



Philbrick, D. J., Hopkins, J. B., Hill, D. C., Alexander, J. C. and Thomson, R. G. (1979). Effects of prolonged cyanide and thiocyanate feeding in rats. *Journal of Toxicology and Environmental Health*. 5: 579-592.

Potter, A. L. (1950). The successful treatment of two recent cases of cyanide poisoning. *British Journal of Industrial Medicine*. 7: 125-130.

Pritsos, C. A. and Ma, J. (1997). Biochemical assessment of cyanide-induced toxicity in migratory birds from gold mining hazardous waste ponds. *Toxicology and Industrial Health*. 13: 203-209.

Pryor AJ, Johnson DE and Jackson NN (1975). Hazards of smoke and toxic gases produced in urban fires. *Combust. Toxicol*. 2:64-112. As cited in NRC 2002.

Purser, D. A., Grimshaw, P. and Berrill, K. R. (1984). Intoxication by cyanide in fires: a study in monkeys using polyacrylonitrile. *Archives of Environmental Health: An International Journal*. 39: 394-400.

Raef, S. F., Characklis, W. G., Kessick, M. A. and Ward, C. H. (1977a). Fate of cyanide and related compounds in aerobic microbial systems—I. Chemical reaction with substrate and physical removal. *Water Research*. 11: 477-483.

Raef, S. F., Characklis, W. G., Kessick, M. A. and Ward, C. H. (1977b). Fate of cyanide and related compounds in aerobic microbial systems—II. Microbial degradation. *Water Research*. 11: 485-492.

Redman, A. and Santore, R. (2012). Bioavailability of cyanide and metal–cyanide mixtures to aquatic life. *Environmental Toxicology and Chemistry*. 31: 1774-1780.

Rieders F (1971). Noxious gases and vapors. I: Carbon monoxide, cyanides, methemoglobin, and sulfhemoglobin. In: DePalma JR (ed). *Drill's Pharmacology in Medicine*, 4<sup>th</sup> ed. New York, NY: McGraw-Hill Book Company, 1180-1205. As cited in ATSDR 2006.

RIVM (2000). The cyanide accident in Barskoon (Kyrgyzstan). Dutch National Institute for Public Health and Environment. Report Number 609026 001. Written by Cleven, RFML and van Bruggen, M. Bilthoven, The Netherlands.  
<http://www.rivm.nl/dsresource?objectid=rivmp:16806&type=org&disposition=inline>.

Rocha-e-Silva, R. C., Cordeiro, L. A. V. and Soto-Blanco, B. (2010). Cyanide toxicity and interference with diet selection in quail (*Coturnix coturnix*). *Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology*. 151: 294-297.

Rubo A, Dickmann A, Gos S (2000). Laboratory simulation of HCN emissions from tailings ponds. *Tailings and Mine Waste '00*: 307-313. As cited in ECETOC 2007.

Sakurai T (1989). Toxic gas tests with several pure and mixed gases using mice. *J. Fire Sci*. 7:22-77. As cited in NRC 2002.

Schulz, V., Bonn, R. and Kindler, J. (1979). Kinetics of elimination of thiocyanate in 7 healthy subjects and in 8 subjects with renal failure. *Klinische Wochenschrift*. 57: 243-247. Abstract only.

Smyth HF, Carpenter CP, Weil CS, et al (1969). Range-finding toxicity data: List VII. *Am Ind Hyg Assoc J* 30:470-476. As cited in ATSDR 2006.



Surleva, A. R., Bancila, S. and Todorova, E. V. (2014). A study on ninhydrin reaction with weak acid dissociable cyanide and its application for toxic cyanide determination. *Science Journal of Analytical Chemistry*. 2: 1-6.

Stence M, Beavers JB, Jaber M (1993a). Sodium cyanide: an LC50 study with the mallard using water borne exposure. Unpublished report HLO 481-93 amended 17 June 1993, project 112-306 by Wildlife International, Easton Massachusetts, USA. Du Pont de Nemours, Haskell Laboratory, Wilmington Delaware, USA; Degussa, Ridgefield New Jersey, USA; FMC, Princeton New Jersey, USA; Cyanco, Salt Lake City Utah USA; ICI (Americas), Wilmington, Delaware, USA. As cited in ECETOC 2007.

Stence M, Beavers JB, Jaber M (1993b). Sodium cyanide: an LC50 study with the northern bobwhite using water borne exposure. Unpublished report HLO 480-93 amended 17 June 1993, project 112-305A by Wildlife International, Easton Massachusetts, USA. Du Pont de Nemours, Haskell Laboratory, Wilmington Delaware, USA; Degussa, Ridgefield New Jersey, USA; FMC, Princeton New Jersey, USA; Cyanco, Salt Lake City Utah USA; ICI (Americas), Wilmington, Delaware, USA. As cited in ECETOC 2007.

Sterner, R. (1979). Effects of sodium cyanide and diphacinone in coyotes (*Canis latrans*): Applications as predacides in livestock toxic collars. *Bulletin of Environmental Contamination and Toxicology*. 23: 211-217.

Trapp, W. G. (1970). Massive cyanide poisoning with recovery: a Boxing-day story. *Canadian Medical Association Journal*. 102: 517.

Trapp, S. A. J. and Christiansen, H. (2004). Phytoremediation of Cyanide-Polluted Soils. In: *Phytoremediation*. John Wiley & Sons, Inc. 829-862.

US EPA (2010). Toxicological review of hydrogen cyanide and cyanide salts. In support of Summary Information on the Integrated Risk Information System (IRIS). United States Environmental Protection Agency. EPA/635/R-08/016F. <http://www.epa.gov/iris/toxreviews/0060tr.pdf>.

Valade, M.P. (1952). Central nervous system lesions in chronic experimental poisoning with gaseous hydrocyanic acid. (in French). *Bull Acad Natl Med (Paris)*. 136: 280-285. As cited in ATSDR 2006.

Vernot, E. H., MacEwen, J. D., Haun, C. C. and Kinkead, E. R. (1977). Acute toxicity and skin corrosion data for some organic and inorganic compounds and aqueous solutions. *Toxicology and Applied Pharmacology*. 42: 417-423.

Weedon FR, Hartzell A, and Setterstrom C (1940). Toxicity of ammonia, chlorine, hydrogen cyanide, hydrogen sulphide and sulfur dioxide gases. *V. Animals Contrib. Boyce Thompson Inst*. 11:365-385. As cited in NRC 2002.

WHO (1997). Poisons Information Monograph 003: Cyanides. World Health Organization, International Programme on Chemical Safety. <http://www.inchem.org/documents/pims/chemical/pimg003.htm>.

WHO (2004). Concise International Chemical Assessment Document 61: Hydrogen cyanide and cyanides: human health aspects. World Health Organization, Geneva. <http://www.inchem.org/documents/cicads/cicads/cicad61.htm>.

WHO (2009). Cyanide in drinking-water. Background document for development of WHO Guidelines for Drinking-water Quality. World Health Organization. WHO/HSE/WSH/09.01/3.



WHO (2011). Guidelines for drinking-water quality. Fourth edition. World Health Organization. Geneva. [http://whqlibdoc.who.int/publications/2011/9789241548151\\_eng.pdf](http://whqlibdoc.who.int/publications/2011/9789241548151_eng.pdf).

Woldemeskel, M. and Styer, E. L. (2010). Feeding behavior-related toxicity due to *Nandina domestica* in cedar waxwings (*Bombycilla cedrorum*). *Veterinary Medicine International*. 818159.



## Appendix A: Derivation of human toxicity reference values

### A1.1 Acute exposure

In 2008 Food Standards Australia New Zealand (FSANZ) conducted a hazard assessment on the cyanogenic glycoside linamarin (the predominant cyanogenic glycoside in cassava). The agency established an acute reference dose (ARfD) based on a NOAEL of 70 mg/kg body weight for clinical signs of cyanide toxicity in hamster dams in a developmental toxicity study following a single dose of linamarin (Frakes et al. 1985)<sup>30</sup>. The agency applied an uncertainty factor of 100 (10x for interspecies extrapolation, 10x for human variability) to give a human ARfD for linamarin of 0.7 mg/kg body weight. The units were converted to an equivalent dose of HCN (i.e. 80 µg HCN/kg bw), the toxic species and the compound measured in analytical assays of cassava (FSANZ 2014).

In 2011, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) re-evaluated the public health implications of cyanogenic glycosides and their derivatives in food (JECFA 2011). Benchmark dose (BMD) modelling was performed on dose-response data from the same study used by FSANZ to establish the ARfD (Frakes et al. 1985). However, JECFA used skeletal defects in the foetuses as the endpoint for modelling. The lower limit of the BMD for a 10% response (BMDL<sub>10</sub>) for linamarin was 85 mg/kg for an increased incidence of skeletal defects in developing hamster foetuses following acute exposure of maternal animals. JECFA applied an uncertainty factor of 100 (presumably 10x for interspecies extrapolation, 10x for human variability) to derive an ARfD for linamarin of 0.9 mg/kg bw (equivalent to 90 µg HCN/kg bw).

Because linamarin was only toxic to foetuses at maternally toxic doses in the Frakes et al (1985) study, and maternal toxicity occurred at lower doses than foetal toxicity, the NOAEL chosen by FSANZ (2014) is more appropriate for calculation of an acute reference dose.

---

<sup>30</sup> In the study, a single linamarin dose of 0, 70, 100, 120 or 140 mg/kg bw was administered by oral gavage to pregnant hamsters on day 8 of gestation, with the foetuses removed on gestation day 15 and examined for internal and external malformations. The top three doses produced signs of cyanide toxicity in a large percentage of dams, with signs appearing within 1 hour of dosing including dyspnea, hyperpnea, ataxia, tremors and hypothermia. Three dams (2 dosed with 140 mg/kg, 1 dosed with 120 mg/kg) died within 2 hours of dosing. Although linamarin was overtly toxic to dams, it had little or no effect on the prenatal survival and growth of foetuses. Linamarin also had no effect on ossification of foetal skeletons or litter size. The top two doses resulted in an increased incidence (statistically significant) of vertebral and rib anomalies (e.g. missing presacral vertebrae, fusion of ribs, etc) as well as the production of encephaloceles (not statistically significant) in offspring. The relationship between maternal intoxication and the production of malformed foetuses in the high dosage groups was examined by the authors (Frakes et al 1985). Out of 13 dams showing signs of intoxication, 11 (85%) produced litters containing abnormal foetuses. In contrast, out of 15 healthy dams, only two (13%) produced an abnormal foetus. The authors conclude that linamarin was only teratogenic at maternally toxic doses.



However, as the rate of release of CN<sup>-</sup> from linamarin ingested in cassava is likely to be different from the rate of release of CN<sup>-</sup> from metallo-complexed WAD cyanide, the applicability of the ARfD for linamarin to Dargues Mine is uncertain.

### A1.2 Subchronic exposure

ATSDR (2006) derived an intermediate-duration (15-364 days) minimal risk level (MRL) of **0.05 mg CN<sup>-</sup>/kg/d** from the No Observed Adverse Effect Level (NOAEL) of 4.5 mg/kg/d in the NTP (1993) rat 13 week oral study with sodium cyanide<sup>31</sup> by applying an uncertainty factor of 100 (10x for interspecies extrapolation, 10x for human variability).

The same point of departure was used by the World Health Organisation (WHO 2009, 2011) to derive a drinking water quality guideline for cyanide. Using the NOAEL of 4.5 mg/kg/d from the NTP (1993) study and applying an uncertainty factor of 100, they derived a tolerable daily intake (TDI) for a short-term exposure (i.e. <5 days) of 0.045 mg/kg body weight/d (i.e. materially the same as the ATSDR intermediate duration MRL). Assuming a 60 kg adult drinking 2 litres of water per day with an allocation of 40% of the TDI to drinking water, WHO (2009, 2011) derived a health-based drinking water guideline of 0.5 mg/L (rounded) for short-term exposure. The guideline applies to total cyanide concentration at the tap. The Australian National Health and Medical Research Council (NHMRC) also derived a drinking water guideline for cyanide (NHMRC 2013). The guideline is 0.08 mg/L and was based on a different study. Since NHMRC have not defined an acute or intermediate exposure timeframe to which the guideline applies, it is assumed it applies to chronic exposure. The derivation of the NHMRC guideline is therefore described in A1.3.

### A1.3 Chronic exposure

In their re-evaluation of the public health implications of cyanogenic glycosides and their derivatives in food, JECFA established a provisional maximum tolerable daily intake (PMTDI) of 20 µg CN/kg/d

<sup>31</sup> NTP (1993) administered NaCN in drinking water for 13 weeks to rats at concentrations of 0, 0.16, 0.48, 1.4, 4.5 and 12.5 mg CN/kg/day in male rats; and 0, 0.16, 0.53, 1.7, 4.9 and 12.5 mg/kg/d in female rats. A statistically significant decrease in cauda epididymis weight (7%) was seen at doses  $\geq 1.4$  mg/kg/day. A 7% decrease in whole epididymis weight (as compared to cauda epididymis) was seen at 12.5 mg/kg/d. At the highest dose tested (12.5 mg/kg/d), epididymis and cauda epididymis weights were decreased by 7 and 13%, respectively. Dose-related decreases in testis weight (8%), number of spermatid heads (14%), and spermatid concentration (14%) were also found to be significant at doses  $\geq 12.5$  mg/kg/d. A statistically significant decrease in epididymal sperm motility was observed at doses  $\geq 1.4$  mg/kg/d, although it did not appear to increase in severity with dose. US EPA (2010) considered 1.4 mg/kg/d a Low Observed Adverse Effect Level (LOAEL), in contrast to ATSDR (2006), who considered 4.5 mg/kg/d to be a NOAEL. ATSDR (2006) considered the reductions in cauda epididymis weights observed at 1.4 and 4.5 mg/kg/d were not biologically significant in the absence of any other significant effect. They also considered the small (<4%) statistically significant, but not dose-related, reductions in sperm motility in the 1.4, 4.5 and 12.5 mg/kg/d groups were within the range of normal values and not biologically significant (ATSDR 2006). DEFRA (2002) came to the same conclusions as ATSDR (2006).



(JECFA 2011). This value was based on a BMDL<sub>10</sub> of 1.9 mg/kg/d for reduced absolute cauda epididymis (in testes) weights from the 13-week NTP drinking water study in rats with sodium cyanide (NTP 1993) and application of an uncertainty factor of 100 (presumably 10x for interspecies extrapolation, 10x for human variability). JECFA deemed it unnecessary to apply an additional uncertainty factor to account for the absence of a long-term study, considering the acute nature of cyanide toxicity and the sensitivity of the effect on which the PMTDI is based.

US EPA (2010) derived a chronic reference dose (RfD) for cyanide. They applied BMD modelling to data from the 13-week NTP (1993) study (see Section 4.3.1) for decreased cauda and whole epididymis weights, decreased testes weight and altered sperm parameters. US EPA (2010) selected the 95% lower confidence limit (BMDL) associated with a 1 standard deviation (SD) decrease in cauda epididymis weight in rats of 1.9 mg/kg/d as the point of departure for derivation of an RfD. It is noteworthy that this is the same value as the BMDL used by JECFA (2011) to derive the PMTDI. However, unlike JECFA (2011) who applied an uncertainty factor of 100, the US EPA (2010) applied a factor of 3,000 (10x for interspecies extrapolation, 10x for human variability, 10x for use of a subchronic study, and 3x for deficiencies in the database) to the point of departure to derive an RfD of 0.0006 mg/kg/d (i.e. 0.6 µg/kg/d).

The National Health and Medical Research Council (NHMRC 2013) used the results of a 6-month feeding study in juvenile pigs (Jackson 1988) to derive a drinking water quality guideline for cyanide. Jackson (1988) orally administered aqueous solutions of inorganic cyanide (KCN) via gavage at doses of 0, 0.4, 0.7, or 1.2 mg CN<sup>-</sup>/kg body weight daily for 24 weeks (~6 months) to juvenile swine. Timed, daily behavioural determinations were made on a range of performance measures and learning events. Cyanide treatments were associated with a significant decrease in dominance behaviour, decreased vocalisation, decreased fighting, increased victimisation, decreased swine investigations of new environments, decreased aggressive feeding patterns, increased distractibility from eating, decreased rooting and water overturning, decreased pica, increased anaesthesia recovery time, increased limping and limb stiffness, and increased vomiting and shivering. The author does not provide a No Effect Level or a NOAEL in the paper (Jackson 1988). Although some of these effects were seen at all dose levels, NHMRC (2013) have used the top dose (1.2 mg/kg/d) in the study as a no-effect level (NOEL) without providing a rationale for this choice. Using this dose combined with a 70 kg assumed body weight, a 2L/day water consumption rate, an uncertainty factor of 100 (10x for interspecies extrapolation, 10x for human variability), and an assumption of 20% of total intake being derived from drinking water, NHMRC (2013) derived a drinking water guideline of 0.08 mg/L for cyanide (presumably to be measured as total, although this is not explicitly stated). US EPA (2010) indicated the biological significance of the behavioural changes observed in the Jackson (1988) study is unclear. In addition, they considered the utility of the study limited for guideline



development due to the use of bolus dosing (US EPA 2010). WHO (2004) came to a similar conclusion with respect to the Jackson (1988) study, citing small numbers of animals and limited statistical analysis as further reasons for the study's limited utility.

This page has intentionally been left blank



ABN: 55 158 303 167

PO Box 316

Darling South, VIC 3145

Tel: 03 9569 3918/ 03 9572 1448

Fax: 03 9563 5330

# Assessment of Ecological and Human Health Impacts Associated with the use of Cyanide at Dargues Gold Mine

Prepared by: Roger Drew, PhD, DABT  
Tarah Hagen, MSc  
ToxConsult Pty Ltd.

Prepared for: Big Island Mining Ltd

ToxConsult document ToxCR051114-RTF2  
June 2015

.....  
**Roger Drew, PhD, DABT, FACTRA**  
(Diplomate American Board of Toxicology)

.....  
**Tarah Hagen, MSc, RACTRA**  
(Environmental Toxicology)



## Document history

Report No.	Date issued	Prepared by	Reviewed by	Document/Revision type
ToxCR051114-RTd1	23/01/2015	R Drew T Hagen	T Hagen R Drew	Original draft for comment
ToxCR051114-RTd2	28/01/2015	R Drew T Hagen	T Hagen R Drew	Draft with additional sections for comment
ToxCR051114-RTF	30/01/2015	R Drew T Hagen	T Hagen R Drew	Final addressing received comments
ToxCR051114-RTF2 (Draft)	07/05/2015	R Drew T Hagen	T Hagen R Drew	Final incorporating new dilution modelling (sent as draft for comment prior to finalisation)
ToxCR051114-RTF2 (Final)	03/06/2015	R Drew T Hagen	T Hagen R Drew	Final incorporating new dilution modelling

## Distribution of Copies

Report No.	Issued to	Sent by	Mode of issue
ToxCR051114-RTd1	J Dornan, Big Island Mining	R Drew	Word document with comments
ToxCR051114-RTd2	J Dornan, Big Island Mining	R Drew	Word document with comments
ToxCR051114-RTF	J Dornan, Big Island Mining	R Drew	pdf via email
ToxCR051114-RTF2 (Draft)	J Dornan, Big Island Mining M Bland, RW Corkery & Co	T Hagen	Word document with track changes & comments
ToxCR051114-RTF2 (Final)	J Dornan, Big Island Mining M Bland, RW Corkery & Co	T Hagen	pdf via email



## Disclaimer

This report was prepared by ToxConsult Pty Ltd as an account of work for Big Island Mining Pty Ltd (the 'Client'). This report should be read, and used in its entirety. The material in it reflects ToxConsult's best judgement in the light of the information available to it at the time of preparation. However, as ToxConsult cannot control the conditions under which this report may be used, ToxConsult will not be responsible for damages of any nature resulting from use of or reliance upon this report. ToxConsult's responsibility for the information herein is subject to the terms of engagement with the client. Information provided by the client has been used in good faith; ToxConsult has not, and was not required to, verify its veracity.

Copyright and any other Intellectual Property associated with this report belongs to ToxConsult Pty Ltd and may not be reproduced in any form without the written consent of ToxConsult. The Client, and only the client, is granted an exclusive licence for the use of the report for the purposes described in the report.

## About ToxConsult Pty Ltd

### About the authors:

#### Dr Drew

Dr Roger Drew is one of the principal consultants of ToxConsult Pty Ltd. He has primary degrees in biochemistry and pharmacology and postgraduate degrees in toxicology. Postdoctoral training was undertaken at the National Institutes of Health, National Cancer Institute in the USA. He has more than 40 years of toxicological and risk assessment experience in academia, industry and consulting. He has served on advisory committees and task forces for Australian Health Departments and the World Health Organization. He has provided advice to a range of industries and Government authorities and has significantly participated in developing risk assessment practice in Australia. Dr Drew is one of just a few toxicologists in Australia certified by the American Board of Toxicology.

Dr Drew is also Adjunct Associate Professor in the Department of Epidemiology and Preventive Medicine, Monash University and teaches various aspects of toxicology and risk assessment to undergraduate and postgraduate students at local Universities. He is a member of professional toxicology societies and is a recognised national and international expert in toxicology and risk assessment. He is currently on the editorial board of the international scientific journal "Regulatory Toxicology and Pharmacology".

#### Ms Hagen

Tarah Hagen is a director and senior consultant at ToxConsult Pty Ltd providing ecotoxicology and risk assessment services to a broad range of industries and government bodies. She has a Masters degree in Environmental Toxicology and Pollution Monitoring, an honours degree in ecotoxicology, and a degree in Applied Science (Biological Sciences).



## Executive Summary

Big Island Mining Ltd (BIM) is proposing to use Carbon In Leach (CIL) processing at the Dargues Gold Mine to extract gold and silver from mined ore. The CIL method relies on complexing the precious metals with cyanide, a process that has been used internationally for many decades. This report is a risk assessment focussed on impacts that may be associated with cyanide containing material discharged into the tailings storage facility (TSF) to wildlife and people living near the mine.

Although a small amount of free cyanide ( $\text{CN}^-$ ) may be present in the discharged effluent, the cyanide is mostly complexed with various metals. The biologically active form is the fraction of total cyanide which can dissociate from the metal complexes in mild acid conditions and release the biologically active  $\text{CN}^-$ . This is called the weak acid dissociable cyanide (WAD). The most prominent form of cyanide in WAD is copper cyanide. Copper cyanide is markedly less toxic than sodium cyanide, the cyanide form ubiquitously used in toxicology investigations to identify hazards to organisms.

Prior to being discharged into the TSF, effluent from the CIL is passed through a cyanide destruction plant so acceptable concentrations of WAD are discharged. International guidelines and many publications in the scientific literature indicate a WAD cyanide of 50 mg/L measured at the spigot discharge of the TSF protects wildlife from cyanosis induced mortality. This is supported by observations undertaken at TSFs. However recent regulatory approved operation licences in NSW have stipulated WAD at the discharge spigots has to be less than 30 mg/L all the time, and less than 20 mg/L for 90% of the time.

This risk assessment has been conducted assuming a TSF water WAD of 30 mg/L. In reality, to meet the 20 mg/L requirement, WAD levels at the discharge point will need to be 10 – 15 mg/L most of the time. In addition WAD in the pond of the TSF, i.e. the TSF water, is usually somewhat less than that discharged at the spigot. This is because further chemical reactions occur to destroy the cyanide. Also once in TSF water, the WAD dissociates to  $\text{CN}^-$  which very quickly reacts with hydrogen ions ( $\text{H}^+$ ) in the water. The hydrogen cyanide (HCN) formed evaporates from the water. This is the major way cyanide is removed from surface water in the environment.

In eco-toxicological risk assessments the major objective is species population and ecosystem viability protection rather than protecting each individual animal from harm. Cyanide does not bioaccumulate in organisms because it is efficiently metabolised in the body to non-toxic compounds. It has very steep dose-response profiles for the effects it produces, and consequently a well-defined threshold exposure level exists that is not associated with adverse effects. It is acute exposure to cyanide that presents greatest risk to wildlife. Since birds and mammals readily and fully recover from



the non-lethal effects associated with acute oral or inhalation cyanide exposure the effect of most concern is mortality. Thus this assessment focuses on the risk of bird and bat mortality should TSF water be drunk. This is consistent with the risk assessments undertaken by competent authorities.

Only birds and bats are considered in the risk assessment because a large fence which extends underground will be built around the TSF. A small mesh size will be incorporated into the bottom of the fence to exclude small animals and amphibians. Thus the only vertebrate wildlife likely to interact with cyanide-bearing tailings at this TSF are flying birds and bats. An assessment of the scientific literature indicates it is a reasonable assumption these species may visit gold mine TSFs.

The theoretical ways which birds may absorb cyanide into their bodies is breathing HCN that is in the air above the TSF, absorbing HCN from the water through their skin when swimming or wading, or from their gastrointestinal tract after drinking the water. Authorities consider inhalation and dermal absorption to be negligible and do not evaluate exposures by these routes. Ingestion is the exposure route of greatest importance and the one of concern. Consistent with the authority deliberations this risk assessment evaluates the risk of mortality to birds should they drink TSF water. Because bats may spend long periods consistently flying through the air above TSF ponds inhalation has been additionally assessed for bats.

#### *Assessment methodology:*

The methodology followed in the risk assessment broadly follows Australian and international guidelines. Estimates of cyanide intake, per drinking event, from the WAD cyanide in TSF water are compared with cyanide intake that does not cause mortality as determined from experiments with sodium cyanide. This is conservative because WAD cyanide is less toxic than sodium cyanide. The dose causing no lethality is the no observed effect level, and is designated as  $NOEL_{mortality}$ . The ratio of the WAD cyanide intake to the  $NOEL_{mortality}$  is an indication of the safety gap and is called the margin of exposure ( $MoE_{mortality}$ ).

#### *Assessment of birds:*

In the risk assessment for birds the  $NOEL_{mortality}$  is derived from experiments with the most sensitive bird tested (mallard duck). It follows that if impacts to this avian species are minimal then other birds are also protected. The calculated  $MoE_{mortality}$  was approximately 2 for duck species observed to be in the mine lease area. On face value this suggests bird populations are adequately protected from mortality if they drink TSF water containing a WAD cyanide concentration of 30 mg/L.



*Assessment of bats:*

As bats search for insects above the TSF (if any insects are present) or make drinking passes over the water they may be exposed by inhalation to HCN volatilising from the TSF water surface. It is noted WAD concentrations greater than approximately 5 mg/L are not conducive for insect breeding. The concentration of HCN above TSF water was estimated using a volatilisation factor for HCN and assuming relatively calm wind conditions. There is very little toxicological data available for bats, only one oral study. Because rats are more sensitive to orally administered sodium cyanide than are bats, inhalation dose-response information from rats was used as a surrogate for bats. With the conditions assumed it was determined bat mortality would be less than 1%, probably much less.

The only data available for effects of cyanide in rats after oral exposure was the dose required to kill 50% of the test population (i.e. an LD<sub>50</sub>). The NOEL<sub>mortality</sub> associated with this LD<sub>50</sub> was estimated by applying the slope of the dose response for birds and other mammals as an adjustment factor. The MoE<sub>mortality</sub> for various bat species observed to be in the mine lease area was calculated to be approximately 3. As for birds, this suggests bat populations are adequately protected from mortality if they drink TSF water containing a WAD cyanide concentration of 30 mg/L.

*Discussion and uncertainties:*

Unfortunately the amount of toxicological or exposure information directly applicable to this risk assessment was limited. Therefore a number of exposure assumptions and toxicological extrapolations have been made. There is uncertainty associated with each of these. While an attempt has been made to be conservative, i.e. err on the side of safety, the conservatism built into the assessment is not excessive.

A MoE<sub>mortality</sub> of 2 -3 is relatively small. While, given the steepness of the mortality dose response, such values indicate population protection, they also suggest a possibility some individual birds or bats may die if they visit the TSF and drink more water than has been assumed in the risk assessment. However as noted above, WAD cyanide is less toxic than sodium cyanide and no, or only few bird deaths have been observed around TSFs which have WAD <50 mg/L. In addition the assessment has assumed a WAD of 30 mg/L, in reality to be able to meet a regulatory target of 20 mg WAD/L 90% of the time, the WAD is likely to be 10 – 15 mg/L, or lower.

The inhalation exposure assessment for bats has much uncertainty which is primarily associated with the assumed volatilisation factor of HCN from the water. This is balanced by significant conservatism in the assumed concentration of free CN<sup>-</sup> in the effluent discharged to the TSF. Due to lack of data for free CN<sup>-</sup> in the discharged effluent from the destruction plant, the concentration was assumed to be the same as in CIL effluent prior to entering the plant.



*Off-site risk to aquatic organisms and downstream humans*

Cyanide exposure to organisms in groundwater or surface water and to humans downstream from the Mine TSF may potentially occur if integrity of the infrastructure handling cyanide containing materials at the site fails or management/safety procedures are not followed. Failures include pipework leakage, TSF seepage, the TSF overtopping, and catastrophic failure of the TSF embankment. A wide range of safety devices will be incorporated into the designs of the gold recovery process and handling of tailings. These are briefly discussed in Sections 2 and 9. The engineering safe guards make off-site exposures negligible. Since in most of these remote exposure circumstances it is not possible to enumerate the concentrations of biologically available cyanide in creek water, quantitative assessment of risk has not been undertaken. That there is negligible risk relies on infrastructure reliability, its appropriate maintenance, and the operational success of safety systems.

The TSF has been designed to withstand earthquake and extreme storm events. To protect the integrity of the TSF embankment wall a drainage ditch around the TSF will direct rain runoff from the surrounding hills away from the TSF. Only rain falling onto the TSF surface will enter the TSF. A spillway is also incorporated into the design. In the event that the spillway runs, i.e. the TSF overtops, TSF water will be much diluted by the rain and will be running into very swollen, flooded creeks. The overall dilution of biologically important cyanide will be large. Dilution modelling of various rare rainfall events undertaken by Knight Piésold at the first point of release from the spillway (Spring Creek) and further downstream indicates potential TSF water dilutions of the order of approximately 320-303,800 times depending on the location in the receiving water system. Since ecological assessments have found the upper reaches of Spring Creek to be moderately to heavily disturbed, the ANZECC (2000) 95% species protection level of 7 µg CN/L (i.e. for slightly-moderately disturbed systems) is appropriate to judge the potential impacts on aquatic organisms in Spring Creek.

Taking into account the modelled dilutions, and assuming 30 mg WAD/L in TSF water, in the event of an overflow the estimated free cyanide concentrations in Spring Creek at the point the TSF overflow enters the creek could be 0.6-33 µg/L, with the maximum estimated concentration being about 5 times higher than the 95% species protection level of 7 µg CN/L. Therefore at the assumed concentration of 30 mg/L WAD in the TSF, it is concluded at Spring Creek there is a potential risk of adverse impacts to aquatic organisms due to the cyanide under certain rainfall scenarios. The risk considerably decreases with higher dilutions downstream from the confluence of Major and Spring Creeks. At these latter locations, predicted concentrations of free cyanide are below the 95% trigger value of 7 µg/L and at or below the 99% protection trigger value of 4 µg/L for pristine waterways. To ensure negligible risk to aquatic organisms in Spring Creek and compliance with a target free cyanide concentration at or below 7 µg/L in Spring Creek, the concentration of WAD cyanide in the TSF pond



would need to be less than or equal to 6.3 mg/L when the TSF water dilution is at the predicted minimum of just 316 times. This worst case (i.e. minimum) dilution coincides with the worst case (i.e. minimum) capacity of the TSF to accept stormwater before overflowing down the spillway. Since TSF capacity to accept stormwater increases as new lifts are added, higher concentrations of WAD cyanide in the TSF at these times may be acceptable for meeting the ANZECC trigger concentrations in downstream receiving waters. This is because the extra TSF freeboard volume increases dilution of TSF water prior to its overflow into Spring Creek.

The modelled dilutions indicate cyanide concentrations in receiving water will be less than the World Health Organization and Australian drinking water standards, therefore risks to human health from drinking cyanide in the water are low.

*Conclusions:*

Taking into account the literature information and the uncertainties in the assessment assumptions it is concluded a target WAD concentration of  $\leq 30$  mg/L would be protective of bird and bat populations around the Dargues mine TSF. However there is a possibility of mortality for individual animals. This risk is low when it is recognised that in order to meet regulatory limits of  $\leq 30$  mg/L all the time and  $\leq 20$  mg/L 90% of the time, the actual concentration of WAD in TSF water will likely be less than 10 – 15 mg/L.

There is a potential risk for adverse impacts to aquatic organisms in nearby surface water. This risk is negligible if WAD cyanide levels in the TSF pond are maintained at levels less than or equal to 6.3mg/L. There is negligible risk to persons using creek water downstream from the mine. Nevertheless as part of its water quality monitoring program the Mine will be monitoring cyanide concentrations in the immediate creeks.



## Contents

Executive Summary .....	4
Contents .....	9
1. Introduction & Scope.....	11
2. Discharges to TSF .....	11
2.1 Process considerations .....	11
2.2 Cyanide destruction in CIL effluent.....	12
2.3 TSF design.....	14
3. Species at risk.....	14
3.1 Terrestrial animals.....	15
3.2 Birds and bats .....	15
3.2.1 Historical aspects.....	15
3.2.2 Bird species at Dargues mine area .....	17
3.2.3 Bat species at Dargues mine area .....	18
3.3 Species sensitivity .....	18
4. Concentration of cyanide(s) in Mine TSF .....	21
5. Important cyanide concentrations.....	23
5.1 ICMI (2009, 2012).....	23
5.2 ECETOC (2007) .....	24
5.3 NICNAS (2010) .....	25
5.4 Licence conditions.....	26
5.5 Presumed WAD target at Dargues Mine .....	26
6. Risk assessment methodology.....	27
6.1 Overview .....	27
7. Bird risk assessment.....	30
7.1 Bird visitations to TSF.....	30
7.2 Bird inhalation assessment.....	30
7.3 Bird oral assessment.....	30
7.3.1 Potential secondary poisoning of birds.....	32
7.3.2 Bird oral assessment uncertainties.....	33
7.3.3 Conclusion .....	35
8. Bat risk assessment.....	36
8.1 Bat visitations to TSF.....	36
8.2 Bat inhalation exposure .....	37
8.2.1 Bat inhalation assessment uncertainties .....	43
8.2.2 Conclusion .....	45
8.3 Bat oral exposure .....	45
8.3.1 Bat oral assessment uncertainties.....	50
8.3.2 Conclusion .....	50
9. Off-site risk assessments to cyanide containing water .....	50
9.1 Exposure scenarios.....	50
9.1.1 Pipework leakage.....	51
9.1.2 TSF seepage .....	51
9.1.3 TSF overtopping .....	52



9.1.4 Catastrophic TSF embankment wall failure .....	57
References .....	58
Appendix A: Birds identified at the Mine .....	62
Appendix B: Bats identified at the Mine.....	65



## 1. Introduction & Scope

Big Island Mining Ltd (BIM), a subsidiary of Unity Mining Limited, owns and operates the Dargues Gold Mine (the mine) at Majors Creek, NSW. The mine is located approximately 60 km southeast of Canberra, 13 km south of Braidwood and immediately north of the village of Majors Creek. It was originally intended the mine, which is predicted to have a 5-year life, would transport gold concentrate to a distant site for processing. However, following a series of optimisation studies and technical reviews BIM intends to submit a modification to its project approval to allow the use of Carbon In Leach (CIL) processing at the Dargues Gold Mine. In CIL processing, cyanide is used to dissolve gold from the crushed ore, and the resulting cyanide-gold solution adsorbed onto carbon. The gold is then retrieved from the carbon by electrolysis. Alternative methods to CIL for leaching gold from ore are less effective. According to the Australian Government Department of Resources Energy and Tourism (DRET), cyanide remains the best industry option for safe and economic extraction of gold (DRET 2008).

Unity Mining (on behalf of BIM) has requested ToxConsult to consider the hazards, and assess the health risks associated with the use of cyanide to wildlife and people living near the mine.

## 2. Discharges to TSF

### 2.1 Process considerations

Cyanide has been used since the late 1800's across the world and in Australia to extract gold from mineral ores. About 80% of the world's gold production uses a cyanide extraction process (DRET 2008, MERG 2001). In the gold mining industry it is used in a variety of forms. These include sodium cyanide (as briquettes or liquid) and calcium cyanide (flake or liquid). At the mine it is intended to have the cyanide transported as small briquettes (made of sodium cyanide and caustic soda) inside specially designed and externally reinforced road tankers, once at the site the cyanide will be solubilised by addition of water. The pH of the solution is >9.5. It is then directly pumped into a storage tank within the processing plant. The tanker, storage tank, and pumping equipment are all on a bunded concrete pad to ensure any unlikely spillage is contained and able to be recovered to put into the storage tank.

The above method of delivering, and preparing the cyanide for use, avoids the hazards and risks associated with storage of dry cyanide on site, and storage and disposal of containers that once had cyanide in them. The tank in which the cyanide is delivered is known as an isotainer and is constructed from 7mm plate steel so in the unlikely advent of an accident or rollover the container is



protected. Additionally should the container be split in an accident the cyanide will not flow out because it is in briquette form.

There are two primary ways of using cyanide to extract gold from mineral ore. Most cyanide environmental impacts seem to be associated with the 'heap leachate' process (Donato et al. 2007, ECETOC 2007, Eisler and Wiemeyer 2004). In this method, alkaline cyanide solutions are sprayed onto the tops of large heaps of finely crushed ore which are in the open. The cyanide solution is allowed to percolate through the heap to complex and dissolve (leach) the gold. This gold-cyanide solution is then collected for further treatment. In this process there can be puddles of concentrated cyanide solution on top, or around the heaps, to which birds or bats have easy access. At the Dargues Mine, a carbon-in-leach extraction method will be used in which leaching is done in a series of open tanks within the processing plant. There is no access by wildlife to concentrated cyanide solutions.

Figure 2.1 summarises the inputs into the tailings storage facility (TSF). There are two effluent streams:

- The tailings from the flotation circuit ("flotation tail").
- The tailings from the concentrate circuit ("concentrate tail") which includes the CIL process.

## 2.2 Cyanide destruction in CIL effluent

Cyanide is used in the CIL plant, therefore only the "concentrate tail" contains cyanide. The effluent from the CIL circuit passes through a 'detoxification' plant where weak acid dissociating (WAD) cyanide is destroyed down to acceptable concentrations (see Section 5).

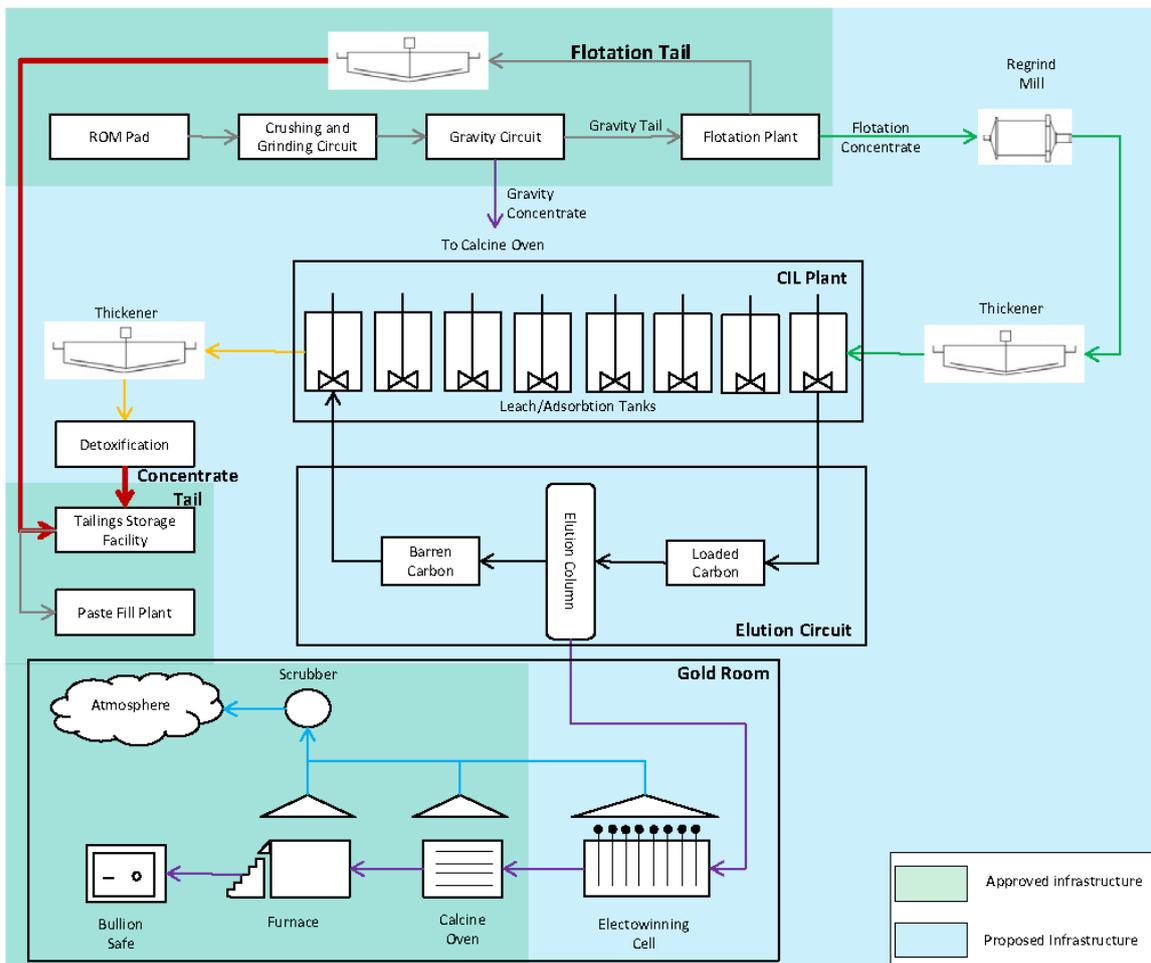
The effluent discharge to the TSF will either be undiluted output from the detoxification unit (the detoxified concentrate tail), or the detoxification effluent diluted approximately ten times with the flotation tail. In this risk assessment it has been assumed there will be no dilution of the detoxification effluent with flotation tailings.

Sodium metabisulphite ( $\text{Na}_2\text{S}_2\text{O}_5$ ) is added to the detoxification unit to provide a source of sulphite for conversion of cyanides (including cyanides weakly complexed with metal ions) to cyanate. The chemical reaction requires air and copper as a catalyst. Copper is only added if it is not already present in adequate amounts in the concentrate tails. The overall reaction is:





Iron complexed cyanides are reduced to the ferrous state and precipitated as insoluble metal-iron-cyanide complexes (Hewitt et al undated, SGS 2005). Residual metals liberated from the WAD cyanide complexes are precipitated as their hydroxides (Hewitt et al undated). The extent of cyanide destruction is dependent on the stoichiometry addition of sulphite relative to WAD in the CIL effluent (CSIRO 2014).



**Figure 2.1: Process flow diagram at the Mine**

Inputs into the TSF (flotation and concentrate tails) are shown by the bolded red arrows. The flotation tails do not contain cyanide.

Adapted from RWC (2015), Figure 8.



### 2.3 TSF design

The TSF will be a cross-valley storage facility with a zoned embankment. Tailings will be discharged via spigots spaced at regular intervals along the embankment crest in order to maximise tailings density and evaporation of water. Tailings deposition will occur upstream from the embankment towards the head of the valley in order to form a supernatant pond towards the north-east perimeter of the facility.

The embankment will be constructed with two zones. The 'structural zone' will provide the structural integrity of the embankment and will be constructed using compacted non-acid forming waste rock. The upstream or inner face of the embankment will be a low permeability zone, which will include a combination clay and high-density polyethylene liner to achieve an average basin permeability of  $2.0 \times 10^{-9}$  m/s to  $3.2 \times 10^{-10}$  m/s. Underdrainage and seepage collection systems will also be installed to minimise seepage and allow collection of infiltrated water at the bottom of the TSF for return to the gold processing plant.

The tailings delivery and decant return pipelines will be equipped with leakage detection and shutoff systems to limit the volume of any spillage that may occur. In addition, the pipelines will be located within a bunded corridor between the process plant and the TSF so any spillage of tailings or TSF decant water that does occur is isolated from the environment and can be collected. The capacity of the TSF will be able to withstand extreme weather, storm and earthquake events<sup>1</sup>. Around the upslope perimeter of the TSF will be constructed a clean water diversion to intercept rain runoff from the surrounding valley walls to prevent rain water ingress into the TSF. That diversion will be constructed to divert the Maximum Probable Flood rainfall event. Rain will only enter the TSF from what falls on the TSF surface. Nevertheless an emergency spillway will be constructed. The TSF is designed to cope with about 705 mm rainfall in a 72 hour period<sup>2</sup>.

### 3. Species at risk

Although wildlife at gold mine tailings storage facilities may be exposed by various routes to material containing cyanide residues, consumption of drinking water is the major route of exposure to cyanides in the TSF (NICNAS 2010, ECETOC 2007). Birds and flying mammals (e.g. bats) are anticipated to be wildlife with the highest potential for exposure.

---

<sup>1</sup> The TSF has been designed to completely contain storm events up to and including annual exceedence probabilities of 1:10,000 on top of predicted maximum average conditions during operations. Therefore, exceeding the storm storage capacity of the facility at any stage of operation is highly unlikely (KP 2011).

<sup>2</sup> Section 2.3 and 4.4 in Knight Piesold (2015).



### 3.1 Terrestrial animals

Livestock and terrestrial animals will not be able to gain direct access to TSF water because it will be surrounded by an animal-proof fence. The fence will exclude terrestrial wildlife, reptiles, and burrowing animals<sup>3</sup> and the fence will be dug below the ground surface and extend ~2 m above. A sloped overhang can further deter kangaroos from jumping over the fence. In addition a small aperture metal mesh may be included at the bottom to exclude small animals including reptiles.

### 3.2 Birds and bats

#### 3.2.1 Historical aspects

There have been incidents of migratory birds being poisoned with cyanide at heap leaching facilities or tailings ponds (MERG 2001).

Only limited published information documenting wildlife deaths from cyanosis at gold mines is available (Donato et al 2007). Observations and incident reports at cyanide containing tailings ponds where avian mortalities had been observed, indicate a weak acid dissociable (WAD) cyanide concentration > 50 mg/L was associated with bird deaths, but at sites where WAD was generally < 50 mg/L, few or no mortalities were observed (MERG 2001, NICNAS 2010, DRET 2008, NT DoME 1998, NPS 1997, Donato et al 2008, Griffiths et al 2014a, Hudson and Bouwman 2009). Some of this information has come from studies investigating bird visitations to tailings storage facilities in the Northern Territory and Western Australia (NT DoME 1998, Donato et al 1997, 2007, 2008, Donato and Smith 2007, Donato 1999). Since these were industry commissioned studies, reports containing the raw data were not available. Henny et al (1994), in a survey of 17 mines, reported bird and wildlife deaths at tailings ponds and heap leach facilities in Nevada containing concentrations of WAD of 62 mg/L, 81 mg/L, and higher, but not at those containing <50 mg/L WAD CN. In Australia's Northern Territory, Donato (1999, as cited in Donato et al 2008) found two mining operations consistently discharging tailings at concentrations <50 mg/L WAD recorded zero wildlife deaths. Another case study recorded significant deaths of a migratory bird species apparently after WAD concentrations exceeded 50 mg/L WAD (Donato et al 2008).

In Nevada, USA, 9512 carcasses of over 100 species were reported at gold mining facilities between 1986 and 1991. This was likely an underestimation due to reporting being voluntary. Birds comprised 80-91% of vertebrate carcasses reported annually (Henny et al 1994). Bats comprised 25% of 665 mammal mortalities (Henny et al 1994). Research conducted between 1980 and 1989 showed that bats and rodents were the most commonly reported mammal mortalities (34 and 35% of 519 deaths,

<sup>3</sup> Personal correspondence with Unity Mining 26<sup>th</sup> August, 2014.



respectively) at cyanide extraction gold mines in California, Nevada and Arizona (Clark and Hothem 1991, as cited in Griffiths et al 2014a).

A questionnaire published by the Minerals Council of Australia in 1996 (Minerals Council of Australia 1996, as cited in Donato et al 2007) reports that:

- 72% of tailings dams in Australia are rarely or never used by wildlife;
- 65% of gold mines in Australia recorded less than five deaths per year;
- 74% of mining operations never experienced 10 deaths in a week;
- migratory birds were not an issue to the extent reported in the USA;
- anecdotal evidence from Australian TSFs is that birds tend to come and go as the TSF offers minimal roosting habitat; and
- there are a number of cases of high WAD cyanide levels where significant bird deaths have not occurred.

Work conducted by the Northern Territory Bird Study Group between 1996 and 1998 (as cited in Donato et al 2007) documented:

- wildlife deaths occurred at 5 of 7 gold mining operations surveyed;
- at-risk wildlife species were frequently recorded at all tailings dams;
- seasonal variation and migratory patterns influenced wildlife abundance;
- 972 wildlife deaths were recorded from four mining operations in a calendar year;
- wildlife deaths were underestimated by mine staff;
- all mining operations which experienced deaths had incidents where more than 20 deaths were recorded in 1 day within one calendar year; and
- all mining operations that discharged at >50 mg/L WAD cyanide experienced wildlife deaths.

Of the birds likely to visit a TSF wading and swimming birds receive the greatest exposure, as they are likely to spend longer periods of time on or near the surface. Raptors are potentially at risk if they are attracted to carrion around the TSF (DRET 2008). Griffiths et al (2014a) found bats to be active above the TSF at a gold mine site in New South Wales, Australia.

Although animals will generally avoid tailings dams if natural water is available, it is acknowledged that some fauna may drink from such facilities. There is no reason to believe birds are able to distinguish between TSFs and any similar area of water formed from precipitation or run-off (Donato et al 2007). However, Donato et al (2008) found counts of either wildlife visitations or deaths at TSFs decreased as the surface area of the supernatant water decreased. Similarly, NT DoME (1998) indicated removal or reduction of supernatant will greatly reduce bird visitation and their exposure to



cyanide. The active decanting of supernatant to decant ponds as will be used at Dargues Mine significantly reduces supernatant size. When constructing TSFs, NT DoME (1998) recommend avoiding uneven floors so islands (favoured roosting sites) do not form as tailings are deposited. The removal of nearby vegetation can also discourage TSF visitation. Should wildlife deaths occur, carcasses near the TSF should be removed quickly so as not to attract raptors (NT DoME 1998).

Observations indicate birds do not develop a taste aversity to drinking cyanide-containing mine waste water (NICNAS 2010). However, birds and other wildlife (including bats) are unlikely to drink from hypersaline waters (>50,000 mg/L total dissolved solids) (Griffiths et al 2014b, NICNAS 2010). With hypersaline water wildlife mortalities due to cyanide do not occur, even when WAD concentrations significantly exceed 50 mg/L (NICNAS 2010, DRET 2008). For example, in one study bird and wildlife interactions with a hypersaline TSF were routinely monitored over 266 days (with an average survey time of one hour per observation) (Donato and Smith 2007). The monitoring methodologies used were consistent with previous case studies<sup>4</sup> where mine waste effluents exceeded the accepted discharge threshold for WAD. The mean concentration of WAD in the facility during the monitoring period was approximately 60 mg/L (n=114 samples), with 90% of samples between 32 and 100 mg/L WAD. Over a thousand wildlife visitations, but no cyanosis deaths were recorded<sup>5</sup>. The authors suggest factors such as hypersalinity, lack of food, or tailings system design may have deterred visiting wildlife from drinking the water or staying for long periods (Donato and Smith 2007).

### 3.2.2 Bird species at Dargues mine area

The bird species identified in the area of the Dargues mine (n=116) are provided in Appendix A. Included are birds considered to be threatened under the NSW Threatened Species Conservation Act (NSW Gov 1995).

---

<sup>4</sup> The reports describing these previous case studies were not available to ToxConsult.

<sup>5</sup> Wildlife monitoring was undertaken at cyanide-bearing water bodies 2-3 times per week. Initially, monitoring was conducted at the process water dam, however, due to low wildlife visitations, formal monitoring at this location stopped and available observational time focused on the central-thickened discharge tailings system (CTD), stormwater/decant pond and CTD ground water interception trenches. Observations were generally conducted in the morning within 3 hours of sunrise. Each monitoring session had a set 30 minute observation period. However observation times varied and were usually 40-60 minutes. During each monitoring session, the observer continually inspected the area using binoculars with 8x magnification. Any wildlife (alive or dead), species and number were recorded. Behavioural (resting, locomotion, feeding, drinking or patrolling by raptors) and habitat (supernatant open water, supernatant beach/wet tails interface, supernatant beach/dry tails interface, aerial, etc) data were also collected. A search for the presence of carcasses within the area of cyanide-bearing water bodies was also conducted. Bat presence and activity was monitored using anabat detectors, placed at the CTD supernatant, dry tails, CTD interception trenches and nearby sewerage ponds.



Ecotoxicology information in Section 3.3 identifies mallard ducks as the most sensitive of avian species tested with cyanide. Hence this risk assessment has been conducted for ducks on the basis that protection of duck populations will also result in the protection of the populations of other bird species.

### **3.2.3 Bat species at Dargues mine area**

The bat species identified in the area of the Dargues mine are provided in Appendix B. Of the 8 species of bats identified, one (the False Pipistrelle) is listed as a threatened species under the NSW Threatened Species Conservation Act (NSW Gov 1995). The inhalational and oral exposure risk to bats found in the Dargues Mine area has been assessed in Sections 8.2 and 8.3 respectively.

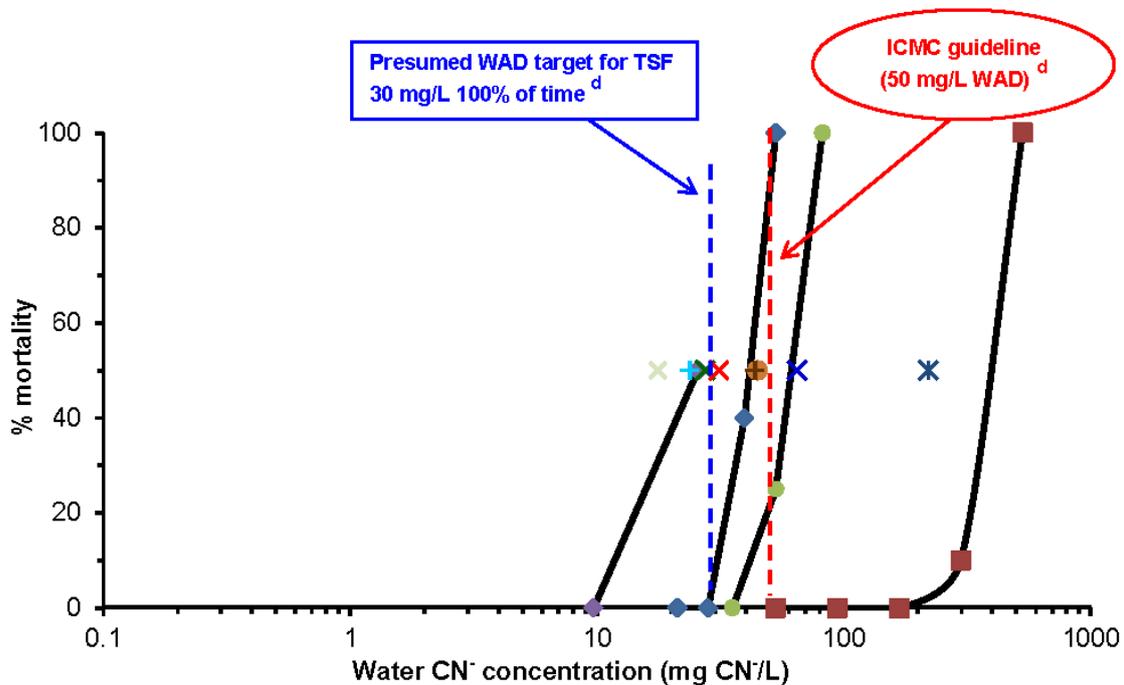
### **3.3 Species sensitivity**

Figure 3.1 summarises dose response information for oral cyanide toxicity in birds. Data displayed in the graph are for mortality, and where required experimental doses (mg/kg bw) were converted to free cyanide concentrations in water (mg CN/L) assuming a birds daily water intake was consumed in a short drinking session<sup>6</sup>. Of the birds for which complete dose response information was available, the mallard duck is the most sensitive. Others have also concluded the mallard is the most sensitive bird species (ECETOC 2007, NICNAS 2010). The figure also shows the presumed WAD target for the TSF of 30 mg/L (see Section 5.4) and the ICMC guideline of 50 mg/L WAD. The risk assessment conservatively assumes the behaviour and toxicological potency of WAD is the same as that of sodium cyanide (i.e. CN<sup>-</sup>) (Sections 7.3 & 8.3).

The relative sensitivity of birds and mammals to the lethality of cyanide, expressed as the LD<sub>50</sub> dose of free cyanide (i.e. mg CN/kg bw) is summarised in Figure 3.2. This also shows, based LD<sub>50</sub>, that the mallard is the most sensitive bird species. Information for the mallard has therefore been used to characterise the risks for birds from oral exposure to cyanide in TSF water (Section 7.3).

---

<sup>6</sup> This assumption is conservative and was only invoked for the purpose of comparing species sensitivity. For the assessment of mortality risk in Section 7 the amount of water drunk per event was assumed to be 15% of the daily requirement. This is consistent with risk assessments performed by the Australian regulatory authority charged with assessing the environmental impact of industrial chemicals (NICNAS 2010).



**Legend:**

- ◆ Mallard duck (Stence et al 1993a)
- ◆ Mallard duck (Henny et al 1994)
- Bobwhite quail (Stence et al 1993a)
- Black vulture (Wiemeyer et al 1986)
- × American kestrel (Wiemeyer et al 1986)
- Eastern screech owl (Wiemeyer et al 1986)
- + Japanese quail (Wiemeyer et al 1986)
- × European starling (Wiemeyer et al 1986)
- × Domestic chicken (Wiemeyer et al 1986)
- × Mallard duck (Clark et al 1991) <sup>a, c</sup>
- + Mallard duck (Fletcher 1986) <sup>b, c</sup>
- × Mallard duck (Fletcher 1986) <sup>b, c</sup>

**Figure 3.1: Dose response curves for oral CN<sup>-</sup> lethality to birds**

Where references reported doses of cyanide salt (i.e. mg/kg bw) (from Tables 3.1-3.3 in the toxicity profile, ToxConsult 2014), these were converted to CN<sup>-</sup> water concentrations using the species mean body weight in the original reference and daily water intake estimated using the allometric equation for birds in US EPA (1993). The allometric equation for estimating drinking water ingestion (WI) for birds is:

$$WI \text{ (L/day)} = 0.059 Wt^{0.67} \text{ (kg)}$$

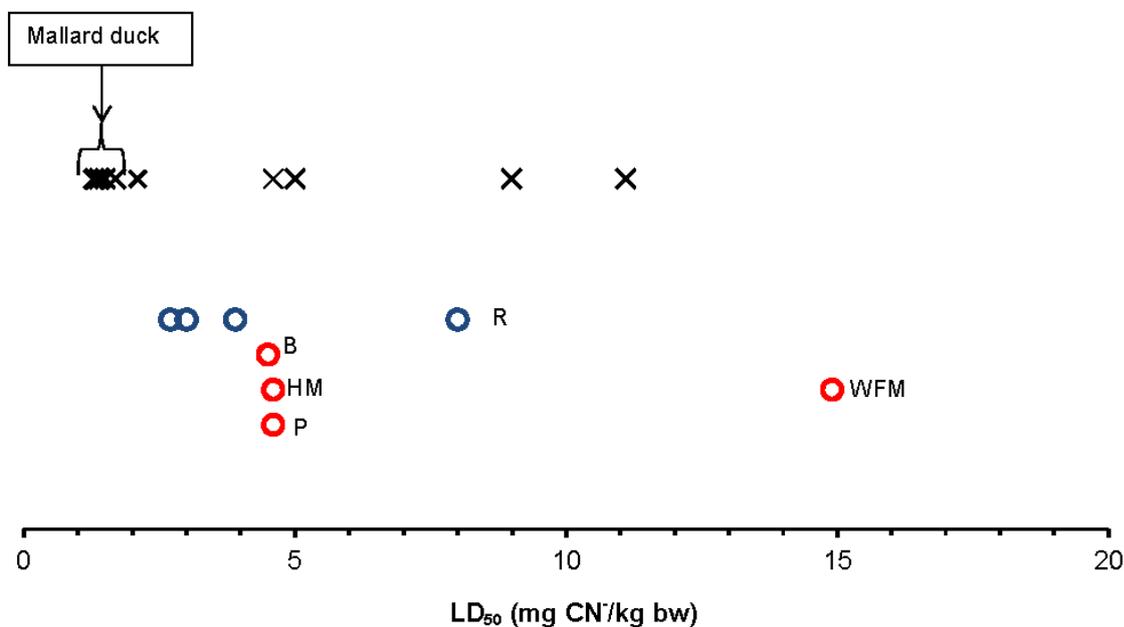
Where Wt equals average body weight (kg)

<sup>a</sup> As cited in ECETOC 2007

<sup>b</sup> As cited in NICNAS 2010

<sup>c</sup> Original reference was not available to ToxConsult. Body weight taken from Henny et al 1994.

<sup>d</sup> Experimental data on graph is for free cyanide. The risk assessment conservatively assumes the behaviour and toxicological potency of WAD will be the same as from the free cyanide ion.



**Legend:**

- X Birds (from left to right: mallard duck (x4), American kestrel, black vulture, Eastern screech owl, Japanese quail, European starling, domestic chicken)
- Blue Circle Laboratory rat (x5)
- Red Circle Other mammals (B = little brown bat; HM = house mouse; P = brushtail possum; WFM = white-footed mouse)

**Figure 3.2: Oral lethality (LD<sub>50</sub>) of CN to birds and mammals**

From Figure 3.2 and Table 3.3 in ToxConsult (2014) bats are less sensitive to the lethal effects of CN than laboratory rats.

- Since limited data is available for the oral toxicity of CN to bats (Clark et al 1991), the oral dose-response information for rats has been used for assessing potential impact to bats drinking TSF water (Section 8.3).
- Inhalation toxicity data for bats are not available, therefore air concentration- inhalation exposure time relationships for rats have been used for assessing the potential lethal effects for bats flying above the TSF (Section 8.2).



#### 4. Concentration of cyanide(s) in Mine TSF

An important consideration relating to free cyanide in particular, but also to WAD, is that they are not persistent in the tailings environment and will degrade through physical, chemical and biological processes, into other less toxic chemicals (Logsdon et al 1999).

The chemical environment of the tailings storage facility (TSF) is likely to be variable due to evaporation of water, irregular decomposition WAD with inconstant sunlight intensity, variable volatilisation of hydrogen cyanide to atmosphere depending on pH and meteorological conditions, presence of varying amounts of ammonia, and other modifying factors (e.g. residual sulphite from the detoxification unit, metal ions in the effluent). Volatilisation is the predominant way cyanide will be lost from the TSF, particularly if the pH of the TSF supernatant drops below 8 or 9 (Logsdon et al 1999, ECETOC 2007, NICNAS 2010). Although the pH of the tailings slurry exiting the 'detoxification' unit is 9.5 (CSIRO 2014) it is currently unknown how this will change when mixed with other waste streams prior to discharge into the TSF, or when stored in the TSF and water volumes change. Metals in other added waste streams when mixed with the cyanide destruction unit waste will change the WAD in the destruction waste. So called heavy metals will tend to form insoluble cyanides which will precipitate and be entrained in the tailings. In addition the actual concentration of WAD in the detoxification unit discharge could be variable; however, the unit will be operated so discharge criteria set by the EPA is not exceeded. As a result of all these factors it is not possible to confidently predict a steady state concentration of cyanide, or cyanide concentration gradients in any given TSF.

Henny et al (1994) studied cyanide concentrations in a number of gold tailings ponds in Nevada. Cyanide concentrations decreased between the discharge and the reclaim area by a factor of 2 to 9. WAD was determined at different parts of 17 tailing ponds.

- At the discharge pipe the concentrations ranged from 8.4 to 216 mg/L (pH mostly between 9.3 and 11.3). Concentrations were lowest in facilities with cyanide destruction units (generally 18-30 mg/L).
- In the interface between discharge and reclaim area WAD was 23 and 132 mg/L (pH 7.2 to 10.5), but this was only measured in 3 of the 17 facilities, none of which incorporated  $\text{SO}_4$  cyanide destruction.
- In the reclaim area WAD was between 7.1 and 86 mg/L (pH 7.8 to 11.3).

CSIRO (2014) have undertaken for the Mine a study of cyanide destruction using a sample of the carbon-in-leach (CIL) tails slurry (i.e. concentrate tails) that was provided by Independent Metallurgical Operations Pty Ltd on behalf of Unity Mining. This slurry is representative of the material



to be treated by the detoxification unit<sup>7</sup>. Analysis of the concentrate tails prior to detoxification revealed the free cyanide concentration was approximately 35% of WAD<sup>8</sup>.

The objective of the study was to determine the destruction reagent requirements to meet the ICMI cyanide code compliance of less than 50 mg/L WAD cyanide (see Section 5.1) in the discharge to the TSF. It is noted the study was very small scale, being undertaken with laboratory bench top glassware. CSIRO recommended an addition of sulphite at 140% stoichiometry to the starting WAD in order to achieve 50 mg/L WAD. In fact this gives a residual WAD of about 25 mg/L. Presumably this level of reduction is required to ensure <50 mg/L most of the time in the detoxification unit effluent, but the statistics supporting this are not in the CSIRO paper. Importantly a higher stoichiometry gives much lower residual WAD (e.g. at 160% sulphite the WAD is reduced to approximately 2.5 mg/L). The higher stoichiometry also produces more precipitate, part of which is likely to be insoluble CuFe cyanides.

The stoichiometry to be used in the detoxification unit at the Mine has not yet been delineated. Operation details of the unit will be determined when it is commissioned. It is apparent from the CSIRO (2014) study that concentrations of WAD lower than 50mg/L in the detoxification unit effluent can be attained. It is assumed operation procedures will be such that the required target cyanide concentrations (Section 5.5) will be achieved.

However this risk assessment (Sections 7, 8 and 9) has been conducted for a potential WAD target of ≤30mg/L 100% of the time, and ≤20 mg/L 90% of the time. To achieve these targets the sulphite:WAD relationship for treating CIL effluent prior to transfer to the TSF will likely need to be higher than the CSIRO recommendation for meeting the 50 mg/L WAD.

Table 4.1 summarises results from the CSIRO testing of WAD destruction. It is apparent that the total cyanide measurements are made up of WAD and that when the supernatant from treating WAD with sulphite is allowed to stand, the WAD concentration decreases. Assuming similar results will be attained from the operational destruction plant treating incoming CIL slurry, it is feasible WAD concentrations of 10 – 20 mg/L can be achieved in water discharged at the spigots, and lower in standing TSF water.

---

<sup>7</sup> Personal correspondence with Unity Mining.

<sup>8</sup> It has been conservatively assumed in this risk assessment the percentage of free cyanide in WAD at the spigot to the TSF (after cyanide destruction) is the same as prior to destruction (i.e. 35%). However, this is very unlikely since the detoxification unit will treat free cyanide preferentially to WAD.



It is further noted that since copper is an integral requirement for cyanide destruction and is the most abundant metal in effluent from the cyanide destruction plant (CSIRO 2014) that most of the WAD will be in the form of copper cyanide. The toxicity of this form of cyanide is much less than that of free cyanide (CN<sup>-</sup>) (Section 7.3).

Table 4.1: Summary of CSIRO results for WAD destruction <sup>a</sup>

Sulphite addition	Filtrate <sup>b</sup>				Slurry <sup>c</sup>			
	Fresh <sup>d</sup>		Standing <sup>e</sup>		Fresh <sup>d</sup>		Standing <sup>e</sup>	
	WAD	Total CN	WAD	Total CN	WAD	Total CN	WAD	Total CN
~ 140%	34	34	21	22	51	58	45	44
~ 160%	2.1	3.4	0.1	0.5	22	23	13	12

<sup>a</sup> Data are from CSIRO (2014). Values in the table are mg/L.

<sup>b</sup> Filtrate is the clear fluid from the slurry effluent of the CIL. This 'filtrate' was reacted with sulphite.

<sup>c</sup> Slurry refers to the effluent from the CIL. Sulphite was reacted with this and then a filtrate from this reaction analysed for cyanide.

<sup>d</sup> Fresh refers to concentrations of WAD and total cyanide in the water immediately after the reaction time (30 min) of sulphite with either CIL filtrate (i.e. clear CIL supernatant) or with CIL slurry.

<sup>e</sup> After measuring the cyanide concentrations immediately after the sulphite reaction, the cyanide containing solutions were allowed to stand in capped vials for several days. The difference between the 'fresh' and 'standing' cyanide concentrations reflects further reactions of WAD with excess sulphite. While the 'fresh' data reflects what is discharged from the spigots into the TSF, the 'standing' results better represent cyanide concentrations in the TSF water.

## 5. Important cyanide concentrations

### 5.1 ICMI (2009, 2012)

The International Cyanide Management Institute (ICMI 2012) developed the International Cyanide Management Code (ICMC), a voluntary initiative for the gold mining industry and for producers and transporters of cyanide used in gold mining. The Code is intended to complement an operation's existing regulatory requirements, and focuses exclusively on the safe management of cyanide produced, transported and used for the recovery of gold. Included is cyanide that may be in tailings effluent and leach solutions.

The Code itself contains brief overarching principles and standards of practice. Standard of Practice number 4 requires signatories to:

*"Manage cyanide process solutions and waste streams to protect human health and the*



*environment.” And “Implement measures to protect birds, other wildlife and livestock from adverse effects of cyanide process solutions.”*

The ICMI have published a companion document to the Code which provides implementation guidance for the Standards of Practice contained in the Code (ICMI 2009). The guidance states a concentration of 50 mg/L WAD cyanide, or lower, in solution is typically viewed as protective against mortality of most wildlife and livestock. However the basis of this statement is not provided in either ICMI documents. The guidance also indicates where birds, wildlife or livestock have access to cyanide containing water measures should be taken to limit WAD cyanide concentration to a maximum of 50 mg/L.

An appraisal of the scientific literature undertaken for the toxicity profile (ToxConsult 2014) and this risk assessment suggests the value of 50 mg/L WAD has basis in observations and incident reports at cyanide containing tailings ponds where avian mortalities had been observed. From the limited information available, it seems a WAD cyanide concentration >50 mg/L was associated with bird deaths, but at sites where WAD was generally <50 mg/L, few or no mortalities were observed (see also Section 3.2.1).

## 5.2 ECETOC (2007)

From the experimental study with mallard ducks conducted by Stence et al (1993a), ECETOC (2007) derived what they considered a “tolerable” water concentration for wildlife of 2 mg CN/L (free cyanide) by applying an assessment factor of 10 to the Low Observed Effect Level of 19.4 mg CN/kg for reduced food and water intake by the birds.

Conservatively assuming there may be approximately 35% free cyanide<sup>9</sup> in the measured WAD, this “tolerable” water concentration of 2 mg CN/L for free cyanide is contained in 5.7 mg/L WAD. However, since a gold mine TSF does not contain a food source for birds, the end point of temporary reduced food and water intake by birds is arguably not the appropriate effect of concern for cyanide in a TSF.

If the free cyanide in the discharged effluent has been reduced to 5% of the measured WAD then the ECETOC (2007) “tolerable” free cyanide concentration is equivalent to 40 mg/L WAD.

---

<sup>9</sup> Analysis of the concentrate tails prior to detoxification revealed the free cyanide concentration was approximately 35% of WAD (CSIRO 2014, see Section 2.1). It has been conservatively assumed in this risk assessment the percentage of free cyanide in WAD at the spigot to the TSF (after detoxification) is the same as prior to detoxification (i.e. 35%). However, this is unlikely since the detoxification unit will treat free cyanide preferentially to WAD.



### 5.3 NICNAS (2010)

For birds NICNAS (2010) estimated an overall water LC<sub>01</sub> (1% mortality) for a WAD of 50 mg/L for repeat exposure. The rationale for the LC<sub>01</sub> was not provided by NICNAS, it is compatible with the above ICMI guidelines. NICNAS (2010) suggested a tailings storage facility concentration of 50 mg WAD/L is appropriate as a general overall maximum concentration target to minimise the risk of significant bird and wildlife death. However as concentrations of WAD may fluctuate<sup>10</sup>, NICNAS indicated it is necessary to set appropriate statistical limits below this so the maximum is not exceeded. An example of 30 mg/L WAD (90<sup>th</sup> percentile) and 50 mg/L WAD (not to be exceeded)<sup>11</sup> was offered. This was partially rationalised by relating the acute toxicity information for mallard ducks to a dose likely to be ingested in a single drinking event (15% of daily water intake). If a duck of 1 kg consumes 8-10 mL of 50 mg WAD/L solution, it would result in a delivered dose of 0.4-0.5 mg CN/kg, which according to NICNAS (2010) is at or below the dose at which no mortality is observed in mallard ducks.

NICNAS (2010) also used data from various studies for acute oral toxicity tests with seven bird species to derive a predicted no effect concentration (PNEC) of ~1mg CN/L, noting there were limitations in the size and quality of the dataset. However, NICNAS (2010) did not provide details of which information was used, or the math underpinning the PNEC derivation. They note that differences in drinking water behaviour between different bird species make the PNEC estimate conservative, and not necessarily appropriate for extrapolation from one species to another. NICNAS (2010) also remarked that this low PNEC may not be justified based on field evidence and difficulties with extrapolating acute toxicity data from laboratory studies to the risk in the field.

NICNAS (2010) also derived an oral toxicity reference value (TRV), equivalent to a No Observed Effect Level (NOEL<sub>any effect</sub>), of 0.14 mg CN/kg bw for the assessment of risks from exposure to cyanide by birds. The TRV was based on an acute LD<sub>50</sub> of 1.4 mg CN/kg bw for mallard ducks from Henny et al (1994), and the application of an assessment (i.e. safety) factor of 10. NICNAS (2010) considered a safety factor of 10 sufficient to convert the LD<sub>50</sub> to a TRV because of cyanide's steep dose response and the nature of cyanide as an acute toxin (i.e. complete recovery from a sublethal dose of cyanide can be expected). The same approach was used by NICNAS (2010) to derive a TRV

<sup>10</sup> Measurement data from gold mines in Australia have shown significant daily fluctuations in WAD concentrations in tailings dams. Perhaps due to photolysis during the day, volatilisation, or depth and location where samples were taken (NICNAS 2010). For this reason, NICNAS (2010) indicate the benchmark targets should be applied to tailings effluent at the point of discharge into the TSF. Henny et al (1994) found WAD levels decreased from the discharge spigots relative to the reclaim area.

<sup>11</sup> NICNAS (2010) indicate this approach is used by the NSW Department of Environment and Heritage (presumably in licenses issued to mines). According to NICNAS (2010) the Department selects lower standards for sites where rare or threatened species may be present, e.g. 20 mg WAD CN/L (90<sup>th</sup> percentile) and 30mgWAD CN/L (maximum).



of 0.23 mg CN/kg bw for mammals. The TRV is based on an acute LD<sub>50</sub> of 2.3 mg CN/kg bw in rabbits (Ballantyne 1987) and an assessment factor of 10.

#### 5.4 Licence conditions

Table 5.1 summarises gold mine licence requirements in Australia for measurement of WAD in effluent discharged to TSFs. The measurements are for release at the spigots and not within the tailings pond itself, nor the decant water. At these latter locations WAD is likely to be lower than at the discharge point (Section 4).

**Table 5.1: Summary of WAD licence requirements at facilities with similar processes as proposed for the Dargues Mine. <sup>a</sup>**

Mine	EPL No.	License date	WAD Cyanide (mg/L)	
			90% Limit	100% limit
Lake Cowal Gold Project	11912	2014	20	30
Manuka Mine	20020	2014	20	30
Tomingley Gold	20169	2013	20	30
Hera Mine	20179	2013	-	10 <sup>b</sup>
			20 <sup>c</sup>	30 <sup>c</sup>
Mt Boppy Gold Mine (Polymetals Pty Ltd)	20192	2013	20	30
Mt Boppy Gold Mine (Peak Gold Mines)	11583	2005	-	50
McKinnons Gold Mine	4982	2003	-	-
May Day Gold Mine	5752	2001	-	-

EPL = Environmental Protection License

- = no limits for cyanide provided in the license.

<sup>a</sup> License information provided by Big Island Mining.

<sup>b</sup> At discharge/monitoring location (i.e. Point) 1.

<sup>c</sup> At discharge/monitoring location (i.e. Point) 2.

#### 5.5 Presumed WAD target at Dargues Mine

As noted in Section 4 the exact concentration of WAD and free cyanide, and their relative proportions, in the Dargues mine TSF water will not be known until the detoxification unit has been commissioned. However it is anticipated the detoxification unit will be operated in a manner to meet a WAD concentration that is safe for wildlife that may visit the TSF. This risk assessment has been undertaken to determine whether recent regulatory imposed limits at mine sites with a similar process (Section 5.4) will be protective for wildlife at the Dargues Mine. These limits are WAD <30mg/L 100% of the time and <20mg/L for 90% of the time. It is worthy to note the maximum concentration which is



not to be exceeded of 30 mg/L WAD under these license conditions is lower than the ICMI (2009, 2012) guidance of 50 mg/L WAD (Section 5.1), but in line with the management strategy proposed by NICNAS (2010) and the NSW Department of Environment and Heritage (Section 5.3). Consequently, it has been assumed in this risk assessment the concentration of WAD at the TSF spigot will be at a maximum of 30 mg/L.

In order to meet these limits the actual WAD concentration at the spigots will need to be lower, around 10-15 mg WAD/L.

## 6. Risk assessment methodology

### 6.1 Overview

In eco-toxicological risk assessments the major objective is species population and ecosystem viability protection rather than protecting each individual from harm (ANZECC 2000, US EPA 2006, DEWHA 2009). Given the very steep dose response for cyanide effects and the lack of bioaccumulation in organisms (Sections 3.3 & 7.3.1, Section 3 in ToxConsult 2014), coupled with infrequent and/or short term wildlife visits to the TSF (if they occur at all), it is acute exposure to cyanide that presents greatest risk and concern. Since birds and mammals readily and fully recover from the non-lethal effects associated with acute oral or inhalation cyanide exposure the effect of most concern is mortality. Thus this assessment focuses on the risk of bird and bat mortality should TSF water be drunk.

This environmental risk assessment follows the general principles and guidelines recommended by competent authorities (ANZECC 2000, WA DEC 2006, 2010; US EPA 2006, NEPC 2013). In brief it entails estimation of potential exposures and characterisation for the risk of mortality by comparison with exposures that do not kill animals. This is a standard margin of exposure (MoE) method. To designate the endpoint of concern the exposure comparator is the No Observed Effect Level for mortality (NOEL<sub>mortality</sub>), consequently the MoE is for mortality, abbreviated as MoE<sub>mortality</sub>.

Figure 6.1 provides a visual outline of the risk assessment. For ease of reading equations used for estimating exposures are located in the sections where they are first used.

Although birds may possibly absorb cyanide through their skin when wading or swimming, or inhale HCN as it evaporates, drinking TSF water is the main route of intoxication (ECETOC 2007, NICNAS 2010). Inhalation exposure was considered to be a negligible exposure pathway by NICNAS (2010) and was not quantitatively included in their generic assessment of cyanide at TSFs. Because there is



insufficient information to assess dermal exposure it has not been included in this risk assessment. This is also consistent with deliberations of NICNAS (2010) and ECETOC (2007), who have also not assessed dermal exposure. Omission of dermal exposure was rationalised by NICNAS because the the exposed skin of birds present an efficient barrier to cyanide being absorbed, as well as observations at gold mine TSFs that no effects are observed in waterfowl on cyanide containing water if the water is not drunk.

Detailed information for the effects of oral cyanide in birds is in the Toxicity Profile companion document to this risk assessment (ToxConsult 2014). Importantly if a bird is not overwhelmed and killed by the cyanide exposure, it recovers relatively quickly with no ill effects. However with a sufficiently large oral dose death occurs between 15 and 30 min. Birds surviving 1 hour usually fully recover (ECETOC 2007). Overall the toxicity of cyanides to birds and other wildlife depends on the dose rate and the conditions of exposure.

The general symptoms observed in birds are essentially the same for all species, although all symptoms may not be exhibited. Symptoms include:

- Slight coordination disturbance,
- Rapid eye blinking,
- Head-bowing,
- Wing-droop.

With lethal doses these early symptoms are followed by:

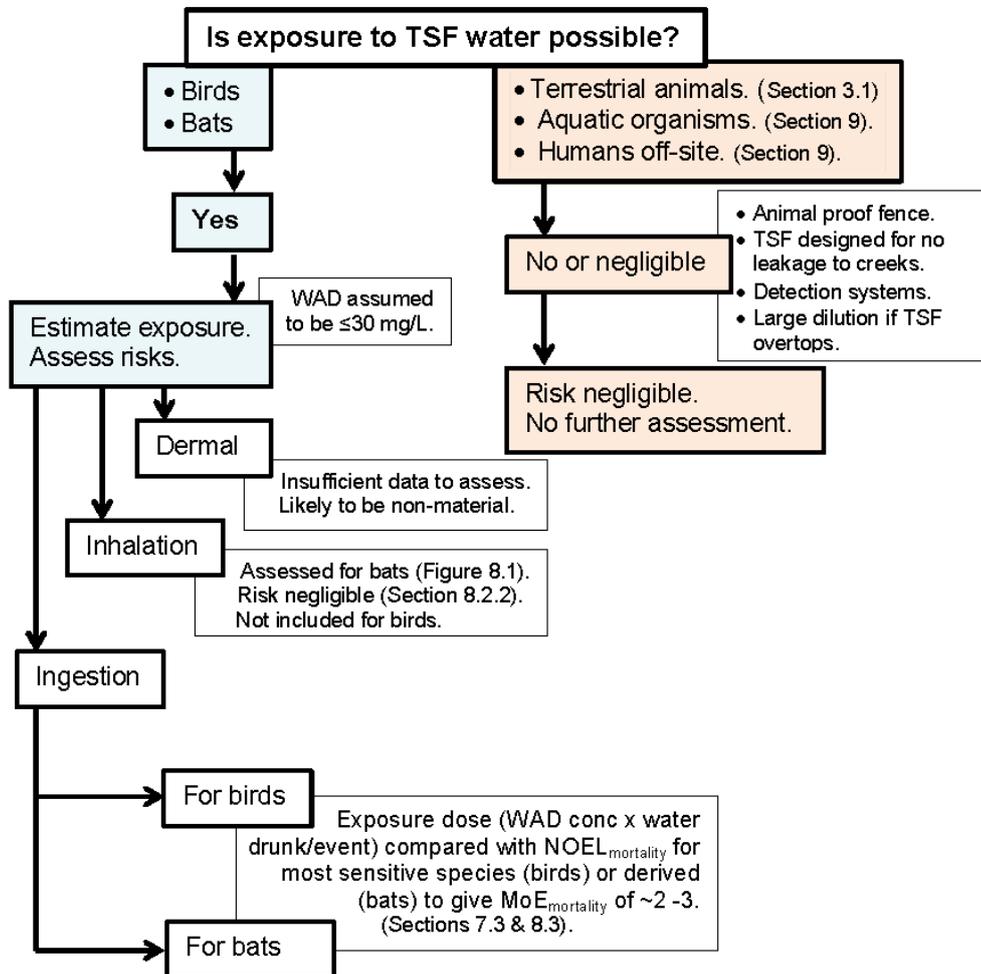
- Loss of coordination,
- Convulsions,
- Tail fanning,
- Breathing disorders followed by death.

As is the case for birds, ingestion of TSF water is likely to be the most important exposure pathway for other animals. Nevertheless because, in addition to possibly visiting the TSF to drink, bats may potentially spend long periods of time above TSF water foraging for insects, inhalation exposure is included in the risk assessment for bats. Its inclusion also facilitates quantitative determination for whether inhalation exposure may materially contribute to cyanide intoxication of other wildlife.

Inhalation toxicity data for bats are not available; therefore air concentration – exposure time relationships for rats have been used for assessing the potential lethal effects to bats flying above the TSF (Section 3.3, 8.2). This is considered appropriate since based on LD<sub>50</sub> values bats are less



sensitive to the lethal effects of CN<sup>-</sup> than are laboratory rats (Section 3.3, Figure 3.2). Because only limited data is available on the oral toxicity of bats (Clark et al 1991) the assessment for potential mortality in bat populations has been assisted by information on the shape of the oral lethality dose-response curves for other species (Section 8.3).



**Figure 6.1: Overview of risk assessment .**  
 WAD = Weak Acid Dissociable cyanide in TSF water (mg/L).  
 NOEL<sub>mortality</sub> = No Observed Effect Level for mortality (mg CN/kg body weight)  
 MoE<sub>mortality</sub> = Margin of Exposure for mortality (i.e. the gap between estimated exposure and dose not causing death).



## 7. Bird risk assessment

### 7.1 Bird visitations to TSF

Birds are known to visit gold mine TSFs (Section 3.2.1). The extent of such visitations is dependent upon a number of factors. Included are the attractiveness of the water body (e.g. size), presence of alternative surface waters, food sources (negligible at a TSF) and roosting locations.

### 7.2 Bird inhalation assessment

The risk assessment for bats showed negligible risk of mortality from inhalation (Section 8.2.2). A similar conclusion can reasonably be made for birds, especially considering HCN will not accumulate near the surface of the TSF because it is lighter than air (i.e. density of 0.94 relative to 1 for air) (NICNAS 2010). This is consistent with deliberations from other agencies indicating the major route of exposure to birds is oral. The Australian agency NICNAS (2010) and ECETOC (2007) have consequently only considered oral exposure for birds. The same approach has been adopted in this risk assessment. Risk to birds from oral exposure to TSF water is assessed in Section 7.3.

### 7.3 Bird oral assessment

Since mallard ducks are the most sensitive of the bird species tested (Section 3.3), data for mallards have been applied to ducks observed in the Dargues Reef area and which hence may visit the TSF. If impacts to the most sensitive avian species (i.e. ducks) are minimal it follows impacts to other species in the area (Appendix A) will also be minimal.

Risk to ducks was characterised by comparing the estimated CN intake with the  $NOEL_{mortality}$  for mallards (i.e. the margin of exposure,  $MoE_{mortality}$  was calculated). The acute  $NOEL_{mortality}$  from bolus dosing mallards with CN was 0.53mg CN/kg bw and the  $LOEL_{mortality}$  1.1 mg CN/kg bw (NICNAS 2010). The experimental acute mallard  $LD_{50}$  for free cyanide ranged from 1.3 – 1.7 mg CN/kg bw.

Equation 7.1 was used to calculate the oral intake for duck species in Table 7.1.

$$Intake_{oral} (mg/kg) = \frac{WI (L/d) \times F \times Target WAD (mg/L)}{BW (kg)} \dots\dots\dots \text{Equation 7.1}$$

Where:

$Intake_{oral}$  = Intake of WAD cyanide from drinking water in TSF (mg/kg).  
WI = Water intake (L/d). This was estimated using the US EPA (1993) daily water intake allometric equation for birds ( $WI = 0.059 Wt^{0.67}$ ), where Wt equals average body weight (kg).



F = Assumed fraction of daily intake ingested per drinking event or over a short time (unitless). This is assumed to be 15%, based on the ratio assumed by NICNAS (2010) for birds<sup>12</sup>.

Target WAD = Target WAD CN concentration at spigot. Assumed to be 30 mg/L (Section 5.5). This assumes all the WAD is free CN<sup>-</sup> and potentially systemically available. Patently these assumptions are conservative.

BW = Body weight of species for which intake is to be calculated (kg) (Table 7.1).

#### Results:

Table 7.1 summarises the MoE<sub>mortality</sub> for ducks in the area of the Dargues mine lease. The MoE<sub>mortality</sub> is simply the NOEL<sub>mortality</sub> divided by the estimated oral CN<sup>-</sup> intake per drinking event assuming 30 mg WAD/L is free CN<sup>-</sup>.

**Table 7.1: Calculated MoE<sub>mortality</sub> for birds**

Common name	Bird weight <sup>a</sup> (kg)	Estimated water intake <sup>b</sup> (mL/d)	Calculated oral intake (mg/kg) <sup>c</sup>	MoE <sub>mortality</sub> <sup>d</sup>
Australian Shelduck	1.4-1.7	74 - 84	0.22 – 0.24	2.2 – 2.4
Australian Wood duck	0.66-0.98	45 - 58	0.27 – 0.31	1.7 – 2.0
Black duck	1.0-1.1	60 – 63	0.25 – 0.26	2.0 – 2.1
Australasian shoveler	0.65	44	0.30	1.7
Grey Teal	0.35 – 0.67	29 – 45	0.30 – 0.37	1.4 – 1.8
Chestnut Teal	0.65	44	0.30	1.7
Hardhead	0.85	53	0.28	1.9

<sup>a</sup> Body weight obtained from various references<sup>13</sup>.

<sup>b</sup> Estimated with US EPA (1993) allometric equation for daily water intake by birds. ( $WI = 0.059 Wt^{0.67}$ , where WI equals water intake (L/day) and Wt equals body weight (kg)). For the risk assessment it was assumed 15% of daily water intake was drunk per event (NICNAS 2010).

<sup>c</sup> Estimated using Equation 7.1.

<sup>d</sup> This is the margin of exposure between the calculated intake and the experimental NOEL<sub>mortality</sub> of 0.53 mg/kg for mallard ducks, the most sensitive avian species (Section 3.3, NICNAS 2010).

MoE between calculated oral intakes and the dose (in mallard ducks) resulting in no mortality (NOEL<sub>mortality</sub>) were all greater than 1 (average 1.9). This suggests bird populations are adequately protected from mortality if exposed to TSF water containing a WAD cyanide concentration of 30 mg/L. However the relatively small MoE's suggest there may be a possibility some birds may die if they visit the TSF and drink more water than has been assumed in the risk assessment. However this notion is countered by the following:

<sup>12</sup> NICNAS (2010) assumed a mallard duck may drink 10 ml per drinking event (i.e. 0.01 L). Using the allometric equation from the US EPA (1993) for birds, a mallard duck weighing 1.26 kg (from Henny et al 1994) drinks 0.0688 L/day. Thus a duck may drink 15% ( $0.01 \div 0.0688$ ) of its daily water intake in one drinking event.

<sup>13</sup> *Australian shelduck*: NZ Birds Online (undated); *Australian Wood duck*: Wildscreen Arkive (undated a); *Black duck*: Bouglouan (undated); *Australasian shoveler*: Birds in Backyards (undated a); *Grey Teal*: Wildscreen Arkive (undated b); *Chestnut Teal*: Birds in Backyards (undated b); *Hardhead*: Birds in Backyards (undated c).



- The assessment assumes the lethality of WAD cyanide in the TSF is the same as NaCN. This is a gross conservative assumption.
- Copper is added to the cyanide destruction process as a catalyst for the chemical reactions. CSIRO (2014) indicates copper is by far the dominant metal<sup>14</sup> in the discharged tails slurry. As a result copper cyanide will be the dominant cyanide species in WAD cyanide measurements.
- In acute experiments with mallard duck (the most sensitive avian species) Link et al (1996) showed at doses of CN from NaCN or KCN causing 50 - 65% mortality no deaths occurred with the equivalent CN dose from copper cyanide.
- It follows the toxicity of WAD cyanide is markedly less than that of NaCN. Therefore a MoE of approximately 2 does not necessarily indicate likelihood that bird deaths will occur.
- The risk assessment is supported by field studies in which no, or only few bird deaths have been observed when TSF is  $\leq 30$  mg WAD/L (MERC 2001, NICNAS 2010, DRET 2008, NT DoME 1998, NPS 1997, Donato et al 2008, Griffiths et al 2014a, Hudson and Bouwman 2009).

Calculations in Table 7.1 are for a target WAD of  $\leq 30$  mg/L 100% of the time. However it is noted recent regulatory licenses for cyanide concentrations in TSFs indicate the WAD concentration should be  $\leq 20$  mg/L for 90% of the time (Section 5.4). At a WAD of 20 mg/L the  $MoE_{mortality}$  will be a third higher, and the risk of mortality lower. Furthermore WAD in TSF water will be less than the concentration at the discharge spigots (Section 4).

Uncertainties and their influence on the risk assessment are further discussed in Section 7.3.2. The potential for secondary poisoning is discussed in Section 7.3.1.

### 7.3.1 Potential secondary poisoning of birds

The potential risk for secondary poisoning of raptors consuming carcasses which died as a result of cyanosis is low (NICNAS 2010). Such exposure is unlikely to affect raptors, since the dose rate of releasable free cyanide from the carcass will be low. Cyanide does not bioaccumulate and is likely to be present in the carcass as thiocyanate, the major metabolite of cyanide. Thiocyanate is significantly less toxic than free cyanide. If free CN is present in the carcass the dose will be and adult raptors will detoxify it. Young birds are more vulnerable by the fact they will receive a larger dose as a result of their lower body weight. However there is no data suggesting young animals may be intrinsically more susceptible.

<sup>14</sup> The CSIRO (2014) experiments for destruction of cyanide show copper is more than approximately 100 times more abundant in the tails slurry from the cyanide destruction plant than other metals.



**7.3.2 Bird oral assessment uncertainties**

This section contains a general qualitative discussion of the major uncertainties for the oral risk assessment for birds and their potential influence on the outcome. Most of the uncertainties associated with assessment of mortality risk to birds from ingesting TSF water are also applicable to bats, therefore both have been discussed in Table 7.2.

Overall the uncertainty analysis suggests the assumptions used are conservative, and for a population of birds tend to over-, rather than under-estimate the mortality risk. The objective of an ecotoxicological risk assessment is to protect species populations and ecosystem viability rather than protecting each individual from harm, and this objective is considered to be met in this risk assessment.

However the margin of safety may be small for an individual animal, which indicates there may be a possibility some birds may die if they visit the TSF and drink more water than has been assumed in the risk assessment. Nevertheless the risk assessment assumes the WAD cyanide in the TSF has the same toxicological potency as NaCN used in the toxicological experiments providing the data for the risk characterisation. WAD cyanide is distinctly less toxic than NaCN. Therefore a small MoE of approximately 2 as has been obtained for birds, does not necessarily indicate likelihood that deaths will occur.

**Table 7.2: Uncertainties in the oral risk assessment for birds at Dargues Mine**

Uncertainty/ Assumption	Comment	Effect on Risk Assessment
<p><b>Concentration of cyanide to which birds may be exposed.</b></p> <p>1) WAD CN in TSF is 30 mg/L</p> <p>2) The WAD to which birds (and bats) are exposed is assumed to be the concentration at release spigot.</p>	<p>1) There is uncertainty regarding the actual concentration of WAD cyanide that will be in the TSF. The concentration leaving the cyanide destruction unit and at the release spigot could be lower than assumed, but will not be higher.</p> <p>Field observations cited in NICNAS (2010) &amp; ECETOC (2007) indicate wildlife mortalities with WAD &gt; 50 mg/L but few or no mortalities at lower concentrations.</p> <p>2) Data from Henny et al (1994) indicate WAD concentrations are likely to be 2-9 fold lower in the TSF pond compared to the spigot.</p>	<p>1) The assumption potentially overestimates the risk of population or individual mortality.</p> <p>2) The assumption potentially overestimates the mortality risk.</p>



Uncertainty/ Assumption	Comment	Effect on Risk Assessment
<p>3) WAD cyanide is biologically available after ingestion and has toxicological potency equal to NaCN (i.e. free cyanide).</p>	<p>3) The majority of WAD cyanide is likely to be cyanide in metal complexes, which may only be released slowly in the gastrointestinal tract before it becomes available for absorption. Thus birds (and bats) may be able to detoxify the cyanide efficiently and a higher dose of WAD CN compared to CN<sup>-</sup> is needed for mortality.</p>	<p>3) The assumption potentially overestimates the mortality risk.</p>
<p><b>Water intake.</b></p> <p>1) Acute intake of TSF water by birds (and bats) is assumed as described by NICNAS (2010) in their semi-quantitative risk assessment (i.e. 15% of daily water intake per drinking event or over a short time).</p> <p>2) Repeat exposure was not quantitatively assessed.</p>	<p>1) This could be either an under- or over-estimation of acute water intake. Intuitively it may have bias to under-estimating the water consumption behaviour of some birds.</p> <p>Since bats obtain small gulps of water each time they mouth-dip into the water surface to drink during flight, the assumed extent of acute water intake may not be unreasonable for the risk assessment of bats. Similarly only small quantities of water are consumed in a short period if the bat 'belly skims' the water and subsequently licks the water off.</p> <p>2) Birds surviving after an acute exposure may drink again, which will increase their overall intake. However, an experiment by Henny et al (1994) with mallards found if they survived the initial two exposures, death rarely occurred after three cycles or after ~1.5 hours of the 4 hour trial. In addition, there does not appear to be significant differences in the LC<sub>50</sub>s of WAD cyanide from single or 5-day repeat oral exposures, as per NICNAS (2010). Cyanide is rapidly detoxified (for example, cyanide blood half-life in mammals is ~15-25 minutes, ECETOC 2007), this means sufficient time between exposures allows detoxification and that cyanide does not bioaccumulate.</p>	<p>1) This potentially over- or under- estimates the risk for birds.</p> <p>2) This is unlikely to materially affect the outcome of the risk assessment.</p>
<p><b>TSF is attractive to birds (and bats).</b></p>	<p>Birds and bats may be attracted to the TSF water if it acts as a potential food source, foraging or rest area. This is considered unlikely since vegetation in the direct vicinity of the TSF will be negligible (as it will be removed), there is unlikely to be significant food sources and roost areas at the TSF are minimal. The TSF cyanide levels are not conducive for insect breeding (see below) and in addition there are more appealing surface water sources in the nearby vicinity.</p>	<p>This potentially overestimates the likelihood of significant bird or bat numbers visiting the TSF and therefore also the mortality risk.</p>



Uncertainty/ Assumption	Comment	Effect on Risk Assessment
<p><b>Species Sensitivity</b></p> <p>1) Use of NOEL<sub>mortality</sub> for mallard duck to assess risk of oral exposure to birds.</p>	<p>1) Available reliable data indicates the mallard is the most sensitive species tested. However, relatively few species have been tested and it is unknown if untested species are more or less sensitive to cyanide.</p>	<p>1) This potentially underestimates the risk if there are more sensitive species than the mallard duck.</p>
<p><b>Toxicity reference value</b></p> <p>1) NOEL for lethality for mallard ducks</p>	<p>1) There is uncertainty with the NOEL<sub>mortality</sub> of 0.53 mg/kg for mallards. It comes from NICNAS (2010) and since the original source was unavailable the value could not be verified. The value could be an over- or under-estimate of the true NOEL<sub>mortality</sub> for the most sensitive tested avian species. However, the NOEL<sub>mortality</sub> of 0.53 mg/kg is 2.6 times less than the LD<sub>50</sub> of 1.4 mg/kg. This ratio is similar to that for other animals for which NOEL<sub>mortality</sub> and LD<sub>50</sub>s have been identified (Section 8.3). Therefore the NOEL<sub>mortality</sub> used is rationalised to be close to the true NOEL<sub>mortality</sub>.</p>	<p>1) This is unlikely to significantly affect the outcome of the risk assessment.</p>
<p><b>Form of cyanide</b></p> <p>It has been assumed the toxicological potency of the forms of cyanide in TSF water is the same as that of sodium cyanide, the toxic form used in experiments.</p>	<p>This is a conservative assumption because free CN<sup>-</sup> dissociating from WAD does so at a slower rate than from NaCN. Consequently cyanide will be absorbed more slowly than from NaCN. The slower release of HCN from WAD and consequent slower absorption rate allows birds and bats the opportunity to detoxify it.</p> <p>This is supported by copper cyanide (the likely dominant form of cyanide in WAD) being markedly less toxic than sodium cyanide.</p>	<p>The assumption of WAD cyanide being toxicologically equivalent to NaCN overestimates the risk.</p>

**7.3.3 Conclusion**

The MoEs between calculated oral intakes and the dose (in mallard ducks) resulting in no mortality (NOEL<sub>mortality</sub>) were all greater than 1 (average 1.9). This suggests bird populations are adequately protected from mortality if exposed to TSF water containing a WAD cyanide concentration of 30 mg/L. The assumed target WAD concentrations in this report are ≤ 30 mg/L 100% of the time, but ≤ 20 mg/L for 90% of the time (Section 5.4). At a WAD of 20 mg/L the MoE<sub>mortality</sub> will be a third higher, and the risk of mortality lower. Since the assumptions used in the calculations are conservative and for a



population of birds tend to over- rather than under-estimate the mortality risk, it is concluded there is negligible risk to bird populations from exposure to the TSF.

## **8. Bat risk assessment**

### **8.1 Bat visitations to TSF**

Henny et al (1994) reported bat deaths in the 1980's at gold mine operations in Nevada.

Griffiths et al (2014a) have observed bats to be active above a TSF at a NSW gold mine<sup>15</sup>.

During the study WAD was measured twice daily. The maximum tailings discharge concentration (at the spigot) was 26 mg/L WAD cyanide with 10 mg/L WAD exceeded only 10% of the time. The maximum cyanide concentration recorded in the central decant pond was 10 mg/L. Apart from the central supernatant pond in the large TSF a small fresh water farm dam 5 km away was also monitored for bat visitations. Over 16 months bat activity was recorded in the airspace above both water bodies on almost all recording nights. Over the entire study period overall bat activity did not differ significantly between the two water bodies but there were considerable differences in any one month or night. There were differences in relative bat activity among seasons at the two water bodies, at both there were much less visitations in June (winter) than November (spring). Donato et al (2008) reported the counts of either wildlife deaths or visitations on tailing dams decrease as the surface area of the supernatant decreases.

Griffiths et al (2014a) concluded the most effective mechanism for preventing cyanide toxicoses to wildlife, including bats, is reducing WAD discharged at the spigots to below 50 mg/L. Griffith et al (2009) also suggest lack of aquatic food resources represents a protective mechanism that limits or prevents cyanide-related wildlife mortality at a TSF. The Griffiths et al (2014a) investigation described above was unable to determine whether the bat visitations at the studied TSF or farm dam were related to drinking or foraging. In relation to the latter it is noted that anticipated cyanide concentrations in the Dargues TSF are not conducive for insect breeding<sup>16</sup>. Thus it is unlikely the TSF will present a food source for bats. Nevertheless, insects may be attracted to the TSF by the mere fact there is water present. The TSF, however, will not have lights thereby decreasing the

---

<sup>15</sup> The gold mine was the Barrick Gold Corporation's Cowal Gold Mine at Lake Cowal, central NSW.

<sup>16</sup> Median lethal concentrations (LC<sub>50</sub>) of free cyanide (CN<sup>-</sup>) for four freshwater insects are reported to be approximately 0.2-0.5 mg CN<sup>-</sup>/L, with one additional species having an LC<sub>50</sub> of 2.5 mg/L (ANZECC 2000, ECETOC 2007). Assuming a free cyanide or WAD concentration of 10 – 30 mg/L in TSF water it is unlikely TSF insect breeding will occur.



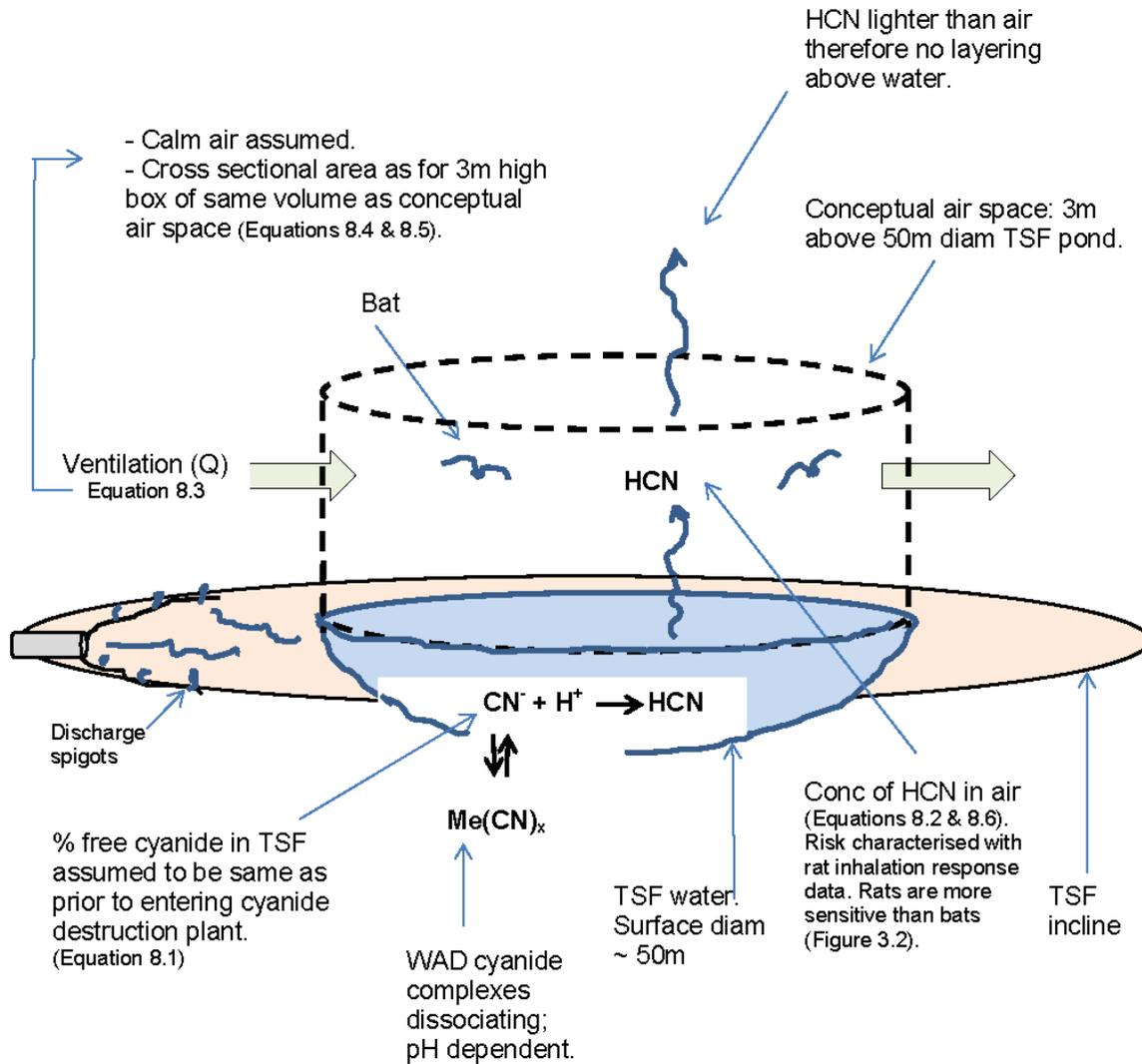
potential attraction, whereas the operating plant (~350 m away) will be lit. Insects are therefore more likely to be present around the operating plant at night.

Overall the above information suggests a reasonable likelihood that bats may visit the Dargues TSF.

## 8.2 Bat inhalation exposure

As bats search for insects above the TSF (if any insects are present) or make drinking passes over the water they may be exposed by inhalation to HCN volatilising from the TSF water surface. It is noted that volatilisation of HCN is the dominant way dissociable cyanides are removed from water.

Figure 8.1 is a conceptual representation of the methodology used to estimate HCN concentrations in air above the TSF. Detailed discussion and equations follow. Uncertainties associated with the major assumptions are explored in Section 8.2.1.



**Figure 8.1: Conceptual representation for estimating inhalation exposure of bats to cyanide at TSF.**



It is only the free CN<sup>-</sup> in water which is subject to volatilisation. At a maximum target WAD CN concentration of 30 mg/L in TSF water (Section 5.5), it has been assumed the concentration of free CN in water may be approximately 10.5 mg/L (see footnote to Equation 8.1).

$$C_{\text{water\_freeCN}} \text{ (mg/L)} = \text{Target WAD (mg/L)} \times \% \text{freeCN} \dots\dots\dots \text{Equation 8.1}$$

Where:

$C_{\text{water\_freeCN}}$  = Free CN<sup>-</sup> concentration at the target WAD concentration (mg/L).

Target WAD = Target WAD concentration at spigot. Assumed to be 30 mg/L, not to be exceeded.

%freeCN = Percentage of free CN<sup>-</sup> in WAD (%) at the spigot entering the TSF.

According to CSIRO (2014), 35% of WAD in the concentrate tails from the CIL process was present as free CN<sup>-</sup> prior to being passed through the cyanide destruction unit. In the absence of data for the detoxification plant effluent it has been assumed the proportion of CN<sup>-</sup> to WAD will be the same as for the input to the detoxification unit.

This assumption is conservative, since the chemical destruction reactions will favour free CN<sup>-</sup> over WAD (although both are destroyed). As the detoxification effluent moves from the detoxification unit to the TSF spigot, residual sulphite will continue to react with free cyanide, reducing the residual free CN<sup>-</sup> concentration further (CSIRO 2014).

The concentration of HCN in air above the TSF was calculated using Equations 8.2- 8.6. The calculation is an adaptation of that from the American Conference of Governmental Industrial Hygienists (ACGIH 2007, Equation 4-2) for calculating dilution ventilation required to maintain steady state concentrations at a given air concentration (Equation 8.2). It is assumed bats fly within a 3m height above the TSF water and the air may be 'calm' (0.5 m/s).

$$C_{\text{air}} \text{ (mg/m}^3\text{)} = \frac{\text{CGR (g/sec)} \times \text{CF (mg/g)}}{Q \text{ (m}^3\text{/sec)}} \dots\dots\dots \text{Equation 8.2}$$

Where:

$C_{\text{air}}$  = Concentration of HCN in air (mg/m<sup>3</sup>).

Q = Ventilation rate (m<sup>3</sup>/sec). Calculated using Equation 8.3.

CGR = Contaminant generation rate (g/sec). Calculated using Equation 8.6.

CF = Conversion factor. 1000 mg/g.

$$Q \text{ (m}^3\text{/sec)} = V \text{ (m/sec)} \times A_{\text{cross}} \text{ (m}^2\text{)} \dots\dots\dots \text{Equation 8.3}$$

Where:

Q = Ventilation rate (m<sup>3</sup>/sec).

V = Air flow (m/sec). Assumed to be that of 'calm' air<sup>17</sup>, i.e. 0.5 m/sec.

$A_{\text{cross}}$  = Cross-sectional area of pond at an assumed height (h) (m<sup>2</sup>). Calculated using Equation 8.4.

<sup>17</sup> The wind velocity of 'calm air' is ≤0.5 m/s. The average annual wind speed for the mine lease is 3.7 m/s with 3.6% of the time the wind speed being calm (BIM 2013).



$$A_{\text{cross}} \text{ (m}^2\text{)} = L \text{ (m)} \times h \text{ (m)} \dots\dots\dots \text{Equation 8.4}$$

Where:

- $A_{\text{cross}}$  = Cross-sectional area of pond at an assumed height (h) (m<sup>2</sup>).
- L = Length of pond equivalent to volume of space with an assumed height of 'h' and radius of 'r'.  
 Calculated using Equation 8.5.
- h = Height of space where mixing of air occurs above pond surface. This is assumed to be 3m.

$$L \text{ (m)} = \frac{\pi r^2 \text{ (m}^2\text{)} \times h \text{ (m)}}{4} \dots\dots\dots \text{Equation 8.5}$$

Where:

- L = Length of pond equivalent to volume of space with an assumed height 'h' and radius of 'r'.
- r = Radius of pond (m). Assumed to be 50 m. The flat size of the assumed pond is not a critical determinant in the calculation, since this parameter cancels itself out, forming part of both the numerator and denominator in Equation 8.2. The predicted concentration in air above the pond will be the same regardless of assumed radius.
- h = Height of space where mixing of air occurs above pond surface. Assumed to be 3m.

CGR (g/sec) =

$$\text{EVP\_rate (mg HCN/cm}^2\text{/sec/ppm)} \times C_{\text{water\_freeCN}} \text{ (ppm)} \times \pi r^2 \text{ (m}^2\text{)} \times \text{CF1 (cm}^2\text{/m}^2\text{)} \times \text{CF2 (g/mg)}$$

.....Equation 8.6

Where:

- CGR = Contaminant generation rate (g/sec).
- EVP\_rate = Evaporation rate of HCN from free CN containing solution.  
 This was assumed to be 0.002mg/cm<sup>2</sup>/sec/ppm from ECETOC (2007) or 0.000027 mg/cm<sup>2</sup>/sec/ppm from Dept of Env (2006).
- $C_{\text{water\_freeCN}}$  = Free CN concentration at the target WAD CN concentration (ppm). Calculated using Equation 8.1.
- r = Radius of pond (m). Assumed to be 50 m. The flat size of the assumed pond is not a critical determinant in the calculation, since this parameter cancels itself out, forming part of both the numerator and denominator in Equation 8.2. The predicted concentration in air above the pond will be the same regardless of assumed radius.
- CF1 = Conversion factor. 10,000 cm<sup>2</sup>/m<sup>2</sup>.
- CF2 = Conversion factor. 1g/1000 mg.



Table 8.1 is a summary of the assumptions used in the calculations described above.

**Table 8.1: Assumptions used to calculate concentration of HCN above TSF**

Abbreviation	Description	Value	Source
$C_{\text{water\_freeCN}}$	Free CN concentration at the target WAD CN concentration (mg/L)	Calculated (Eq. 8.1)	-
Target WAD	Target WAD CN concentration at spigot (mg/L).	30 mg/L	Section 5.5
%freeCN	Percentage of free CN in WAD CN at the spigot entering the TSF.	35%	CSIRO 2014
$C_{\text{air}}$	Concentration of HCN in air ( $\text{mg}/\text{m}^3$ ).	Calculated (Eq. 8.2)	-
Q	Ventilation rate ( $\text{m}^3/\text{sec}$ ).	Calculated (Eq. 8.3)	-
CGR	Contaminant generation rate ( $\text{g}/\text{sec}$ ).	Calculated (Eq. 8.6)	-
V	Air flow ( $\text{m}/\text{sec}$ ) for 'calm' air.	0.5 $\text{m}/\text{sec}$	BIM (2013)
$A_{\text{cross}}$	Cross-sectional area of pond at an assumed height (h) ( $\text{m}^2$ ).	Calculated (Eq. 8.4)	-
L	Length of pond equivalent to volume of space with an assumed height of 'h' and radius of 'r'.	Calculated (Eq. 8.5)	-
h	Height of space where mixing of air occurs above pond surface.	3 m	Realistic judgement.
r	Radius of pond (m).	50 m	Realistic judgement.
EVP_rate	Evaporation rate of HCN from free CN containing solution.	0.002 $\text{mg}/\text{cm}^2/\text{sec}/\text{ppm}$ or 0.000027 $\text{mg}/\text{cm}^2/\text{sec}/\text{ppm}$	ECETOC (2007)  Dept of Env (2006)

Using the evaporation rate of HCN from free CN containing solution from ECETOC (2007), and applying Equations 8.2-8.6, the predicted concentration<sup>18</sup> of HCN in air is **187  $\text{mg}/\text{m}^3$**  (rounded). Intuitively the emission rate quoted by ECETOC (2007) seems very high and ToxConsult has been unable to identify how it was generated. Similarly the emission rate from the Department of Environment (Dept of Env 2006) cannot be substantiated. However, using this value a concentration<sup>19</sup> of **2.5  $\text{mg}/\text{m}^3$**  is obtained, this is inherently a more logical outcome when compared to the air

<sup>18</sup>  $[(0.002 \times 10.5 \times 7,853.98 \times 10,000 \times 1/1000) \text{ g}/\text{sec} \times 1000 \text{ mg}/\text{g}] \div (0.5 \times 17,671.46) \text{ m}^3/\text{sec} = 186.7 \text{ mg}/\text{m}^3$ .

<sup>19</sup>  $[(0.000027 \times 10.5 \times 7,853.98 \times 10,000 \times 1/1000) \text{ g}/\text{sec} \times 1000 \text{ mg}/\text{g}] \div (0.5 \times 17,671.46) \text{ m}^3/\text{sec} = 2.52 \text{ mg}/\text{m}^3$ .



concentration of HCN of 34 mg HCN/m<sup>3</sup> immediately above the water surface predicted by Henry's Law constant and not incorporating ventilation<sup>20</sup>.

From Section 3.3, rats are more sensitive to CN<sup>-</sup> than tested bats. ECETOC (2007) provide an equation for calculating the probit<sup>21</sup> for the concentration-time lethality response to inhaled HCN. The equation is:

$$\text{Probit} = b \times \ln(C^N \times t) - a$$

Where:

b = slope; 0.70 for rats as per ECETOC (2007).

C = concentration of HCN in air (mg/m<sup>3</sup>).

N = exponent; 1.64 for rats as per ECETOC (2007).

t = exposure time (480 minutes, i.e. 8 hours).

a = intercept; 3.27 for rats as per ECETOC (2007).

The probit for a 1% response (i.e. 1% chance of mortality) is 2.67 (Derelanko 2008). Assuming bats may be flying in air above the TSF for 8 hours a night<sup>22</sup>, the calculated probit at the estimated HCN air concentration (2.5 mg/m<sup>3</sup>) is 2.11. This probit indicates less than 1% response, indicating very low risk of bat deaths from inhalation.

---

<sup>20</sup> Using Henry's law constant for HCN at a pH of 9 predicts a steady state (no ventilation) air concentration of 34 mg HCN/m<sup>3</sup>. This does not incorporate diluting factors such as volume of the air into which HCN will be mixed or ventilation (i.e. air flow). The equation is:

$$C_{\text{air}} (\text{mg/m}^3) = C_{\text{water\_freeCN}} (\text{mg/L}) \times V\% \times H \times CF (\text{L/m}^3).$$

Where:

C<sub>air</sub> = Concentration of HCN in air (mg/m<sup>3</sup>).

C<sub>water\_freeCN</sub> = Free CN concentration at the target WAD CN concentration (mg/L). Calculated using Equation 8.1.

V% = Percentage of free CN subject to volatilisation (%). This varies with pH, i.e. the higher the pH the less volatilisation will occur. The pH of concentrate tails is adjusted to a pH of 9 during detoxification (CSIRO 2014), therefore it can be reasonably anticipated the pH of the tails at the TSF spigot will also be around 9. Assuming a pH of 9, the percentage of free CN subject to volatilisation at the spigot is 60% (Dept of Env 2006).

H = Henry's law constant for HCN (unitless), i.e. the dimensionless ratio of HCN concentration in air and water. This is 0.00544, as per ECETOC (2007).

CF = Conversion factor. 1000 L/m<sup>3</sup>.

<sup>21</sup> Probits are used to analyse dose-response or binomial (i.e. death/no death) response experiments in a variety of fields. Once a probit has been calculated, this can be translated to a corresponding percentage of a response (e.g. % mortality) using published percentage response-probit conversion tables (Derelanko 2008).

<sup>22</sup> Griffiths et al (2014a) surveyed bat activity in the air space above water bodies at a gold mine using echolocation calls. Highest activity occurred within 4-5 hours of sunset, then steadily decreasing towards dawn. It is unknown if the same bat would be active for the full duration of this time at the same water body, nevertheless an exposure time of 8 hours has been assumed in this risk assessment.



**8.2.1 Bat inhalation assessment uncertainties**

The risk assessment process involves a number of steps (e.g. exposure assessment, toxicity assessment and risk characterisation), each of which incorporates the use of assumptions and simplifications to manage uncertainty or lack of knowledge about the ‘true’ value. Without such assumptions and simplifications it would not be possible to quantitatively evaluate the potential for adverse effects. Although uncertainties in the risk assessment may influence its accuracy, the assumptions used to cope with unknown data for specific parameters tend to err on the side of safety and therefore bias the evaluation to over estimation of health risk. It must be realised the conservatism embedded in any one value is at least additive, most times multiplicative, with conservatisms in other values such that the cumulative or compound conservatism incorporated into the assessment can be large. This is especially so when gross, unrealistic default parameters are used *in lieu* of measured data.

This section contains a general qualitative discussion of the major uncertainties for the inhalational risk assessment for bats and their potential influence on the outcome. Uncertainties in the oral risk assessment are described in Sections 7.3.2 and 8.3.1.

**Table 8.2: Inhalation risk assessment uncertainties for bats.**

Uncertainty/ Assumption	Comment	Effect on Risk Assessment
<p><b>Concentration of WAD cyanide to which bats may be exposed.</b></p> <p>1) WAD CN in TSF will be 30 mg/L</p> <p>2) WAD in TSF water assumed corresponds to concentration at spigot entering TSF.</p>	<p>1) There is uncertainty regarding the actual concentration of WAD cyanide that will be in the TSF. To achieve a WAD concentration of 20 mg/L for 90% of the time at the discharge spigots the concentration leaving the detoxification unit would need to be lower than the 30 mg/L assumed, but will not be higher. Many publications report a WAD ≤50 mg/L protects wildlife from cyanosis. In reality to meet a possible regulatory target of ≤20 mg/L 90% of the time the WAD released at spigots will likely be less than 10 – 15 mg/L most of the time.</p> <p>2) Data from Henny et al (1994) indicate WAD concentrations could be 2-9 fold lower in the TSF pond compared to the spigot.</p>	<p>1) The assumption overestimates the risk.</p> <p>2) The assumption potentially overestimates the risk.</p>



Uncertainty/ Assumption	Comment	Effect on Risk Assessment
3) Free CN in TSF is 35% of WAD CN	3) It was assumed free cyanide was at the same proportion relative to WAD CN as in CIL effluent prior to destruction. In actual fact, it will be anticipated free cyanide when leaving the detoxification plant would be very much reduced, perhaps <1%.	3) The assumption significantly overestimates the risk.
<p><b>Calculation of HCN air concentration &amp; estimation of risk.</b></p> <p><i>Calculation assumes:</i></p> <p>1) 'Calm' air (0.5 m/s)</p> <p>2) Uniform mixing</p> <p>3) Height of theoretical air space is 3m</p> <p>4) 8hr flying time for bats</p> <p>5) Concentration-response for rats is applicable to bats</p> <p>6) Evaporation rate assumed to be for a pH of 9</p>	<p>1) At the mine site 'calm' air occurs for 3.6% of the year. The annual average wind speed is 3.7 m/s. At this ventilation rate the HCN above TSF water is estimated to be 0.34 mg/m<sup>3</sup> and the corresponding probit is 0 for lethality. This indicates the mortality for bats visiting the TSF is much less than 1%.</p> <p>2) Because HCN is lighter than air it is unlikely there will be a significant concentration gradation over the distance from the surface of the water to the height of the assumed air volume in which bats will be flying.</p> <p>3) This may be an underestimate of the air space in which bats will be occupying their time.</p> <p>4) Likely to be conservative, since Griffiths et al (2014a) recorded highest bat activity occurred over 4 -5 hrs. It is unlikely the same bat would be active for the full 8 hours at the same water body.</p> <p>5) Based on oral lethality data, rats appear more sensitive to cyanide than bats (Section 3.3). However there are no data for cyanide inhalation in bats, or the relative sensitivity of different bat species.</p> <p>6) It is uncertain what the pH in the TSF water will be. If it is pH of 9, the estimated concentration used in the risk assessment</p>	<p>1) The assumption potentially overestimates the HCN air concentration and hence the risk.</p> <p>2) Unlikely to materially under- or over-estimate risk.</p> <p>3) This potentially overestimates the risk.</p> <p>4) This potentially overestimates the risk.</p> <p>5) This potentially over- or under-estimates the risk.</p> <p>6) Unlikely to significantly affect the risk assessment</p>



Uncertainty/ Assumption	Comment	Effect on Risk Assessment
	is appropriate. If there is a lower pH (e.g. 7), more HCN will evaporate from the water surface resulting in a higher HCN concentration in air.	outcome.
<b>TSF is attractive to bats.</b>	Bats may be attracted to the TSF water if it acts as a potential food source for insects. This is considered unlikely since vegetation in the direct vicinity of the TSF will be sparse (as it will be removed). This means there is unlikely to be significant food sources for insects. Nevertheless bats are known to visit gold mine TSFs containing cyanide.	This potentially overestimates mortality risk to bat populations.
<b>Form of cyanide</b>	All toxicity experiments with bats and birds have been conducted with NaCN. It has been assumed in the risk assessment all WAD will behave like and have the same toxicological potency as free cyanide (CN <sup>-</sup> ). This is a conservative assumption because copper cyanide (the form most likely to be present in measured WAD) is decidedly less toxic than sodium cyanide.	This overestimates the risk.

**8.2.2 Conclusion**

The risk assessment for bat inhalation exposure above the surface of the TSF has shown there is negligible risk of bat deaths from inhalation of HCN. A number of assumptions have been made in arriving at this conclusion which tend to over- rather than under- estimate the risk.

**8.3 Bat oral exposure**

Inhalation exposure by bats has been quantitatively assessed in Section 8.2, and was found to be negligible and so is not considered to meaningfully contribute to the overall exposure of bats to cyanide in the TSF. As with birds oral exposure from drinking TSF water is the exposure route of concern. Not combining inhalation and oral exposure simplifies the risk assessment.

For birds mortality risk was characterised by comparing the NOEL<sub>mortality</sub> with the estimated intake of cyanide (assuming WAD cyanide is toxicologically equivalent to CN<sup>-</sup>). The same margin of exposure (MoE) risk characterisation method has been used for oral exposure of bats to TSF water. However



since NOEL<sub>mortality</sub> data were not available for bats, the LD<sub>50</sub> of 4.5 mg CN<sup>-</sup>/kg from Clark et al (1991) for little brown bats has been used to inform the potential impact of oral exposure to cyanide in the Dargues TSF. A NOEL<sub>mortality</sub> for bats has been derived by adjusting the bat LD<sub>50</sub> using the slope of the mortality dose response.

In a variety of birds and mammals the dose response for cyanide mortality is steep. This is also the case for bats (Clark et al 1991). The steepness of the dose response curve is demonstrated by the relatively small gap between experimental oral doses of CN<sup>-</sup> at which no mortality is observed (NOEL<sub>mortality</sub>) and doses resulting in death of half the test animals (LD<sub>50</sub>). The LD<sub>50</sub>:NOEL<sub>mortality</sub> ratio derived from various studies is presented in Table 8.3 and ranges from 1.1-2.6.

Table 8.3: Ratio of LD(C)<sub>50</sub>:NOEL(C)<sub>mortality</sub> in various studies

ORAL			
Species	LD <sub>50</sub> (mg CN/kg bw) <sup>a</sup>	NOEL <sub>mortality</sub> (mg CN/kg bw) <sup>a</sup>	Ratio
Mallard duck	1.4 <sup>b</sup>	0.53 <sup>c</sup>	2.6
Mallard duck	18 <sup>d</sup>	16.4 <sup>d</sup>	1.1
	(43 mg CN/L)	(28 mg CN/L)	1.5
Black vulture	2.5 <sup>e</sup>	1.6 <sup>e</sup>	1.6
Bobwhite quail	69.0 <sup>f</sup>	35.0 <sup>f</sup>	2.0
	(374 mg CN/L)	(168 mg CN/L)	2.2
INHALATION			
Species	LC <sub>50</sub> (mg HCN/m <sup>3</sup> )	NOEC <sub>mortality</sub> (mg HCN/m <sup>3</sup> )	Ratio
Rat	556 <sup>g</sup>	313 <sup>g</sup>	1.8
Rat	408 <sup>h</sup>	302 <sup>h</sup>	1.4
Rat	217 <sup>h</sup>	122 <sup>h</sup>	1.8
Rat	154 <sup>h</sup>	84 <sup>h</sup>	1.8
Mouse	357 <sup>g</sup>	221 <sup>g</sup>	1.6

<sup>a</sup> All oral experiments listed were conducted with NaCN. For this table, oral doses of NaCN have been converted by ToxConsult to equivalent doses of cyanide ion, i.e. mg CN/kg body weight by dividing by a conversion factor of 1.88.

<sup>b</sup> Data from Henny et al (1994).

<sup>c</sup> Data from Hagelstein and Mudder (1997a), as cited in NICNAS (2010).

<sup>d</sup> Data from Stence et al (1993a), as cited in ECETOC (2007). The doses in the table for the Stence et al. (1993a) study were calculated by ECETOC (2007) using measured water concentrations. The measured concentrations of CN<sup>-</sup> in this study were lower (by approximately a factor of 10 or more) than the nominal concentrations and as a result provide a more conservative estimation of the effect concentrations of cyanide than if the nominal concentrations were used in the calculations. Because the assumptions for calculating the doses from the measured water concentrations were not provided by ECETOC (2007), there is some uncertainty in the resulting doses. As a result, LD<sub>50</sub>:NOEL<sub>mortality</sub> ratios have been calculated using both the doses calculated by ECETOC (2007) and the measured water concentrations in the study. The measured water concentrations are provided in brackets in the table.

<sup>e</sup> Data from Wiemeyer et al (1986).

<sup>f</sup> The doses in the table are from Stence et al (1993b) as calculated by ECETOC (2007). ECETOC (2007) appears to have determined the intake doses based on body weight and average drinking water consumption rates over the test period, however the assumptions used in the calculations are not provided. LD<sub>50</sub>:NOEL<sub>mortality</sub> ratios in this table have been calculated using both the doses derived by ECETOC (2007) and the nominal water concentrations in the study (in brackets).

<sup>g</sup> Data from Higgins et al (1972).

<sup>h</sup> Data from Du Pont (1981), as cited in NRC (2002).

Surprisingly oral NOEL<sub>mortality</sub> information for mammals (e.g. rats and mice) is not readily available, however inhalation data suggests the inhalation dose response curve in mammals is as steep as the



oral dose response for mortality in birds. The  $LC_{50}:NOEC_{mortality}$  ratios from various inhalation studies in rats and mice range from 1.4-1.8 (Table 8.3).

The mode of action by which cyanide causes mortality is the same for all species, the primary detoxification process and tissue distribution is also essentially the same in all species. Hence there is no *a priori* reason for the slope of the dose response curve for cyanide mortality in bats to be markedly different from other species. It is also very steep, as was noted by Clark et al (1991), with the  $LD_{50}:NOEL_{mortality}$  ratio likely to be within the range observed for other animals (1.1-2.6), the midpoint of the range is 1.8. Thus for characterising the mortality risk to bats in the locality of Dargues Reef an assessment (i.e. adjustment) factor of 2 has been used to convert the little brown bat  $LD_{50}$  of 4.5 mg CN/kg bw (Clark et al 1991) to a  $NOEL_{mortality}$  of 2.25 mg CN/kg bw. This  $NOEL_{mortality}$  has been used to characterise the mortality risk to bats visiting the TSF and drinking the water.

Because the reported data in any given study is sometimes less than ideal for determining the slope of the mortality dose response there is uncertainty regarding the calculated slope (i.e. the  $LD_{50}:NOEL_{mortality}$  or  $LC_{50}:NOEC_{mortality}$  ratio) for some studies. Nevertheless, regardless of whether cyanide exposure is by ingestion or inhalation there is good concordance in the calculated slopes for the various studies in Table 8.3. Consequently we consider the derived bat  $NOEL_{mortality}$  to be a reasonable approximation of the likely true  $NOEL_{mortality}$ .

To characterise the risk of bat deaths from drinking the TSF water, estimated CN intake was compared with the  $NOEL_{mortality}$  derived above (i.e. the margin of exposure,  $MoE_{mortality}$  was calculated).

Equation 7.1 was used to calculate the oral intake for bats (Table 8.4) except the US EPA (1993) allometric equation for daily water intake by mammals was applied<sup>23</sup>. Assumptions are:

- As no information could be found for how much water a bat may drink in a short time, the same percentage of the daily intake in birds (15%) was also assumed to be ingested over a short time for bats (see also Section 7.3).
- In calculating the cyanide intake (mg CN/kg bw) from the assumed target WAD cyanide of 30 mg/L it was assumed all the WAD was free CN. Patently this is conservative.

#### Results:

Table 8.4 summarises the  $MoE_{mortality}$  for bats in the area of the Dargues mine lease. The  $MoE_{mortality}$  is simply the  $NOEL_{mortality}$  divided by the estimated oral CN intake assuming 30 mg WAD/L is free CN.

<sup>23</sup> The US EPA (1993) allometric equation is  $WI = 0.099 Wt^{0.9}$ , where WI equals water intake (L/day) and Wt equal body weight (kg).

Table 8.4: Calculated MoE<sub>mortality</sub> for bats

Common name	Bat weight <sup>a</sup> (g)	Estimated water intake <sup>c</sup> (mL/d)	Calculated oral intake (mg/kg) <sup>d</sup>	MoE <sub>mortality</sub> <sup>e</sup>
Little forest bat	4 – 4.5	0.7 – 0.8	0.79-0.8	2.9
Large forest bat	6	1.0	0.75	3.0
Southern forest bat	5 – 5.5	0.8 – 0.9	0.72-0.73	3.1
Chocolate wattle bat	14 - 15	2.1 – 2.3	0.68-0.69	3.3
Gould's wattled bat	14 - 15	2.1 – 2.3	0.68-0.69	3.3
Gould's Large-eared bat	11.5 <sup>b</sup>	1.8	0.70	3.2
Lesser Large-eared bat	8 - 10	1.3 – 1.6	0.72-0.73	3.1
False Pipistrelle <sup>b</sup>	19 - 21	2.8 – 3.1	0.66	3.4

<sup>a</sup> Body weights from Parks Tas (2008).

<sup>b</sup> Body weight from Turbill and Geiser (2006).

<sup>c</sup> Estimated with US EPA (1993) allometric equation for mammalian daily water intake ( $WI = 0.099 Wt^{0.9}$ , where WI equals water intake (L/day). Consistent with the water intake per drinking event for birds (NICNAS 2010) 15% of daily intake was assumed per event.

<sup>d</sup> Estimated using Equation 7.1. If there was a range of body weights and water intakes for a particular species, the mid-point of the range was used for calculating the intake.

<sup>e</sup> This is the margin of exposure between the calculated intake and the NOEL<sub>mortality</sub> of 2.25 mg/kg for little brown bats. See text for derivation of the NOEL<sub>mortality</sub>.

The MoE between the estimated oral intakes of cyanide by bats drinking TSF water and the dose resulting in no mortality (NOEL<sub>mortality</sub>) were all greater than 1 (average 3.2). This suggests bat populations are adequately protected from mortality if exposed to TSF water containing a WAD cyanide concentration of 30 mg/L. The relatively small MoEs suggest there may be a possibility some bats may die if they visit the TSF and drink more water than has been assumed in the risk assessment. However the risk assessment assumes the WAD cyanide in the TSF has the same toxicological potency as NaCN used in the toxicological experiments providing the data for the risk characterisation. In fact, as described in the assessment for birds (Section 7.3) WAD cyanide is distinctly less toxic than NaCN. Therefore a MoE of approximately 3 does not necessarily indicate likelihood that bat deaths will occur.

Calculations in the table are for a target WAD of  $\leq 30$  mg/L 100% of the time. However it is noted recent regulatory licenses for cyanide concentrations in TSFs indicate the WAD concentration should be  $\leq 20$  mg/L for 90% of the time (Section 5.4). At a WAD of 20 mg/L the MoE<sub>mortality</sub> will be a third higher, and the risk of mortality lower.

Uncertainties and their influence on the risk assessment are further discussed in Section 8.3.1.



### 8.3.1 Bat oral assessment uncertainties

Most of the uncertainties associated with assessment of mortality risk to bats from ingesting TSF water are the same as for birds. Therefore to reduce repetition the uncertainties are discussed in Section 7.3.2 of the bird risk assessment.

A few differences from the bird risk assessment are the toxicity reference value for bats associated with oral exposure and the assumption that the slope of the oral dose response for bats is the same as for birds:

- Bats in the Dargues mine lease area are assumed to be as sensitive as little brown bats. LD<sub>50</sub> data for little brown bats is the only data available for bats, so it has been used. It is unknown if local species are more or less sensitive. This potentially underestimates the risk if more sensitive species than the little brown bat drink from the TSF.
- The slope of the oral dose response for bats has been assumed to be similar to birds. The chemical mode of action for cyanide lethality and cyanide detoxification is the same for all species. Thus it is reasonable to assume the relationship between the LD<sub>50</sub> and NOEL<sub>mortality</sub> will be similar between species. This is evinced by the information in Section 8.3. Therefore, this assumption is unlikely to materially over- or under-estimate the risk.

### 8.3.2 Conclusion

The MoEs between calculated oral intakes and the dose (in bats) resulting in no mortality (NOEL<sub>mortality</sub>) were all greater than 1 (average 3.2). This suggests bat populations are adequately protected from mortality if exposed to TSF water containing a WAD cyanide concentration of 30 mg/L. The assumed target WAD concentrations in this report are  $\leq 30$  mg/L 100% of the time, but  $\leq 20$  mg/L for 90% of the time (Section 5.4). At a WAD of 20 mg/L the MoE<sub>mortality</sub> will be a third higher, and the risk of mortality lower. Since the assumptions used in the calculations are conservative and for a population of bats tend to over- rather than under-estimate the mortality risk, it is concluded there is negligible risk to bat populations from exposure to the TSF.

## 9. Off-site risk assessments to cyanide containing water

### 9.1 Exposure scenarios

Exposure of organisms to water containing cyanide outside the TSF requires such water to find its way into groundwater and/or surface waters downstream from the TSF. These exposure pathways essentially require mechanical failure of the infrastructure handling cyanide process water or effluent containing cyanide. They include leakage from pipework or tanks, seepage underground from the



TSF, the TSF overtopping and catastrophic failure of the TSF embankment wall. Each is discussed below. For most off-site (i.e. non-TSF) exposures it is not possible to enumerate concentrations of biologically available cyanide to which organisms will be exposed. The risk is therefore addressed qualitatively in the context of infrastructure safeguards and in some instances semi-quantitatively assuming dilutions by rainfall and receiving waters.

#### 9.1.1 Pipework leakage<sup>24</sup>

There are areas of the mine site where cyanide will be used for gold extraction, or where tailings containing cyanide will be treated in preparation for discharge to the TSF. These areas will be bunded to contain and facilitate collection of spilled or leaked materials.

Pipework transferring tailings from the main process areas will run within bunded areas or be double skinned. Alarm systems will be in place to detect leaks in the internal pipes, and will be coupled with automatic shutdown of pumps. Thus leakage of material containing cyanide will be prevented and cyanide entry into the environment very unlikely.

Because it is not possible to predict concentrations of cyanide that might be in the local environment should pipework leakage occur, risk to organisms and persons from this exposure pathway has not been considered further. That there is negligible risk relies on infrastructure integrity, its appropriate maintenance and operational success of safety systems.

#### 9.1.2 TSF seepage

With regards to seepage, leaching into groundwater could theoretically be a potential exposure route for stygofauna (groundwater organisms) and aquatic organisms (if the groundwater recharges the nearby waterways) (see EIA). Migration into groundwater primarily occurs below unlined TSFs, TSFs with poorly designed and constructed liners, or where liner integrity has been violated (NICNAS 2010). Seepage of water from the Dargues TSF is considered unlikely due to the TSF design. The TSF design incorporates:

- an impermeable plastic membrane (smooth high density polyethylene) above
- a heavily compacted low permeability layer of water conditioned soil.
- Hydraulic pressure above the membrane and compacted soil barriers driving potential seepage will be reduced by an underdrainage system comprising an array of perforated drainage pipes in a permeable drainage medium that will collect water percolating through

---

<sup>24</sup> The term 'pipework leakage' includes leakage from storage and process tanks, or vehicles unloading cyanide caustic solution, pipework between them as well as the pipework transferring tailings to the TSF.



TSF solids<sup>25</sup>. The water will be directed towards a sump where it is reclaimed and pumped back into the gold recovery process.

- The likelihood of seepage reaching groundwater or surface water will be further reduced by a seepage interception and detection system below the membrane, which will capture any seepage in the unlikely event it occurs. Water that may have passed through the linings is collected and pumped back into the gold recovery process.

In seepage flow modelling, which took into consideration flow from partially saturated tailings, an equivalent overall basin permeability of  $3.2 \times 10^{-10}$  to  $2 \times 10^{-9}$  m/s was estimated by Knight Piésold (KP 2015). These levels are about the same or lower than the  $1 \times 10^{-9}$  m/s proposed by BIM. Thus, the potential for, and rate of, seepage to underlying strata from the TSF at the Mine will be negligible.

Free cyanide in water that does seep into the underlying basement geology would become non-biologically available as a result of complexation with trace metals in the basement rocks and/or microbial degradation. This is evinced by free cyanide being rarely detected in groundwater; where it has been detected, this was associated with spills from equipment failure, damage, or poor dam construction and from uses not relevant to this particular assessment (DEFRA 2002, ATSDR 2006, ECETOC 2007, NICNAS 2010).

Because the potential for seepage from the TSF will be negligible, this seepage exposure scenario has not been considered further in the off-site risk assessments.

Despite the above engineering design precautions, and in order to provide for adaptive management of seepage-related discharge, the Mine will monitor cyanide concentrations in Spring Creek, Majors Creek and in an array of monitoring bores surrounding the TSF.

### **9.1.3 TSF overtopping**

KP (2011) discusses the likelihood of the TSF overtopping in an extreme storm event.

The TSF is designed to hold water from an extreme rainfall event that may deposit 705mm of water in 72 hours (i.e. a 1 in 1,000 rainfall event). To protect the integrity of the TSF embankment there will be a clean water diversion dug around the TSF perimeter that will divert runoff water from the surrounding hillside and prevent it flowing into the TSF. Thus rainwater entering the TSF will only be from what falls directly onto the TSF surface. Nevertheless an emergency spillway from the TSF will

---

<sup>25</sup> Dry TSF solids have the consistency of coarse sand.



allow control of discharge from the TSF should the TSF be in danger of overtopping should such a rare rainfall event take place.

In circumstances of TSF spillway flow, not only will the cyanide in the TSF water be extremely diluted by rain, but the receiving Spring Creek, and those further downstream will also be swelled with water and further dilute any overflow from the TSF. KP (2015) has undertaken an analysis of TSF water dilution potentially discharged from the TSF emergency spillway to Spring Creek and further downstream. The modelling predicted the dilution resulting from a 1 in 2000, 1 in 10000 and 1 in 10 million year rare rainfall events. These rainfall events correspond to 789, 936 and 1,728 mm of rain in a 72-hour period, respectively. The modelling indicates a TSF water dilution of approximately 320 – 18,200 times where it enters Spring Creek in the event of an overflow, the dilution at the confluence of Spring and Majors Creeks is estimated to be approximately 2,400 - 146,700 times, and approximately 3.5 km downstream of the Project Site boundary the dilution is predicted to be approximately 4,900 - 303,800 times.

#### 9.1.3.1 Aquatic risk assessment

This section evaluates the potential impact of cyanide concentrations in overflow water.

As discussed above, in the unlikely occasion of water running down the spillway, TSF cyanide concentrations will be significantly diluted by rain and by receiving creek water. Modelling by KP (2015) indicates the dilution will range from approximately 320 – 303,800 times depending on the rainfall scenario and location. Furthermore the TSF cyanide will most likely be present as metallo-cyanides (e.g. copper cyanide), which, as discussed in the companion toxicity profile to this risk assessment (ToxConsult 2014), are significantly less toxic to aquatic organisms than free cyanide (NPS 1997, ANZECC 2000, Little et al 2007, NICNAS 2010). HCN and free cyanide, rather than metal cyanides, are the principal toxic forms to aquatic organisms. Free cyanide concentration is considered the most appropriate indicator of risk to aquatic organisms (Redman and Santore 2012). Consequently national and international guidelines protecting aquatic organisms are for free cyanide, rather than total cyanide or WAD (e.g. ANZECC 2000).

Fish are the most sensitive aquatic organisms to free cyanide, followed by invertebrates (NPS 1997, MERG 2001, Eisler and Wiemeyer 2004, NICNAS 2010). Amphibians are less sensitive than fish<sup>26</sup>.

<sup>26</sup> Acute toxicity data for amphibians were sourced from the United States EPA Ecotox database (US EPA 2015). Data were available for 9 different species of amphibians, and 96-hour LC<sub>50</sub> values ranged from 192 to 11,472 µg/L CN<sup>-</sup>, whereas for fish LC<sub>50</sub>'s are lower ranging from 27 to 1,200 µg/L CN<sup>-</sup>. Although only two NOECs were available for amphibians (<48h hatched tadpoles) (32.5 µg/L and 172.5 µg/L CN<sup>-</sup>), these were also higher than NOECs for sublethal effects in fish (1 µg/L to 29 µg/L CN<sup>-</sup>).



Algae and aquatic plants are comparatively tolerant to cyanide. Thus if fish are adequately protected it follows that other aquatic organisms are also protected.

Acute toxicity of free cyanide to fish as 96-hour LC<sub>50</sub>s (i.e. concentrations lethal to 50% of the test population) ranges from 27 to 1,200 µg/L depending on the experimental design and species tested (ECETOC 2007, ANZECC 2000). Similar to mammals and birds, the dose-response for lethal effects is very steep in fish. Cyanide concentrations without lethality (LC<sub>0</sub>) or up to 10% mortality (LC<sub>10</sub>) are close to the LC<sub>50</sub>, particularly for sensitive species such as rainbow trout (ECETOC 2007). In chronic toxicity studies ranging in duration from 20-256 days, No Observed Effect Concentrations (NOECs) for sublethal effects range from 1 to 29µg/L for fish (ECETOC 2007, Eisler and Wiemeyer 2004). Sublethal effects of cyanide on fish include impaired swimming and reproduction (e.g. spawning, egg production, spermatogenesis) (NICNAS 2010, NPS 1997, Eisler and Wiemeyer 2004).

A range of acute and chronic toxicity data were used in the development of the ANZECC water quality guidelines for cyanide (ANZECC 2000). The cyanide freshwater trigger value for protection of 95% of aquatic species is 7 µg CN/L, and for protection of 99% of species is 4 µg CN/L.

Target WAD concentrations for the TSF are assumed to be ≤20 mg (90% of the time) and ≤30 mg/L (100% of the time) (Section 5.5). However to account for operation variability of the cyanide destruction plant, to achieve these targets the WAD cyanide in effluent from the plant will need to be lower.

Assuming a maximum WAD concentration of 30 mg/L at the TSF spigot:

- If the concentration of free cyanide is conservatively assumed to be 35% of WAD (see Section 8.2), i.e. the same as for the CIL effluent input to the destruction unit, the free cyanide concentration at the discharge point into the TSF is potentially 10.5 mg/L.
- The prospective concentrations of free cyanide at various points in the Deua River system can be estimated using the dilution factors predicted by Knight Piésold (2015). The estimated free cyanide concentrations are provided in Table 9.1.

**Table 9.1: Predicted free cyanide concentration (µg/L) at modelling locations in Deua River system**

Location	Predicted dilution <sup>a</sup>	Predicted free cyanide concentration (µg/L)
Spring Creek (where overflow meets creek)	316 – 18,220	0.6 - 33



Location	Predicted dilution <sup>a</sup>	Predicted free cyanide concentration (µg/L)
Confluence of Major & Spring Creeks	2,386 – 146,739	0.07 – 4.4
Majors Creek; 3.5 km downstream from confluence	4,917 – 303,813	0.03 – 2.1

<sup>a</sup> Dilution factors as provided by KP (2015)

Aquatic ecology assessments conducted by Cardno Pty Ltd in 2011 and 2013 identified habitat quality within the upper reaches of the study area (Spring Creek) and within the boundary of the Project Site to be moderately to heavily disturbed and below that of a reference control site. It is therefore appropriate to use a 95% species protection trigger value (i.e. for slightly-moderately disturbed systems) for assessing potential aquatic risks in Spring Creek.

In the event of an overflow the estimated free cyanide concentrations in Spring Creek, where the overflow meets the creek, are 0.6 – 33 µg/L, the highest of this range being about 5 times higher than the ANZECC (2000) 95% species protection level of 7 µg CN/L. Therefore at an assumed concentration of 30 mg/L WAD in the TSF, it is concluded at Spring Creek there is a potential risk of adverse impacts to aquatic organisms under certain rainfall conditions. The risk considerably decreases with higher dilutions downstream.

The concentrations of free cyanide at each of the other modelled points in the Deua River system are below the 95% trigger value of 7 µg/L and at or below the 99% trigger value of 4 µg/L for pristine waterways. It is therefore concluded the potential risk of cyanide impacts on aquatic organisms as a result of TSF overflow at locations downstream from the confluence of Major and Spring Creeks is low.

To ensure negligible risk to aquatic organisms in Spring Creek and compliance with a target free cyanide concentration at or below 7 µg/L in Spring Creek, the concentration of WAD cyanide in the TSF pond would need to be less than or equal to 6.3 mg/L<sup>27</sup> when the TSF water dilution is at the predicted minimum of just 316 times.

This assessment is conservative because:

- This worst case (i.e. minimum) dilution coincides with the worst case (i.e. minimum) capacity of the TSF to accept stormwater before overflowing down the spillway. Since TSF capacity to accept stormwater increases as new lifts are added, higher concentrations of WAD cyanide in the TSF at these times may be acceptable for meeting the ANZECC trigger concentrations in

<sup>27</sup> 7 µg CN/L x 316 dilution (i.e. lowest estimated dilution) ÷ 0.35 [assumed ratio of free cyanide to WAD cyanide] = 6,320 µg/L WAD cyanide (i.e. 6.3 mg/L)



downstream receiving waters. This is because the extra TSF freeboard volume increases dilution of TSF water prior to its overflow into Spring Creek.

- The chemical reactions in the detoxification unit will favour free  $CN^-$  over WAD (although both are destroyed). As the detoxification effluent moves from the detoxification unit to the TSF spigot, residual sulphite will continue to react with free cyanide, reducing the residual free  $CN^-$  concentration below that assumed in this assessment (CSIRO 2014).
- The assessment assumes WAD concentration in the TSF standing water is equivalent to that at the discharge spigot. However, studies have shown WAD concentrations are 2-9 fold lower in the TSF pond compared to the spigot (Henny et al 1994, Griffiths et al 2014a).

#### **9.1.3.2 Off-site human health risk assessment**

In the event of an extreme rainfall event, spillway water from the TSF will flow into Spring Creek and then into Majors Creek. Water from Majors Creek may be utilised by persons downstream of the Mine as a source of drinking water, for irrigation, or filling swimming/wading pools. Of these use patterns, drinking water use is the more sensitive from a human health perspective. Consequently the risk assessment addresses this potential exposure on the basis that if there is low risk from drinking water from the creek the health risk associated with other uses will be lower and will not require formal assessment.

Target WAD concentrations for the TSF are  $\leq 20$  mg (90% of the time) and  $\leq 30$  mg/L (100% of the time) (Section 5.5).

- In the event the TSF overtops it is feasible Majors Creek will receive diluted overflow for a relatively short time. The World Health Organization drinking water guideline for short term exposure ( $\leq 5$  days) is 0.5 mg/L (total cyanide) (WHO 2011). At a target maximum of 30 mg/L in the TSF water, it only requires a 60 fold dilution to achieve the WHO drinking water guideline.
- The Australian drinking water guideline for long term consumption is 0.08 mg/L (NHMRC 2013). At a TSF concentration of 30 mg/L the chronic drinking water guideline is achieved with a 375 times dilution.

Both the WHO and NHMRC drinking water guidelines have been developed to protect human health.



The dilutions estimated (Table 9.1) by Knight Piesold (2015) at the relevant locations where water abstraction may occur (i.e. Majors Creek) result in water concentrations less than the short term and long term WHO and NHMRC drinking water guidelines. Therefore the risks of human health impacts from cyanide in the event of the TSF overtopping are low. It is however likely that other aspects of water quality (e.g. turbidity) will render the water unsuitable and unpalatable for drinking.

#### **9.1.4 Catastrophic TSF embankment wall failure**

An assessment of the consequences of a breach of the TSF embankment wall has been undertaken by Knight Piesold (KP 2011). It was recognised the consequences of the downstream rural/productive environment could be significant if there was a breach of the wall. The dam has therefore been designed, assuming worst case conditions, to ensure such an occurrence is highly unlikely. The presence of the clean water diversion and the spillway protects the integrity of the TSF embankment wall.

KP (2011) undertook flood inundation modelling for both dam “failure” and “non-failure” scenarios. It addresses the likelihood and consequences of a dramatic failure of the TSF wall by an earthquake event.

Other than noting the town of Majors Creek is not expected to be impacted by a breach of the facility and the risk from cyanide exposure in a tailings slide event would be low relative to other risks, this scenario is not considered further in the off-site risk assessments.



## References

- ACGIH (2007). Industrial Ventilation 26<sup>th</sup> Edition. A Manual of Recommended Practice. American Conference of Governmental Industrial Hygienists, Ohio.
- ANZECC (2000). Australian and New Zealand guidelines for fresh and marine water quality. Australian and New Zealand Environment and Conservation Council.
- BIM (2013). Air quality and greenhouse gas management plan for the Dargues gold mine. Big Island Mining Limited and Unity Mining Limited. Document number: DAR-HSE-PO-QU-PLN-5904.
- Birds in Backyards (undated a). Australasian shoveler (*Anas rhynchotis*). Birds Australia. [Accessed 15/12/2014]. <http://www.birdsinbackyards.net/species/Anas-rhynchotis>.
- Birds in Backyards (undated b). Chestnut teal (*Anas castanea*). Birds Australia. [Accessed 15/12/2014]. <http://www.birdsinbackyards.net/species/Anas-castanea>.
- Birds in Backyards (undated c). Hardhead (*Aythya australis*). Birds Australia. [Accessed 15/12/2014]. <http://www.birdsinbackyards.net/species/Aythya-australis>.
- Bouglouan, N. (undated). Pacific black duck (*Anas superciliosa*). Oiseaux (Birds). [Accessed 15/12/2014]. <http://www.oiseaux-birds.com/card-pacific-black-duck.html>.
- Cardno (2011). Dargues Reef Gold Project Aquatic Ecological Assessment. Job Number EL1112025. November 2011.
- Cardno (2013). Dargues Gold Mine 2013 Aquatic Ecology Monitoring Report. Job Number NA49913133. 6 January 2014.
- Clark Jr, D. R. and Hothem, R. (1991). Mammal mortality at Arizona, California and Nevada gold mines using cyanide extraction. Calif Fish Game. 77: 61-69. As cited in Griffiths et al 2014a.
- Clark DR, Hill EF, Henry PFP (1991). Comparative sensitivity of little brown bats *Myotis lucifugus* to acute dosages of sodium cyanide. Bat Res News 32: 68 [Abstract]. Abstracts of Papers presented at the 21<sup>st</sup> Annual North American Symposium on Bat Research.
- CSIRO (2014). Cyanide Destruction for the Dargues Reef Project. By D Hewitt. Document EP 143597.
- Cummings, J., Duke, G. and Jegers, A. (1975). Corrosion of bone by solutions simulating raptor gastric juice. Raptor Research. 10: 55-57.
- Dept of Env (2006). National Pollutant Inventory Emission estimation technique manual for gold ore processing, Version 2.0. Department of the Environment and Heritage. <http://www.npi.gov.au/sites/www.npi.gov.au/files/resources/9dbbea8f-b557-edc4-a103-92ecc881d463/files/gold.pdf>.
- DEWHA (2009). Environmental risk assessment guidance manual for industrial chemicals. Prepared by Chris Lee-Steere. Department of the Environment, Water, Heritage and the Arts.
- Donato, D. (1997). Current bird usage patterns on Northern Territory mine water impoundments and their management to reduce mortalities. MCA, Minerals Council of Australia environmental workshop. Adelaide. As cited in Donato et al 2007.



Donato, D. (1999). Bird usage patterns on northern territory mining water tailings and their management to reduce mortalities. Darwin, Northern Territory: Public Report Department of Mines & Energy. As cited in Donato et al 2007.

Donato, D. B., Nichols, O., Possingham, H., Moore, M., Ricci, P. F. and Noller, B. N. (2007). A critical review of the effects of gold cyanide-bearing tailings solutions on wildlife. *Environment International*. 33: 974-984.

Donato, D. B. and Smith, G. B. (2007). Summary of findings: ACMER project 58, Sunrise Dam Gold Mine Sponsor's Report, AngloGold Ashanti Australia. <http://www.cyanidecode.org/sites/default/files/pdf/AngloSunriseStudyFindingsPeerReviews.pdf>.

Donato, D., Ricci, P. F., Noller, B., Moore, M., Possingham, H. and Nichols, O. (2008). The protection of wildlife from mortality: Hypothesis and results for risk assessment. *Environment International*. 34: 727-736.

DRET (2008). Cyanide management: leading practice sustainable development program for the mining industry. Department of Resources Energy and Tourism. <http://www.ret.gov.au/resources/documents/lpsdp/lpsdp-cyanidehandbook.pdf>.

ECETOC (2007). Cyanides of hydrogen, sodium and potassium, and acetone cyanohydrin (CAS No. 74-90-8, 143-33-9, 151-50-8 and 75-86-5). Volumes I and II. JACC No. 53. European Centre for Ecotoxicology and Toxicology of Chemicals. ISSN-0773-6339-53. Brussels, September 2007.

Eisler, R. and Wiemeyer, S. N. (2004). Cyanide hazards to plants and animals from gold mining and related water issues. *Reviews of Environmental Contamination and Toxicology*. 183: 21-54.

Gaia Research (2014). Summer 2013 fauna monitoring for the Dargues Gold Mine. Prepared by Garry Daly from Gaia Research Pty Ltd for Big Island Mining Pty Ltd. January 2014.

Gaia Research (2013). 2013 fauna monitoring for the Dargues Gold Mine. Prepared by Garry Daly from Gaia Research Pty Ltd for Big Island Mining Pty Ltd. March 2013.

Griffiths, S. R., Smith, G. B., Donato, D. B. and Gillespie, C. G. (2009). Factors influencing the risk of wildlife cyanide poisoning on a tailings storage facility in the Eastern Goldfields of Western Australia. *Ecotoxicology and Environmental Safety*. 72: 1579-1586.

Griffiths, S., Donato, D., Coulson, G. and Lumsden, L. (2014a). High levels of activity of bats at gold mining water bodies: implications for compliance with the International Cyanide Management Code. *Environmental Science and Pollution Research*. 21: 7263-7275.

Griffiths, S. R., Donato, D. B., Lumsden, L. F. and Coulson, G. (2014b). Hypersalinity reduces the risk of cyanide toxicosis to insectivorous bats interacting with wastewater impoundments at gold mines. *Ecotoxicology and Environmental Safety*. 99: 28-34.

Henny, C., Hallock, R. and Hill, E. (1994). Cyanide and migratory birds at gold mines in Nevada, USA. *Ecotoxicology*. 3: 45-58.

Hewitt, D., Breuer, P. and Jeffery, C. (undated). Cyanide detoxification of cyanideation tails and process streams. CSIRO Minerals Down Under National Research Flagship, Australia. pg. 62-75. <https://publications.csiro.au/rpr/download?pid=csiro:EP123765&dsid=DS3>.



Hudson, A. and Bouwman, H. (2009). Birds associated with the tailings storage facility and surrounds of a South African gold mine. Abstract of the International Mine Water Conference. 19-23rd October, 2009. Pretoria, South Africa. [http://www.imwa.info/docs/imwa\\_2009/IMWA2009\\_Hudson.pdf](http://www.imwa.info/docs/imwa_2009/IMWA2009_Hudson.pdf)

ICMI (2009). Implementation guide for the International Cyanide Management Code. International Cyanide Management Institute. <http://www.cyanidecode.org/sites/default/files/IGRevisions09.pdf>.

ICMI (2012). The International Cyanide Management Code. International Cyanide Management Institute. <http://www.cyanidecode.org/sites/default/files/pdf/thecode.pdf>.

KP (2011). Cortona Resources Limited. Dargues Reef Gold Project. Tailings Storage Facility Final Design. Knight Piesold Pty Ltd. Ref. PE801-00139/05. November 2011.

KP (2014). Memorandum RE: Dargues Gold Project – Concentrate TSF Conceptual Design. Knight Piesold Pty Ltd. Ref: PE14-00930. 23<sup>rd</sup> September 2014.

KP (2015). Dargues Gold Project Tailings Storage Facility Final Design Update (Report PE801-00139/10). Dated June 2015.

Little, E., Calfee, R., Theodorakos, P., Brown, Z. and Johnson, C. (2007). Toxicity of cobalt-complexed cyanide to *Oncorhynchus mykiss*, *Daphnia magna*, and *Ceriodaphnia dubia*. *Environmental Science and Pollution Research - International*. 14: 333-337.

Logsdon, M. J., Hagelstein, K. and Mudder, T. I. (1999). The management of cyanide in gold extraction. *International Council on Metals and the Environment*. <http://static.squarespace.com/static/516c6bb1e4b0e4e26c1d8e53/t/51b9546ae4b003975d41bbca/1371100266892/ICMMCY~1.PDF>.

MERG (2001). Cyanide - The facts. By Laberge Environmental Services. For Mining Environment Research Group. MERG Report 2001-2. Whitehorse, Yukon. [http://www.geology.gov.yk.ca/pdf/MPERG\\_2001\\_2.pdf](http://www.geology.gov.yk.ca/pdf/MPERG_2001_2.pdf).

NEPM (2013). National Environment Protection (Assessment of Site Contamination) Measure 1999, as amended 16 May 2013. Prepared by the Office of Parliamentary Counsel, Canberra. Federal Register of Legislative Instruments F2013C00288.

NHMRC and NRMCC (2013). National Water Quality Management Strategy. Australian Drinking Water Guidelines 6. Version 2.0 Updated December 2013. National Health and Medical Research Council and Natural Resource Management Ministerial Council. [https://www.nhmrc.gov.au/files/nhmrc/publications/attachments/eh52\\_aust\\_drinking\\_water\\_guidelines\\_update\\_131216.pdf](https://www.nhmrc.gov.au/files/nhmrc/publications/attachments/eh52_aust_drinking_water_guidelines_update_131216.pdf).

NICNAS (2010). Priority Existing Chemical Assessment Report No. 31: sodium cyanide. National Industrial Chemical Notification Assessment Scheme. [http://www.nicnas.gov.au/data/assets/pdf\\_file/0018/4392/PEC\\_31\\_Sodium-Cyanide\\_Full\\_Report\\_PDF.pdf](http://www.nicnas.gov.au/data/assets/pdf_file/0018/4392/PEC_31_Sodium-Cyanide_Full_Report_PDF.pdf).

NPS (1997). Environmental contaminants Encyclopedia: entry on cyanide(s) in general. Compiled/edited by Roy J. Irwin, National Park Service, with assistance from Colorado State University. Colorado, USA. <http://www.nature.nps.gov/hazardssafety/toxic/cyanide.pdf>.

NSW Gov (1995). Threatened Species Conservation Act 1995. As at 17 October 2014. New South Wales Consolidated Acts. [http://www.austlii.edu.au/au/legis/nsw/consol\\_act/tsca1995323/](http://www.austlii.edu.au/au/legis/nsw/consol_act/tsca1995323/).



NT DoME (1998). Best Practice Guidelines . Reducing Impacts of Tailings Storage Facilities on Avian Wildlife in the Northern Territory of Australia. Northern Territory Department of Mines and Energy. [http://www.nt.gov.au/d/Minerals\\_Energy/Content/File/Forms\\_Guidelines/AVIAN\\_WILDLIFE\\_GUIDELINES.pdf](http://www.nt.gov.au/d/Minerals_Energy/Content/File/Forms_Guidelines/AVIAN_WILDLIFE_GUIDELINES.pdf).

NZ Birds Online (undated). Paradise shelduck (*Tadoma veriegata*). New Zealand Birds Online (Digital Encyclopaedia of New Zealand Birds). [Accessed 15/12/2014]. <http://nzbirdsonline.org.nz/species/paradise-shelduck>.

Parks Tas (2008). Bats. Parks & Wildlife Service Tasmania. <http://www.parks.tas.gov.au/index.aspX?base=4905>.

Redman, A. and Santore, R. (2012). Bioavailability of cyanide and metal–cyanide mixtures to aquatic life. *Environmental Toxicology and Chemistry*. 31: 1774-1780.

Rhoades, D. D. and Duke, G. E. (1975). Gastric function in a captive American bittern. *The Auk*. 92: 786-792.

RWC (2015). Environmental Assessment for the Dargues Gold Mine – Modification 3. RW Corkery & Co Pty Limited.

SGS (2005). Cyanide destruction. SGS Minerals Services - T3 SGS 018. <http://www.sgs.com.au/~media/Global/Documents/Flyers%20and%20Leaflets/SGS-MIN-WA017-Cyanide-Destruction-EN-11.pdf>.

Stence M, Beavers JB, Jaber M (1993a). Sodium cyanide: an LC50 study with the mallard using water borne exposure. Unpublished report HLO 481-93 amended 17 June 1993, project 112-306 by

ToxConsult (2015) Toxicity profile for cyanide. ToxConsult document ToxCR030914-TRF.

Turbill, C. and Geiser, F. (2006). Thermal physiology of pregnant and lactating female and male long-eared bats, *Nyctophilus geoffroyi* and *N. gouldi*. *Journal of Comparative Physiology B*. 176: 165-172.

US EPA (1993). Wildlife Exposure Factors Handbook, Volumes I and II. EPA/600/R-93/187a&b. Office of Health and Environmental Assessment, Office of Research and Development, United States Environmental Protection Agency.

US EPA (2006). Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments - Interim Final. Waste and Cleanup Risk Assessment, Office of Solid Waste and Emergency Response. U.S. Environmental Protection Agency.

US EPA (2015). US EPA ECOTOX database. Search results for “cyanide”. United States Environmental Protection Agency. [http://cfpub.epa.gov/ecotox/advanced\\_query.htm](http://cfpub.epa.gov/ecotox/advanced_query.htm).

WA DEC (2006). The use of risk assessment in contaminated site assessment and management. Guidance on the overall approach. Government of Western Australia, Department of Environment and Conservation.

WA DEC (2010). Assessment levels for soil, sediment and water. Western Australia Department of Conservation. Contaminated Sites Management Series. Version 4, revision 1.

WHO (2011). Guidelines for drinking-water quality. Fourth edition. World Health Organization. Geneva. [http://whqlibdoc.who.int/publications/2011/9789241548151\\_eng.pdf](http://whqlibdoc.who.int/publications/2011/9789241548151_eng.pdf).



Wildscreen Arkive (undated a). Australian wood duck (*Chenonetta jubata*). [Accessed 15/12/2014]. <http://www.arkive.org/australian-wood-duck/chenonetta-jubata/>.

Wildscreen Arkive (undated b). Grey teal (*Anas gracilis*). [Accessed 15/12/2014]. <http://www.arkive.org/grey-teal/anas-gracilis/>.

## Appendix A: Birds identified at the Mine

Bird species identified by Gaia Research (2014, 2013).

Common name	Species
Brown quail	<i>Coturnix ypsilophora</i>
Black swan	<i>Cygnus atratus</i>
Australian Shelduck	<i>Tadorna variegata</i>
Australian Wood duck	<i>Chenonetta jubata</i>
Black duck	<i>Anus superciliosa</i>
Australasian shoveler	<i>Anus rhynchotis</i>
Grey Teal	<i>Anus gracilis</i>
Chestnut Teal	<i>Anus castanea</i>
Hardhead	<i>Aythya australis</i>
Australasian Grebe	<i>Tachybaptus novaehollandiae</i>
Hoary-headed Grebe	<i>Tachybaptus poliocephalus</i>
Little Pied Cormorant	<i>Phalacrocorax melanoleucos</i>
Little Black Cormorant	<i>Phalacrocorax sulcirostris</i>
Australian Pelican	<i>Pelecanus conspicillatus</i>
White Faced Heron	<i>Egretta novaehollandiae</i>
Little Egret	<i>Egretta garzetta</i>
Pacific Heron	<i>Ardea pacifica</i>
Australian White Ibis	<i>Threskiornis molucca</i>
Straw-necked Ibis	<i>Threskiornis spinicollis</i>
Yellow-billed Spoonbill	<i>Platalea flavipes</i>
Black-shouldered Kite	<i>Elanus axillaris</i>
Brown Goshawk	<i>Accipiter fasciatus</i>
Wedge-tailed Eagle	<i>Aquila audax</i>
Little Eagle <sup>a</sup>	<i>Hieraaetus morphnoides</i>
Brown Falcon	<i>Falco berigora</i>
Australian Hobby	<i>Falco longipennis</i>
Black Falcon <sup>a</sup>	<i>Falco subniger</i>
Peregrine Falcon	<i>Falco peregrinus</i>
Nankeen Kestrel	<i>Falco cenchroides</i>
Dusky Moorhen	<i>Gallinula tenebrosa</i>
Eurasian Coot	<i>Fulica atra</i>
Common Sandpiper	<i>Actitis hypoleucos</i>
Black-fronted Dotterel	<i>Euseyornis melanops</i>
Masked Lapwing	<i>Vanellus miles</i>
Spotted Turtle-Dove	<i>Streptopelia chinensis</i>
Brown Cuckoo-dove	<i>Macropygia amboinensis</i>
Common bronzewing	<i>Phaps chalcoptera</i>
Crested pigeon	<i>Ocyphaps lophotes</i>
Wonga pigeon	<i>Leucosarica melanoleuca</i>



Common name	Species
Yellow-tailed black cockatoo	<i>Calyptorhynchus funereus</i>
Gang-gang cockatoo <sup>a</sup>	<i>Callocephalon fimbriatum</i>
Galah	<i>Cacatua roseicapilla</i>
Little Corella	<i>Cacatua sanguinea</i>
Sulphur-crested cockatoo	<i>Cacatua galerita</i>
Australian King Parrot	<i>Alisterus scapularis</i>
Crimson Rosella	<i>Platycercus elegans</i>
Eastern Rosella	<i>Platycercus eximius</i>
Pallid Cuckoo	<i>Cuculus pallidus</i>
Fan-tailed Cuckoo	<i>Cacomantis pyrrhophanus</i>
Brush Cuckoo	<i>Cacomantis variolosus</i>
Southern Boobook <sup>a</sup>	<i>Ninox novaeseelandiae</i>
Tawny Frogmouth	<i>Podargus strigoides</i>
White-throated needletail	<i>Hirundapus caudacutus</i>
Fork-tailed swift	<i>Apus pacificicus</i>
Laughing Kookaburra	<i>Dacelo novaeguineae</i>
Sacred Kingfisher	<i>Todiramphus sancta</i>
Dollarbird	<i>Eurystomus orientalis</i>
White-throated treecreeper	<i>Climacteris leucophaea</i>
Superb Fairy-wren	<i>Malurus cyaneus</i>
Spotted Pardalote	<i>Pardalotus punctatus</i>
Striated Pardalote	<i>Pardalotus striatus</i>
White-browed scrubwren	<i>Sericornis frontalis</i>
White-throated gerygone	<i>Gerygone olivaceae</i>
Buff-rumped Thornbill	<i>Acanthiza reguloides</i>
Yellow-rumped thornbill	<i>Acanthiza chrysorrhoa</i>
Brown Thornbill	<i>Acanthiza pusilla</i>
Yellow Thornbill	<i>Acanthiza nana</i>
Striated Thornbill	<i>Acanthiza lineata</i>
Weebill	<i>Smicronis brevirostris</i>
Red Wattlebird	<i>Anthochaera carunculata</i>
Little Wattlebird	<i>Anthochaera chrysoptera</i>
Noisy Friarbird	<i>Philemon corniculatus</i>
Noisy Miner	<i>Manorina melanocephala</i>
Lewin's Honeyeater	<i>Meliphaga lewinii</i>
White-naped Honeyeater	<i>Melithreptus lunatus</i>
Yellow-faced Honeyeater	<i>Lichenostomus chrysops</i>
White-eared Honeyeater	<i>Lichenostomus leucotis</i>
Brown-headed Honeyeater	<i>Melithreptus brevirostris</i>
Crescent Honeyeater	<i>Phylidonyris pyrrhoptera</i>
New Holland Honeyeater	<i>Phylidonyris novaehollandiae</i>
Eastern Spinebill	<i>Acanthorhynchus tenuirostris</i>
Jacky Winter	<i>Microeca leucophaea</i>
Eastern Yellow Robin	<i>Eopsaltria australis</i>
Scarlet Robin <sup>a</sup>	<i>Petroica boodang</i>
Flame Robin <sup>a</sup>	<i>Petroica phoenicea</i>
Eastern Whipbird	<i>Psophodes olivaceus</i>
Spotted Quail-thrush	<i>Cinclosoma punctatum</i>
Golden Whistler	<i>Pachycephala pectoralis</i>
Rufous Whistler	<i>Pachycephala rufiventris</i>



Common name	Species
Grey Shrike-thrush	<i>Colluricincla harmonica</i>
Grey Fantail <sup>a</sup>	<i>Rhipidura fuliginosa</i>
Willie Wagtail	<i>Rhipidura leucophrys</i>
Magpie Lark	<i>Grallina cyanoleuca</i>
Black-faced Monarch	<i>Monarcha melanopsis</i>
Black-faced Cuckoo-shrike	<i>Coracina novaehollandiae</i>
White-winged Triller	<i>Lalage sueurii</i>
Dusky Woodswallow	<i>Artamus cyanopterus</i>
Grey Butcherbird	<i>Cracticus torquatus</i>
Australian Magpie	<i>Gymnorhina tibicen</i>
Pied Currawong	<i>Strepera graculina</i>
Grey Currawong	<i>Strepera visicolor</i>
Australian raven	<i>Corvus coronoides</i>
Little raven	<i>Corvus mellori</i>
White-winged Chough	<i>Corcorax melanorhamphos</i>
Satin Bowerbird	<i>Ptilonorhynchus violaceus</i>
Skylark	<i>Alauda arvensis</i>
Richard's Pipit	<i>Anthus novaeseelandiae</i>
Red-browed Firetail	<i>Neochmia temporalis</i>
Diamond Firetail <sup>a</sup>	<i>Stagonopleura guttata</i>
European Goldfinch	<i>Carduelis carduelis</i>
Welcome Swallow	<i>Hirundo neoxena</i>
Tree Martin	<i>Hirundo nigricans</i>
Brown Songlark	<i>Cincloramphus cruralis</i>
Silvereye <sup>a</sup>	<i>Zosterops lateralis</i>
Common blackbird	<i>Turdus merula</i>
Common starling	<i>Sturnus vulgaris</i>

<sup>a</sup> Listed as a threatened species under the NSW Threatened Species Conservation Act (NSW Gov 1995).



## Appendix B: Bats identified at the Mine

Bat species identified by Gaia Research (2014, 2013).

Common name	Species
Little forest bat	<i>Vespadelus vulturnus</i>
Large forest bat	<i>V. darlingtoni</i>
Southern forest bat	<i>V. regulus</i>
Chocolate wattle bat	<i>Chalinolobus morio</i>
Gould's wattled bat	<i>C. gouldii</i>
Gould's Large-eared bat <sup>b</sup>	<i>Nyctophilus gouldii</i>
Lesser Large-eared bat	<i>N. geoffroyi</i>
<b>False Pipistrelle<sup>a</sup></b>	<i>Falsistrellus tasmaniensis</i>

<sup>a</sup> Listed as a threatened species under the NSW Threatened Species Conservation Act (NSW Gov 1995).

This page has intentionally been left blank