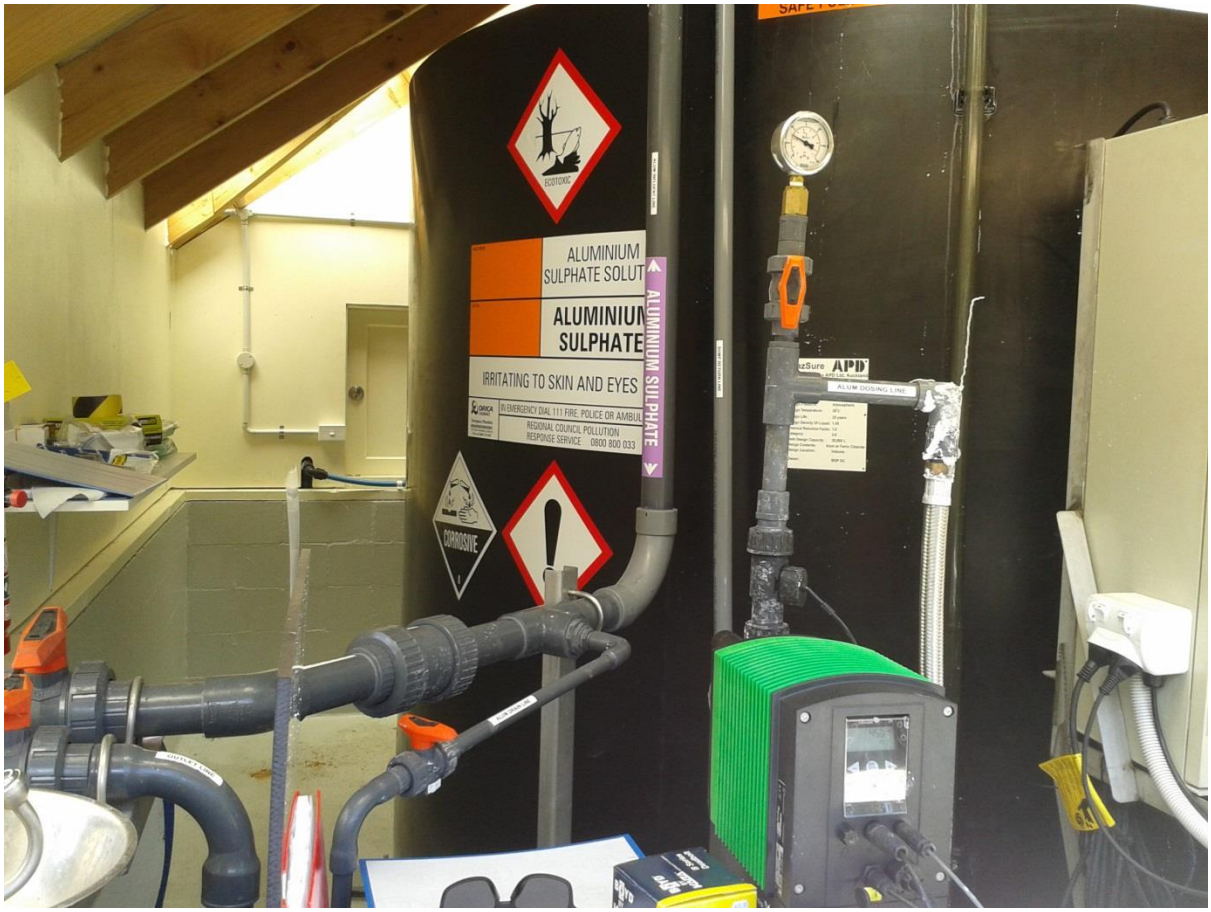
Ecotoxicological Review of Alum Applications to the Rotorua Lakes



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Lay Summary

Alum dosing is a highly effective method for removing phosphorus from freshwater systems, and is currently being employed by the Bay of Plenty Regional Council to help meet water quality targets for lakes Rotorua, Rotoehu and Okaro. Alum chemically binds both suspended particles and dissolved phosphorus before settling to the lake bed, a process known as flocculation. Over time, phosphorus bound alum becomes buried and unavailable for algal growth, resulting in improved water quality. This literature review was prepared in order to address a number of concerns regarding the toxicity of alum to aquatic organisms and the fate of alum in lake sediments.

The phosphorus binding component of alum is aluminium. Aluminium is a common element in the Earth's crust and under neutral pH conditions forms a white, non-toxic, solid substance. However, under acidic (low) or basic (high) pH, aluminium dissolves and becomes toxic to aquatic animals. Fish are the most susceptible group to aluminium toxicity effects, with first clogging of the gills and then disruption of their ability to regulate water content in the body occurring as pH decreases. This is of concern, as the addition of alum to water causes the water to become acidic and aluminium to become toxic. Therefore, the quantity of aluminium that can be safely added to a water body is dictated by its ability to resist the change in pH, otherwise known as buffering capacity. Most of the Rotorua lakes, and in particular Lake Rotorua, have very low buffering capacities, and therefore have limited resistance to acidification by alum. It is recommended that to preclude lethal effects in the Rotorua lakes total Al does not exceed 200 µg l^{-1} at pH >6.0.

The limited buffering capacity of the Rotorua lakes has meant that current alum application rates by the Bay of Plenty Regional Council are appropriately conservative. If alum dosing rates were to be increased, serious consideration must be given to the use of buffering agents such as sodium aluminate to prevent toxic conditions forming. The limited buffering capacity also means there will be little environmental resistance to the formation of toxic conditions should acidification of the dosing sites result from unforeseen natural processes (i.e. geothermal activity) or human error.

There is little evidence in support of aluminium passing through the food chain, as rates of transfer between predator and prey appear to be relatively limited. However, it should be noted that the long term effects of aluminium exposure are poorly studied and a cautious approach is recommended. Flocculation can improve water clarity, but if there are large amounts of suspended particles, organisms living on the lake bed may become buried under flocculated material following alum application. Scientific studies of alum applications rarely report the depth of flocculated material following treatments, or the effects on organisms living on the lake bed. However, the restricted alum application rates in the Rotorua lakes means that it is unlikely that heavy deposits of flocculated material will occur.

Following application, the aluminium component of alum forms the mineral gibbsite in a process taking up to a year. Currently, it is unknown if a large shift in lake pH will dissolve this mineral causing the release of aluminium and phosphorus back into the lake, and further research is recommended. It is concluded that alum dosing is of minimal risk in the short-term as current application rates are conservative. However, significant uncertainties and risks exist in regard to long-term and increased use of alum as a water quality management tool.

Executive Summary

The use of alum (aluminium sulphate) has become a recognised technique for the restoration of freshwater systems. When added to water, alum dissociates and dissolved aluminium undergoes a series of hydrolysis reactions resulting in the formation of aluminium hydroxide ($Al(OH)_3$) which adsorbs dissolved phosphorus and coagulates suspended solids. The resulting flocculent sequesters dissolved and particulate phosphorus, reducing primary production, thereby improving water clarity.

Aluminium hydroxide is a relatively benign substance with peak abundance occurring at pH 6.3; above and below this point, soluble, more toxic aluminium species predominate. For example, under alkaline conditions (>pH 8.5) toxic $Al(OH_4)^-$ forms, while below pH 4.5 free monomeric aluminium (Al^{3+}) becomes prevalent. The hydrolysis reaction of aluminium (Al) causes the release of H^+, lowering pH and potentially causing the formation of toxic aluminium species. It is therefore critical that application rates do not exceed the buffering capacity of the treated system. In addition, eutrophic systems often experience photosynthetic driven alkaline pH shifts, resulting in Al solubilisation and the formation of the toxic Al species ($Al(OH)_4^-$).

The Bay of Plenty Regional Council has initiated several alum dosing programmes in the Rotorua lakes district in an effort to reduce lake trophic levels. Currently, continuous alum dosing is undertaken on the Puarenga and Utuhina Streams discharging to Lake Rotorua and the Waitangi Soda Stream discharging to Lake Rotoehu. In addition, seven discrete alum applications have been conducted on Lake Okaro since 2003. This has resulted in the total applications of 444.2, 55.2, and 2.8 tonnes of Al to lakes Rotorua, Rotoehu and Okaro respectively. Current estimated continuous lake water dose rates for Lake Rotoehu 6.72 µg Al l^{-1} and Lake Rotorua 2.62 µg Al l^{-1} are low by international standards and the estimated maximum dose applied to Lake Okaro of 0.22 mg Al l^{-1} lake water was also conservative.

A literature review was undertaken to provide guidance on a number of concerns associated with alum dosing of the Rotorua lakes. This includes; (1) the likely concentration thresholds for acute toxicological effects from Al dosing and whether current dosing programmes are likely to exceed them; (2) the fate of flocculated Al in lake sediments and whether they pose an ecological hazard; (3) the risk of the buffering capacity of water in the Rotorua lakes being exceeded leading to release of toxic Al species; and (4) the risk to biota of burial by Al-floc and its potential to disrupt lake processes.

The toxicity of Al is closely associated with pH, and acute toxic effects are likely to be a combination of physiological responses to both acidic pH and Al. Fish appear to be the most susceptible group to Al toxicity with respiratory disruption initially occurring at pH 6.0 due to gill irritation by colloidal Al. This is followed by increasing levels of osmoregulatory disruption peaking at pH 3.0 where Al^{3+} is the predominant species. Toxic effects may also manifest under alkaline conditions (pH >8.5), and although the precise mechanism is unknown it is theorised that the gills may be the primary site of action. As well as pH, susceptibility to Al toxicity is dependent on a number of factors including species, life stage, and even whether the organism has had previously exposure to Al. In addition, a number of chemical components have an ameliorating influence; foremost amongst these are dissolved organic matter (DOM), silica and calcium concentrations.

Due to the complexity and large influence of pH in determining Al toxicity, a conservative approach to selecting toxic threshold values was adopted. It is recommended that total Al does not exceed 200 µg l^{-1} at pH >6.0, 75 µg l^{-1} at pH 5.0 - 6.0 and 25 µg l^{-1} at pH 4.0 - 5.0 to avoid acute lethal effects. These proposed threshold values are based on soft water conditions, with low DOM and silica concentrations. Current alum dose rates and measured water column total Al concentrations are below these proposed threshold values.

The buffering capacity of the Rotorua lakes is low. This restricts the quantity of alum that can be applied either continuously or as a discrete dose. Current application rates to the Rotorua lakes are conservative, but the low buffering capacity provides little capacity to increase dose rates without initiating environmental acidification. As well as direct toxicological effects environmental acidification also carries the risk of mobilising toxic Al species from sedimented Al-floc. This may be somewhat mitigated by the fact that Al has a higher affinity for silica minerals which are located deeper in the sediment, compared to humic (organic) substances that predominate in the surface layers. This results in Al diffusing through surface sediment and forming aluminosilicate complexes deeper in the sediment thereby providing a limited separation buffer from transient acidic events in the water column. In addition, flocculated $Al(OH)_3$ undergoes an aging process, changing from a colloidal amorphous solid to microcrystals, and then to the mineral gibbsite, although this process may take up to a year. In this final stage, Al is more resistant to mobilisation by acidification and is less likely to bioaccumulate.

Current research suggests that the risks of Al bioaccumulation are relatively low. Most animals accumulate Al through incidental ingestion and the majority is subsequently excreted. Increased tissue concentrations of Al have been reported in koura (*Paranephrops planifrons*) and common bully (*Gobiomorphus cotidianus*) in the Utuhina Stream but no toxicological effects have been observed. There is little evidence in support of Al biomagnification through the food chain, as rates of trophic transfer appear to be relatively limited. It should be noted that chronic effects of Al exposure are not as well studied and a cautious approach is recommended.

The depth of flocculent formation following alum application is rarely reported and relatively little is known about the effect of Al-floc on lake processes. It has been reported that sustained (12 months) high dose rate alum applications (>8 mg Al l^{-1}) can result in flocculent accumulation to a depth of 50 cm and localised hypoxia. However, the comparatively low dose rates utilised in the Rotorua lakes will result in minimal floc formation. Further research is needed to determine the effect of flocculent deposition on benthic organisms and whether bio-geochemical processes are disrupted by alum dosing.

The current alum dosing programme for the Rotorua lakes is appropriately conservative due to the low buffering capacity of the lakes. The risk of acute Al toxicity is therefore minimal under the current regime. The risk of bioaccumulation and biomagnification also appear to be minimal provided pH levels are maintained above 6.0. However, the low buffering capacity of the lakes means there is little capacity to significantly increase application rates and serious consideration should be given to the use of buffering agents such as sodium aluminate if there is a move towards increased dosing rates. There is relatively little information examining the ecological effects of Al-floc formation and deposition. Current dose rates to the Rotorua lakes are unlikely to form significant quantities of Al-floc but adverse effects of floc deposition are poorly studied.

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Introduction

Description of Alum

Alum is both a specific group of chemical compounds, characterised by double sulphate groups, and the specific chemical compound aluminium sulphate ($Al_2(SO_4)_3$). Aluminium sulphate is used extensively in water purification due to its ability to coagulate and flocculate impurities which can then be filtered out (Cooke et al. 2005). Another characteristic of aluminium sulphate is that when added to water under neutral-alkaline conditions, it forms a chemical precipitate, aluminium hydroxide ($Al(OH)_3$) which adsorbs dissolved phosphorus forming a flocculent which settles to the bottom of the water column (Harper 2013). This has value for lake managers tasked with lake restoration; by treating a system with alum, the availability of phosphate is reduced, restricting plant growth and harmful algal blooms, leading to improved water quality (Wetzel 2001). The coagulation and adsorption properties of alum result in reported nutrient removal efficiencies from stormwater runoff of 65 – 73% for total nitrogen and 86 – 96% for total phosphorus, depending on dose rate (Harper 2013).

Mode of Action

In solution, aluminium sulphate dissociates, with aluminium (Al) hydrolysing water molecules to form a number of chemical species such as Al^{3+}, $Al(OH)_2^+$, $Al(OH)^{2+}$, $Al(OH)_3$ and $Al(OH)_4^-$ depending on pH conditions (Figure 1). Under circumneutral pH (6-8), insoluble $Al(OH)_3$ predominates while at higher pH $Al(OH)_4^-$ occurs. Alternatively, as pH drops below 6, first $Al(OH)_2^+$ and then more toxic $Al(OH)^{2+}$ and Al^{3+} species form (Kennedy and Cook 1982). Typically, most eutrophic systems are neutral-alkaline and $Al(OH)_3$ is the dominant product rather than monomeric Al. Phosphate removal from the water column occurs through both adsorption of dissolved phosphate or coagulation and flocculation of bound particulate phosphorus (Kennedy and Cook 1982). Coagulation of suspended particles occurs due to the positive electrical charge of aluminium hydroxide molecules neutralising the negative charge of the colloidal particles suspended in the water column. Coagulation proceeds until particles reach a size where the effect of gravity dominates over resuspension forces and the particles settle from the water column (Engelhardt 2014). Once the phosphorus bound Al-flocs have settled from the water column they become crystallised, and over time become buried preventing the release of phosphorus (Auvray et al. 2006). An additional property of $Al(OH)_3$ is that adsorbed phosphorus is not released back into the water column under reducing conditions (Cooke et al. 2005).

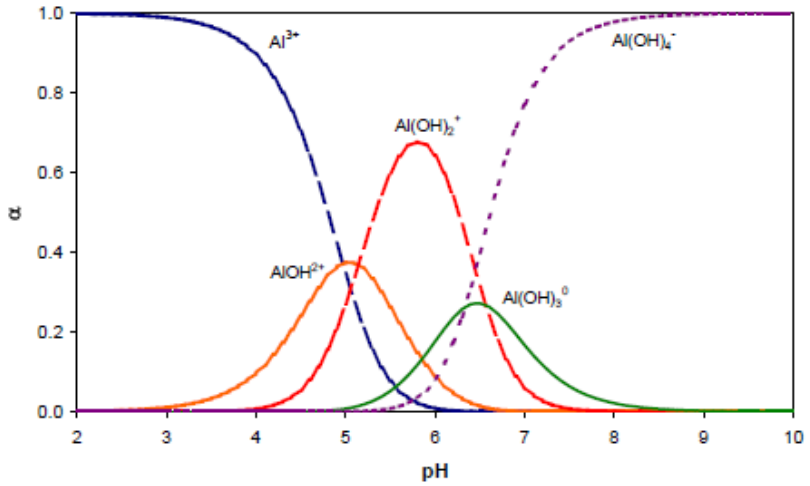

Figure 1. Distribution of aqueous aluminium hydroxide species in relation to pH (reproduced from Cerqueira and Regina da Costa Marques 2012).

Risks of Alum Dosing

Aluminium is the most common metal in the Earth's crust, accounting for approximately 8.1% of the crust by weight. Thus, it is ubiquitous in soils and is often found bound to organic molecules, especially oxalic, humic and fulvic acids (Sparling and Lowe 1996). As Al forms insoluble $Al(OH)_3$ at pH 6-8 it is essentially biologically inactive and only of toxicological concern should pH drop below 5.5 (Sparling and Lowe 1996). Below pH 5.5, soluble free monomeric Al (Al^{3+}) and $Al(OH)^{2+}$ begin to form, both of which have a disrupting effect on ionoregulartory processes in aquatic animals. Similarly, above pH 8.5 the formation of $Al(OH)_4^-$ also causes disruptions in homeostasis. The relative biological activity and toxicity of these compounds varies with composition and concentration. For example, aluminium hydroxides are generally considered less toxic than monomeric free Al ions, as this form is more chemically and biologically active than other bound forms (Sparling and Lowe 1996). A significant amount of research has been published regarding the acute toxicity of Al under acidic conditions, which is known to disrupt respiratory and osmoregulatory processes in aquatic organisms (Driscoll and Schecher 1988; Klöppel et al. 1997; Gensemer and Playle 1999; Allin and Wilson 2000; Berthon 2002; Pilgrim and Brezonik 2005; Eriksen et al. 2009). However, further research examining Al toxicity under alkaline conditions and chronic exposure effects in terms of bioaccumulation in a wide range of organisms is still required. This becomes highly relevant in systems where continuous dosing with alum or related aluminium flocculants (i.e. aluminium chloride) occur.

Alum induced flocculation of suspended material may also have detrimental effects on aquatic biota. Potential risks may include inducement of anoxic conditions due to decomposition of flocculated biological material. This in turn may inhibit microbial nitrification and denitrification processes (Gibbs et al. 2011). It has also been suggested that the gills or feeding structures of invertebrates may become clogged or irritated by flocculating particles (Clearwater et al. 2014), or that pelagic zooplankton may become captured by coagulating particles under conditions of intense flocculation (Jančula et al. 2011).

Report Objectives

This report attempts to address a number of questions related to the alum dosing program conducted by the Bay of Plenty Regional Council in the Rotorua lakes district. Continuous and one-off alum dosing has been utilised in a number of lakes and inflows in the Rotorua district, with no major adverse effects reported (Quinn et al. 2004; Ling and Brijs 2009; Landman and Ling 2011; Ling 2014). However, several questions relating to potential adverse ecological effects to the Rotorua lakes remain; these include:

1) What are the likely concentration thresholds for acute toxicological effects from free aluminium ions (Al^{3+}) to a range of biota likely to be present in the Rotorua lakes and are these likely to be reached by current alum dosing programmes?

2) What is the fate of aluminium in bottom sediments, does it accumulate, and if so what are the ecological consequences?

3) What is the risk of the buffering capacity of water in the Rotorua lakes district being exceeded during an alum dosing event, thereby triggering a toxicological event?

4) What are the risks of biota burial and/or interference with their functional roles as a result of elevated levels of deposition of alum-rich flocculated sediments?

Alum Dosing of the Rotorua Lakes

In the Rotorua lakes district, alum was first trialled in December 2003 by discrete dosing of Lake Okaro. An alum solution (47% $Al_2(SO_4)_3.14H_2O$) was applied to the surface of the lake by spraying from a moving boat, producing an estimated concentration of 0.6 mg Al l^{-1} in the epilimnion (0-3 m) (Paul et al. 2008). Further discrete applications were completed in 2010, 2012, 2013 and 2014 (Table 1). Continuous alum dosing to the Utuhina Stream was started in mid-2006, and the Puarenga Stream in mid-2010, both of which discharge into Lake Rotorua. In mid-2011, a third continuous alum dosing station was commissioned for the Waitangi geothermal spring waters discharging into Lake Rotoehu. Dosing rates for the continuous applications have been variable, with the Utuhina Stream receiving 0 – 100 kg Al day^{-1} and the Puarenga Stream 0 – 300 kg Al day^{-1} depending on discharge (Hamilton et al. 2015). A calculated rolling average by Hamilton et al. (2015) showed that the combined average aluminium dose for the Utuhina and Puarenga Streams was c. 202 kg Al day^{-1} with peaks of up to c. 400 kg Al day^{-1} for the period July 2010 – June 2014. In contrast, mean alum dose rates to the Waitangi spring waters were lower at c. 41 kg Al day^{-1} (July 2011 – June 2014). Total alum loads to Lakes Rotorua, Rotoehu and Okaro have been supplied by the Bay of Plenty Regional Council (Table 1).

The ANZECC (2000) trigger value for dissolved Al in freshwater systems is 0.055 mg l^{-1}, which is higher than the mean total Al concentrations in the water columns of Lake Rotoehu (0.01 mg l^{-1}) (Figure 2), Lake Rotorua (0.02 mg m^{-1}) (Figure 3) and Lake Okaro (0.03 mg l^{-1}) (Figure 4) since alum dosing was initiated at each lake. Water column total aluminium concentrations in Lakes Rotorua (Figure 3) and Okaro (Figure 4) do occasionally exceed the ANZECC guidelines; however these guidelines are for dissolved aluminium and occasional exceedances for total aluminium are not concerning (ANZECC 2000).

Table 1. Total alum (tonnes) delivered to Lake Rotorua, Lake Rotoehu and Lake Okaro. Tonnes of aluminium are given in parentheses. Alum applications to Lakes Rotorua and Rotoehu occurred by continuous dosing of inflows, while Lake Okaro applications were lake surface discrete applications. Data supplied by the Bay of Plenty Regional Council.

	Lake Rotorua (tonnes)	Lake Rotoehu (tonnes)	Lake Okaro (tonnes)
1/7/2003 to 30/6/2004			17.2 (0.7)
1/7/2006 to 30/6/2007	826.5 (34.7)		
1/7/2007 to 30/6/2008	694.0 (29.1)		
1/7/2008 to 30/6/2009	518.0 (21.8)		
1/7/2009 to 30/6/2010	640.0 (26.9)		12.0 (0.5)
1/7/2010 to 30/6/2011	1,772.3 (74.4)		
1/7/2011 to 30/6/2012	2,354.2 (98.9)	520.3 (21.9)	
1/7/2012 to 30/6/2013	1,503.2 (63.1)	246.1 (10.3)	22.6 (0.9)
1/7/2013 to 30/06/2014	1,566.2 (65.8)	300.0 (12.6)	14.8 (0.6)
1/7/2014 to 31/12/2014	701.4 (29.5)	248.5 (10.4)	
Total	**10,057.8 (444.2)**	**1314.9 (55.2)**	**66.6 (2.8)**
Total kg m^{-2} lake area	**0.125 (0.005)**	**0.164 (0.007)**	**0.215 (0.009)**

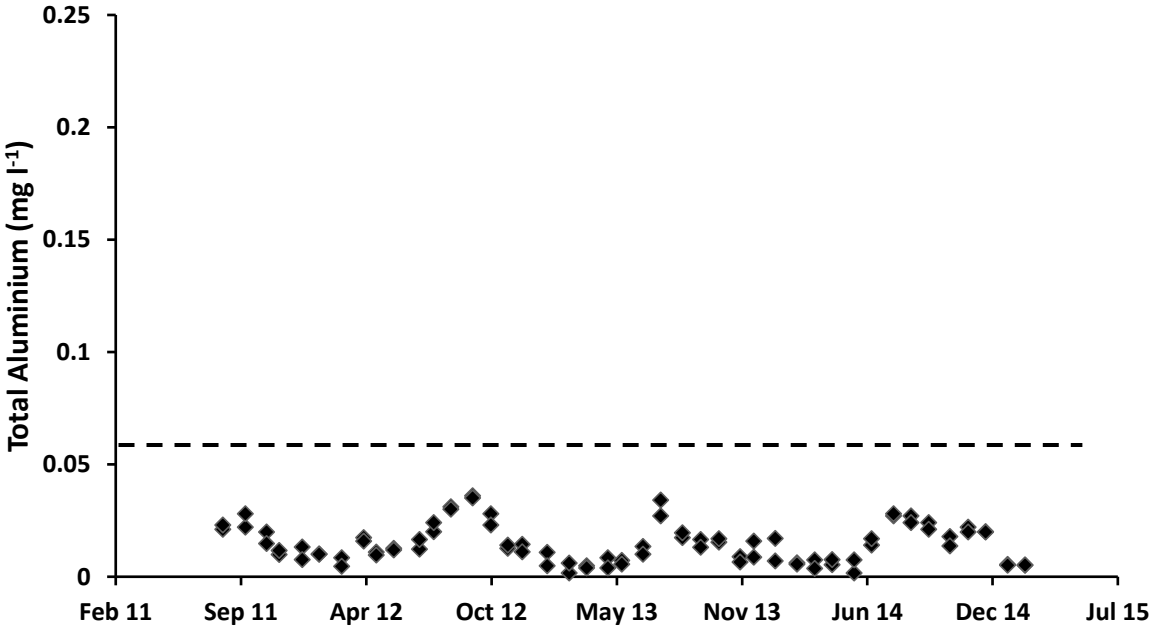

Figure 2. Lake Rotoehu total aluminium concentrations. Dashed line indicates the ANZECC trigger value for sustained toxic exposure to dissolved aluminium. Data supplied by the Bay of Plenty Regional Council.

A survey of Lake Rotorua sediments by Özkundakci et al. (2013) reported total Al concentrations of 4.8 to 10.9 g kg^{-1} DW with an average 6.4 g kg^{-1} DW. As expected, Al levels were elevated in sediments near the Utuhina Stream outlet and declined towards the main basin. However, comparison with an earlier survey of Lake Rotorua sediments by Pearson (2007) found while Al levels were elevated near the outlet of the streams dosed with alum, concentrations measured in 2012 were lower in the main basin of the lake compared to the

2006 survey. The authors suggest that the lower 2012 Al concentrations may be due to increased bioturbation by macroinvertebrates in the main basin (Özkundakci et al. 2013); however a comparison of invertebrate densities between the two surveys could not be made.

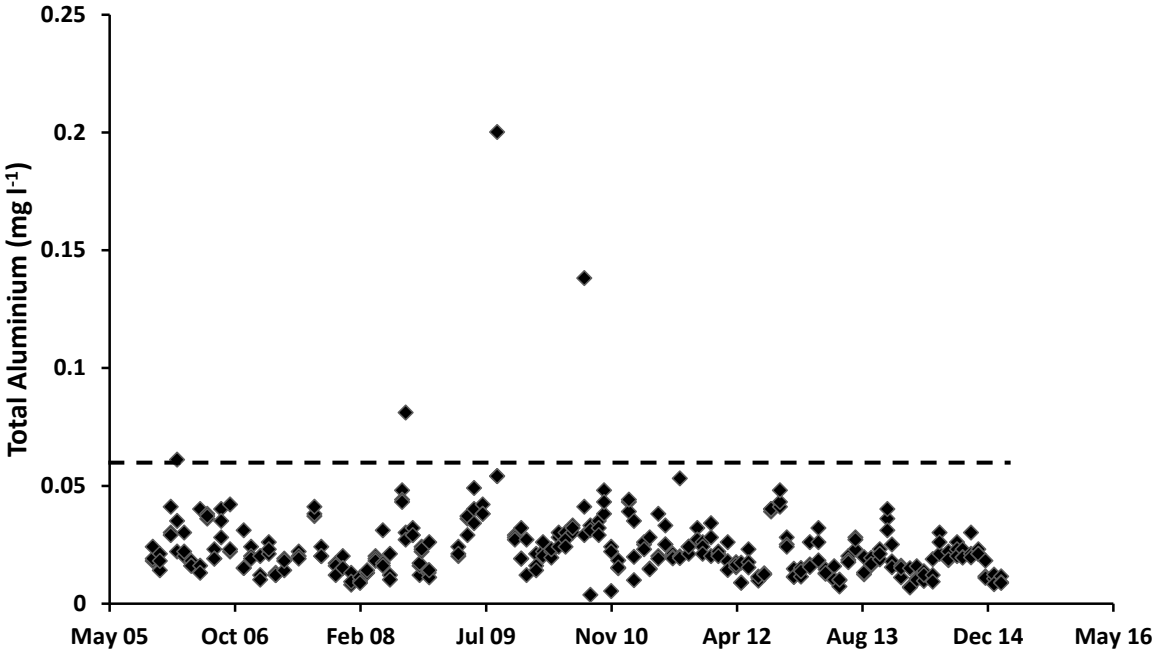

Figure 3. Lake Rotorua total aluminium concentrations. Dashed line indicates the ANZECC trigger value for sustained toxic exposure to dissolved aluminium. Data supplied by the Bay of Plenty Regional Council.

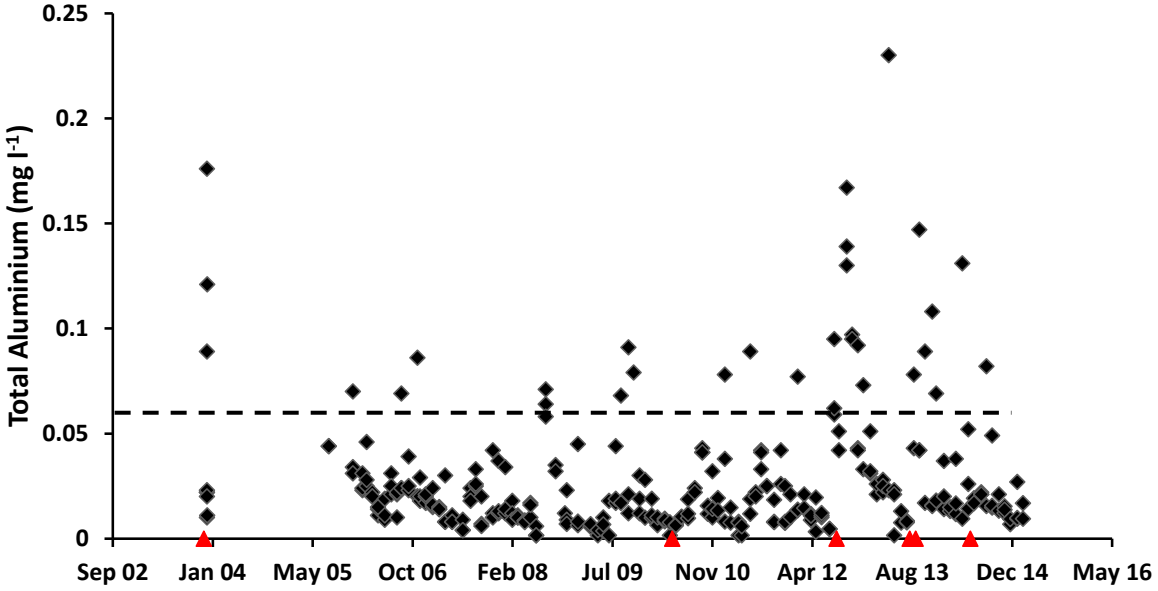

Figure 4. Lake Okaro total aluminium concentrations. Dashed line indicates the ANZECC trigger value for sustained toxic exposure to dissolved aluminium. Red triangles indicate timing of alum applications to the lake. Data supplied by the Bay of Plenty Regional Council.

Toxicity of Alum/Aluminium

Alum Toxicity

As a solid, alum (aluminium sulphate) may have a minor irritating effect to skin and eyes of humans but is considered relatively benign (ATSDR 2008). Alum has a reported oral toxicity LD_{50} value of 980 mg kg^{-1} for mice (Ondreička et al. 1966), notably higher than the safe daily intake of some aluminium-containing medications (12 -71 mg kg^{-1}) (Pennington and Schoen 1995) and significantly higher than the normal daily dietary intake for adult humans (0.10 – 0.12 mg kg^{-1}) (ATSDR 2008).

In freshwater environments the toxicity of alum is intimately associated with the solubility of its Al component, which in turn is dictated by environmental pH. Aluminium is soluble and biologically available in acidic (pH <5.5) soils and waters, but relatively insoluble and therefore innocuous in circumneutral (pH 6.0-8.0) conditions (Sparling and Lowe 1996). Above pH 8.5, aluminium again becomes soluble, forming $Al(OH)_4^-$ which can potentially interfere with ionoregulatory processes (Gundersen et al. 1994). Water column concentrations of dissolved Al are therefore highly variable due to the influence of pH. For example, total Al levels can range from a few µg l^{-1} to over 300 µg l^{-1} in Ontario lakes and as high as 9000 µg l^{-1} in river waters (Wren and Stephenson 1991). For North American lakes, typical ambient aluminium levels in non-acidified lakes are 50 µg l^{-1} (Havas and Likens 1985), but may reach 1,000 µg L^{-1} in some acidified lakes (Havas 1986). As a result, it is often difficult to discriminate between the direct toxic effects of AI and factors associated with reduced pH. In addition, the effects of Al on plant species are further complicated by the binding of phosphorus, reducing its availability for growth (Sparling and Lowe 1996).

Toxicity Pathways

The effects of Al on biota are highly variable. In fish, mortality is primarily due to either asphyxiation at pH levels c. 5.5 – 6.5 or interference with electrolyte balance below pH 5.5 or above 8.5 (Gundersen et al. 1994; Sparling and Lowe 1996). The relative importance of asphyxiation or ionoregulation is dependent on pH, Al level, and the magnitude of the pH change of water irrigating the gills (Playle and Wood 1991). Asphyxiation is usually due to precipitation of $Al(OH)_3$ on the gill surface causing excessive mucus production and inflammation of the gill surface blocking oxygen and carbon dioxide diffusion across the gill (Neville and Campbell 1988). It has also been proposed that $Al(OH)_x$ and Al^{3+} may form complexes with the gill membrane interfering with metabolic processes (Neville and Campbell 1988). The disruption in electrolyte regulation outside of pH range 6.5–8.5 results from AI displacement of Ca^{2+} from the gill surface, allowing passive diffusion outward of Na^+ and Cl^- and inward movement of H^+ through the gills. However, under laboratory conditions it has been observed that once pH drops below 4.0, AI seems to have reduced toxicity. It has been suggested that this is due to H^+ concentrations becoming more detrimental to the fish than Al^{3+} (Sparling and Lowe 1996). Water hardness, and in particular Mg^{2+} and Ca^{2+} concentrations have an increasing ameliorating effect as concentrations of the ions rise presumably due to reduced Ca^{2+} displacement (Gundersen et al. 1994).

In amphibians, the earlier the life stage, the more susceptible it is to Al toxicity (Freda and McDonald 1990). The most prominent toxic effects are disruption of the embryo

membranes or hatching proteins resulting in reduced hatching or severe deformations of the tail and spine leading to mortality (Sparling and Lowe 1996). Other chronic effects include retarded growth, osmoregulatory disruption, and skin irritation (Freda and McDonald 1990). There is comparatively little published literature regarding toxic effects of Al to reptiles. Most studies have focused on Al body concentrations of marine turtles as part of a wider examination of heavy metals and dietary sources of contaminants (e.g. Camacho et al. (2013)). No studies were found to examine the potential toxicological pathways of Al in reptiles, although presumably the risk is reduced compared to amphibians and freshwater fish as even aquatic reptiles nest on land. Given New Zealand has no native aquatic reptiles, no further examination of this group will be undertaken.

Birds are most likely to be exposed to Al through their diets. Fortunately, only a fraction of the ingested metal is assimilated, and faecal excretion is relatively efficient. Thus, birds are less at risk than fish, amphibians, or aquatic invertebrates (Sparling and Lowe 1996). Toxic effects of high dietary Al typically are associated with problems in calcium and phosphorus metabolism. The metal binds with phosphorus in the gut to form an insoluble complex and decreases the availability of phosphorus in the diet. Signs of toxicity, therefore, are consistent with phosphorus deficiency and include reduced growth, impaired eggshell quality, loss of appetite, decreased laying, feather moult, and rickets (Sparling and Lowe 1996).

Aquatic macroinvertebrate species have a wide range of tolerances to Al. Although the physiological effects of Al have not been as widely examined as those in vertebrates, toxic effects appear to be similar to those observed in fish. At higher pH (5.5 – 6.5), mortality is primarily driven by disruption of respiration, while below pH 5.5, mortality is associated with osmoregulatory disturbance (Burton and Allan 1986; Wren and Stephenson 1991).

Identification of Al toxicity pathways in aquatic plant species is complex. Plants adapted to live in acidic conditions appear to be extremely tolerant of high Al concentrations (Sparling and Lowe 1996). In addition, it has been suggested that Al may have an ameliorating effect at low pH (<3.5) as it competes for binding sites with protons (Rout et al. 2001). Aluminium has been shown to interfere with uptake and transport of some essential nutrients as well as with cell division in roots, to increase cell wall rigidity (cross-linking pectins), to alter the structure and function of plasma membrane by binding to phospholipids, and to alter activities of many enzymes and metabolic pathway involved in cellular repair mechanisms (Radić et al. 2010). In addition, aluminium likely reduces phosphorus uptake rates in algae, by inhibition of the acid phosphatase enzyme (Gensemer and Playle 1999).

Toxic Effects of Aluminium on Biota

The relative toxicity of Al is modified by a number of factors including life stage of the organism, species, exposure time, Ca^{2+} concentration, dissolved organic matter (DOM) concentration, temperature and pH. Dissolved organic matter has a large influence on Al toxicity as it readily forms ligands with Al^{3+} reducing toxic interactions with biota (Burton and Allan 1986; Sparling and Lowe 1996). For example, mortality of juvenile Atlantic salmon (*Salmo salar*) was two to three times greater in the presence of Al^{3+} alone, compared to fulvic acid and Al^{3+} across a range of Al concentrations (11 – 44 uM Al) (Roy and Campbell 1997). Silica plays a similar role to DOM by complexing with more toxic Al species (Al^{3+} and $Al(OH)_2^+$) and forming benign aluminosilicate complexes (Pilgrim and Brezonik 2005). No

single study could hope to adequately encompass all these variables; however, an examination of published survival rates from toxicological tests provides sufficient information to formulate a hazard assessment for a given Al concentration in a specified pH range.

Fish

The majority of toxicological studies examining the acute effects of Al to fish have focused on salmonid species (Sparling and Lowe 1996). Salmonids, which are also sensitive to low pH (Henriksen et al. 1984; Baldisserotto and Mancera Romero 2007), such as rainbow trout (*Oncorhynchus mykiss*) and brown trout (*Salmo trutta*), are important introduced recreational fishing species in the Rotorua lakes. In contrast, no acute toxicological studies have been published regarding the sensitivity of native New Zealand fish species to Al. However, given the dependence of Al toxicity on pH and indications that Al tolerance is associated with low pH tolerance (Sparling and Lowe 1996), pH tolerance may give some indication as to Al toxicity in New Zealand native freshwater fish species.

Greig et al. (2010) examined native fish distribution in relation to acid mine drainage in the South Island of New Zealand. The authors demonstrated that five *Galaxias* species, longfin eel (*Anguilla dieffenbachii*) and shortfin eel (*Anguilla australis*) were found in more acidic waters (pH <5), while bluegill bullies (*Gobiomorphus hubbsi*) and torrentfish (*Cheimarrichthys fosteri*) were less tolerant of low pH (minimum pH 6.2 and 5.5, respectively). Further analysis found that dissolved metal concentrations (Fe, Zn, Mn, Ni and Al) rather than pH were more important in predicting fish diversity, density and biomass (Greig et al. 2010). From this evidence and pH preferences tests by West et al. (1997), native species such as common bully (*Gobiomorphus cotidianus*) and common smelt (*Retropinna retropinna*), common to the Rotorua lakes, are likely to have similar tolerances to Al as those reported for rainbow trout and brown trout.

A safe threshold value for dissolved Al concentration in neutral to basic water of 100 µg l^{-1} for rainbow trout to grow and survive was first proposed by Freeman and Everhart (1971). Subsequent studies under acidic conditions revealed that Al becomes increasingly toxic as pH declines and that more conservative acute threshold values should be adopted under acidic conditions. Aluminium toxicity also varies with life stage; a review of the literature reveals that Al is most toxic to newly hatched fish that have not reached independence from the yolk sac (termed the alevin stage); embryos (egg stages)are the next most susceptible life stage followed by parr and smolt stages, and then adults (Sparling and Lowe 1996).

A traffic light scale for increasing expected mortality with regard to life stage, acidic pH and total aluminium concentration has been constructed from published toxicological data for 14 North American freshwater fish species (Figure 5) of which two (rainbow trout and brown trout) are present in the Rotorua lakes. This approach was adopted as it best communicates the inherent variation in toxicological data but also provides guidance as to the expected effects under a given set of conditions. Acute lethal effects have been selected as a trigger point as this is the most commonly reported toxicological data with a consistent, easily identifiable end point. For consistency only toxicological tests that utilised the following conditions were used; Al exposure time of 1 – 4 days, temperature 15°C, soft water conditions (i.e. $CaCO_3$ concentration of <17 mg l^{-1}), use of water with low DOM. Therefore, consideration to modification of toxic thresholds would be needed under hard

water or high DOM conditions. Furthermore, below pH 4.0, extensive mortality (>50%) would be expected from all life-stages even at near zero Al concentrations due to osmoregulatory disruption by H^+.

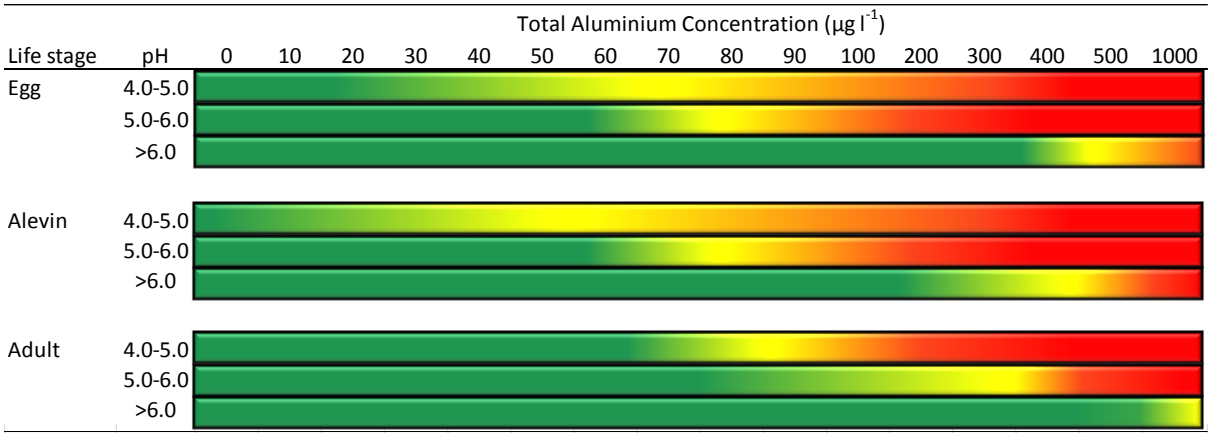

Figure 5. Water column total aluminium concentrations and indicative acute lethal toxic effects with regard to life stage and environmental pH based on 14 North American freshwater fish species, including the two salmonid species present in the Rotorua lakes. Green indicates no expected mortality (0%), yellow low mortality (1 – 10%), orange moderate mortality (10-50%) and red high mortality (>50%) based on published literature values.

From the data used to construct Figure 5 the following no expected mortality threshold values are proposed for fish based on total Al concentrations. As toxicity varies with pH, threshold values have been assigned to pH bands and total Al concentrations should not exceed 200 µg l^{-1} at pH >6.0, 75 µg l^{-1} at pH 5.0 - 6.0 and 25 µg l^{-1} at pH 4.0 - 5.0 if mortality is to be avoided.

The toxicological effect of Al to fish under alkaline conditions has not been extensively investigated. Juvenile Atlantic salmon exposed to 350 µg Al l^{-1} at pH 9.5 displayed no acute effects, however large increases in blood glucose (+300%) and blood haematocrit (+30%) were observed after 3 weeks exposure (Poléo and Hytterød 2003). The authors concluded that the combination of elevated pH and Al did induce a stress response, but was unlikely to represent a serious problem unless exposure continued for an extended period of time (Poléo and Hytterød 2003). At higher pH (>10) the toxicological effects of pH begin to dominate over those of Al. For example, survival rates of juvenile rainbow trout were similar for groups exposed to pH 10 water compared to groups exposed to pH 10 and 80 µg l^{-1} Al (Winter et al. 2005)

Calcium appears to have a mitigating effect on Al toxicity under alkaline conditions, although the mechanism for this cannot be explained. Gundersen et al. (1994) examined acute Al toxicity to juvenile rainbow trout under neutral and mildly alkaline conditions. Under acute conditions (96 h), no mortality was observed at near-neutral pH (7.1 4–7.64) and total Al concentrations up to 8 mg l^{-1}. Low mortality (<10%) was observed at pH 8.34, total Al concentration 4000 µg l^{-1}; and high mortality (>50%) at pH 8.58, total Al concentration of 8000 µg l^{-1}. Some protection was afforded by increasing water hardness. For example, mortality decreased from 95% to 65% as water hardness increased from 37.7

to 83.3 mg l^{-1} $CaCO_3$ respectively at pH 8.58 (Gundersen et al. 1994). This effect is likely due to reduced displacement of Ca^{2+} on the gill surface by Al species (Wilson 2011).

The few studies conducted on the toxicological effects of Al to fish at high pH suggest that no expected mortality threshold values of 200 µg Al total l^{-1} up to pH 9.0 and 100 µg total Al l^{-1} between pH 9.0 and 10.0 should be employed; beyond this the toxic effects from high pH are likely to dominate. As with proposed acidic pH threshold values, these levels are for limited exposure time (1 – 4 days), temperature ~15°C, soft water conditions. Due consideration should also be given to the limited number of species and life stages that have been tested.

Amphibians

New Zealand native frogs are highly threatened and only two of the four species are present on mainland North Island (Newman et al. 2010). Given the highly restricted distribution and limited aquatic phase of native New Zealand frogs, alum toxicity is not a threat. Introduced species such as the southern bell frog (*Litoria raniformis*) and the green and golden bell frog (*Litoria aurea*) have been reported in the Rotorua lakes district (Pickard and Towns 1988). Both of these species have obligate aquatic life stages that can last up to a year (Zweifel 1998) making them susceptible to Al toxicity.

Information on Al toxicity in amphibians is incomplete and confounded by the toxicity of low pH. As a general rule, elevated levels of Al are likely to be toxic when pH drops below 4.5, although some species show signs of toxicity within the pH range of 4.5–5.5. Lethal levels may be as low as 150 µg l^{-1}, but LC_{50} levels are commonly several times higher (Sparling and Lowe 1996). As the southern bell frog and the green and golden bell frog are both considered to be vulnerable to extinction in Australia, few toxicological studies have been performed on these species (Mann and Bidwell 1999). Both species appear to have some avoidance of low pH (<5.6) (Hamer et al. 2002; Pyke 2002), however no information regarding tolerance to low pH or Al could be located. Similarly, no studies could be found examining Al toxicity at high pH.

Ambient levels of soluble Al are acutely toxic at 250 µg Al l^{-1} for some amphibian species, and physiological problems appear above 100 µg Al l^{-1}. Newly hatched tadpoles tend to be the most sensitive amphibian life stage, followed by embryos and older tadpoles. For example, LC_{50} values for leopard frog (*Rana pipiens*) embryos, young tadpoles, and older tadpoles at pH 4.8 were 403, <250, and >1000 µg Al l^{-1}, respectively (Freda and McDonald 1990). These levels are similar to those reported for some fish species, and adoption of the fish threshold values should afford sufficient protection for amphibian species.

Aquatic Invertebrates

Most studies of Al toxicity on invertebrates concern freshwater species, especially crustaceans and larval or nymphal stages of insects. Although some studies have examined effects of Al at pH around neutral, most have focused on effects at acidic pH (Sparling and Lowe 1996).

Invertebrates appear to be more robust against Al toxicity than fish species; however they are more sensitive to low pH. In fact, Al may have an ameliorative effect against H^+ toxicity

by reducing osmotic loss of sodium and chloride (Sparling and Lowe 1996). In many studies, adding Al to acidified water produced few differences compared to acidity alone. Exposure to 400–500 µg l^{-1} soluble Al at a pH of 4.0 had no effect beyond that of H^+ on clams (*Pisidium* sp.), amphipods (*Hyalella* sp.), isopods (*Asellus* sp.), snails (*Amnicola* sp., *Physella* sp.), or insect larvae (*Enallagma* sp., *Lepidostoma* sp., *Pycnopsyche* sp.) (Burton and Allan 1986; Mackie 1989). Similarly, exposure to pH 4.28 and 350 µg Al l^{-1} did not increase mortality of larval *Chironomus* sp., *Hydropsyche* sp., or *Dinocras* sp. (Ormerod et al. 1987). Aluminium levels as high as 1000 µg l^{-1} did not kill larvae of *Chaoborus* sp. and *Chironomus* sp. midges at pH 4.0 (Havas and Likens 1985).

At circumneutral pH, 1020 µg Al l^{-1} significantly increased mortality in cladocerans (Havas and Hutchinson 1982). Cladocerans also appear to be sensitive to Al under acid conditions, but these organisms are also highly sensitive to low pH making determinations of toxicity due to Al difficult. Cladocerans experienced severe mortality at pH below 5.0, but, as with other invertebrate species (e.g. amphipod *Gammarus* sp. (Ormerod et al. 1987; McCahon et al. 1989)), elevated Al appeared to prolong life compared to acidity alone. However, all cladocerans tested at low pH were dead after 24 h, regardless of Al concentration (range 0 – 20 000 µg l^{-1}) (Havas and Hutchinson 1982).

Studies specifically related to the toxicological effects of Al to New Zealand macroinvertebrate species are limited. Winterbourn et al. (2000) examined the effect of acid mine drainage on macroinvertebrate body Al concentrations. They found that Al body burdens varied considerably, suggesting that groups of insects differ considerably in their uptake of toxic metals. The authors also note that taxonomic richness and abundance was severely reduced in streams with low pH (<5.0) (Winterbourn et al. 2000). In the Rotorua lakes district, monitoring of macroinvertebrates in the Utuhina Stream by Ling and Brijs (2009) found no obvious differences in community structure upstream and downstream of the alum dosing station.

Particular mention should also be made of koura (*Paranephrops planifrons*) due to its cultural significance (Kusabs and Shaw 2008) and use as a bioindicator species. Koura abundance and catch per unit effort were both higher in the Utuhina stream following commencement of alum dosing (Ling and Brijs 2009). In addition, there were no detectable differences in Al concentrations in the flesh and heptopancreas of koura upstream and downstream of the alum dosing station (Ling and Brijs 2009). Although there appears to be a notable absence of information regarding the sensitivity of freshwater decapods to Al, this data suggests that koura are not adversely affected by Al under circumneutral pH conditions.

Invertebrates are more tolerant of Al than fish species and adoption of the Al concentration thresholds for fish should provide more than sufficient protection for invertebrate species. However, it should be noted that a number of invertebrate species are highly sensitive to acidification which can occur during alum dosing.

Birds

Birds are most likely to be exposed to Al through their diets. Thus, birds are less at risk than fish, amphibians, or aquatic invertebrates. Aluminium binds with phosphorus in the gut to form an insoluble complex and decreases the availability of phosphorus in the diet, thereby

disrupting phosphorus and calcium metabolism. Harmful effects are most apparent when diets are deficient in phosphorus or when Al levels exceed 50% of phosphorus (Scheuhammer 1991). Sparling and Lowe (1996) stated that diets with less than 1000 mg kg^{-1} Al should not be considered harmful to birds. However, this figure appears conservative and need only be adopted if there are concurrent low levels of dietary calcium (<7000 mg kg^{-1}) and phosphorus (<4500 mg kg^{-1}) (Scheuhammer 1991).

Insectivorous birds may be more at risk in acidified environments as insects have been observed to contain Al levels up to 5000 mg kg^{-1} dry wt (Sadler and Lynam 1988), and similar levels have also been reported in daphnia (Havas and Hutchinson 1982). Many insectivorous birds, especially those that probe or dabble in mud may also consume substantial amounts of Al-rich soil with their foods. In acidified environments, much of this metal may be biologically active (Sparling and Lowe 1996).

Herbivorous birds, including most waterfowl at certain times of the year, may have diets with high Al levels. Havas (1986) presented Al bioaccumulation data for U. S. and Canadian lakes, and macrophytes accumulated from less than 40 to 32,000 mg Al g^{-1} dry wt, with differences being observed as a function of season, location, portion of plant analysed, and species tested. A diet consisting largely of these species could be toxic, but there have been no studies dealing with secondary toxicity of Al in birds. It appears that if birds are at risk, the greatest probability of toxicity would be in rapidly growing chicks when the rate of bone deposition and phosphorus requirements are greatest (Scheuhammer 1991). As chicks approach adult size, their tolerance appears to increase. There is little evidence that Al presents much risk to adult birds, even during egg laying, but reproduction may be impaired (Sparling and Lowe 1996; Gensemer and Playle 1999). Further research on the assimilation of Al and its competition with phosphorus and calcium uptake in chicks and juvenile birds is necessary to assess possible population effects.

In New Zealand, few studies have been published regarding metals in native bird species (refer to (Lock et al. 1992; Thompson and Dowding 1999) and none with regard to Al. Given the high endemism and threatened status of many New Zealand water fowl, ascertaining at least baseline Al levels for a number of species would seem prudent. Despite this, the risk of Al toxicity to waterfowl appears low, unless accompanied by low dietary levels of calcium and phosphorus. High Al and low calcium and phosphorus generally only occur under conditions of persistent environmental acidification (pH <5.5) (Gensemer and Playle 1999). While some geothermal areas in the Rotorua lakes do have persistent low pH (e.g. Sulphur Bay in Lake Rotorua), phosphorus and calcium levels are likely adequate to compensate for increased Al^{3+} levels in these areas.

Plants

Studies on the toxicological effects of Al to algae have used a wide assortment of growth media, methods of culture, and biological responses, making it difficult to generalise which algal taxonomic groups are most tolerant of Al exposure. The single study to use cultures from several algal divisions concluded that desmids and diatoms were particularly sensitive, whereas Chrysophytes along with some (but not all) Chlorophyceae could tolerate total Al exposures up to 400 µg l^{-1} at pH 5.5 before significant growth inhibition occurred (Gensemer and Playle 1999). The variation in Al tolerance from a number of studies also makes it difficult to provide guidance on threshold levels. For example, two Chlorophyceae species

were tested at an Al concentration of 400 µg l^{-1} and separate pH values of 4.8 and 6.8. In response, *Monoraphidium dybowskii* showed little growth inhibition, while *M. griffithii* showed 50% and 60% growth inhibition respectively (Hörnström et al. 1995). As with other biota, algal species that are tolerant of low pH also tolerate higher levels of Al. For example, several species of algae (*Charcium* spp., *Euglena mutabilis*, and *Pinnularia acoricola*) have been reported growing at pH <3.0 and dissolved Al above 25 000 µg l^{-1} (Havas 1986).

Given the importance of macrophytes to the structure and function of the littoral zone of freshwater systems, it is surprising that so few experimental exposures of Al to macrophytes exist. Most studies examining macrophyte responses to Al found that acute effects (reduced growth or loss of biomass) only occurred at concentrations >5000 µg l^{-1} and results were often complicated by concordant negative responses to acidic pH (Sparling and Lowe 1996; Gensemer and Playle 1999). The likelihood of increased sensitivity to H^+ over Al^{3+} by macrophytes is also supported by two field studies that examined the distribution of aquatic plant species in response to a range of environmental factors. Both concluded that pH was the primary factor limiting plant distribution, whereas Al was only secondary (Ormerod et al. 1987; Jackson and Charles 1988).

Alum dosing also has an important secondary effect on plant growth resulting from the binding and flocculation of phosphorus, reducing its availability to primary producers. In terms of lake restoration this is a desired effect, reducing phytoplankton abundance and improving lake water clarity. Surprisingly, alum dosing appears to have no effect on macrophyte growth despite reducing phosphorus concentrations in both the water column and interstitial pore water (Jacoby et al. 1983; Mesner and Narf 1987; Malecki-Brown et al. 2010). Some reduction in submerged macrophyte biomass in response to alum (total addition of 68 g Al m^{-2}) was reported by Malecki-Brown et al. (2010) but the authors were unsure as to whether this was due to reduced phosphorus availability or Al toxicity as emergent macrophyte species showed no reduction in growth. There are few studies examining long-term alum dosing effects, and it is possible that macrophytes are able to source phosphorus from deeper in the benthic layer through their root systems (Rout et al. 2001). As alum dosing continues over time, reduced phosphorus availability may impact macrophyte growth, but this remains speculative.

Aquatic plants appear to be sufficiently robust to Al toxicity that implementation of the threshold values for fish will avoid acute toxic effects. Impacts to algae are more likely to result from changes in pH, phosphorus availability, coagulation and flocculation. There is no evidence that phosphorus binding by aluminium impacts macrophyte growth, but this area does require further study, especially with regard to long-term phosphorus availability. No studies could be found examining Al toxicity at high pH.

Sediment Accumulation of Aluminium

Aluminium Deposition and Potential Release

There is a notable absence in the published literature of the long-term fate and effects of anthropogenic Al in sediments. Despite alum first being used for lake restoration in Europe from 1968 (Lake Långsjön) (Cooke et al. 2005) and the United States in 1970 (Horseshoe Lake) (Garrison and Knauer 1984), no applied or theoretical research has been published

examining the effects of high Al deposition over sustained time periods (i.e. >10 years). This includes both the ultimate fate of aluminium in sediments and effects on biota.

After formation, Al floc undergoes an aging process altering its density and mineralogical form. Depending on water chemistry and temperature this process may take up to a year (Cooke et al. 2005). The aging process of Al floc in water from two Californian lakes was investigated by Berkowitz et al. (2005). Initially, soluble amorphous $Al(OH)_3$ transformed to a lower solubility, more crystalline phase over a period of 30-80 days. During this time it was easily resuspended, redistributed and buried beneath other sediments. Testing at 150 days following Al treatment, the $Al(OH)_3$ crystals had reordered to form the mineral gibbsite or in waters with silica levels >6.17 mg l^{-1}, aluminosilicate minerals (Berkowitz et al. 2005).

The amorphous white $Al(OH)_3$ floc that forms following alum application eventually settles at the sediment surface and then into the benthic layer (Cooke et al. 2005). Initially this floc is mobile and sensitive to resuspension and redistribution due to its low density (Egemose et al. 2009). Whether the floc permanently settles into the bottom sediments or is flushed from the lake will depend on individual lake characteristics such as depth, wind velocity and fetch, bioturbation rate, vegetation coverage, and stormflows. A laboratory study using intact sediment cores from previously Al-treated Lake Möllen, Germany, found Al floc surface sediments took 2 months to return to pre-treatment stability levels (Egemose et al. 2009).

Other research has found that early stage Al floc is often transported and focused to deeper parts of the lake or even washed out of the lake. Horizontal redistribution of Al towards deeper areas was found in four of six Danish lakes treated with Al (Egemose et al. 2013). While these lakes were comparatively shallow (range of mean depths 1.3 - 5.0 m), deeper lakes were more likely to have greater Al accumulations with increasing depth. Time since treatment appeared to indicate that Al redistribution mainly occurred during the initial floc low density phase, with the distribution of Al showing the same trend 4 years after treatment as 1 month following treatment (Egemose et al. 2013). Evidence for horizontal redistribution of Al floc to deeper areas was also observed by Welch and Cooke (1999) who examined 21 Al-treated lakes across the United States.

As well as horizontal redistribution, Al floc sediments are readily incorporated into the deeper benthic layers. This appears to be facilitated by bioturbation, physical settling through sediment due to density difference, and burial with new sediment (Welch and Cooke 1999). While Al enriched sediments are always identifiable, the relative distribution of the Al layers appears to be highly variable between lakes with both concentrated, easily identifiable layers (Jacoby et al. 1983; Lewandowski et al. 2003) and more diffuse layers (Welch and Cooke 1999; Egemose et al. 2013) reported. However, none of these studies directly examined the processes that have led to either concentrated or diffuse Al sediment layers, but rather relied on generalised explanations of bioturbation or calculated sedimentation rates.

One identifiable risk is that sedimented $Al(OH)_3$ floc could be mobilised by environmental acidification and toxic species (i.e. $Al(OH)_2^+$ and Al^{3+}) released into the water column. However, geochemical processes appear to provide some moderation of this risk. Studies examining lake sediment Al concentrations (including Lake Rotorua) typically report increasing Al abundance with increasing sediment depth (Carignan and Nriagu 1985; Dauvalter 1995; Einax et al. 2004; Özkundakci et al. 2013) suggesting downward diffusion of

Al. This process appears to be facilitated by aluminium's lower affinity for humic (organic) substances that predominate in sediment surface layers (in comparison to elements such as lead and mercury) and higher affinity for silica minerals which are more prevalent in deeper sediments (El Bilali et al. 2002). The diffusion of aluminium away from surface sediments provides some protection against aluminium toxicity by episodic acidification. In addition, sediment pH is somewhat resistant to modification by overlying waters due to the reduced mixing rate between sediment pore water and the overlying water column.

A laboratory study by Driscoll et al. (2014) of alum treated Kensico Reservoir (New York) sediments provides a detailed examination of Al release under acidic conditions. Important factors in determining Al mobilisation were pH, temperature, buffering capacity, and the amount of Al-complexing ligands such as DOM, silica and fluoride. In the case of Kensico Reservoir sediments (total buffering capacity 12.2 mg l^{-1} CaCO3; DOC 1.31 mg l^{-1} C; Fluorine 22.8 µg l^{-1} F^-), toxic concentrations of Al^{3+} (~ 50 µg l^{-1}) were mobilised at pH 5.2, 25°C (Driscoll et al. 2014). However, while water total Al concentrations were provided (59 µg l^{-1}), sediment concentrations were not stated and the authors could not differentiate between natural and anthropogenic (i.e. alum) Al sources.

There are a limited number of studies examining the potential release of Al from in-situ lake sediments under acidifying conditions. However, from published data it appears that once $Al(OH)_3$ has settled, a significant sustained acidification event (pH <5) would be required before release of toxic Al species would be triggered (Nelson and Campbell 1991). For example, both Kahl and Norton (1983) and Renberg (1985) reported that aluminium deposition increased in lake sediments following lake acidification (pH 4.5), concluding that sediment was not a source of aluminium and sediment chemistry of aluminium was not significantly altered by acidification. The effects of acidification on Al budgets of five lakes near Sudbury, Ontario were described by Dillon et al. (1988). Lake pH ranged from 4.4 to 7.1 and all lakes were net sinks for Al, although the more acidic lakes had lower Al retention rates (Dillon et al. 1988).

Published research on Al dynamics in freshwater sediments is comparatively scarce, with most research focusing on interactions with phosphorus. Once formed, $Al(OH)_3$ floc ages from an amorphous solid to microcrystals, and then to the mineral gibbsite. During the early stages of aging, it may be transported and redeposited to deeper areas by various lake processes. Following settling, Al-floc is readily incorporated into lake sediments and diffuses away from the sediment surface-water interface. Once incorporated into the sediment, it appears to be more resistant to remobilisation by environmental acidification as it forms inert complexes with silicate minerals. Rates of Al remobilisation will be dependent on pH, temperature, buffering capacity and concentrations of Al-complexing ligands.

Bioaccumulation and Chronic Toxicity

Continuous and repeated applications of alum will increase Al deposition to lake sediments beyond normal background levels (Welch and Cooke 1999; Cooke et al. 2005; Pilgrim and Brezonik 2005). This can lead to increased ingestion, absorption and accumulation of Al in body tissues of benthic organisms such as koura (Ling and Brijs 2009), freshwater snail (*Lymnaea stagnalis*) (Elangovan et al. 1997), freshwater clams *Anodonta anatina* and *Unio pictorum* (Pynnönen 1990) and benthic insects (Herrmann and Frick 1995). Despite strong evidence for bioaccumulation, several studies and review papers have concluded that there

is no evidence of biomagnification of Al in the food chain (Wren and Stephenson 1991; Herrmann and Frick 1995; Sparling and Lowe 1996). This appears to abrogate much of the risk of Al toxicity to vertebrate species such as waterfowl which predate on benthic organisms.

The amount of a toxic metal accumulated by an organism is often taken as an indicator of potential toxicity (Borgmann 2000). Studies investigating chronic toxicity of Al are limited in comparison to acute toxicity tests, but evidence suggests that bioaccumulation of Al is not a predictor of toxicity (Wren and Stephenson 1991; Sparling and Lowe 1996; Walton et al. 2009). In vertebrates, toxic effects due to Al accumulation from ingestion manifest only when associated with low levels of calcium and phosphorus (Scheuhammer 1987; Scheuhammer 1991; Sparling and Lowe 1996). In addition, invertebrates appear to be resistant to dietary Al accumulation; Walton et al. (2010) reported that only 17% of trophically available Al in the tissues of snails (*L. stagnalis*) was transferred when they were fed to freshwater crayfish (*Pacifastacus leniusculus*).

Aluminium is typically concentrated in the digestive and excretory organs of invertebrate species, compared to the gills and bones of vertebrates (Scheuhammer 1991; Sparling and Lowe 1996). Aluminium appears to readily depurate from invertebrates. For example, freshwater crayfish (*P. leniusculus*) fed Al aluminium-spiked pellets (420 µg Al g^{-1}) over 28 days had cleared 54% of Al from the hepatopancreas 10 days after resumption of uncontaminated food. Aluminium caused inflammation in the hepatopancreas but did not affect the number of circulating haemocytes, haemolymph ion concentrations or protein levels. The authors concluded that crayfish were able to accumulate, store and excrete Al from contaminated food with only localised toxicity (Woodburn et al. 2011).

Current evidence suggests that bioaccumulation and biomagnification of Al are not significant hazards. There is little evidence to support biomagnification of Al through the food chain, as rates of trophic transfer appear to be relatively limited. Bioaccumulation of Al is typically associated with incidental ingestion of detritus and sediment, but there is inconsistent evidence that bioaccumulation leads to significant deleterious effects. Further research regarding chronic exposure to Al is needed to ascertain species specific threshold levels for exposure. Presumably, continuous alum dosing regimens such as the Utuhina and Puarenga programmes constitute a greater risk of chronic effects compared with the discrete applications to Lake Okaro, where high depuration rates by benthic invertebrate species are likely to mitigate a significant proportion of the risk.

Risk of an Acidification Event

Acidification by Alum

It is well established that Al is minimally toxic under circumneutral conditions and that toxicity rises exponentially with increasing H^+ concentration (Rosseland et al. 1990; Sparling and Lowe 1996; Rout et al. 2001). A consequence of treating freshwater systems with alum is the release of H^+ during hydrolysis, resulting in environmental acidification if the system has insufficient buffering capacity (Kennedy and Cook 1982). Hydrolysis of Al resulting in the release of H^+ proceeds via the following reactions:

$$Al^{3+} + H_2O \leftrightarrow Al(OH)^{2+} + H^+$$

$$Al(OH)^{2+} + H_2O \leftrightarrow Al(OH)_2^{+} + H^+$$

$$Al(OH)_2^{+} + H_2O \leftrightarrow Al(OH)_3\ (s) + H^+$$

where (s) = solid precipitate (Cooke et al. 2005)

Despite recommendations by Kennedy and Cook (1982) that determination of lake alkalinity and buffering capacity is necessary to ensure an effective, non-toxic alum treatment programme, especially in low alkalinity lakes, very few studies report these values. Determination of alkalinity is critical in lakes with low buffering capacity, as it restricts the quantity of alum that can be applied before pH drops to an undesirable level (i.e. pH <6.0). This in turn dictates the amount of phosphorus that will be adsorbed and the likelihood of attaining a sustained improvement in water quality. Determination of lake buffering capacity will also provide guidance in determining the risk of phosphorus release and mobilisation of toxic Al species should an acidification event occur following lake treatment.

Buffering Capacity of Rotorua Lakes

Cooke et al. (2005) recommended a maximum Al dose for low alkalinity waters (<50 mg l^{-1} as $CaCO_3$) of 5 mg Al l^{-1}. It is therefore critical that environmental managers utilising alum as a management tool are aware of the hydrolysis chemistry of alum and the chemistry of the receiving waters. Data provided by the Bay of Plenty Regional Council shows that the majority of the Rotorua lakes are soft and generally have a low alkalinity (Table 2). Alkalinity for the Puarenga Stream and Hamurana Stream has also been reported as 12 mg l^{-1} and 27 mg l^{-1} (as $CaCO_3$), respectively (Ho 2004). Limited tests by Ho (2004) of Hamurana and Puarenga Stream waters confirm that alkalinity and pH declined markedly at alum dosing rates of 5 mg Al l^{-1}. A report on the buffering capacity of water from Lake Rotorua concluded that there was only sufficient buffering capacity against the addition of 0.1 mg Al l^{-1}, while applications greater than 0.5 mg Al l^{-1} would result in a significant change in lake water alkalinity and buffering capacity exhausted at application rates >1.25 mg Al l^{-1} (Browne 2011). In assessing the risk of a toxicological event from alum dosing, the low alkalinity and buffering capacity of Lake Rotorua (and many of the other Rotorua lakes) is of concern. The extent of a significant toxic event resulting from environmental acidification or anthropogenic Al^{3+} release is likely to be greater in these poorly buffered systems.

Table 2. Mean (±SEM) pH, alkalinity and water hardness of eleven lakes in the Rotorua district. Samples were taken in either July of August from 2009-2014. Data supplied by Bay of Plenty Regional Council.

Lake	*n*	pH	Alkalinity (mg l^{-1} $CaCO_3$)	Hardness (mg l^{-1} $CaCO_3$)
Tikitapu	6	7.1 ± 0.11	4.3 ± 0.23	2.8 ± 0.04
Rotorua	10	7.0 ± 0.07	8.3 ± 0.37	14.8 ± 0.25
Rotoiti	18	7.2 ± 0.04	21.1 ± 0.60	17.7 ± 0.26
Rerewhakaaitu	6	7.4 ± 0.08	24.3 ± 1.02	16.6 ± 0.48
Okareka	6	7.6 ± 0.05	30.3 ± 0.76	18.7 ± 0.61
Okaro	6	7.4 ± 0.08	32.3 ± 0.61	23.5 ± 0.43
Rotoehu	6	7.8 ± 0.06	67.0 ± 1.71	30.3 ± 0.61
Tarawera	5	7.7 ± 0.04	111.6 ± 0.24	59.8 ± 0.97
Rotoma	6	7.1 ± 0.06	182.3 ± 0.88	76.0 ± 0.19
Rotomahana	6	7.1 ± 0.05	182.3 ± 1.69	76.0 ± 1.73

From July 2010 to June 2014, Al loading to Lake Rotorua from alum dosing of the Utuhina and Puarenga Streams averaged c. 200 kg Al day^{-1} with peaks of up to c. 400 kg Al day^{-1} (Hamilton et al. 2015). If the assumption is made that pH of Lake Rotorua should not drop below 6.0, then based on the buffering capacity tests conducted by Browne (2011) the maximum amount of Al that can be applied to Lake Rotorua as a single dose is approximately 554.2 tonnes. In comparison, the current mean daily Al loading to Lake Rotorua from alum dosing of the Puarenga and Utuhina Streams equates to 0.202 tonnes Al day^{-1}. However, it should be noted that the rate of $Al(OH)_3$ sequestration will be limited by the rate of $Al(OH)_3$ mineralisation to gibbsite and lake water residence time (Berkowitz et al. 2005), limiting the scope for increasing alum dose rates.

The buffering capacity of Lake Rotoehu has not been reported; while the water alkalinity is greater than Lake Rotorua (Table 2), this is somewhat countered by the higher volumetric dosing rate to Lake Rotoehu (i.e. Rotoehu 6.72 µg Al l^{-1} day^{-1} cf. Rotorua 2.62 µg Al l^{-1} day^{-1}). Between December 2003 and June 2014, Lake Okaro received seven alum treatments ranging from 190 to 722 kg Al. As the alum treatments of Lake Okaro were discrete, the volumetric doses were considerably larger with the largest being 0.22 mg Al l^{-1} day^{-1}. This dose was reflected in the measured Al concentration of 0.6 mg Al l^{-1} four days after treatment, which was 0.3 mg Al l^{-1} higher than pre-treatment levels (Paul et al. 2008). There was considerable variation in pH in the hypolimnion and epilimnion, both pre-and post-treatment. While a decrease in pH was observed following alum treatment, the values fell within the typical range for the lake (Paul et al. 2008). The shifts in pH were more likely the result of photosynthetic activity and respiration causing fluctuations in dissolved CO_2 concentrations (Talling 1976) rather than hydrolysis of Al.

Risk of Alum Dosing

Although there are few robust studies of alum dosing effects on the Rotorua lakes, current application rates to Lakes Rotorua, Rotoehu and Okaro are duly conservative, even compared to other low alkalinity lakes such as Lake Courtille, France (alkalinity: 22.7 mg l^{-1} $CaCO_3$; alum dose rate: 1.5 mg Al l^{-1}) (Hullebusch et al. 2002) and Lake Moray, USA (alkalinity: 35-45 mg l^{-1} $CaCO_3$; alum dose rate: 12 mg Al l^{-1}) (Smeltzer et al. 1999). Following from this conservative approach, no significant detrimental effects have been reported for the Rotorua lakes (Ling and Brijs 2009; Landman and Ling 2011; Ling 2014). However, the risk of a toxic event resulting from human error or natural hazards should be acknowledged.

Environmental acidification and release of Al^{3+} can have both natural and anthropogenic origins (Gorham et al. 1986). In higher northern latitudes, increasing industrialisation leading to acid rain and snow has been recognised as having significant detrimental impact on freshwater systems for more than 50 years (Schindler 1988). Changes in catchment vegetation leading to soil acidification have also influenced catchments with base-poor geology, although this mainly occurs over moderate time scales (10s to 100s of years) (Gorham et al. 1986). In the Rotorua lakes area, one of the most relevant potential initiators of lake acidification is volcanic or geothermal activity.

Sulphur Bay is a well-known geothermal area on the southern side of Lake Rotorua. The environment in the bay is a naturally occurring simulation of conditions likely to occur during a major Al toxicological event. The pH levels in the bay are significantly lower (c. pH 3.5) compared to the main body of the lake (c. pH 7.0) (Landman and Ling 2008). This, in turn contributes to the elevated Al levels within the bay (c. 1000 µg Al l^{-1}), derived either from leaching of Al of soil and sediment or from Al rich geothermal fluids (Landman and Ling 2009). Biota within the bay is restricted to acid and Al tolerant *Chironomus* species, while at the perimeter, where circulation from the main body of the lake begins to take effect, kakahi (*Echyridella menziesi*) and *Eleocharis* sp are present. Fish species appear to be entirely excluded from the bay (Landman and Ling 2009). The geothermal activity of the Rotorua area is fluidic, resulting in the sudden initiation or cessation of extreme thermal and chemical environmental conditions, including pH changes. It is possible that acidification from geothermal activity could result in the mobilisation of toxic Al species from flocculated alum.

Undesirable Al levels resulting from mechanical faults in continuous alum treatment systems have been reported for Lake Moray, Vermont, and Long Lake, Wisconsin, with a fish kill associated with the latter. Both of these lakes were considered soft water, with the primary cause of failure at Long Lake being a fault in the delivery of the sodium aluminate buffer (Cooke et al. 2005). These cases highlight the importance of failsafe systems and regular monitoring, especially when treating low alkalinity systems. There is also an identifiable risk that should an unintended release of alum occur from the continuous treatment stations on the Utuhina and Waitangi Streams, it is likely to have significant adverse environmental impacts, both from the resulting acidification and the production of toxic aluminium species.

Treatment of soft water systems with alum must be done with caution, as the buffering capacity can easily be exhausted leading to acidification and the production of toxic Al species. The majority of the Rotorua lakes have very low alkalinity and the application of alum has been appropriately cautious. Buffering agents such as sodium aluminate are often

employed when treating such soft water systems as they allow greater alum application rates and improve phosphorus inactivation (Cooke et al. 2005). Use of such buffering agents is recommended if alum application rates above 2.5 mg Al l^{-1} are initiated. In addition, high frequency monitoring of pH levels near lake discharge sites for the Utuhina and Waitangi Streams is recommended. Such monitoring will provide early detection of environmental acidification and allow the dosing stations to be shut down before Al concentrations reach toxic levels.

Risk to Biota from Flocculated Particles

Impact of Alum Flocculent

There is a growing recognition that use of alum for lake restoration may have adverse effects on biota from the production of flocculated material (Pilgrim and Brezonik 2005). Alum flocculation occurs through the neutralisation of electric charge surrounding fine suspended particles. This allows them to coagulate and form larger particles which then settle to the bottom of the water column. In some cases this settled flocculent can be tens of centimetres deep (Pilgrim and Brezonik 2005). Benthic organisms may be adversely affected by the settling flocculent due to burial, modification of habitat, and oxygen depletion from bacterial decomposition of sedimented organic particles (Pilgrim and Brezonik 2005). In addition, the insoluble $Al(OH)_3$ precipitate that forms under circumneutral conditions can have an irritating effect on fish gill surfaces, leading to inflammation of the gill tissue, excessive mucus secretion, reduced blood oxygenation and even death (Sparling and Lowe 1996; Gensemer and Playle 1999).

Unlike the toxic effects of aqueous Al species which primarily occur at pH <5.0, inflammation and gill mucification leading to asphyxiation from particulate $Al(OH)_3$ is more prevalent at pH 6.0-8.0 (LaZerte et al. 1997). Rainbow trout exposed to total Al concentrations of 76 µg l^{-1} at a range of pH's (4.0 – 6.5) had maximum gill ventilation rates and minimum blood oxygen levels at pH 6.1 (Neville 1985). This was attributed to either adhesion of colloidal $Al(OH)_3$ on the gill surface or formation of gill-ligand-$Al(OH)_X$ interfering with oxygen uptake (Neville and Campbell 1988). In other cases, reduced blood oxygenation may have been responsible for the decreased activity observed in smallmouth bass (*Micropterus dolomieui*) exposed to Al of 200 µg l^{-1} at pH 7.3 for 30 days (Kane and Rabeni 1987), and rainbow trout exposed to Al of up to 5200 µg l^{-1} at pH 7.0 for 6 weeks (Freeman and Everhart 1971). For discrete alum applications, the problem of colloidal $Al(OH)_3$ adhesion to gills may be limited by the fact that increasingly larger floc particles form over time and fall from the water column. While continuous alum applications provide a constant source of colloidal particles, alum dose rates are typically much lower, reducing the likelihood of adverse effects. For example, continuous exposure to 52 µg Al l^{-1} at a range of pH (7-9) produced no physiological or behavioural responses in rainbow trout (Freeman and Everhart 1971).

A drawback of continuous exposure experiments is they do not provide a realistic test of the impact of an alum treatment on fish; in actual practice, fish are exposed only when the $Al(OH)_3$ floc falls through the water column, unless the floc settles on early life stages (Cooke et al. 2005). In the case of the Rotorua lakes, the stream velocity of the Utuhina, Puarenga and Waitangi Streams makes it unlikely that Al-floc will accumulate and adult fish

are more likely to avoid areas where the streams discharge into the lake and velocities decrease enough to allow the floc to settle. Quinn et al. (2004) suggested that the initial alum dosing of Lake Okaro in 2003 may have resulted in decreased abundance of common bully, but this could not be definitively attributed to the alum treatment. The effects of Al floc are more likely to impact benthic organisms that are either immobile or have limited mobility. For example, in a mesocosm study, application of alum (total Al 159 mg l^{-1}) resulted in a significant reduction of fingernail clam (*Sphaerium novaezelandiae*) survival after 2 months (23%), compared to controls (92%). In contrast, no significant effects were observed in mesocosms containing freshwater cray fish and common bully at the same dose rate (Clearwater et al. 2014). The authors suggested that the reduced fingernail clam survival was due to lack of bioturbation by macroorganisms resulting in clogging of gills and feeding appendages and that this was unlikely to occur under natural conditions (Clearwater et al. 2014).

As pH is lowered below or raised above 6.3, more Al soluble species are formed, and less insoluble $Al(OH)_3$ is produced at a given dose. Therefore, the amount of flocculent formed is largely dependent on pH and Al dose (Pilgrim and Brezonik 2005; Browne 2011). Unsurprisingly, the size of flocculated particles is also related to pH and Al dose rate (Pilgrim and Brezonik 2005; Browne 2011). In column tests using water from Hurley Wetland (Minnesota), the amount and size of flocculent particles was found to be greatest at 8 mg Al l^{-1} and pH 6.1 by Pilgrim and Brezonik (2005). Tests conducted using Lake Rotorua water (pH not stated) found no settled floc at or below 0.5 mg Al l^{-1} and notable flocculation only occurred at dose rates above 0.75 mg Al l^{-1} (Browne 2011).

While a substantial body of research has been published on the toxic effects of Al, little research has been conducted on the potential negative effects of Al floc. Adverse effects appear to be restricted to invertebrate fauna. Alum dosing of the Cuyahoga River, Ohio, of up to 17 m^3 alum day^{-1} (discharge not provided) resulted in large accumulations of flocculent, over 0.5 m deep within 4 weeks. Following cessation of treatment, large increases in abundance of oligochaetes were observed along with decreases in all other taxa, particularly chironomids (Barbiero et al. 1988). It should be noted that some declines in invertebrate abundance were observed at several monitoring sites prior to flocculent accumulation, resulting in the authors being unable to distinguish between the effects of toxic Al species and those of flocculent deposition.

Following 12 months of alum treatment (8 mg Al l^{-1}) of water entering Fish Lake (Minnesota) (mean discharge 3970 m^3 day^{-1}) by way of a settling pond, flocculent had accumulated to a depth of approximately 0.5 m in the settling pond. This resulted in elimination of all Chironomidae, Gastropoda and Chaoboridae and macrophyte species in the settling pond; all of which were abundant in the discharge area to Fish Lake. In comparison, a dose rate of 1 mg Al l^{-1} in the previous year had no effect on benthic invertebrate community composition or abundance in the settling pond. The authors attribute the effects on benthic invertebrate community in 2000 to physical displacement of suitable habitat (Pilgrim and Brezonik 2005).

It is difficult to assess the risks of alum flocculent accumulation given such data is rarely reported. Flocculent accumulation depths of 0.5 m are likely to be the extreme upper end of the scale and it is unsurprising that negative effects such as hypoxia and habitat disruption are associated with such accumulations. Lamb and Bailey (1981) studied the effect of alum floc on benthic invertebrates in a laboratory setting to determine if proposed whole lake

alum treatments would have adverse effects on benthic invertebrates. They reported the formation of floc layers up to 4 mm in which chironomids (*Tanytarsus dissimilis*) readily formed tubes. While heavy Al doses (6.5 to 77.7 mg Al l^{-1}) caused more floc formation and significant mortality with time, it was concluded that at doses <77 mg Al l^{-1}, floc did not have a negative influence on behaviour and survival (Lamb and Bailey 1981).

Risk of Oxygen Depletion

A concern with Al floc formation is that bacterial respiration during the breakdown of trapped organic particles may generate anoxic conditions. Only one study has directly measured oxygen depletion due to Al floc accumulation. As reported above, alum dosing the Cuyahoga River resulted in the formation of 0.5 m deep floc deposits in some locations. Reduced oxygen levels were occasionally associated with these deposits, with some locations producing DO levels of <1.0 mg l^{-1} (Barbiero et al. 1988). The 0.5 m of Al floc that accumulated in the settling pond at Fish Lake was associated with an unpleasant hydrogen sulphide odour indicating anoxic conditions (Pilgrim and Brezonik 2005), however the authors did not provide any direct measurement of DO levels.

Interference of Natural Processes

The desired outcome of alum dosing is a reduction in phosphorus availability resulting in reduced primary productivity. Ideally, this will shift the system to a lower trophic level causing a reduction in total biomass and changes in community species composition. A number of unintended disruptions to lake systems have been noted or proposed in the literature associated with alum dosing. These appear to be either isolated incidents or areas where further research is required.

Following discrete alum dosing, flocculation of algal cells is a common and desirable result for lake managers as immediate improvements in water quality are apparent (Sparling and Lowe 1996). There have also been associated decreases in zooplankton abundances, which have either been attributed to reduced food abundance (Gensemer and Playle 1999) or from entrapment in flocculent (Schumaker et al. 1993). Such effects are transient and declines in zooplankton abundance temporary but subsequent changes in taxonomic composition should be expected (Schumaker et al. 1993). Whether such fluctuations in abundance and species composition have flow-on effects to the rest of the food-chain have not been investigated.

As established previously, Al and Al-floc are unlikely to impact on macroinvertebrate communities unless Al dosing is extremely high (Lamb and Bailey 1981; Barbiero et al. 1988; Doke et al. 1995). The effects on microorganisms are less defined. Bulson et al. (1984) established that alum treatment of Liberty Lake (Washington) resulted in flocculation of up to 95% of the total culturable bacterial population from the water column. The authors warned that the resulting floc could pose a health risk due to the concentration of enteric organisms, including pathogens. In addition, investigations of alum as a sediment capping agent suggest that it may suppress bacterial nitrification resulting in enhanced release of ammonium (NH_4-N) by 70% (Gibbs et al. 2011). Gibbs et al. (2011) attributed this to the capping layer thickness reducing the diffusion of oxygen to nitrifying bacteria. Further

investigation of this result is needed as observations were conducted under highly modified laboratory conditions, and may not be translatable to real world conditions.

Current published literature provides only limited guidance as to the ecological effects of Al floc deposition. However, current alum dosing rates to the Rotorua lakes are unlikely to generate Al floc deposition of sufficient volume to cause notable hypoxia or adverse impacts on benthic macroinvertebrates. Sustained exposure to colloidal $Al(OH)_3$ (>75 µg Al l^{-1}) precipitate does cause inflammation of gill surfaces, mucification and respiratory distress in fish, but alum dosing rates to the Rotorua lakes are unlikely to generate such sustained levels of exposure. In addition, many species are likely to actively avoid such conditions. Few conclusions can be made regarding potential effects of Al-floc formation to natural processes in the Rotorua lakes as only very limited exists. Further research is needed in areas of food web modification, community composition and bio-geochemical processes.

Conclusions

The restricted alum dosing rates currently employed in the Rotorua lakes district appear to have had little identifiable impact on local biota. Current alum dosing rates are well below proposed threshold values for acute Al toxicity for a wide range of organisms. It is recommended that total Al concentrations do not exceed 200 µg l^{-1} at pH >6.0, 75 µg l^{-1} at pH 5.0 - 6.0 and 25 µg l^{-1} at pH 4.0 - 5.0. However, it is likely that DOM and silica will provide further protection due to their propensity to form complexes with toxic Al species. Despite this, a conservative approach is still recommended as chronic exposure to Al, especially under conditions of continuous application, has not been extensively examined. In addition, many of the Rotorua lakes have extremely low alkalinity, which indicate that their buffering capacity would become exhausted at Al application rates greater than 5 mg l^{-1}, likely resulting in environmental acidification and release of toxic Al species. Restraint should also be taken with regards to proposed threshold values for high pH conditions: at pH 9.0, levels of 200 µg Al total l^{-1} should not be exceeded and at >pH 10 a threshold value of 100 µg total Al l^{-1} should be employed.

Bioaccumulation of Al occurs primarily through ingestion or, in fish, adsorption on to the gill surface. However, the relationship between bioaccumulation and toxicity appears to be highly variable and species specific. This is likely related to the fact that relatively little Al is absorbed through the digestive system and there is little evidence for biomagnification. Associated with this is the fact that depuration rates for Al are relatively high preventing excessive accumulations of Al under discrete application regimes. However, continuous alum application programmes are of more concern and further research is needed in this area.

The formation of large accumulations of Al-floc is associated with high alum dose rates (>8 mg Al l^{-1}) over extended periods of time. Current alum dose rates to the Rotorua lakes are unlikely to generate significant quantities of Al-floc. The risk of hypoxia and disruption of natural lake processes from Al-floc therefore appear to be minimal, but further research is recommended. Al-floc initially has a low density and is readily disturbed, but also readily diffuses through sediment surface layers and over time ages to form the mineral gibbsite that is resistant to mobilisation.

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