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**TOXICITY REDUCTION EVALUATION
CASE SUMMARY
FOR THE CHLOR-ALKALI INDUSTRY**

**Parsons Engineering Science
R&D Technical Report P29**

Toxicity Reduction Evaluation Case Summary for the Chlor-Alkali Industry

Parsons Engineering Science

Supported by:

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**Research Contractor:
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R&D Technical Report P29

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Statement of use

This report reviews the causes of toxicity in discharges from the Chlor-Alkali Industry and the actions taken to reduce this toxicity. Case studies from the USA and Canada where toxicity controls have been in place for 10 to 15 years were utilised as sources for this review. It was intended that this information would assist both the regulator and industry in toxicity identification and reduction programmes here in the UK resulting from the current toxicity-based licencing initiative.

Research contractor

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1. SUMMARY

Information on U.S. toxicity reduction evaluation (TRE) studies is provided to assist the chlor-alkali industry in addressing new toxicity criteria for use in consenting to be considered soon by the Environment Agency. These studies identified causes of toxicity that may be common to chlor-alkali plant discharges: chlorine, total dissolved solids (TDS), and an imbalance in effluent concentrations of calcium carbonate. The toxicity of the chlorine and TDS in chlor-alkali plant effluents can be readily determined through bench-scale testing using U.S. Environmental Protection Agency (EPA) toxicity identification evaluation (TIE) procedures. Ion imbalance is a concern when effluent toxicity tests utilise crustaceans such as estuarine shrimp (e.g. *Mysidopsis bahia*), which require minimum concentrations of calcium carbonate for survival, growth and reproduction. Other potential causes of effluent toxicity are noted based on experience at similar U.S. industries.

2. INTRODUCTION

2.1 Background

The environmental regulators are developing toxicity criteria to help identify and control the potentially toxic discharges to UK controlled waters. The purpose is to address toxic effects caused by complex effluents that are not readily explained by data on chemical concentrations or by the interaction of effluent constituents in receiving waters. Direct toxicity assessment (DTA) using aquatic organisms in standardised toxicity tests will be applied to evaluate the net toxic effect of whole effluents.

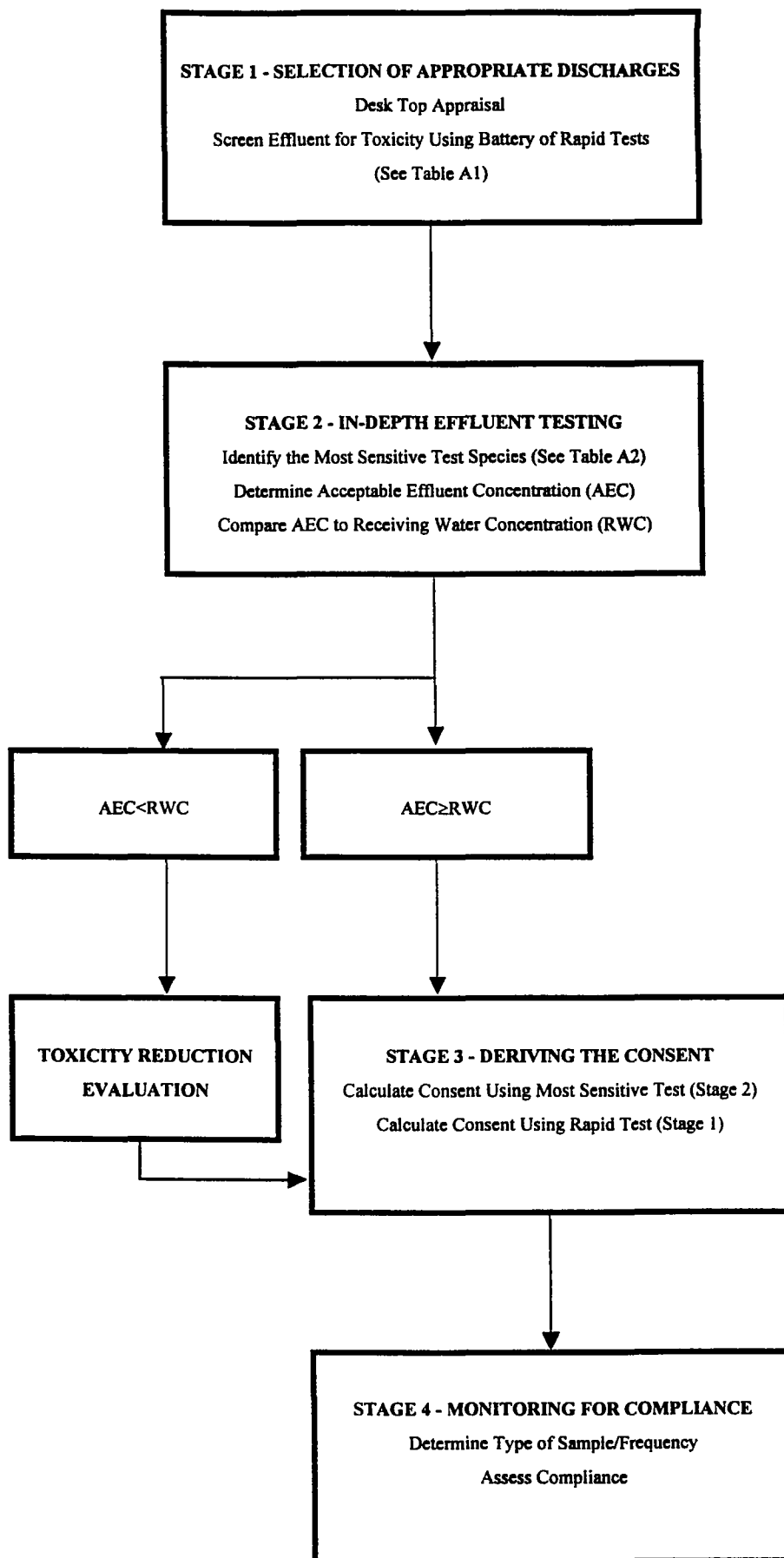
A protocol for deriving consents containing toxicity criteria is being evaluated. These studies will evaluate the available toxicity testing procedures (including low cost, rapid tests) and approaches for deriving TBCs that protect water quality. Comparisons of the test procedures will be made to determine the most appropriate tests to apply to particular types of effluents (e.g. metal fabricating, pulp and paper, and municipal) and receiving waters (freshwater versus marine). The magnitude and frequency of effluent toxicity and the available dilution and mixing conditions, will be used to determine consents containing toxicity criteria.

The regulators draft protocol for TBC derivation is illustrated in Figure 1. Toxicity tests currently proposed for effluent monitoring by the regulators are shown in Appendix A.

2.2 Case Summary Information

Preliminary results of the studies indicate a potential for toxicity in effluents from chlor-alkali facilities. This report provides information that can be used by chlor-alkali facilities to identify and control the discharge of toxicity. Information on toxicity reduction studies performed in the U.S. over the last five years has been summarised with emphasis on the methods used, types of toxicants identified, and control measures successfully implemented. This report is intended to be a general guide on TRE approaches that may

Figure 1. The Environmental Regulators' Protocol For Deriving And Monitoring Compliance With Toxicity-Based Consents



apply to chlor-alkali plants. Each facility has unique manufacturing processes, effluent constituents, and receiving water conditions, which will influence the selection and use of TRE methods. Therefore, facility managers should develop a facility-specific plan, preferably in consultation with an experienced team of toxicologists, engineers and chemists.

2.3 **TRE Process**

Since the early 1980s, toxicity monitoring has been applied to effluents of nearly all types of industries in the U.S, including the chlor-alkali industry. Dischargers with permits issued under National Pollutant Discharge Elimination System (NPDES) are required to perform a TRE if their effluents are suspected of causing unacceptable instream toxicity. Several guidance documents have been prepared by EPA to assist dischargers in conducting TRE studies (USEPA, 1993a, 1993b, 1992, 1991, 1989a and 1989b). These documents describe why it is necessary to use a toxicity-based approach that relies on toxicity tests (instead of chemical measurements) to identify the cause(s) and source(s) of toxicity. This approach will ensure that corrective measures will achieve compliance with the toxicity-based limit.

A generalised schematic of the TRE process is presented in Figure 2. Major steps in the TRE process are summarised as follows:

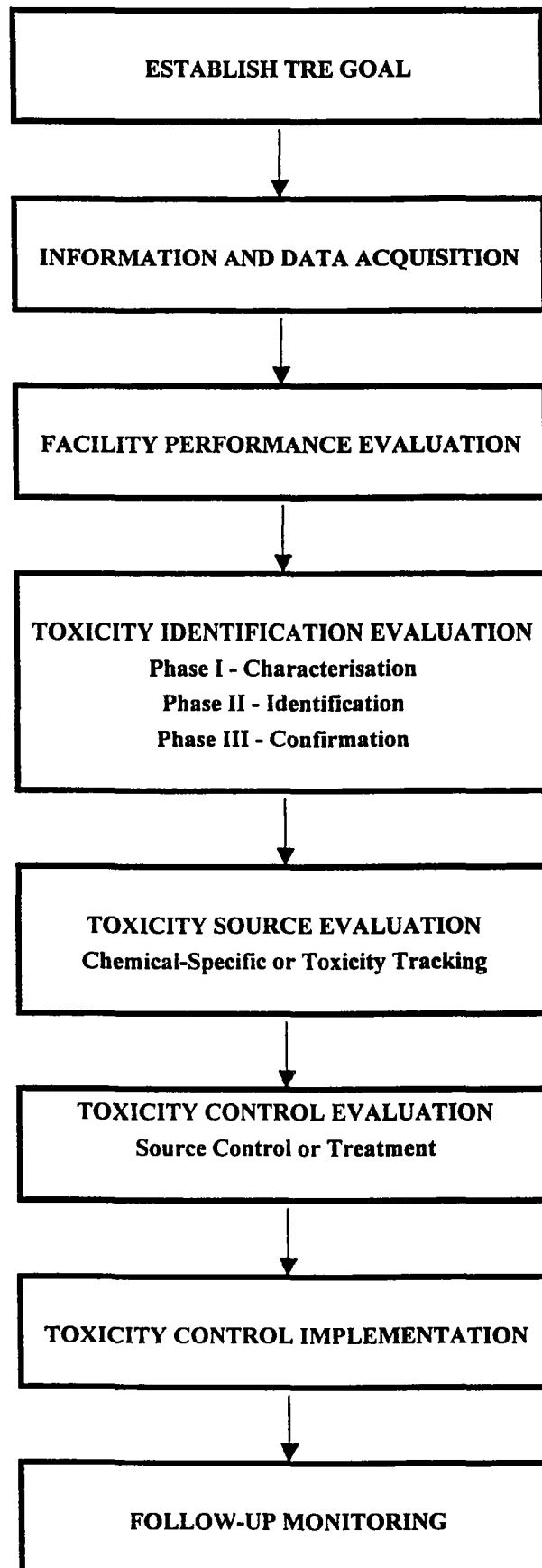
Establish the TRE Goal - so that the effluent compliance level and the duration of the TRE are understood. The goal and schedule of the TRE should be reviewed and agreed upon with the regulatory authority before studies are initiated.

Information and Data Acquisition - regarding facility activities and effluent monitoring is needed to prepare a plan of study. This information should include data on chemical use management, waste generating activities, and wastewater treatment operations and performance. TREs are an iterative process; therefore, the plan will change depending on the outcome of each step of the study.

Facility Performance Evaluation - involves a review of the manufacturing processes and wastewater treatment system to identify problem areas that may be contributing to effluent toxicity. This evaluation usually does not establish a "cause and effect" relationship between a suspect toxicant and effluent toxicity; therefore, simple, low cost corrective measures (e.g., waste minimisation, chemical use optimisation, and improved operational strategies) that have the potential to reduce toxicity are generally recommended. This step is most effective in cases where facility deficiencies or concentrations of known toxicants appear to be related to effluent toxicity.

Toxicity Identification Evaluation - is generally performed in three phases: toxicity characterisation (Phase I), toxicant identification (Phase II), and toxicant confirmation (Phase III). Phase I characterises the types of effluent toxicants by testing the toxicity of aliquots of effluent sample that have undergone bench-top treatments (USEPA, 1992 and 1991). Treatment steps include pH adjustment, filtration, aeration, oxidant reduction,

Figure 2. Generalised Flow Chart For Toxicity Reduction Evaluation



chelation of metals, and removal of non-polar organic compounds by a C₁₈ solid phase extraction (SPE) column. Removal of toxicity by one or more of these steps provides information on the class of the toxicants (e.g. ammonia, chlorine, metals, non-polar organic compounds, etc.). Phases II and III involve further treatments in conjunction with chemical analyses to identify and confirm the compound(s) causing effluent toxicity (USEPA, 1993a and 1993b). It is not always necessary to identify the actual toxicant(s) if toxicity can be effectively removed through source control or other means.

Source Identification - involves sampling and analysis of samples of individual waste lines to locate the source(s) of toxicity. Chemical tracking is recommended when the toxicants have been identified and confirmed in the TIE. However, toxicity tracking may be needed if the TIE results are inconclusive. In this latter approach, samples are first collected from the main sewer lines and results of toxicity tests are used to identify toxic tributary waste streams and, ultimately, the specific sources of toxicity. Samples must be subjected to the same treatment as is practiced in the facility's treatment process to provide an accurate measure of the toxicity that passes through in the final effluent.

Toxicity Control Evaluation - selects the most appropriate control method based on a thorough review of the technical and cost considerations of the available alternatives. The selected control method(s) is implemented and follow-up monitoring is conducted to ensure that it reduces effluent toxicity to compliance levels. Many TREs in the U.S. have identified relatively low cost operational or chemical use changes, rather than large-scale treatment plant modifications or additions, as the preferred toxicity control methods.

3. INDUSTRY DESCRIPTION

3.1 Process Types

Chlorine is mainly produced by the electrolysis of sodium chloride brine or, occasionally, potassium chloride brine. Manufacturing utilises mercury, diaphragm or membrane cell processes. Methods of production are well documented and detail need not be repeated here.

In the U.K., 90% of the chlorine is currently produced by the mercury cell process. Most manufacturers are planning to convert to less environmentally sensitive processes such as diaphragm cell or membrane cell technology, but progress has been slow. Manufacturers claim that the conversion is not cost effective and cite research carried out which apparently shows that mercury discharges from chlor-alkali plant are insignificant compared to mercury discharges from industry as a whole. Instead, investment has focused on reducing mercury discharges from existing equipment, generally via improved effluent interception and treatment.

Basic information on chlor-alkali manufacturers suggest that the four or five manufacturers operate plants ranging from 42 000 to 750 000 tonnes per annum peak output. However, some of these plants run at reduced capacity as a result of lessened product demand.

3.2 **Chemicals Used**

Raw materials for mercury cell process:

Mercury
Sodium chloride brine
Sulphuric acid
R12 refrigerant
Water treatment chemicals
Hydrochloric acid *
Sodium Hydroxide*

Raw materials for the membrane/diaphragm process:

Sodium chloride brine
Sulphuric acid
Hydrochloric acid*
Asbestos
Nickel sulphate
R12 refrigerant
Water treatment chemicals
Nitrogen
Filter aid

*Note that chlor-alkali plants may include other related processes such as hydrochloric acid, sodium hydroxide or alkyl lead production.

3.3 **Wastewater Treatment Processes**

3.3.1 **Mercury Plants**

Plant A - operates 110 mercury cells with a peak capacity of 90 000 tonnes per year. Effluent from the process is sent to a recovery plant of 9 m³/hr capacity. Here oxidation and acidification dissolve all the elemental mercury, followed by sand filters, activated carbon filters and finally ion exchange.

Effluent from this plant is sent to the main treatment plant for the site (on which other processes take place). The main processes here are; pH neutralisation, sodium thiosulphate addition (to eliminate free chlorine), oil removal, settling and final clarification. Treated effluent is discharged to a canal.

Plant B - operates 250 cells with a peak of 740 tonnes/year. Effluent treatment involves mercury recovery by mixed media filters followed by dechlorination and pH adjustment.

The company states that in case of failures within the treatment plant, effluent is sent to an emergency outfall and sodium thiosulphate and caustic are added to eliminate chlorine

and precipitate the mercury before discharge to the canal.

3.3.2 Membrane/Diaphragm Plants

The main UK plant operating this technology employs two separate effluent systems; the first called the acid/alkali system which runs hypochlorite free, the second called the miscellaneous system which contains small quantities of hypochlorite.

The waste streams from these two systems are combined before being discharged to a limebed system which allows settlement and neutralisation. The overflow from the limebeds falls to a local river.

3.4 Discharge Characteristics

3.4.1 Potential Release Routes

Potential releases to water include:

- a) Waste brine from the cells (brine is purged from membrane cells to reduce the levels of sodium sulphate and/or sodium chlorate in the cells).
- b) Chlorine-contaminated water containing residual dissolved free chlorine or hypochlorite.
- c) Spent sulphuric acid from chlorine drying, unless recycled.
- d) Waste scrubber liquor (hypochlorite in caustic) from the absorption plant.
- e) Brine mud, filter washings and waste liquor from the brine purification plant.
- f) Mercury contamination may be present in any liquid effluent discharges of storm water from mercury plants.
- g) Water treatment chemicals.

3.4.2 Discharge Requirements

Discharge requirements for representative facilities are presented in Tables 1, 2, and 3. The allowable effluent concentrations of mercury, chlorine and ammonia for these facilities are high enough to cause acute toxicity to freshwater and marine organisms (USEPA, 1994). Therefore, depending on how future TBCs are derived, it may be necessary to meet lower limits for these as well as other parameters.

As noted above, chlor-alkali plants may include other manufacturing processes, such as hydrochloric acid, sodium hydroxide or alkyl lead production. These processes may

generate other wastes of concern, such as organo-lead compounds from alkyl lead production.

Table 1. Discharge requirements for a plant operating mercury cells of 90 000 tonnes/year peak output

Parameters	Limit
Mercury (concentration)	100 µg/l per 24 hours 50 µg/l averaged over 30 consecutive days
Mercury (load)	1 kg/day 7.3 kg/month
Free Chlorine	10 mg/l
Suspended solids	35 mg/l

Table 2. Discharge requirements for a plant operating mercury cells of 740 000 tonnes/year peak

Parameter	Limit
Suspended solids	50 mg/l
pH	5-10 range
Mercury - concentration - load	500 µg/l 7.3 kg/month
Free Chlorine	10 mg/l
Suspended solids	35 mg/l

Table 3. Discharge requirements for a plant operating diaphragm cells and membrane cells (75:25 split) of 90 000 tonnes/year peak output

Parameter	Limit
Suspended solids	300 mg/l
Ammoniacal Nitrogen	150 mg/l (as N)
pH	5-11 range
Temperature	<30 deg C
Volume	22 Megalitres per day (dry weather flow)
Rate	500 litres/second

4. TRE CASE STUDIES

Published information on the toxicity and potential toxicants in chlor-alkali plant effluents is limited. A primary concern is the discharge of mercury from plants using the mercury cell process; however, mercury has not been specifically related to effluent toxicity. In fact, substances other than mercury have been indicated as causing effluent toxicity at a chlor-alkali facility in India (Shaw *et al.*, 1990, 1989 and 1988).

Unpublished information on two TRE studies was made available through a chlor-alkali company in the U.S. TRE information on ASHTA Chemical's facility in Ashtabula, Ohio was also summarised from Kircher and Tallon (1994). In addition, results of a literature review are provided as follows.

4.1 Literature Review

Mercury pollution related to chlor-alkali plants has been well documented (Gobeil and Cosa, 1993; Winger *et al.*, 1993; Cosa, 1990; Bothner *et al.*, 1980 and Loring and Bewers, 1978). In the early 1970s, some commercial fisheries in the US and Europe were closed due to mercury contamination linked to chlor-alkali plant discharges. More recently, waste minimisation and improved treatment have significantly reduced mercury levels in these discharges. However, the long-term accumulation of mercury in sediments continues to be a concern because of *insitu* microbial methylation and the subsequent release of methylmercury (Callister and Winfrey, 1986 and Bothner *et al.*, 1980), a form of mercury that has been found to be very toxic to aquatic life.

Studies of a chlor-alkali plant in India (Shaw *et al.*, 1990, 1989 and 1988) have shown the effluent to be toxic to a blue green algae (*Westiellopsis prolifica*) and two freshwater fish (*Anabas scandens* and *Tilapia mossambica*). Both pigment content (chlorophyll, phaeophytin, and carotenoid) and oxygen evolution were inhibited in the algae. Twenty-four hour LC₅₀ values for *A. scandens* and *T. mossambica* were 39.4 and 31.3 percent

effluent, respectively. In each case, a substance other than mercury was indicated to be causing the observed toxicity. This conclusion was based on the low mercury concentration in the effluent as compared to the reported lethal levels for the algae (0.04 mg/l) and fish (24-hour LC₅₀ = 1.38 mg/l for both fish species).

Shaw and Panigrahi (1990) also found acetylcholinesterase (AChE) activity, a measure of brain function, to be impaired in several fish species in the Rushikulya River estuary (India) receiving the chlor-alkali plant discharge. Inhibition of AChE activity was significantly correlated with mercury concentration in the fish brains.

In Brunswick, Georgia, sediments and pore water in a tidal stream near a chlor-alkali plant discharge were found to be toxic to Microtox™ and an amphipod, *Hyalella azteca* (Winger *et al.*, 1993). Microtox™ EC₅₀ values ranged from <5 to <20 percent pore water in close proximity to the plant outfall. Percent mortality and feeding rate of *H. azteca* in 10 day toxicity tests of sediment pore water varied from 55 to 75% and 0.4 to 0.65 mg/animal/day, respectively. These values were significantly different than those observed for an unaffected reference site (i.e. percent mortality = 5 and feeding rate = 0.9 mg/animal/day). Percent mortality of *H. azteca* in sediment from the impacted stations (45 and 55%) was also significantly different than the reference site sediment (75%).

Although several metals were found in relatively high concentrations in the sediment pore water, Winger *et al.*, (1993) surmised that the presence of sufficient quantities of acid volatile sulfide (AVS) would cause the metals to be bound and, hence, not biologically available. Therefore, they suggested that metals not bound by AVS or organometals, such as methylmercury, were contributing to toxicity.

Although the literature review did not provide direct information on causes of effluent toxicity at chlor-alkali plants, there is some evidence that substances other than mercury may be a concern. Based on the U.S. experience in conducting industrial TREs, the cause(s) of toxicity may be different for each facility.

4.2 TRE Case Examples

Each TRE case study focused on TIE testing to identify the cause(s) of effluent toxicity. In two cases, the identified toxicants were easily controlled. The third study involved extensive modifications, which were required to meet both toxicity and chemical permit limits. Key elements of each study are presented in Table 4. A description of the studies is provided below.

4.2.1 Northeast US Facility

New effluent toxicity requirements were anticipated for a chlor-alkali facility in the northeastern U.S. The company decided to monitor the effluent in advance of the requirements to determine the magnitude of toxicity and, if necessary, undertake corrective proactive actions.

A series of acute toxicity tests were performed using a water flea (*Ceriodaphnia dubia*) and a minnow (*Pimephales promelas*). Results showed toxicity to both species; therefore, effluent samples were collected for evaluation with EPA's TIE Phase I procedures (USEPA, 1991).

As noted above, the Phase I treatment steps include pH adjustment, filtration, aeration, oxidant reduction, chelation of metals, and removal of non-polar organic compounds by a C₁₈ solid phase extraction (SPE) column. Due to concern about potentially toxic chlorine levels in the effluent, the TIE tests in this study focused on the oxidant reduction step, which is used to evaluate the presence of toxic oxidants, such as chlorine. Sodium thiosulphate (Na₂S₂O₃) was added in varying amounts to aliquots of an effluent sample and toxicity tests using *Ceriodaphnia* were performed on the treated aliquots and the original untreated sample. The results showed a trend of decreasing toxicity with higher thiosulphate additions up to a point where residual chlorine would be expected to be fully reduced. Results of oxidant reduction tests and total residual chlorine (TRC) analyses performed on additional effluent samples confirmed chlorine as the primary cause of toxicity.

Based on these results, the company decided to add a final chlorine reduction step to the wastewater treatment system. The process involves mixing sulphur dioxide into the final effluent stream to reduce TRC to levels that are not acutely toxic to *Ceriodaphnia* or fathead minnows.

It is important to note that several cationic metals, including mercury, may be complexed and effectively removed by thiosulphate addition (USEPA, 1991). As a result, toxicity reduction by thiosulphate addition may not provide conclusive evidence that toxicity was caused solely by chlorine. Another TIE Phase I step, the addition of a chelating agent [ethylenediaminetetraacetate (EDTA)], can be used to differentiate between chlorine and metals toxicity. If toxicity is removed by both thiosulphate and EDTA addition, metals (such as mercury) may be the toxicants. If toxicity is removed by thiosulphate addition, but not EDTA addition, an oxidant such as chlorine would be the indicated toxicant.

Table 4. Key elements of the TRE case studies.

Facility	Goal (Permit Limit)	Toxicants Identified	Sources Identified	Control Method
Northeast US	LC ₅₀ =100% using <i>Ceriodaphnia dubia</i> and <i>Pimephales promelas</i>	Residual chlorine	Manufacturing process	Dechlorination
Southwest US	LC ₅₀ =100% using <i>Mysidopsis bahia</i>	Calcium carbonate imbalance	Not determined	Addition of dry sea salts to toxicity test samples
ASHTA Chemicals	Chronic toxicity limit (not specified) using <i>Ceriodaphnia dubia</i> and <i>Pimephales promelas</i>	Total dissolved solids (TDS)	Manufacturing process	Zero discharge

4.2.2 Southwest US Facility

Routine monitoring of a chlor-alkali plant effluent in the southwest U.S. indicated unacceptable levels of acute toxicity to the estuarine shrimp, *Mysidopsis bahia*. Based on these results, the regulatory authority required the company to perform a TRE study. Preliminary analyses of the effluent indicated very low levels of calcium carbonate (CaCO₃), an essential compound for mysid growth and reproduction. The low calcium levels were related to treatment (softening) of waste brine, which effectively removed calcium from the plant effluent. Although calcium and other ions were added to dilution water used to prepare the concentration series for mysid toxicity tests (e.g., 12.5, 25, 50, 75 percent effluent), the 100 percent effluent concentration did not receive these added ions. As a result, the undiluted effluent had relatively low levels of calcium and mysid mortality exceeded the permissible limit (i.e. 50 percent mortality in 100 percent effluent).

Calcium and carbonate, in proper balance with other natural ions, are essential for the formation of new exoskeleton for mysids and other crustaceans. At low calcium carbonate levels (i.e. 15 mg/l CaCO₃), Ward (1989) observed sixty percent mortality in mysids between the 48 hour and 72 hour exposure periods, which corresponds well with the mysid molting cycle. Low CaCO₃ levels also appear to enhance mysid sensitivity to other toxicants. Ward (1989) observed a significant increase in the toxicity of cadmium to mysids when calcium carbonate levels were reduced.

Based on this information, the company performed tests to determine the effect of adding calcium carbonate, in the form of dry sea salts, to undiluted effluent samples. Lower mysid mortality was observed for samples adjusted with a synthetic sea salt mixture as compared to unadjusted samples. On the strength of this evidence, the company submitted a request to modify the toxicity test procedure to allow the addition of a commercial sea salts mixture to effluent samples used for mysid testing. The regulator approved the request, and since then the company has consistently met its permit limit for toxicity.

4.2.3 ASHTA Chemicals Facility

ASHTA Chemicals, Inc. (ASHTA) operates a chlor-alkali facility in Ashtabula, Ohio that produces potassium hydroxide, potassium carbonate, chlorine, hypochlorite bleach, hydrogen, and chloropicrin. Untreated process wastewater contains part per million (ppm) levels of mercury and percent concentrations of total dissolved solids, primarily as potassium chloride. Treatment of this waste stream includes sulphide precipitation to remove mercury to part per billion levels.

In 1989, ASHTA initiated a TRE to identify and control the causes of effluent toxicity at the facility. In TIE tests using *Ceriodaphnia dubia* and fathead minnows, potassium chloride (KCl) was found to be the primary cause of toxicity. Based on the test results, the maximum allowable effluent concentration of KCl was determined to be 75 mg/l. The treated process wastewater contained 20 000 to 100 000 mg/l of KCl; therefore, substantial facility modifications would be required to reduce the salt concentrations to below the toxicity threshold. Even diluting the process waste stream with the facility's non-contact cooling water would reduce the KCl concentration to only 500 mg/l. As shown in Table 5, further testing identified relatively high TDS levels in many individual process waste streams.

In the recent discharge permit, ASHTA Chemicals also received a maximum discharge limit of 0.012 $\mu\text{g/l}$ (ppb) of mercury. State-of-the-art treatment technologies were evaluated; however, no single process or combination of processes were expected to achieve compliance with this mercury limit.

Table 5. Summary of waste stream characterisation data - ASHTA Chemicals, Inc.

Waste Stream	Average Flow (gpd)	Total Dissolved Solids (ppm)	Total Mercury (ppb)	pH
Effluent sump	5 500	107 370	19 800	>14
Brine cell flushing	750	84 060	18 800	<3
Boiler blowdown	14 400	1 250	<0.2	-1
Potassium hydroxide storage tank area	6 600	19 100	101	>10
Deionised water regenerant	3 700	11 190	53	3 - 12
Leachate	6 400	27 210	59	>10
Chlorinated water	18 000	900	35	<2.5
Storm water	18.6	697	8.6	>8.0

N/A - not applicable

Source: Kircher and Tallon (1994).

Faced with permit limits that would stretch the limits of technology, ASHTA decided to evaluate the possibility of eliminating its discharge. Various combinations of closed-looped systems were reviewed, including evaporators, reverse osmosis and physical separation processes. Based on water balance calculations, it was determined that a closed-looped cooling tower system could be implemented; however, mercury and KCl would need to be removed before the process water could be used in the cooling tower. Treatment of the process water focused on three promising technologies: sulphur-impregnated carbon, ion exchange, and reverse osmosis. Results of treatability tests, shown in Table 6, demonstrated that reverse osmosis would achieve the best removal of both mercury and KCl.

Table 6. Treatability test results for candidate mercury and potassium chloride recovery processes -ASHTA Chemicals, Inc.

System	Influent TDS (ppm)	Influent Mercury (ppb)	KCl Rejection (%)	Mercury Rejection (%)
Impregnated carbon	1 128	2.4	N/A	99.4
Ion exchange	1 118	N/A	79.1	N/A
	4 957	N/A	49.9	N/A
Reverse osmosis	1 118	4.1	85.7	97.6
	4 957	6.64	72.2	91.7

N/A - not applicable

Source: Kircher and Tallon (1994).

The new closed-loop system will consist of a recirculating cooling water tower and a mercury and KCl recovery process. The new recovery process will greatly reduce the need for the existing sulphide precipitation system used for mercury removal. This will minimise the generation of mercuric sulphide waste sludge, which is classified as hazardous in the U.S. and must undergo a costly disposal process. Additional steps are being implemented to reduce TDS levels in the process waste streams before they enter the recovery process. These steps include recycling process streams that have high salt levels, modifying operating procedures, improving housekeeping, automating process control, and minimising and recovering product losses. ASHTA is also implementing a comprehensive storm water collection and storage system and a leachate management process for its on-site landfill.

Although the new water management system will require a substantial capital outlay, ASHTA anticipates several immediate and long-term benefits, including:

- Significant cost savings through recycling and reuse of recovered materials, including mercury and salts.
- Virtual elimination of hazardous waste sludge, which will obviate the need for a retort to recover mercury (required because mercury contaminated sludge has been banned from landfills).
- Elimination of the surface water discharge permit and its associated monitoring costs and potential liabilities.
- Successful completion of the TRE.

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6. REFERENCES

Bothner, M H, Jahnke, R A, Peterson, M L and Carpenter, R (1980). Rate of mercury loss from contaminated estuarine sediments. *Geochim. Cosmochim. Acta* 44:273-285.

Callister, S M and Winfrey, M R (1986). Microbial methylation of mercury in upper Wisconsin River sediments. *Water Air Soil Pollut.* 29:453-465.

Cosa, D (1990). Chemical contaminants in the St. Lawrence estuary and Saguenay fjord. p. 239-262. In El-Sabh, M I and Silverberg, N [ed.] *Oceanography of a large-scale estuarine system: the St. Lawrence*. Springer-Verlag, Berlin, 434 p.

Gobeil, C and Cosa, D (1993). Mercury in sediments and sediment pore water in the Laurentian trough. *Can. J. Fish. Aquat. Sci.* Vol. 50 pp. 1794-1800.

Kircher, T P and Tallon, J T (1994). Achieving zero discharge: ASHTA Chemicals' zero discharge process water recovery system. *Pollution Prevention Review*, Autumn, 1994, pp 371-385.

Loring, D H and Bowers, J M (1978). Geochemical mass balances for mercury in a Canadian fjord. *Chem. Geol.* 22:309-330.

Shaw B P, Sahu, A and Panigrahi, A K (1990). Comparative toxicity of an effluent from a chlor-alkali industry and HgCl₂. *Bull. Environ. Contam. Toxicol.* 45:280-287.

Shaw B P, Sahu, A and Panigrahi, A K (1989). Effect of the effluent from a chlor-alkali factory on a blue-green algae: changes in the pigment content. *Bull. Environ. Contam. Toxicol.* 43:618-626.

Shaw, B P, Sahu, A and Panigrahi, A K (1988). Effect of the effluent from a chlor-alkali factory on a blue-green alga: changes in the oxygen evolution rate. *Microbios Letters* 37:89-96.

Shaw B P and Panigrahi, A K (1990). Brain AchE activity studies in some fish species collected from a mercury contaminated estuary. *Water, Air and Soil Pollution* 53:327-334.

U.S. Environmental Protection Agency (1994). Water quality standards handbook. Second Edition. Office of Water, Washington, D.C. EPA-823-B-94-005a.

U.S. Environmental Protection Agency (1993a). Methods for aquatic toxicity identification evaluations: phase II toxicity identification procedures for samples exhibiting acute and chronic toxicity. EPA/600/R-92/080.

U.S. Environmental Protection Agency (1993b). Methods for aquatic toxicity identification evaluations: phase III toxicity confirmation procedures for samples exhibiting acute and chronic toxicity. EPA/600/R-92/081.

U.S. Environmental Protection Agency (1992). Toxicity identification evaluation: characterisation of chronically toxic effluents, phase I. EPA/600/6-91/005F.

U.S. Environmental Protection Agency (1991). Methods for aquatic toxicity identification evaluations: phase I toxicity characterisation procedures. Second edition. EPA/600/6-91/003.

U.S. Environmental Protection Agency (1989a). Toxicity reduction evaluation protocol for municipal wastewater treatment plants. EPA/2-88/062.

U.S. Environmental Protection Agency (1989b). Generalised methodology for conducting industrial toxicity reduction evaluations. EPA/600/2-88/070.

Ward, S H (1989). The requirements for a balanced medium in toxicological experiments using *Mysidopsis bahia* with special reference to calcium carbonate. Aquatic Toxicology and Hazard Assessment: 12th volume. ASTM STP 1027, Cowgill, U M and Williams, L R [eds.], American Society for Testing and Materials, Philadelphia, pp. 402-412.

Winger, P V, Lasier, P J and Geitner, H (1993). Toxicity of Sediments and Pore Water From Brunswick Estuary. Arch. Environ. Contam. Toxicol. 25:371-376.

APPENDIX A

Table A-1. Screening tests proposed for use in selecting appropriate effluents for toxicity-based control.

Toxicity Test	Receiving Water	
	Freshwater	Marine Waters
5-30 minute Microtox™ (<i>Photobacterium phosphoreum</i>) bioluminescence test (Butler <i>et al</i> 1991)	+	+
24 hr water flea (<i>Daphnia magna</i>) immobilisation test (OECD 1984)	+	
24 hr oyster (<i>Crassostrea gigas</i>) embyro-larval development test (ICES 1991)		+

Table A-2. Algae, invertebrates, and fish proposed for use in the testing of the toxicity of effluents discharged to fresh and marine waters.

Type of Organism	Freshwaters	Marine Waters
ALGAE	<i>Selenastrum capricornutum</i> 72 hr inhibition of growth (OECD 1984)	<i>Phaeodactylum tricornutum</i> <i>Skeletonema costatum</i> 96 hr inhibition of growth (ISO 1988)
INVERTEBRATES	<i>Daphnia magna</i> (water flea) 48 hr immobilisation (OECD 1984)	<i>Crassostrea gigas</i> (Pacific oyster) embryos 24 hr inhibition of development (ICES 1991)
FISH	<i>Salmo trutta</i> (Brown trout) <i>Oncorhynchus mykiss</i> (Rainbow trout) 96 hr mortality (OECD 1984)	<i>Pleuronectes platessa</i> (Plaice) <i>Scophthalmus maximus</i> (Turbot) 96 hr mortality

