and Bull, 1990).

Groups of 5 male and female B6C3F<sub>1</sub>-mice each were daily administered by gavage 100, 250, 500 or 1000 mg TCA/kg bw in corn oil for a period of 11 days. 24 hours after the last application, the animals were intraperitoneally administered [<sup>3</sup>H]thymidine. The incorporation of radioactively labeled thymidine into the liver-DNA was significantly increased in all treated animals. Only marginal changes in the liver, similar to a nodular cell proliferation, could be determined histopathologically even in the highest dosage group. The authors assume that the increased DNA-synthesis and the increased rate of division of the liver cells contribute to the tumorigenic effect of TCA (Dees and Travis, 1994).

The initiating and promoting properties of TCA were studied in a short-term carcinogenicity test on the male Sprague-Dawley rat. This test is based on the detection of preneoplastic lesions in form of enzyme-altered foci in the rat liver. 24 hours after a 2/3 hepatectomy, the rats obtained either a single oral application of 1500 mg/kg bw or 5000 ppm TCA in drinking water (ca. 350 mg/kg bw) for 10, 20 or 30 days. Two weeks after ending the treatment, the animals were treated for 3 or 6 month with 500 ppm phenobarbital in drinking water as a promoter. TCA had no effects as an initiator, because it did not induce any gamma-glutamyl-transpeptidase (GGT)-positive foci in the rat liver (Parnell et al., 1986).

Furthermore, male Sprague-Dawley rats (6 animals per group) were orally treated after a 2/3 hepatectomy with a single dose of 10 mg/kg bw of the initiator diethylnitrosamine. They then received 50, 500 or 5000 ppm TCA in drinking water (ca. 4, 40 or 350 mg/kg bw) for 3 or 6 month. In all dosage groups, there was a significant increase in GGT-positive foci in the rat liver after 6 month. A slight stimulation (10-20 % above the control) of cyanide-insensitive palmitoyl-CoA-oxidation could be observed simultaneously only in the highest dosage group. Hepatomegaly was not observed. According to the authors, TCA seems to have weakly promoting properties and thus seems to possibly be a weak epigenetically effective carcinogen (Parnell et al., 1986).

#### Human experience:

The herbicide sodium trichloroacetate increases chromosome aberration frequency in the culture of human peripheral lymphocytes.

#### Conclusion:

Overall, on balance, the available evidence does not suggest that TCA is a mutagen. This conclusion is consistent with the view that the liver tumours observed in mice treated with TCA (see carcinogenicity comments) arose by a non-genotoxic mechanism (peroxisome proliferation).

Recommendation: In view of these considerations further testing is not necessary.

Priority setting: NTP has not conducted genetic toxicology

### 3.2 Ecotoxicity

#### 3.2.1 Aquatic organisms

Many test results with aquatic organisms are available. Not all of the results related in the SIDS could be checked on validity due to lack of data in the respective publications. Only the test results considered to be valid are described here. Unless otherwise stated, the tests were performed with the neutralized acid or with the sodium salt (the mass conversion from the TCA-Na-salt to the free acid was not performed; the concentrations marked "\*" are related to the TCA-Na-salt).

##### a) toxicity to fish

Leuciscus idus	48h-LC50	>10000 mg/l
Poecilia reticulata	48h-LC50	9160 mg/l
Oryzias latipes	48h-LC50	277 mg/l
(the test medium was not neutralized)		
Alburnus alburnus	96h-LC50	9300 mg/l
Pimephales promelas	96h-LC50	2000 mg/l

The highest toxicity to fish was recorded with *Oncorhynchus tshawytscha* (48h-LC50 = 10 mg/l) but no information is available on the test conditions, especially if the test medium was neutralized or not. The LC50-values for neutralized solutions are consistently high. The lowest acute toxicity in a valid test was recorded at 2000 mg/l. Furthermore the results from a long-term test are available:

Cyprinus carpio	63d-LOEC	7 mg/l *
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The weight loss was ca. 10% compared to the controls. No other behavioural or clinical changes were observed. Histopathological changes were muscular atrophy and hyaline degeneration as well as cell necrosis in the gills.

b) toxicity to invertebrates

Daphnia magna	24h-EC50	8370 mg/l
	24h-EC50	>10000 mg/l*
	48h-EC50	2000 mg/l
Daphnia magna (the test medium was not neutralized)	24h-EC50	110 mg/l
Nitocra spinipes	48h-EC50	4800 mg/l
Thamnocephalus platyurus	24h-EC50	16.9 mg/l
Streptocephalus proboscideus (no information about neutralization; Centeno et al. 1995)	24h-EC50	1.5 mg/l

The EC50-values for neutralized solutions are consistently high. The lowest acute toxicity in a valid test was recorded at 2000 mg/l. No long-term test results are available. Sublethal effects were determined with Dragonfly nymphs (several different species: *Somatochlora cingulata*, *Aeschna umbrosa* and species of genera *Aeschna* and *Basiaeschna*); significant effects were recorded on the oxygen consumption rate at 0.01 mg/l and 0.1 mg/l and the ammonia excretion rate at 0.01 mg/l. This is the lowest recorded effect concentration with TCA.

c) toxicity to algae

Scenedesmus quadricauda	7d-TT	200 mg/l*
Effect: growth inhibition (biomass); TT = toxicity threshold		
Ankistrodesmus minutissimus	14d-EC50	98 mg/l*
Chlorella pyrenoidosa	14d-EC50	0.3 mg/l*
	14d-NOEC	0.01 mg/l*
Chlorella mucosa	14d-EC50	0.46 mg/l*
	14d-NOEC	0.01 mg/l*
Chlorococcum sp.	14d-EC50	1.2 mg/l*
Dictosphaerium pulchellum	14d-EC50	7 mg/l*
Scenedesmus acutus	14d-EC50	8.8 mg/l*
Effect: growth inhibition (biomass)		

mixed culture of Chlorococcales	24h-EC10	> 1000 mg/l*
Effect: inhibition of oxygen production		

As expected (cf. SIDS for Monochloroacetic acid CAS Nr. 79-11-8), algae are very sensitive to TCA. Only results from non standardized tests are available. The strongest effects were recorded in a 14-day test with the species *Chlorella pyrenoidosa* and *Chlorella mucosa* (9). Although the recorded effects are based on biomass after the exponential growth phase, an analysis of the growth curves reported in the publication shows that the effect concentrations derived from biomass during the exponential growth phase are of the same order as those reported after 14 days.

#### d) Toxicity to microorganisms

##### Protozoae:

Entosiphon sulcatum	16h-TT	800 mg/l*
Effect: growth inhibition (biomass); TT = toxicity threshold		

##### Cyanobacteria:

Microcystis aeruginosa	8d-TT	250 mg/l*
Anabaena variabilis	14d-EC50	8 mg/l*
Spirulina platensis	14d-EC50	5 mg/l*
Effect: growth inhibition (biomass); TT = toxicity threshold		

##### Bacteria:

Photobacterium phosphoreum	15min-EC50	35 mg/l
Effect: inhibition of bioluminescence		
Pseudomonas putida	16h-TT	>1000 mg/l*
Effect: growth inhibition (biomass); TT = toxicity threshold		
Pseudomonas cepacia	24h-MIC	1000 mg/l*
Aeromonas hydrophilia	24h-MIC	1000 mg/l*
Bacillus Subtilis	24h-MIC	1000 mg/l*
Effect: growth inhibition (cell count): MIC = Minimum Inhibition concentration		
Mixed culture of heterotrophic bacteria	24h-EC10	> 1000 mg/l*
Effect: inhibition of oxygen consumption		
Activated sludge, industrial	24h-EC20	> 750 mg/l
Effect: inhibition of oxygen consumption		

#### **Determination of PNEC<sub>aqua</sub>**

The lowest aquatic effect concentrations were determined with algae (14d-NOEC = 0.01 mg NaTCA/l = 0.0086 mg TCA/l) and with invertebrates (significant effects at 0.01 mg/l). The acute sublethal effects recorded with the dragonfly nymphs underline the high sensibility of certain

organisms with TCA. On the other hand, as this type of recorded effects do not fit the usual assessment schemes, the effect data from algae are used for the assessment here.

According to the EU-Technical Guidance Documents (2), a safety factor of  $F = 50$  applied to the lowest NOEC would be appropriate, as long-term tests are available only for species from two trophic levels. It could be argued that a NOEC is available for the most sensitive species, and that a safety factor of 10 is sufficient. On the other hand, the very high acute to long-term toxicity ratio found in fish (nearly 300) lessens this probability.

$$PNEC_{\text{aqua}} = 8.6 \mu\text{g/l} / 50 = 0.17 \mu\text{g/l}$$

### 3.2.2 Terrestrial organisms

#### a) toxicity to plants

Many results on acute toxicity to plants are available:

Avena sativa (2 test results)	14d-EC50	< 1 - 23 mg/kg dw
Brassica rapa	14d-EC50	308 mg/kg dw
Triticum aestivum (17)	sandy loam 15d-EC50 (Ø of 10 test results)	1.46 mg/kg dw
organic soil	15d-EC50 (Ø of 3 test results)	2.79 mg/kg dw

(effect: growth only, effect on germination not determined)

The following test results reflect effects on germination as well as growth:

Avena sativa	humic sand	14d-EC50	8.1 mg/kg dw
	loam	14d-EC50	4.6 mg/kg dw
	aqueous solution	14d-EC50	19 mg/l
Lactuca sativa	humic sand	14d-EC50	560 mg/kg dw
	loam	14d-EC50	37 mg/kg dw
	aqueous solution	14d-EC50	48 mg/l
Lycopersicum	humic sand	14d-EC50	124 mg/kg dw
	esculentum loam	14d-EC50	154 mg/kg dw
	aqueous solution	14d-EC50	59 mg/l

Long-term tests with NaTCA were performed with pine and spruce seeds (species not indicated). At 4 mg/kg dw, the germination was inhibited in 2 - 15 % of the shoots and 27 - 61 % at 64 mg/kg dw. The most sensitive parameter was the weight of the roots: an inhibition of 34% for pine and 60% for spruce, compared to the controls was observed at 1 mg/kg dw after 60 days. No NOECs were determined by the author. For pine, a dose-response curve for weight reduction in the roots can be established by probit analysis, and a 60d-EC10 of 0.14 mg/kg dw can be derived. For spruce, which was even more sensitive to TCA than pine, no dose-response curve can be established. The 60d-EC10 of 0.14 mg/kg corresponds to an EC10 of 0.12 mg/kg related to TCA.

#### b) toxicity to invertebrates



$$PNEC_{\text{soil}} = 1.46 \text{ mg/kg dw} / 1000 = 1.46 \text{ } \mu\text{g/kg dw}$$

Both values are in the same order of magnitude. For the further assessment the  $PNEC_{\text{soil}}$  of  $2.4 \text{ } \mu\text{g/kg dw}$  is used, as long-term tests have a higher priority for the risk assessment of existing chemicals.

## **4. Initial Assessment**

### **4.1 Human toxicity**

TCA is a relatively strong organic acid. Acute exposure shows mild to moderate skin and eye burns, but it is not readily absorbed through the skin.

In high concentrations TCA causes peroxisome induction in mice. For many such substances it is known that they typically lead to liver tumours in male B6C3F1 mice (but not in rats) and with a lower incidence in female mice. Through a regenerative process at hepatotoxic doses TCA clearly stimulates the mitosis rate and thus DNA synthesis in the liver.

Point-mutation tests were predominantly negative. In-vivo tests of chromosome mutations were mostly positive, but effects only appeared after high loading of the animals. The SCE test in mice was negative. The results of a micronucleus test in mice are apparently not reproducible. The end point of the sperm anomaly test is not necessarily due to genetic damage.

The validity of the positive test results described for a clastogenic effect in mice suffers from the partly insufficient experimental procedure.

As an overall conclusion the available data do not suggest any genotoxic mechanism. This is supported by carcinogenicity studies in which no indications of a carcinogenic effect were observed aside from an increased incidence of hepatic tumours in the male mouse. The tumorigenic action in male mouse liver corresponds to the type which leads to liver tumours preferentially in male mice via peroxisome proliferation, hepatotoxicity and liver-cell proliferation.

The corrosive property of TCA prompts workers to dramatically limit the potential exposure to this chemical. An OSHA PEL of  $7 \text{ mg/m}^3$  has been established for TCA. The extent to which TCA is used in consumer products is unknown. The potential for consumer exposure to TCA has not been estimated.

### **4.2 Assessment of environmental hazards**

#### **a. aquatic compartment**

In the following table, the PEC/PNEC ratios for the different exposure scenarios are presented:

<b>Scenario</b>	<b>PEC<sub>local</sub> + PEC<sub>regional</sub> [<math>\mu\text{g/l}</math>]</b>	<b>PEC/PNEC</b>
formulation of textile dyes (generic)	0.37 + 0.12	2.88
formulation of textile dyes (site)	0.04 + 0.12	0.94

use as auxiliary in textile dyes	7 + 0.12 resp. 27 + 0.12	41 resp. 159
waste water from electroplating facilities	1 + 0.12	6.6
textile washing	3.3 + 0.12	20
pulp mills	56 - 370	329 - 2176

For most configurations, the ratio PEC/PNEC is greater 1. A risk for the aquatic compartment has to be expected.

#### b. terrestrial compartment

As the PEC<sub>soil</sub> can not be modelled satisfactorily, the risk assessment is performed with the monitoring data. The PEC<sub>soil</sub> was estimated to 8 - 150 µg/kg dw (cf. 2.2.3.3). With a PNEC<sub>soil</sub> of 2.4 µg/kg dw, the following ratios are calculated:

	PEC [µg/kg dw]	PEC/PNEC
estimated concentration (min)	8	3.3
estimated concentration (max)	150	63

With the measured concentrations in soil, a risk to the terrestrial ecosystem can clearly be deduced.

In addition it has to be kept in mind that plants are not only exposed to TCA via the soil (uptake by the roots) but also via the atmosphere (uptake via the needle or leaf surface). Therefore it means to underestimate the risk for plants if only the uptake of TCA via the soil is considered. However, with the available data it is not possible to quantify the amount of TCA taken up by plants via the needles or leaves from the atmosphere and to compare it with the uptake by the roots.

Mono- and dichloroacetic acid were found as metabolites of TCA in soil. These compounds are more toxic than TCA. In addition, all three compounds have a similar mode of action and therefore additive effects can be assumed. This means that the real risk seems to be underestimated.

## **5. Conclusions and Recommendations**

### **Conclusion**

The environmental hazard assessment showed that TCA represents a risk both to the hydrosphere as well as the soil compartment.

OSHA established a PEL-TWA of 1 ppm for trichloroacetic acid. OSHA concluded that this limit would protect workers from the significant risk of skin and eye irritation associated with exposure at levels above the PEL. The OSHA PEL is consistent with the recommended ACGIH TLV.

### **Recommendations**

Before recommending emission reductions, it should be checked whether the database can be improved. For the aquatic compartment, the following endpoints might be revised:

- toxicity towards algae: the highest effects were recorded in a non standard test. In a thorough evaluation of the publication, the validity of the results could be ascertained though. A result from a standard test with *Chlorella pyrenoidosa* would probably not improve the assessment.
- long-term toxicity towards daphnids: a reproduction tests with daphnids could lower the safety factor. A lower NOEC than for the algae is not expected though. However, the use of a safety factor of 10 would not change the overall result of the assessment.
- use as auxiliary in textile dyes: the PEC from this use is estimated. Can the releases be analytically monitored ?

For the terrestrial compartment, the risk assessment results that there is a risk for the terrestrial ecosystem. This risk is caused by TCA which is formed in the atmosphere mainly from tetrachloroethylene. As the PER emissions have decreased in the last years, measurements could be performed to actualize the soil pollution. The substance should be preferredly measured in forest soils, as it is known that these soils are highly polluted.

A revision of the  $PNEC_{soil}$  by the conduction of further biotests is not possible with justifiable expense. No guideline exists for the conduction of a real chronic study with conifers that considers both the long lifetime of a tree and the high bioaccumulation potential of TCA in plants.

**References: (not presented in the SIDS)**

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## 1. Appendix

### Sources of TCA in the atmosphere

There are no direct emissions of TCA in the atmosphere during its production or use. However, the substance is formed due to the photooxidation of tetrachloroethylene and 1,1,1-trichloroethane. In the following, an attempt is made to estimate the relative importance of both TCA-precursors.

#### Formation of TCA from 1,1,1-trichloroethane (111-T)

1,1,1-trichloroethane is mainly used as a solvent for industrial cleaning techniques and degreasing of metallic surfaces. The global production is reported to 678,000 and 726,000 t/a for 1988 and 1990, respectively (12). As the substance causes the depletion of the stratospheric ozone, the substance was subjected to regulation by the Montreal Protocol which aims to stop its production until 2005.

Because of its long lifetime, 1,1,1-trichloroethane is ubiquitous in the atmosphere. In (8), many monitoring values are cited, generally they are in the range of 0.5 to 2  $\mu\text{g}/\text{m}^3$ . In appendix III, monitoring values from 3 different locations in Germany are cited.

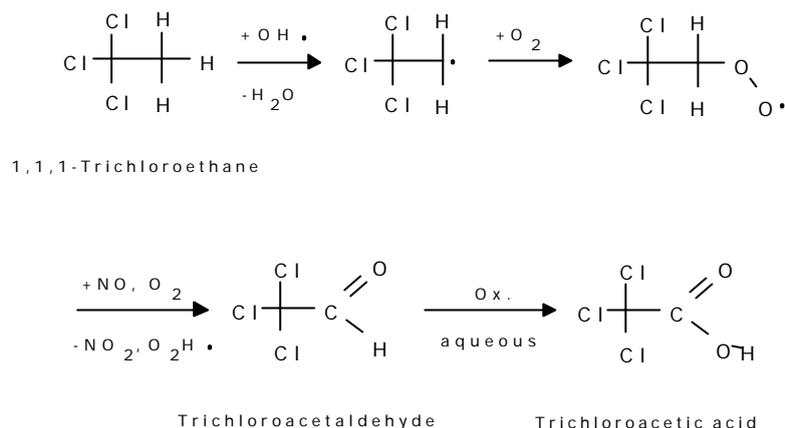
In the troposphere, 1,1,1-trichloroethane is degraded by reaction with photochemically formed hydroxyl radicals. Reactions in the stratosphere (where the substance causes ozone depletion) are not considered here. In laboratory tests, predominantly smog chamber experiments, chlorine radicals, chlorides, phosgene, HCl, TCA, trichloroacetyl chloride, acetyl chloride, acetic acid, formaldehyde, CO and CO<sub>2</sub> were determined as the reaction products (8).

There are several investigations on the rate constant for the reaction of 1,1,1-trichloroethane with hydroxyl radicals. In reference 8, values for the second order rate in the range of  $0.0080 \cdot 10^{-12}$  to  $0.0280 \cdot 10^{-12} \text{ cm}^3 \cdot \text{molecule}^{-1} \cdot \text{s}^{-1}$  are reviewed. With an OH-radical concentration of  $5 \cdot 10^5 \text{ molecules} \cdot \text{cm}^{-3}$ , half-lives between 1.6 and 5.5 years are calculated. In this assessment, the calculations are performed with a second order reaction rate of  $0.01 \cdot 10^{-12} \text{ cm}^3 \cdot \text{molecule}^{-1} \cdot \text{s}^{-1}$  (first order rate  $k = 5 \cdot 10^{-9} \text{ s}^{-1}$ ;  $t_{1/2} = 4.4 \text{ a}$ ), as in recent investigations values around the lower limit of the range are preferred.

The pathway of 1,1,1-trichloroethane degradation is branched off leading to different end products. For this assessment, the overall yield of the TCA formation is an important parameter. EAWAG reports 10-50% for the TCA yield (11).

Recent investigations show that the atmospheric breakdown leads essentially to C<sub>1</sub> products, and based on model calculations a TCA yield of only 0.06% was estimated (10). This value may be underestimate the yield under natural conditions as only gas phase reactions are considered. Especially the last reaction step should happen in the aerosol.

In the following figure, only the degradation pathway which leads to TCA is presented (11). The reactions leading to other products are omitted:



### Formation of TCA from tetrachloroethene (PER)

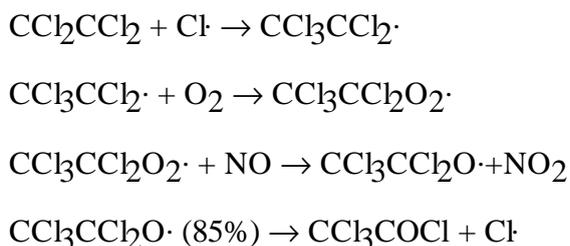
At present, a comprehensive risk assessment report on tetrachloroethylene is prepared by UK in the frame of the European Regulation on Existing Substances (13). In this paper, the formation of TCA from tetrachloroethylene is elaborated in detail, thus only the most important results are cited here:

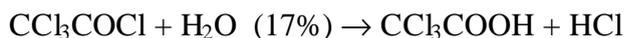
Tetrachloroethylene is mainly used as a solvent in dry cleaning, metal degreasing and extraction processes. The European production volume of tetrachloroethylene is 164,000 t/a in 1994 (13). According to (4), the world production was 553,000 t/a in 1984.

Tetrachloroethylene is ubiquitous in the atmosphere, however with a strong concentration gradient from the emission areas to the global background pollution. Typical concentrations for rural areas are 0.1 to 0.5  $\mu\text{g}/\text{m}^3$ , and for cities 0.5 to 15  $\mu\text{g}/\text{m}^3$  (4). In appendix III, monitoring data from 3 different locations in Germany are cited.

In the troposphere, tetrachloroethylene reacts both with OH-radicals and with Cl-radicals. Only the last reaction leads to the formation of TCA. The best available values for the second order reaction rates are  $k_{\text{OH}} = 1.23 \cdot 10^{-13} \text{ cm}^3 \cdot \text{molecule}^{-1} \cdot \text{s}^{-1}$  and  $k_{\text{Cl}} = 4 \cdot 10^{-11} \text{ cm}^3 \cdot \text{molecule}^{-1} \cdot \text{s}^{-1}$  corresponding first order rate constants of  $k_{\text{OH}} = 6.15 \cdot 10^{-8} \cdot \text{s}^{-1}$  and  $k_{\text{Cl}} = 2.0 \cdot 10^{-8} \cdot \text{s}^{-1}$ , respectively, and half-lives of 4.3 months ( $C_{\text{OH}} = 5 \cdot 10^5 \text{ molecules} \cdot \text{cm}^{-3}$ ) and 13 months ( $C_{\text{Cl}} = 500 \text{ molecules} \cdot \text{cm}^{-3}$ ). With these figures, 25% of the tetrachloroethylene are degraded by the pathway starting with chlorine atom addition, which leads with 15% yield to TCA (13).

In the following, only the degradation pathway which leads to TCA is presented (13). The reactions leading to other products are omitted:





### Determination of the major source of atmospheric TCA

During the production or use of TCA, no emissions into the atmosphere occur. The only sources of the atmospheric TCA are 1,1,1-trichloroethane and tetrachloroethylene from which it is formed by photolytically induced reactions as described above. The substance was analytically detected in the rain water. With its low Henry's law constant, the substance is transferred from the gas phase into the rain water and reaches the terrestrial compartment where it was measured in high concentrations (cf. 2.2.2). The relative contribution of both sources has to be analysed.

In the common risk assessment documents no guidance for such an analysis is described. Thus, we propose the following model:

The amount of a product formed from a precursor is proportional to both the concentration of the precursor, the (pseudo-first order) rate constant of the reaction leading to the product, and the yield of this reaction. These assumptions are evident, and no further justification is needed. Thus, the following equation can be laid down:

$$f_{111-T} = K \cdot C_{111-T} \cdot k_{111-T} \cdot Y_{111-T} \quad (1)$$

With:

- $f_{111-T}$ : fraction of the total environmental TCA which is formed from 1,1,1-trichloroethane
- $C_{111-T}$ : concentration of 1,1,1-trichloroethane in the atmosphere
- $k_{111-T}$ : pseudo-first order reaction constant of the degradation of 1,1,1-trichloroethane
- $Y_{111-T}$ : yield of TCA formation by this reaction
- $K$ : proportionality constant

The proportionality constant  $K$  includes many physical and physico-chemical parameters as properties of atmosphere and soil, the fate of TCA in the atmosphere (e.g. precipitation) and in soil (e.g. "dilution", mobility, degradation), etc.. If all these parameters were known precisely, the TCA concentrations in rain water and in soil could be modelled giving reliable results. For our purpose it is sufficient that  $K$  does not include any characteristics of the precursor 1,1,1-trichloroethane.

Analogously, the equation for the formation of TCA from tetrachloroethylene (PER) is

$$f_{PER} = K \cdot C_{PER} \cdot k_{PER} \cdot Y_{PER} \quad (2)$$

As the proportionality constant  $K$  includes only parameters describing the environment and the environmental fate of TCA, and no properties of 1,1,1-trichloroethane and tetrachloroethylene, in both equations  $K$  must be equal. It can be eliminated by division of equations 1 and 2:

$$\frac{f_{111-T}}{f_{PER}} = \frac{C_{111-T} \cdot k_{111-T} \cdot Y_{111-T}}{C_{PER} \cdot k_{PER} \cdot Y_{PER}} \quad (3)$$

With the elimination of  $K$ , most of the uncertainties of the analysis are eliminated.

As 1,1,1-trichloroethane and tetrachloroethylene are the only sources of TCA in the atmosphere and rural soils, the sum of  $f_{1,1-T}$  and  $f_{PER}$  is equal to the environmental TCA concentrations, which are known from the measurements (cf. 2.2.2).

An apparent contradiction is that the model describes the formation of TCA as an end product of a reaction chain, while reaction constants only for the primary transformation reactions of the precursors are used. In the paragraphs above the reaction pathways and the intermediates are given. The reaction pathways were investigated in smog chamber experiments, and 1,1,1-trichloroethane, tetrachloroethylene as well as TCA were detected, but not the intermediates. It can be concluded that the intermediates have a very short lifetime, so the TCA formation rate is nearly equal with the rates for the primary degradation steps. The only intermediate found is trichloroacetylchloride (TCAC), which occurs because dry air was used in the laboratory experiments. Under natural conditions, the aerosol is a sink of TCAC where it hydrolyses to TCA.

A further problem is that the atmospheric concentrations especially of tetrachloroethylene are fluctuating dependent on the sampling site and time. These fluctuations become less important when values from sites are used at which both precursors were measured simultaneously. In Appendix III, the results of three sampling sites in Germany are given. The quotients of the yearly averaged values are calculated and the tendency during the last 10 years is shown. Instead of accidental measured values, the quotients  $C_{111-T} / C_{PER}$  are used in the calculations to get more reliable results.

The sampling site Offenbach is representative for an urban air pollution. In the last years, the  $C_{111-T} / C_{PER}$  ratio is about 2.5 (cf. Appendix III).

Deuselbach is located near a densely populated region. The ratio is about 1, except the last year. We assume a fault in the PER value, therefore this value is ignored.

Schauinsland is a summit in the Black Forest, far away from industrialized regions. The ratio is decreasing slightly in the last years indicating that the emissions of 1,1,1-trichloroethane are nearly stopped, while the PER pollution was constant in the last 4 years. The ratio  $C_{111-T} / C_{PER}$  amounts about 4.5.

The parameters for the solution of equation 3 are summarized:

$C_{111-T} / C_{PER}$ :	4.5 [-]
$k_{111-T}$ :	$5 \cdot 10^{-9} \text{ s}^{-1}$
$Y_{111-T}$ :	0.06% / 10% / 50%
$k_{PER}$ :	$2.0 \cdot 10^{-8} \cdot \text{s}^{-1}$
$Y_{PER}$ :	15%

As large differences for the formation yield of TCA from 1,1,1-trichloroethane are found in the literature, the calculation is done with all values. The calculation of the relative contribution of both sources is done with a concentration ratio  $C_{111-T} / C_{PER}$  of 4.5 which is characteristic for rural regions. The results are:

$Y_{111-T}$	$f_{111-T} / f_{PER}$	$f_{111-T} [\%]$	$f_{PER} [\%]$
0.06%	$4.5 \cdot 10^{-3}$	0.45%	99.55%
10%	0.75	43%	57%
50%	3.8	79%	21%

The result of the analysis above reveals that in most cases tetrachloroethylene is the major source of TCA formed in the atmosphere. In rural regions, where the PER concentration is relatively low, 1,1,1-trichloroethane could be the main source if it is transformed to TCA with a high yield. In urban areas PER is the main source of the TCA even when a high transformation yield for 1,1,1-trichloroethane is assumed.

**EXTRACT FROM IRPTC LEGAL FILES**

File: 17.01 LEGAL rn : 20687

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TRICHLOROACETIC ACID  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : CHE type : REG

```
-----
|subject|specification|descriptor|
|-----+-----+-----|
| AIR   |   OCC   |   MAK   |
|-----+-----+-----|
-----
```

TWA: 7MG/M3 (1PPM)  
 entry date: DEC 1987

original : ILO , , , , ,  
 amendment: ZWACH\*, ZULAESSIGE WERTE AM ARBEITSPLATZ(PERMITTED VALUES IN  
 THE WORKPLACE), , , , 1987

\*\*\*\*\*

File: 17.01 LEGAL rn : 21563

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TRICHLOROACETIC ACID  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : NLD type : REC

```
-----
|subject|specification|descriptor|
|-----+-----+-----|
| AIR   |   OCC   |   MAK   |
|-----+-----+-----|
-----
```

TWA: 1MG/M3.  
 entry date: JUN 1987

original : ILO , , , , ,  
 amendment: NMACN\*, NATIONALE MAC-LIST(NATIONAL MAC-LIST), , , , 1986

\*\*\*\*\*

File: 17.01 LEGAL rn : 26238

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TRICHLOROACETIC ACID  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : EEC type : REG

```
-----
|subject|specification|descriptor|
|-----+-----+-----|
| GOODS |           |   PRO   |
|-----+-----+-----|
-----
```

SUBSTANCE WHICH MUST NOT FORM PART OF THE COMPOSITION OF COSMETIC  
 PRODUCTS. THE MARKETING OF COSMETIC PRODUCTS CONTAINING THE SUBSTANCE IS  
 PROHIBITED. EEC DIRECTIVE 76/768/EEC - OJEC L262,169,1976- AS LAST  
 AMENDED BY THE REFERENCE GIVEN.

entry date: SEP 1987 effective date: 1JAN1988

amendment: OJEC\*\*, Official Journal of the European (Communities)/Union,  
 L56 , , 20 , 1987

\*\*\*\*\*

File: 17.01 LEGAL rn : 40958

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TRICHLOROACETIC ACID  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : AUS type : REC

subject	specification	descriptor
AIR	OCC	TLV

TWA: 5MG/M3 (1PPM)  
 entry date: MCH 1985

amendment: AOHGN\*, APPROVED OCCUPATIONAL HEALTH GUIDE THRESHOLD LIMIT  
 VALUES, , , , 1983

\*\*\*\*\*

File: 17.01 LEGAL rn : 51019

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TCA  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : GBR type : REG

subject	specification	descriptor
USE	PESTI	RSTR
USE	AGRIC	RSTR

Active ingredient of pesticide products approved for professional use  
 only.

entry date: NOV 1992 effective date: 01NOV1991

title: Pesticides 1992: Pesticides approved under the Control of  
 Pesticides Regulations 1986.

original : PACPR\*, , 500 , , , 1992

\*\*\*\*\*

File: 17.01 LEGAL rn : 100081

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TRICHLOROACETIC ACID  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : ARG type : REG

subject	specification	descriptor
AIR	OCC	MPC

8H-TWA: 7MG/M3 (1PPM)  
 entry date: OCT 1991

effective date: 29MAY1991

title: LIMIT VALUES FOR CHEMICAL SUBSTANCES IN THE WORKING

ENVIRONMENT-RESOLUTION NO. 444/1991 OF THE MINISTRY OF WORK AND SOCIAL SECURITY (AMENDING REGULATION DECREE NO. 351/1979 UNDER LAW NO. 19587/1972: HYGIENE AND SAFETY AT WORK)

original : ARGOB\*, Boletin Oficial de la Republica Argentina(Argentinian Official Bulletin), 24170 , I , 1 , 1979

amendment: ARGOB\*, Boletin Oficial de la Republica Argentina(Argentinian Official Bulletin), 27145 , I , 4 , 1991

\*\*\*\*\*

File: 17.01 LEGAL

rn : 234946

systematic name:Acetic acid, trichloro-

common name :TCA

reported name :TCA

cas no :76-03-9

rtecs no :AJ7875000

area : BRA

type : REG

subject	specification	descriptor
FOOD		AL
USE	AGRIC	PRMT
USE	CONSM	PRO
CLASS		CLASS

SPECIFIED PLANT PRODUCTS: 0.01-0.5MG/KG; SECURITY INTERVAL: 60-120 DAYS; PROVISIONAL TOXICITY CLASS: III (I = MOST TOXIC; IV = LEAST TOXIC); USE IN AGRICULTURE: AUTHORIZED; USE IN DOMESTIC SANITATION: NOT AUTHORIZED.  
entry date: AUG 1985

title: SUBSTANCIAS COM ACAA TOXICA SOBRE ANIMAIS E/OU PLANTAS  
(SUBSTANCES TOXIC TO ANIMALS AND/OR PLANTS)

original : SBTAP\*, Substancias Comacao Toxicas sobre Animais e/ou Plantas(Substances Toxic to Animals and/or Plants), , , 8 , 1984

\*\*\*\*\*

File: 17.01 LEGAL

rn : 300361

systematic name:Acetic acid, trichloro-

common name :TCA

reported name :TRICHLOROACETIC ACID

cas no :76-03-9

rtecs no :AJ7875000

area : CAN

type : REG

subject	specification	descriptor
AIR	OCC	TLV

TWA: 1 PPM, 7 MG/M3. PRESCRIBED BY THE CANADA OCCUPATIONAL SAFETY AND HEALTH REGULATIONS, UNDER THE CANADA LABOUR CODE (ADMINISTERED BY THE DEPARTMENT OF LABOUR). THE REGULATIONS STATE THAT NO EMPLOYEE SHALL BE EXPOSED TO A CONCENTRATION OF AN AIRBORNE CHEMICAL AGENT IN EXCESS OF THE VALUE FOR THAT CHEMICAL AGENT ADOPTED BY ACGIH (AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS) IN ITS PUBLICATION ENTITLED: "THRESHOLD LIMIT VALUE AND BIOLOGICAL EXPOSURE INDICES FOR 1985-86".  
entry date: MCH 1991 effective date: 13MCH1986

amendment: CAGAAK, Canada Gazette Part II, 120 , 6 , 1105 ,

\*\*\*\*\*

File: 17.01 LEGAL rn : 301515

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TRICHLOROACETIC ACID  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : CAN type : REG

subject	specification	descriptor
TRNSP		CLASS
LABEL		RQR
PACK		

SOLID. PIN (PRODUCT IDENTIFICATION NO.): UN1839. CLASS (8): CORROSIVE. SPECIAL PROVISIONS: 46. PACKING GROUP II, (I=GREAT DANGER, III=MINOR DANGER). MAXIMUM AMOUNT PER PACKAGE THAT MAY BE TRANSPORTED ON A PASSENGER AIRCRAFT OR VEHICLE: 15 KG. MAXIMUM AMOUNT PER PACKAGE THAT MAY BE TRANSPORTED ON A CARGO AIRCRAFT: 50 KG. PRESCRIBED BY THE TRANSPORTATION OF DANGEROUSGOODS REGULATIONS, UNDER THE TRANSPORTATION OF DANGEROUS GOODS ACT (ADMINISTERED BY THE DEPARTMENT OF TRANSPORT). THE ACT AND REGULATIONS ARE INTENDED TO PROMOTE SAFETY INTHE TRANSPORTATION OF DANGEROUS GOODS IN CANADA, AS WELL AS PROVIDE ONE COMPREHENSIVE SET OF RULES APPLICABLE TO ALL MODES OF TRANSPORT ACCROSS CANADA. THESE ARE BASED ONUNITED NATIONS RECOMMENDATIONS. THE ACT AND REGULATIONS SHOULD BE CONSULTED FOR DETAILS. RECORDS ARE ENTERED UNDER THE PROPER SHIPPINGNAME FOUND IN THE REGULATIONS; THIS MAY INCLUDE VERY GENERAL GROUPS OF CHEMICAL SUBSTANCES.

entry date: OCT 1991 effective date: 06DEC1990

amendment: CAGAAK, Canada Gazette Part II, 124 , 26 , 5523 ,

\*\*\*\*\*

File: 17.01 LEGAL rn : 301516

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TRICHLOROACETIC ACID  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : CAN type : REG

subject	specification	descriptor
TRNSP		CLASS
LABEL		RQR
PACK		

SOLUTION. PIN (PRODUCT IDENTIFICATION NO.): UN2564. CLASS (8): CORROSIVE. SPECIAL PROVISIONS: 110. PACKING GROUP II, (I=GREATDANGER, III=MINOR DANGER). MAXIMUM AMOUNT PER PACKAGE THAT MAY BE TRANSPORTED ON A PASSENGER AIRCRAFT OR VEHICLE: 1 L. MAXIMUMAMOUNT PER PACKAGE THAT MAY BE TRANSPORTED ONA CARGO AIRCRAFT: 30 L. PRESCRIBED BY THE TRANSPORTATION OF DANGEROUS GOODS REGULATIONS, UNDER THE TRANSPORTATION OF DANGEROUS GOODS ACT (ADMINISTERED BY THE DEPARTMENT OF TRANSPORT). THE ACT AND REGULATIONS ARE INTENDED TO PROMOTE SAFETY INTHE TRANSPORTATION OF DANGEROUS GOODS IN CANADA, AS WELL AS PROVIDE ONE COMPREHENSIVE SET OF RULES APPLICABLE TO ALL MODES OF TRANSPORT ACCROSS

CANADA. THESE ARE BASED ON UNITED NATIONS RECOMMENDATIONS. THE ACT AND REGULATIONS SHOULD BE CONSULTED FOR DETAILS. RECORDS ARE ENTERED UNDER THE PROPER SHIPPING NAME FOUND IN THE REGULATIONS; THIS MAY INCLUDE VERY GENERAL GROUPS OF CHEMICAL SUBSTANCES.

entry date: OCT 1991

effective date: 06DEC1990

amendment: CAGAAK, Canada Gazette Part II, 124 , 26 , 5523 ,

\*\*\*\*\*

File: 17.01 LEGAL

rn : 302406

systematic name: Acetic acid, trichloro-

common name : TCA

reported name : TRICHLOROACETIC ACID

cas no : 76-03-9

rtecs no : AJ7875000

area : CAN

type : REC

subject	specification	descriptor
AQ	DRINK	GL MAC

TABLE 2 - UNDER REVIEW FOR POSSIBLE ADDITION TO THE GUIDELINES. THESE LEVELS ARE SET ACCORDING TO GUIDELINES FOR CANADIAN DRINKING WATER QUALITY - 1989. THE GUIDELINES ARE PREPARED BY THE FEDERAL-PROVINCIAL SUBCOMMITTEE ON DRINKING WATER OF THE FEDERAL-PROVINCIAL ADVISORY COMMITTEE ON ENVIRONMENTAL AND OCCUPATIONAL HEALTH AND PUBLISHED BY AUTHORITY OF THE MINISTER OF NATIONAL HEALTH AND WELFARE; THEY SPECIFY RECOMMENDATIONS AND LIMITS FOR SUBSTANCES AND CONDITIONS WHICH AFFECT THE QUALITY OF DRINKING WATER.

entry date: NOV 1991

effective date: 1989

amendment: GWQUEQ, GUIDELINES FOR CANADIAN DRINKING WATER QUALITY, , , ,

\*\*\*\*\*

File: 17.01 LEGAL

rn : 302479

systematic name: Acetic acid, trichloro-

common name : TCA

reported name : TRICHLOROACETIC ACID

cas no : 76-03-9

rtecs no : AJ7875000

area : CAN

type : REG

subject	specification	descriptor
SALE IMPRT GOODS LABEL	CONSM	PRO RQR

IT IS PROHIBITED TO SELL, ADVERTISE OR IMPORT INTO CANADA A PRODUCT PACKAGED AS A CONSUMER PRODUCT THAT CONTAINS A CORROSIVE CHEMICAL, INCLUDING TRICHLOROACETIC ACID AND HAS A PH OF 2.5 OR LESS OR 11.5 OR MORE i) BEFORE IT IS PREPARED FOR USE, OR ii) WHEN IT IS PREPARED FOR USE ACCORDING TO THE DIRECTIONS OR IN THE MANNER THAT IS CUSTOMARY OR USUAL, UNLESS DETAILED LABELLING REQUIREMENTS ARE MET. THIS PROHIBITION IS PRESCRIBED BY SCHEDULE I OF THE HAZARDOUS PRODUCTS ACT (HPA), ADMINISTERED BY THE DEPARTMENT OF CONSUMER AND CORPORATE AFFAIRS. IT

AUTHORIZES THE PROHIBITION OF PRODUCTS THAT ARE LIKELY TO BE OF DANGER TO THE HEALTH AND SAFETY OF THE PUBLIC.

entry date: MCH 1991

effective date: 01NOV1988

amendment: CAGAAK, Canada Gazette Part II, 122 , 24 , 4625 ,

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File: 17.01 LEGAL

rn : 304074

systematic name:Acetic acid, trichloro-

common name :TCA

reported name :TRICHLOROACETIC ACID

cas no :76-03-9

rtecs no :AJ7875000

area : CAN

type : REG

subject	specification	descriptor
USE	OCC	RQR
STORE		
LABEL		

INGREDIENT DISCLOSURE LIST CONCENTRATION 1% WEIGHT/WEIGHT. THE WORKPLACE HAZARDOUS MATERIALS INFORMATION SYSTEM (WHMIS) IS A NATIONAL SYSTEM TO PROVIDE INFORMATION ON HAZARDOUS MATERIALS USED IN THE WORKPLACE. WHMIS IS IMPLEMENTED BY THE HAZARDOUS PRODUCTS ACT AND THE CONTROLLED PRODUCTS REGULATIONS (ADMINISTERED BY THE DEPARTMENT OF CONSUMER AND CORPORATE AFFAIRS). THE REGULATIONS IMPOSE STANDARDS ON EMPLOYERS FOR THE USE, STORAGE AND HANDLING OF CONTROLLED PRODUCTS AND ADDRESS LABELLING AND IDENTIFICATION, EMPLOYEE INSTRUCTION AND TRAINING, AS WELL AS THE UPKEEP OF A MATERIALS SAFETY DATA SHEET (MSDS). THE PRESENCE IN A CONTROLLED PRODUCT OF AN INGREDIENT IN A CONCENTRATION EQUAL TO OR GREATER THAN SPECIFIED IN THE INGREDIENT DISCLOSURE LIST MUST BE DISCLOSED IN THE SAFETY DATA SHEET.

entry date: APR 1991

effective date: 31DEC1987

amendment: CAGAAK, Canada Gazette Part II, 122 , 2 , 551 ,

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File: 17.01 LEGAL

rn : 500518

systematic name:Acetic acid, trichloro-

common name :TCA

reported name :TRICHLOROACETIC ACID

cas no :76-03-9

rtecs no :AJ7875000

area : DEU

type : REC

subject	specification	descriptor
AQ		CLASS
USE	INDST	RQR

THIS SUBSTANCE IS CLASSIFIED AS SLIGHTLY HAZARDOUS TO WATER (WATER-HAZARD CLASS: WGK 1). (THE DIFFERENT CLASSES ARE: WGK 3 = VERY HAZARDOUS; WGK 2 = HAZARDOUS; WGK 1 = SLIGHTLY HAZARDOUS; WGK 0 = IN GENERAL NOT HAZARDOUS.) THE CLASSIFICATION FORMS THE BASIS FOR WATER-PROTECTION REQUIREMENTS FOR INDUSTRIAL PLANTS IN WHICH WATER-HAZARDOUS SUBSTANCES ARE HANDLED.

entry date: DEC 1991

title: ADMINISTRATIVE RULES CONCERNING WATER-HAZARDOUS SUBSTANCES

(VERWALTUNGSVORSCHRIFT WASSERGEFAEHRDENDE STOFFE)

original : GMSMA6, Gemeinsames Ministerialblatt. Joint Ministerial  
Papers, , 8 , 114 , 1990

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File: 17.01 LEGAL rn : 501451

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TCA  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : DEU type : REG

subject	specification	descriptor
USE	PESTI	RSTR

PLANT PROTECTANTS CONSISTING OF OR CONTAINING THE SUBSTANCE MAY NOT BE USED IN PROTECTED WATER AREAS AND MINERAL SPRING AREAS UNLESS THEY ARE MARKETED IN WEED-STICKS, IN SPRAYERS READY FOR USE, OR AS STICKS OR PILLS FOR USE IN POTTED PLANTS. THE SUBSTANCE MAY NOT BE USED IN NATURAL PRESERVES, NATIONAL PARKS, NATURAL MONUMENTS AND OTHER PROTECTED AREAS UNLESS THE USE IS EXPLICITLY PERMITTED.

entry date: DEC 1991 effective date: 29MCH1991

title: ORDINANCE ON THE USE FOR PLANT PROTECTION  
 (PFLANZENSCHUTZ-ANWENDUNGSVERORDNUNG)

original : BGZBAD, Bundesgesetzblatt (Federal Law Gazette), , I , 1196 ,  
1988amendment: BGZBAD, Bundesgesetzblatt (Federal Law Gazette), , I , 796 ,  
1991

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File: 17.01 LEGAL rn : 503145

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TRICHLOROACETIC ACID  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : DEU type : REC

subject	specification	descriptor
AIR	OCC	MAK

EXPERIENCE IN HUMANS OR IN ANIMAL EXPERIMENTATION HAS NOT PROVIDED SUFFICIENT INFORMATION FOR ESTABLISHMENT OF A MAK VALUE.

entry date: JAN 1992

title: MAXIMUM CONCENTRATIONS AT THE WORKPLACE AND BIOLOGICAL TOLERANCE VALUES FOR WORKING MATERIALS (MAXIMALE ARBEITSPLATZKONZENTRATIONEN UND BIOLOGISCHE ARBEITSTOFFTOLERANZWERTE)

original : MPGFDF, MITTEILUNG DER SENATSKOMMISSION ZUR PRUEFUNG  
GESUNDHEITSSCHAEDLICHER ARBEITSTOFFE (DEUTSCHE  
FORSCHUNGSGEMEINSCHAFT), XXVII , , 17 , 1991

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File: 17.01 LEGAL rn : 510579

systematic name:Acetic acid, trichloro-

common name :TCA  
 reported name :TRICHLOROACETIC ACID  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : DEU type : REG

subject	specification	descriptor
CLASS		CLASS
LABEL		RQR
PACK		RQR

CLASSIFICATION AND LABELLING IN GERMANY IS GENERALLY THE SAME AS FOR THE EEC (SEE OJEC\*\* L180, 1991). HOWEVER, SLIGHT MODIFICATIONS MAY BE INTRODUCED FOR SOME SUBSTANCES IN THE GERMAN LEGISLATION.

entry date: APR 1992 effective date: 15JUN1991

title: ORDINANCE ON HAZARDOUS SUBSTANCES. (GEFAHRSTOFFVERORDNUNG)  
 original : BGZBAD, Bundesgesetzblatt (Federal Law Gazette), , I , 1931 , 1991

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File: 17.01 LEGAL rn : 606167

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TCA  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : GBR type : REG

subject	specification	descriptor
USE	PESTI	PRMT

APPROVED AS A SOIL-ACTING HERBICIDE. SPECIFIC USES, LIMITATIONS AND SAFETY PRECAUTIONS ARE LISTED

entry date: 1983

title: APPROVED PRODUCTS FOR FARMERS AND GROWERS 1983  
 original : APFG\*\*, APPROVED PRODUCTS FOR FARMERS AND GROWERS, , , 89 , 1983

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File: 17.01 LEGAL rn : 1120995

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TRICHLOROACETIC ACID  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : RUS type : REG

subject	specification	descriptor
AIR	OCC	MAC CLASS

CLV: 5.0MG/M3 (VAPOUR, AEROSOL) HAZARD CLASS: III  
 entry date: MAY 1990 effective date: 01JAN1989

amendment: GOSTS\*, GOSUDARSTVENNYI STANDART SSSR(STATE STANDARD OF

USSR), 12.1.005 , , , 1988

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File: 17.01 LEGAL rn : 1123780

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TCA  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : RUS type : REG

subject	specification	descriptor
USE	AGRIC	PRMT

SUBSTANCE IS APPROVED AS HERBICIDE FOR AGRICULTURAL USE. APPLICATION DOSE, MODE AND TREATMENT FREQUENCY IS SPECIFIED.

entry date: NOV 1987 effective date: 1JAN1986

amendment: SCHSB\*, SPISOK KHIMICHESKIKH I BIOLOGICHESKIKH SREDSTV RAZRESHENNYKH DLIAPRIMENENIA V SELSKOM KHOZIAISTEVE (LIST OF CHEMICALS & BIOLOGICAL MEANS APPROVED FOR AGRICULTURAL USE), 1986-90 , , , 1987

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File: 17.01 LEGAL rn : 1123834

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TCA  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : RUS type : REG

subject	specification	descriptor
USE	AGRIC	PRMT

SUBSTANCE IS APPROVED AS PESTICIDE FOR HERBS TREATMENT. APPLICATION DOSE AND TREATMENT CONDITIONS ARE SPECIFIED.

entry date: NOV 1987 effective date: 1JAN1986

amendment: SCHSB\*, SPISOK KHIMICHESKIKH I BIOLOGICHESKIKH SREDSTV RAZRESHENNYKH DLIAPRIMENENIA V SELSKOM KHOZIAISTEVE (LIST OF CHEMICALS & BIOLOGICAL MEANS APPROVED FOR AGRICULTURAL USE), 1986-90 , , , 1987

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File: 17.01 LEGAL rn : 1315006

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TRICHLOROACETIC ACID  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : USA type : REG

subject	specification	descriptor
TRNSP		PRMT
PACK		CNTRL
LABEL		RQR

SOLID: MAY BE TRANSPORTED IN PASSENGER AIRCRAFT AND PASSENGER RAILCAR NOT TO EXCEED 25 POUNDS/PACKAGE. MAY BE TRANSPORTED IN CARGO AIRCRAFT NOT TO EXCEED 100 POUNDS/PACKAGE. MAY BE TRANSPORTED IN CARGO VESSELS ON AND BELOW DECK AND IN PASSENGER VESSELS ON DECK. ALL SHIPMENTS MUST BE LABELED CORROSIVE. SOLUTION: MAY BE TRANSPORTED IN PASSENGER AIRCRAFT AND PASSENGER RAILCAR NOT TO EXCEED 1 QUART/PACKAGE. MAY BE TRANSPORTED IN CARGO AIRCRAFT NOT TO EXCEED 1 QUART/PACKAGE. MAY BE TRANSPORTED IN CARGO AND PASSENGER VESSELS ON AND BELOW DECK. FOR VESSEL SHIPMENTS GLASS CARBOYS IN HAMPERS NOT PERMITTED UNDER DECK. ALL SHIPMENTS MUST BE LABELED CORROSIVE.; Summary - THIS REGULATION LISTS AND CLASSIFIES THOSE MATERIALS WHICH THE DEPARTMENT OF TRANSPORTATION HAS DESIGNATED AS HAZARDOUS MATERIALS FOR SHIPPING PAPERS, PACKAGE MARKING, LABELING, AND TRANSPORT VEHICLE PLACARDING APPLICABLE TO THE SHIPMENT AND TRANSPORT OF THOSE HAZARDOUS MATERIALS.

entry date: NOV 1991

effective date: OCT1991

title: HAZARDOUS MATERIALS REGULATIONS, PART 172--HAZARDOUS MATERIALS TABLES AND HAZARDOUS MATERIALS COMMUNICATIONS REGULATIONS

original : CFRUS\*, Code of Federal Regulations, 49 , 172 , 101 , 1984

amendment: CFRUS\*, Code of Federal Regulations, 49 , 172 , 101 , 1990

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File: 17.01 LEGAL

rn : 1323225

systematic name:Acetic acid, trichloro-

common name :TCA

reported name :TRICHLOROACETIC ACID

cas no :76-03-9

rtecs no :AJ7875000

area : USA

type : REG

subject	specification	descriptor
CLASS	PESTI	RQR
MANUF	PESTI	PRMT
FOOD	ADDIT	RQR

CASE NAME TCA,AND SALTS; Summary - THIS SUBSTANCE IS INCLUDED ON A LIST OF ACTIVE INGREDIENTS CONTAINED IN A PRODUCT FIRST REGISTERED BEFORE NOVEMBER 1, 1984, FOR WHICH A REGISTRATION STANDARD HAS NOT BEEN ISSUED. PUBLICATION OF THIS LIST INITIATES AN ACCELERATED REREGISTRATION AND DATA C ALL-IN FOR PRODUCTS CONTAINING THE LISTED ACTIVE INGREDIENTS.

entry date: JAN 1992

effective date: 1989

title: FEDERAL INSECTICIDE, FUNGICIDE, AND RODENTICIDE ACT PESTICIDES REQUIRED TO BE REREGISTERED; LIST D

original : FEREAC, Federal Register, 54 , 204 , 43388 , 1989

amendment: FEREAC, Federal Register, 54 , 204 , 43388 , 1989

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File: 17.01 LEGAL

rn : 1340715

systematic name:Acetic acid, trichloro-

common name :TCA

reported name :TRICHLOROACETIC ACID

cas no :76-03-9

rtecs no :AJ7875000

area : USA

type : REC

subject	specification	descriptor
AIR	OCC	TLV

Time Weighted Avg (TWA) 1 ppm, 6.7 MG/M3; Summary - THIS THRESHOLD LIMIT VALUE IS INTENDED FOR USE IN THE PRACTICE OF INDUSTRIAL HYGIENE AS A GUIDELINE OR RECOMMENDATION IN THE CONTROL OF POTENTIAL HEALTH HAZARDS.  
 entry date: DEC 1991 effective date: 1989

title: THRESHOLD LIMIT VALUES  
 original : ACGIH\*, Threshold Limit Values and Biological Exposure Indices, , , 11 , 1989  
 amendment: ACGIH\*, Threshold Limit Values and Biological Exposure Indices, , , 11 , 1991

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File: 17.01 LEGAL rn : 1421922

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TRICHLOROACETIC ACID  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : EEC type : REG

subject	specification	descriptor
CLASS		CLASS
LABEL		RQR
PACK		RQR

CLASS: C - CORROSIVE; CAUSES SEVERE BURNS (R 35). LABEL: C - CORROSIVE; CAUSES SEVERE BURNS (R 35); AVOID CONTACT WITH SKIN AND EYES (S 24/25); IN CASE OF CONTACT WITH EYES, RINSE IMMEDIATELY WITH PLENTY OF WATER AND SEEK MEDICAL ADVICE (S 26). CLASSIFICATION OF PREPARATIONS CONTAINING THE SUBSTANCE IN CONCENTRATION RANGE: ABOVE 10%: C - CORROSIVE; CAUSES SEVERE BURNS (R 35). FROM 5% TO 10%: C - CORROSIVE; CAUSES BURNS (R 34). FROM 1% TO 5%: XI - IRRITANT; IRRITATING TO EYES AND SKIN (R 36/38).  
 entry date: APR 1992 effective date: 1JUL1992

title: COUNCIL DIRECTIVE 67/548/EEC OF 27 JUNE 1967 ON THE APROXIMATION OF THE LAWS, REGULATIONS AND ADMINISTRATIVE PROVISIONS RELATING TO THE CLASSIFICATION, PACKAGING AND LABELLING OF DANGEROUS SUBSTANCES  
 original : OJEC\*\*, Official Journal of the European (Communities)/Union, 196 , , 1 , 1967  
 amendment: OJEC\*\*, Official Journal of the European (Communities)/Union, L 180 , , 79 , 1991

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File: 17.01 LEGAL rn : 1646960

systematic name:Acetic acid, trichloro-  
 common name :TCA  
 reported name :TRICHLOROACETIC ACID  
 cas no :76-03-9 rtecs no :AJ7875000  
 area : IMO type : REC

subject	specification	descriptor
TRNSP	MARIN	CLASS
LABEL		
PACK		

HAZARD CLASS: 8 = CORROSIVE. PACKING GROUP: II = MEDIUM DANGER (I=GREAT

DANGER - III=MINOR DANGER). UN NO. 1839  
entry date: JAN 1991

amendment: !IMCOC\*, International Maritime Dangerous Goods Code, , ,  
10004 , 1990

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File: 17.01 LEGAL rn : 1646961

systematic name:Acetic acid, trichloro-  
common name :TCA  
reported name :TRICHLOROACETIC ACID  
cas no :76-03-9 rtecs no :AJ7875000  
area : IMO type : REC

subject	specification	descriptor
TRNSP	MARIN	CLASS
LABEL		
PACK		

HAZARD CLASS: 8 = CORROSIVE. PACKING GROUP: II = MEDIUM DANGER (I=GREAT  
DANGER - III=MINOR DANGER). (APPLIES TO TRICHLOROACETIC ACID, SOLUTION).  
UN NO. 2564  
entry date: JAN 1991

amendment: !IMCOC\*, International Maritime Dangerous Goods Code, , ,  
10004 , 1990

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File: 17.01 LEGAL rn : 1744668

systematic name:Acetic acid, trichloro-  
common name :TCA  
reported name :TRICHLOROACETIC ACID  
cas no :76-03-9 rtecs no :AJ7875000  
area : UN type : REC

subject	specification	descriptor
TRNSP		CLASS
LABEL		
PACK		

HAZARD CLASS: 8 = CORROSIVE. PACKING GROUP: II = MEDIUM DANGER (I=GREAT  
DANGER - III=MINOR DANGER). UN NO. 1839  
entry date: AUG 1990

amendment: !UNTDG\*, UN Transport of Dangerous Goods, Recommendation  
prepared by the Committee of Experts on the Transport of  
Dangerous Goods, , , 15 , 1989

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File: 17.01 LEGAL rn : 1744964

systematic name:Acetic acid, trichloro-  
common name :TCA  
reported name :TRICHLOROACETIC ACID  
cas no :76-03-9 rtecs no :AJ7875000

area : UN type : REC

subject	specification	descriptor
TRNSP		CLASS
LABEL		
PACK		

HAZARD CLASS: 8 = CORROSIVE. PACKING GROUP: II = MEDIUM DANGER (I=GREAT DANGER - III=MINOR DANGER). (APPLIES TO TRICHLOROACETIC ACID, SOLUTION).

UN NO. 2564

entry date: AUG 1990

amendment: !UNTDG\*, UN Transport of Dangerous Goods, Recommendation prepared by the Committee of Experts on the Transport of Dangerous Goods, , , 15 , 1989

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File: 17.01 LEGAL

rn : 1860107

systematic name: Acetic acid, trichloro-

common name : TCA

reported name : TRICHLOROACETIC ACID

cas no : 76-03-9

rtecs no : AJ7875000

area : WHO

type : REC

subject	specification	descriptor
AQ	DRINK	GL

100 ug/l (provisional guideline value). The substance is a chemical of health significance in drinking water.

entry date: OCT 1992

title: WHO GUIDELINES FOR DRINKING-WATER QUALITY

original : WHODW\*, GUIDELINES FOR DRINKING WATER QUALITY, VOLUME 1 - RECOMMENDATIONS, , , , 1983

amendment: WHODW\*, GUIDELINES FOR DRINKING WATER QUALITY, VOLUME 1 - RECOMMENDATIONS, , , , 1992