

## Ecotoxicology of actively treated mine waters

Andrew J Harford, David R Jones, Rick A van Dam

*Environmental Research Institute of the Supervising Scientist (eriss), Department of Sustainability, Environment, Water, Population and Communities, GPO Box 461, Darwin, Northern Territory 0801, Australia. andrew.harford@environment.gov.au*

### Abstract

Mining operations are increasingly using active water treatment to improve water quality prior to its discharge to the environment. Our experience of assessing treated mine waters using a suite of biological toxicity tests and Toxicity Identification Evaluations (TIE) has highlighted that environmental risks can still exist for these treated waters. We present examples of this based on mine waters that were treated using a high density sludge-microfiltration-reverse osmosis (HDS/MF/RO) process or brine concentration (BC). Whilst active treatment undoubtedly reduces the toxicity of mine waters, the subsequent management strategies for treated water need to recognise that toxicity risks may exist and will be different to those of untreated water.

**Keywords:** ecotoxicology, biological toxicity tests, Toxicity Identification Evaluation, water treatment, water management, environmental impacts.

### Introduction

Depending on the mineral resource being extracted, mining operations may produce large volumes of saline, acidic, turbid and/or metal contaminated mine and process waters (Lottermoser 2010). A mine site's water inventory often represents a significant environmental and financial liability for the operation. Operations that need to discharge untreated and/or treated mine waters to the environment are usually required to ensure there will be no unacceptable measureable impact on adjacent ecosystems (Moore and Luoma, 1990) before such discharge occurs.

There are numerous potential passive and active water treatment options available to mine operators, with the ultimate selection of the most appropriate technology or combinations of technologies depending on economic, environmental and technical factors (Gunther and Mey 2006). Key considerations need to be the extent to which the water quality is actually improved and what residual effects may remain to be managed. Some costly active water treatment options have the ability to produce waters that, whilst being significantly improved compared to the feed water, may still contain residual contaminants and/or may not sufficiently support the needs of local ecosystems due to a lack of essential elements. There is a surprising paucity of published literature concerning the potential impact of treated mining/industrial waters on aquatic ecosystems, despite some industries potentially producing gigalitres annually of highly-treated waters (e.g. the coal seam gas industry; Takahashi et al 2011), of which a proportion may require discharge into the environment. Ecotoxicological assessments of the treated water can determine the residual, or acquired, toxicity

of the water through biological toxicity test protocols, including identifying the cause(s) of toxicity using Toxicity Identification Evaluations (TIE) procedures.

Our research, which is primarily focused on the Ranger Uranium Mine operation in tropical Northern Australia, provides examples of the potential of treated waters to impact the environment if directly released. The Ranger mine has been in operation since the early 1980s and the lease is surrounded by the Kakadu World Heritage area and is upstream of the Ramsar-listed Magela Creek floodplain. Consequently, the mine is required to manage discharges under a strict regulatory regime to ensure such releases result in no measurable detrimental impact to the downstream environment. In the early 2000s the operation recognised that the increasing pond (PoW) and process (PrW) water inventories were a significant risk to the business (Topp et al 2003). Process water represents the poorest water quality on site, with key water quality characteristics typically as follows: pH: 3.7–4.0; electrical conductivity (EC): 22 000–27 000  $\mu\text{S}/\text{cm}$ ; sulfate ( $\text{SO}_4$ ): 24 000–34 000 mg/L; uranium (U): 18–25 mg/L; and total ammonia ( $\text{NH}_3$ ): 780–950 mg/L N. Pond water consists of water from the run-off from ore stockpiles and is characterised by pH: 7.0–8.2; EC: 1 190–1 660  $\mu\text{S}/\text{cm}$ ;  $\text{SO}_4$ : 720–960 mg/L; U: 4–5 mg/L; and  $\text{NH}_3$ : <0.01–0.08 mg/L N (ERA, Water Management Plans 2006–2011, unpublished). Since identifying the need to treat PoW and PrW water, the mine has invested in a high density sludge-microfiltration-reverse osmosis (HDS/MF/RO) water treatment plant, and a brine concentration (BC) facility is currently under construction. Both of these treatment methods result in orders of magnitude improvement in water quality. However, some residual inorganic contaminants remain depending on the feed water type and treatment method.

The ecotoxicology research team at the Environmental Research Institute of the Supervising Scientist (eriss) has conducted numerous ecotoxicological assessments on various pilot- and full-scale plants waters in order to determine the suitability of the waters for release into high-value ecosystems and the need for any further manipulations of water quality prior to release. The aim of this paper is to summarise the findings from the various treated mine water assessments and to highlight the need for mine operators to remain cognisant of the potential environmental risks of treated waters.

## Methods

Over the course of 10 years, toxicity assessments have been undertaken for five different treated waters produced by the Ranger mine using various pilot and full-scale plants to treat pond or process water (Table 1). Two of these waters were permeates produced from process water using HDS/MF/RO systems, two were permeates from the treatment of pond water using MF/RO systems without HDS, while the most recent water was a distillate produced by a pilot-scale brine concentrator (Table 1).

The toxicity of the treated waters was assessed using up to five Australian tropical freshwater species (Table 1). The full suite of 5 species included; the unicellular green alga (*Chlorella* sp.); the duckweed (*Lemna aequinoctialis*); the green hydra (*Hydra viridissima*); the cladoceran (*Moinodaphnia macleayi*); and the Northern

trout gudgeon (*Mogurnda mogurnda*). All the organisms were originally isolated from soft surface waters in Kakadu National Park and have been cultured continuously at (eriss) over 10–25 years, depending on the species. The test methods are described in detail by Riethmuller et al (2003). A natural soft water (Magela Creek Water; MCW) collected from upstream of the Ranger mine was used as the control/diluent for all tests.

**Table 1** Summary of treated waters assessed for ecotoxicity

Study #	Water Treatment	Water Type	Date tested	Testing-suite	Key Chemistry	Species affected	Effect of raw water (%)
1	HDS/MF/RO (pilot)	Process	Dec 2001	3 species a	EC = 170 $\mu$ S/cm NH <sub>3</sub> = 18 mg/L	<i>Cladoceran</i> <i>Hydra</i>	100 100
2	MF/RO (full-scale)	Pond	Dec 2005	5 species BTT	EC = 20 $\mu$ S/cm U = 4 $\mu$ g /L	<i>Cladoceran</i>	50
3	MF/RO (mobile plant)	Pond	July 2007	5 species BTT	EC = 19 $\mu$ S/cm U = 8 $\mu$ g /L	<i>Cladoceran</i> <i>Hydra</i>	50 10
4	HDS/MF/RO (full-scale)	Process	Oct 2009	5 species BTT	EC = 91 $\mu$ S/cm NH <sub>3</sub> = 7 mg/L N	<i>Cladoceran</i> <i>Hydra</i> <i>Duckweed</i>	80 100 40
5	Brine concentrator (pilot)	Process	Oct 2011	5 species BTT 1 species TIE	EC = 12-17 $\mu$ S/cm Mn = 130–230 $\mu$ g/L	<i>Hydra</i> <i>Cladoceran</i>	50- 100 <sup>b</sup> 10

<sup>a</sup> *Hydra*, the cladoceran and the fish; <sup>b</sup> Two different batches were tested with different toxicities that was attributed to differences in major ion concentrations.

A TIE procedure was conducted to determine the cause(s) of adverse effects on one species (*H. viridissima*) following exposure to brine concentrator distillate (BC-PrW). There are three phases that may be included in a TIE: Phase I (Characterisation) involves manipulations of the effluent that enable only a broad screening of the toxic constituents; Phase II (Identification) involves manipulations that specifically identify the toxicants of interest that have been implicated in Phase I; and Phase III (Confirmation) involves the re-introduction of the toxicants to confirm the toxicity of the suspected contaminants in the effluent (Mirenda and Hall 1992). All TIE tests included a control water (MCW or Synthetic Soft water, SSW) and distillate that were treated (as described in Table 2), as well as untreated control water (MCW and/or SSW) and distillate.

## Results and Discussion

Similar feed water and treatment process combinations produced products of similar composition and, toxicities. Specifically, the HDS/MF/RO treatment of process water (RO-PrW) produced water that was elevated in  $\text{NH}_3$  (~10–20 mg/L N) and EC (90–170  $\mu\text{S}/\text{cm}$ ) in comparison to the other treated waters. The treated pond water (RO-PoW) had a lower EC (~20  $\mu\text{S}/\text{cm}$ ) and negligible  $\text{NH}_3$  but higher concentrations of residual U (4–8  $\mu\text{g U}/\text{L}$ ). The process water that was treated by the brine concentrator (BC-Pr) had the lowest EC (12–17  $\mu\text{S}/\text{cm}$ ), low  $\text{NH}_3$  (~1 mg/L N) and U (1.0–1.5  $\mu\text{g}/\text{L}$ ) but residual manganese (Mn; 110–220  $\mu\text{g}/\text{L}$ ) concentrations (Table 1).

**Table 2** Toxicity Identification Evaluation toxicity tests for brine concentrator distillate using *Hydra viridissima* (from Harford et al 2012.)

TIE test	Test solution manipulation	Reason for manipulation	Result
Graduated pH	MCW and Distillate adjusted to pH (nominal) 5.5 and 7.5	Differentially alters speciation and toxicity of chemicals	Better growth rate at higher pH indicates that Mn more likely toxicant
EDTA <sup>a</sup> addition	0, 2.8, 5.5 and 11.0 mg/L EDTA added to MCW and distillate	EDTA binding reduces cationic metal bioavailability	No recovery in growth rates indicates that Mn is not the toxicant
$\text{NH}_3$ stripping	MCW and distillate adjusted to pH (nominal) 11 and aerated for 18 h. pH re-adjusted to 6.5 prior to testing.	Removes toxicity due to $\text{NH}_3$	No recovery in growth rates indicates that $\text{NH}_3$ is not the toxicant
C18 SPE	MCW and distillate post-C18 column water tested. Eluate of distillate tested in MCW	Tests for toxicity of organic compounds	No recovery in growth rates indicates that an organic is not the toxicant
Ca addition	0, 0.2, 0.5 mg/L Ca concentrations tested in synthetic soft water (SSW) and distillate	Reintroduction of an essential element	64% recovery in growth rate indicates lack of Ca is a major factor in adverse effects
Major ion addition	0, 50 and 100% proportions (compared to SSW <sup>b</sup> ) of Na, Ca and K added to SSW and distillate	Reintroduction of essential elements	100% recovery indicates that all adverse effects may be due to lack of major ions

<sup>a</sup> Ethylenediamine tetraacetic acid; <sup>b</sup> Synthetic Soft Water contains 0.5, 1.0 and 0.4 mg/L of calcium (Ca), sodium (Na) and potassium (K), respectively.

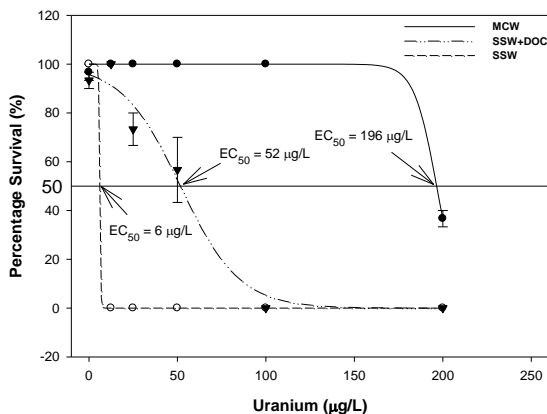
Although the toxicities of all of the treated product waters were substantially reduced from the feedwater, residual effects remained for all waters. The cause(s)

of adverse effects from the treated waters were hypothesised to be due to three key mechanisms of action, alone or in combination, as discussed below

### Increased bioavailability of contaminants

Dissolved organic carbon (DOC) can bind with trace metals, reducing their ability reach target sites and cause toxic effects (Trenfield et al. 2010). Removal of DOC can, therefore, remove this protective mechanism. Although, concentrations of U in the RO-PoW were considerably lower than those that were known to cause toxicity in natural surface waters (i.e. Inhibitory Concentration causing 50% effect ( $IC_{50}$ ) > 30  $\mu\text{g/L}$ ), exposure to this water (studies 2 and 3) reduced the reproduction of the cladoceran by 50%. Uranium induced toxicity was hypothesised to be a causative factor because the reduction of DOC in the treated water would have increased the proportion of bioavailable, and toxic, U. This hypothesis was investigated, and was supported by a reduction of acute U toxicity to the cladoceran in SSW dosed with 3.8 mg/L DOC (Figure 1). Moreover, the toxic effects of U in the SSW with no added DOC (Figure 1) were observed at concentrations similar to those found in the RO-PoW (i.e.  $\sim 6 \mu\text{g/L}$ ).

The concentration and composition of major ions can also affect the bioavailability of residual metals. Major cations (e.g.  $\text{Ca}^{2+}$ ,  $\text{Mg}^{2+}$ ,  $\text{Na}^+$ ) may compete with trace metals for cell target sites and are known to modify the toxicity of certain metals (Markich and Jeffree 1994; van Dam et al. 2010). Additionally, major anions (e.g.  $\text{SO}_4^{2-}$ ,  $\text{HCO}_3^-$ ) bind with metals and, as with DOC, can reduce their ability reach target sites. Hence, the reduction of major ions in treated waters has the potential to increase the bioavailability of trace metals. Although this mechanism could be important in some contexts, it was not evident in the work that has been done on treated Range waters.



**Figure 1** Acute toxicity of uranium to the cladoceran (*Moinodaphnia macleayi*, 48-h survival) in three water types: (i) synthetic Magela Creek water (SMCW) only; (ii) SMCW + 3.8 mg/L DOC (as IHSS Suwannee River Fulvic Acid Standard 1); and (iii) natural Magela Creek water (NMCW; containing 3.7 mg/L natural DOC) (from Hogan et al. 2009)

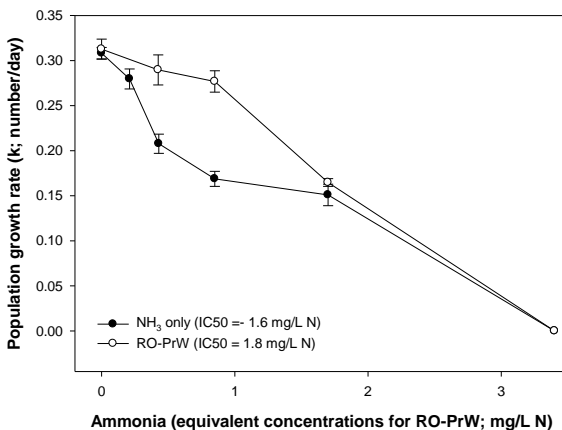
## Reduced essential elements

Water treatment can result in removal of essential ions such that their residual concentrations are insufficient for normal physiological functioning of some organisms (i.e. the treated water is too pure). For example, exposure to the BC-PrW markedly inhibited the population growth rate of hydra. Initial assessment of the BC-PrW water indentified the key residual contaminants as Mn, NH<sub>3</sub> or an unknown organic compound. A TIE was conducted using hydra in order to determine the cause(s) of the growth inhibition by the BC-PrW. Phase I TIE tests indicated that none of the identified contaminants were the primary cause of toxicity (Table 2). Hence, the focus turned to the hypothesis of essential ion deficiency. Firstly, Ca was investigated due to its importance in the physiological processes of hydra (Gitter et al. 1994). The addition of 0.2 and 0.5 mg/L Ca to the distillate resulted in a 61% and 66% recovery relative to the control, respectively, suggesting Ca deficiency as a major reason for the effect of BC-PrW on hydra (Table 2). A subsequent experiment where Na and K, which were also present at below background concentrations, were supplemented as well as Ca resulted in full recovery of hydra population growth rates (Table 2). This strongly indicated that majority of the adverse effect from the BC-PrW on hydra was due to major ion deficiency issue rather than a chemical toxicity. Hence, ion deficiency should be considered as a potential stressor when assessing the risk of discharging high purity treated waters, and may be the primary factor causing adverse effects.

Another essential element to consider is DOC, which is a nutritional source for many aquatic organisms. Hence, in addition to DOC's ability to reduce trace metal bioavailability, the removal of DOC may also impact some aquatic organisms' ability to grow and reproduce.

### *Residual contaminants*

Even highly efficient treatment technologies may not remove to all of the more highly toxic contaminants that that were initially present in the source water at high concentrations environmentally desirable concentrations. For example, residual concentrations of ammonia following HDS/MF/RO treatment of PrW were also sufficient to cause toxicity to some species (Table 1; studies 1 and 4). Exposure to RO-PrW markedly reduced the reproduction and growth rates of the cladoceran and the hydra with almost complete inhibition observed when exposed to the undiluted RO-PrW. This effect was attributed to the residual NH<sub>3</sub> that remained in the permeate, despite >99.9% removal of NH<sub>3</sub> during the treatment process (i.e. from ~900 mg/L to ~7 mg/L NH<sub>3</sub>-N). This hypothesis was supported by the hydra's response to NH<sub>3</sub> only in a synthetic soft water (Figure 2). Specifically, the IC<sub>50</sub> estimates for the NH<sub>3</sub> only and RO-PrW were 1.6 and 1.8 mg/L N, respectively. Hence, it was concluded that ammonia was the primary cause of toxicity in the RO-PrW water.



**Figure 2** Chronic toxicity of ammonia only and from HDS/MF/RO treated process water (plotted in terms of the concentration of ammonia in the treated water) to hydra.

## Conclusions

Whilst treatment can substantially reduce the toxicity of mine waters, it needs to be recognised that risks due to toxicity may still exist and be of a different nature to those of untreated water, and that management strategies for treated water need to take these residual risks into account. This paper has presented examples of water treatment processes producing products that: 1) increased bioavailability of contaminants due to physiochemical changes (i.e. reduced concentrations of major ions/water hardness, reduced DOC); 2) contained residual contaminants and ; 3) were too low in essential ions to be able to sustain certain freshwater species.

Ion deficiency clearly needs to be considered as a potential primary stressor in risk/impact assessments of the discharge of high purity treated waste waters, given the large future volumes of MF/RO treated water forecast to be produced by components of the mining industry. In this context, we have found only one other published study (RO-treated sewage water; Griffith and Biddulph 2010) and one government report (RO-treated CSG water; Takahashi et al 2011) that have previously reported ion deficiency as being the primary cause of adverse biological effects from RO-treated waters. Ultimately, treated waters should not be assumed *a priori* to be of a sufficient quality for direct and uncontrolled release to the environment, even if their water quality is comparable to existing water quality guidelines.

## Acknowledgements

Thanks to the *eriss* ecotoxicology technical team for their performance of the laboratory work; Alicia Hogan, Melanie Trenfield, Kim Cheng and Claire Costello.

---

## References

- Gitter AH, Oliver D and Thurm U (1994) Calcium-and voltage-dependence of nematocyst discharge in *Hydra vulgaris*. *J Comp Physiol A* 175(1), 115-122.
- Gunther P and Mey W (2006) Selection of mine water treatment technologies for the eMalahleni (Witbank) Water Reclamation Project, Proc Water Inst of Sth Africa Conf, 21-25 May, Durban, South Africa.
- Griffith M and Biddulph S (2010) Replacement of Environmental Flows in the Hawkesbury-Nepean River-Assessing the influence of low conductivity recycled water on freshwater aquatic biota. *Water* 37(1), 166-172.
- Harford AJ, Hogan AC, Cheng K, Costello C, Houston M and van Dam RA (2009) Preliminary assessment of the toxicity of manganese to three tropical freshwater species. In: *eriss research summary 2007-2008*. Supervising Scientist Report 200, Supervising Scientist, Darwin NT, pg 12-19.
- Hogan A, van Dam R, Harford A and Costello C (2009) Screening level ecotoxicological assessment of treated pond water from Ranger uranium mine to five local freshwater species. In: *eriss research summary 2007-2008*. Jones DR & Webb A (eds), Supervising Scientist Report 200, Supervising Scientist, Darwin NT, pg 20-24
- Lottermoser B (2010) Mine wastes: characterization, treatment and environmental impacts, Springer-Verlag, Berlin, Germany.
- Markich SJ and Jeffree RA (1994) Absorption of divalent trace metals as analogues of calcium by Australian freshwater bivalves: an explanation of how water hardness reduces metal toxicity. *Aquatic toxicology* 29(3-4), 257-290.
- Mirenda R & Hall W 1992. The application of effluent characterization procedures in toxicity identification evaluations. *Water Sci Technol* 25 (3), 39-44
- Moore JN and Luoma SN (1990) Hazardous wastes from large-scale metal extraction. *Environ Sci Technol* 24(9), 1278-1285.
- Peters A, Lofts S, Merrington G, Brown B, Stubblefield W and Harlow K (2011) Development of biotic ligand models for chronic manganese toxicity to fish, invertebrates, and algae. *Environ Toxicol Chem* 30 (11), 2407-2415.
- Riethmuller N, Camilleri C, Franklin N, Hogan AC, King A, Koch A, Markich SJ, Turley C and van Dam RA (2003). Ecotoxicological testing protocols for Australian tropical freshwater ecosystems. Supervising Scientist Report 173, Supervising Scientist, Darwin NT.
- Takahashi E, McGregor G and Rogers S (2011) Stream ecosystem health response to coal seam gas water release: Direct toxicity assessment. Queensland Department of Environment and Resource Management, Brisbane.
- Topp H, Russell H, Davidson J, Jones D, Levy V, Gilderdale M, Davis S, Ring R, Conway G and Macintosh P (2003) Process water treatment at the Ranger uranium mine, Northern Australia. *Water Sci Technol* 47(10), 155.
- van Dam RA, Hogan, AC, McCullough CD, Houston MA, Humphrey CL and Harford AJ (2010) Aquatic toxicity of magnesium sulfate, and the influence of calcium, in very low ionic concentration water. *Environ Toxicol Chem* 29(2), 410-421.