Estuarine Acidification: Impacts on Aquatic Biota of Draining Acid Sulphate Soils

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Acid sulphate soils form when iron pyrite oxidises to sulphuric acid on atmospheric exposure, lowering soil pH below 4. In the Richmond River estuary, northern New South Wales, flood mitigation, drainage works and floodplain excavations have augmented acid sulphate soil formation by increasing pyrite availability for oxidation. These engineering works have facilitated the transport of acidified water and have impeded recovery from tributary acidification. The increased frequency and duration of acidification are associated with elevated concentrations of inorganic monomeric aluminium and dissolved iron, and major iron hydroxide precipitation, with deleterious effects on exposed aquatic biota and habitat. Fish kills and an outbreak of epizootic ulcerative syndrome (EUS) have been recorded from acidified sites in the estuary.

Major soil acidification in Australia occurs in Holocene estuarine sediments of coastal floodplains. Drainage from acid sulphate soils contained in these sediments is the dominant cause of acidification in estuarine waterways (Willett *et al.*, 1993). Coastal floodplains have been modified through drainage and excavation to facilitate a range of human activities dominated by agriculture and urbanisation. Consequently, these modifications increase the production and transport of acidified water (Willett *et al.*, 1993). Both freshwater and brackish water environments are affected, as the output of acidity from these soils may at times exceed the neutralising capacity of the waterway (Dent and Bowman, 1993).

The acidification of estuarine tributaries is a major threat to the recreational, commercial and conservation value of aquatic ecosystems. Fish kills are an immediate and recognisable response to many acidification events. However, there are many direct and indirect impacts of acidification that are more significant than fish kills, but generally less visible. To date, the actual and potential impacts of estuarine acidification have not been fully

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appreciated or have been blamed on a variety of other factors. Although there is much to learn about estuarine acidification and its impacts, there are sufficient data to demonstrate that this perturbation urgently requires management.

We present, in this paper, observations and preliminary data implicating acidified water derived from acid sulphate soils as a major cause of ecosystem degradation in the estuarine tributaries of the lower Richmond River, northern New South Wales (Fig. 1). Estuarine acidification can be spatially extensive and causes major habitat loss and negatively affects aquatic flora and fauna. We present evidence associating acidification with fish kills and outbreaks of fish disease and discuss the probable deleterious effects of acidification on reproduction and recruitment in fish.

The role of drainage in the acidification of estuarine tributaries

Acid sulphate soils form when sediments contain so much naturally accumulated iron pyrite (FeS_2), that the sulphuric acid produced through its oxidation exceeds the acid-neutralising capacity of carbonates in the soil. Pyrite is preserved under anaerobic conditions, typically found below the watertable, and only presents an environmental threat when exposed to air and moisture (Lin and Melville, 1992). Pyrite may oxidise following excavation and relocation of sediments into aerobic environments, or by lowering of the watertable, either naturally through evapotranspiration processes or through artificial drainage (Lin *et al.*, 1995). Aluminium and iron are mobilised from the sediments by the increased acidity, and along with sulphate, are flushed into nearby drains and waterways during wet periods (Willett *et al.*, 1993).

On the north coast of NSW, flows from many estuarine tributaries have been modified by floodgates, and natural levees have been breached by artificial drains. These modifications occurred in stages. In the early 1900s, small drains and floodgates were built by drainage groups and individual farmers with the general purpose of expanding grazing land (Middleton *et al.*, 1985). In 1957, a government-subsidised flood mitigation programme was established to provide protection to flood-prone towns in NSW, and to reduce the frequency of flood-related losses to rural production (Bodycott, 1993). Currently, floodgates and barrages (multiple floodgates) are installed at selected tributary junctions; they are designed to open outward during low tide and to close during high tide, thereby restricting the inflow of tidal water and assisting in the removal of floodwater from the floodplain. In addition, these modifications lower the long-term equilibrium elevation of shallow watertables in backswamps, expanding the area of land available for dryland activities. Consequently, the lowering of the watertable allows increased pyrite oxidation during drier periods, but the oxidation products are usually transported only during wet periods (Willett *et al.*, 1993).

Artificial drainage systems also modify the habitat upstream of the control structures. With restricted tidal inflow, the upstream reaches become less saline and, therefore, less buffered than the tidal reaches downstream. Floodgates also dampen water level fluctuation in the upstream reaches. Freshwater habitat is expanded at the expense of important brackish water habitat, and the floodgated reaches are more susceptible to acidification.

Drainage and Acidification of Tuckean Swamp

At Tuckean Swamp, on the left bank of the lower Richmond River, water discharges through the Bagotville Barrage into the Tuckean Broadwater (Fig. 1), a 4.5 km channel flanked by tidal flats. Tuckean Swamp is dissected by a system of artificial drains which has increased drainage density and changed the drainage pattern of the swamp (Fig. 2). These modifications have increased the discharge capacity of the drainage basin. The drainage system occurs within extensive areas of acid sulphate soils (typically pH 4 down to ~ 2). The 1:5 soil:water pH profile (Fig. 3) shows the

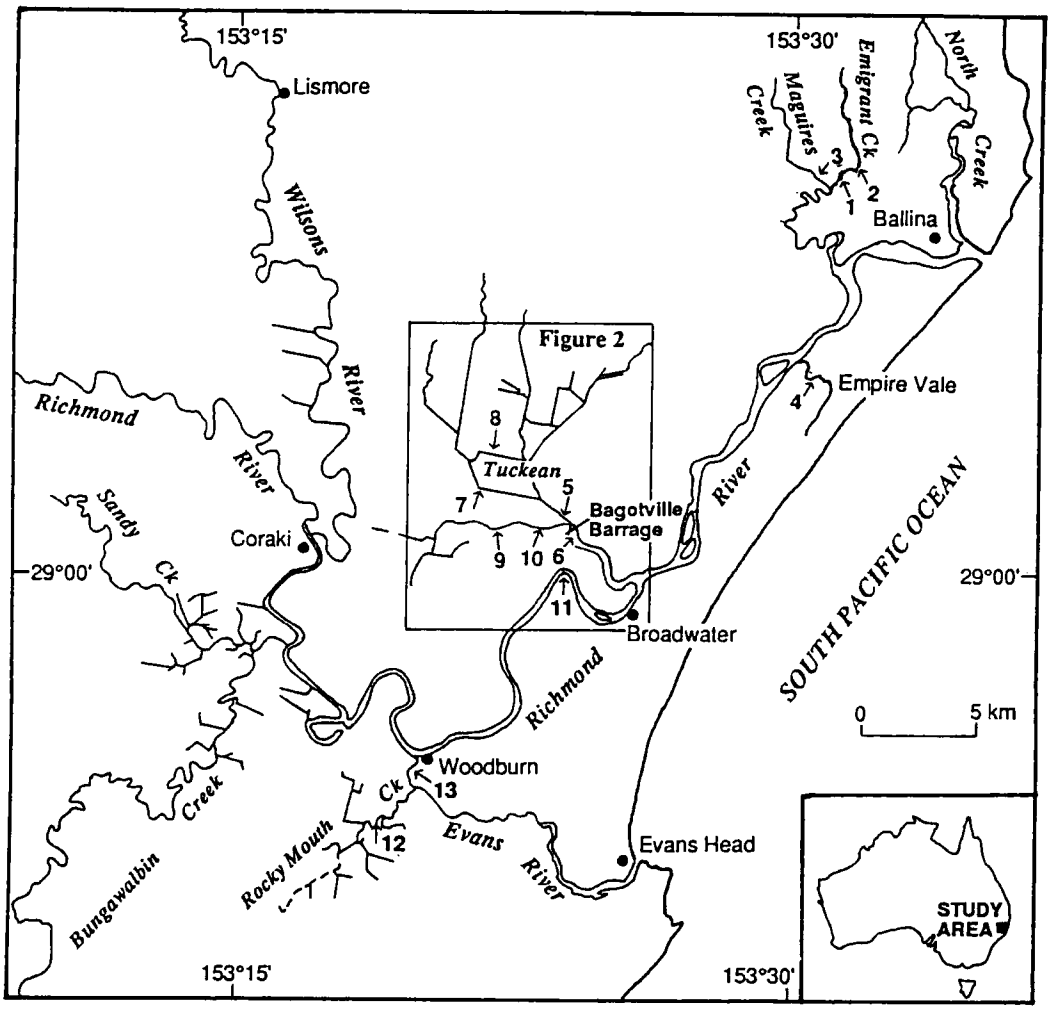

Fig. 1 Study area location and water quality sampling points for Table I data

depth location where soluble acidity exists in greatest concentration (low pH) at an example soil profile site. This occurs above the profile location of maximum peroxide-oxidisable sulphuric acidity (POSA). The parameter POSA, is a measure of the concentration of iron pyrite in the profile and, therefore, a measure of the total potential acidity (Lin and Melville, 1993).

The peak of POSA in the profile (Fig. 3) represents part of the original Holocene estuarine sediment surface, possibly under mangrove vegetation, where pyrite accumulation was maximal at the time that the present sea level was reached. Pyrite has been oxidised above this profile elevation (and therefore POSA lessened) although it is not clear how much low pyritic freshwater sediment has been added

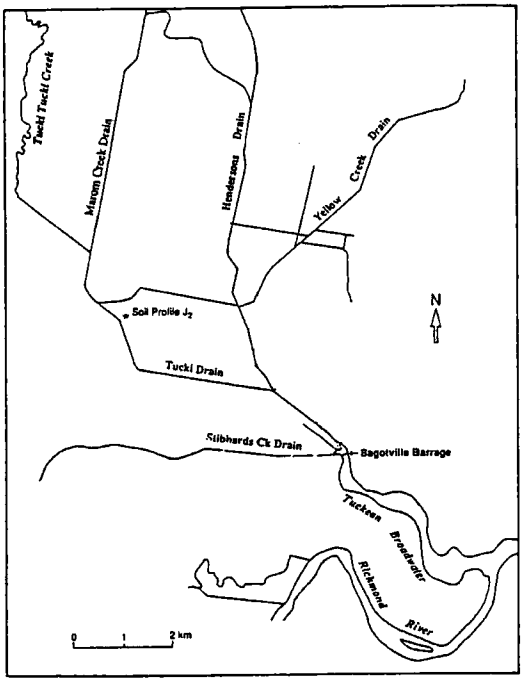

Fig. 2 Major drains of Tuckean Swamp and location of J2 soil profile

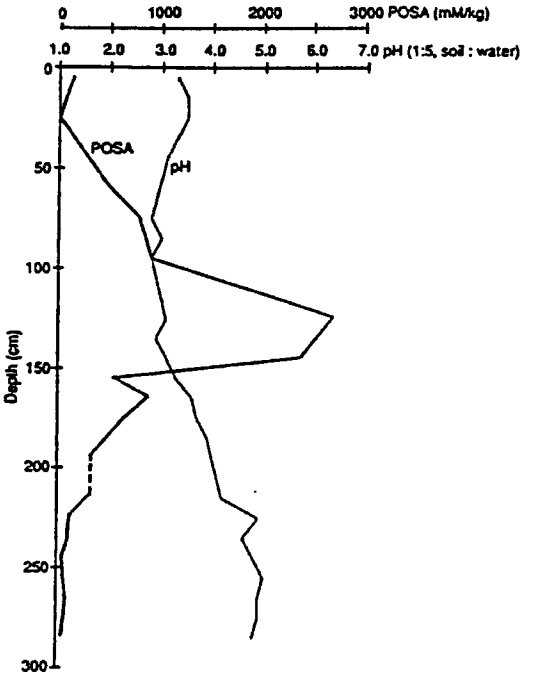

Fig. 3 Depth characteristics of profile J2 soil properties (1:5, soil:water pH) and peroxide oxidisable sulphuric acidity

towards the profile surface. The pyrite oxidation produces soluble acidity that decreases pH at a profile location but which also moves upwards through the profile during subsequent watertable rises. The low POSA below about 150 cm occurs because estuarine sedimentation was too rapid to allow much pyrite accumulation in the time prior to present sea level being reached.

Acidity in Tweed River canefield drains enters from immediate channel-bank runoff during most rainfall events. Major acidity inputs from the acid sulphate soil landscape occur during large rainfall events, which raise the watertable above the ground surface and thereby enable acidic runoff into drains (Lin *et al.*, 1995). Such changes in drainwater pH can be very rapid. Similar processes have been observed in Tuckean Swamp and other sites on the lower Richmond River system.

A series of water quality measurements in Tuckean Swamp was made in July 1993 (Fig. 4) along the major drains feeding into the Bagotville Barrage (Fig. 2). The pH measurements of drain water on 5 July 1993 showed that all drains were acidified in the monitored reaches and provided acidified drainage water which discharged through the barrage. The very acidic conditions in Stibbards Creek Drain and Hendersons Drain reflect the low ground-surface elevations and low or absent levee banks that otherwise impede acidic runoff during most rainfall events. Hendersons Drain can remain acidic in its upstream reaches for periods greater than 21 months. Tucki Drain has more extensive levee-bank protection but can become very acidic (pH < 3.5), particularly when moderate flooding exceeds the capacity of levees to impede acidic runoff. In March 1994, which regionally was the wettest on record, acidic

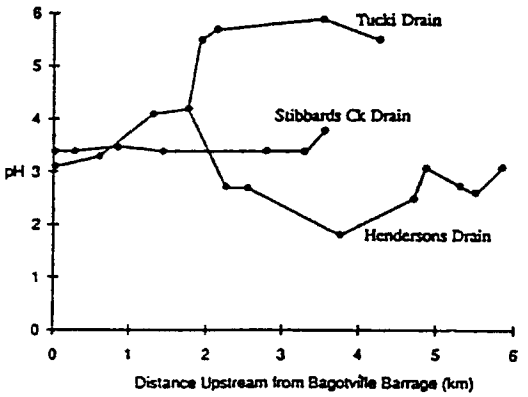

Fig. 4 pH measurements along the major drains upstream of Bagotville Barrage on 5 July 1993

floodwaters breached the levees in Tucki Drain and acidified the drain (pH < 4) for approximately four kilometres. Our ongoing sampling programme has shown that the spatial and temporal extent, as well as the level of acidity in the Tuckean Swamp drains, vary because of differences in hydrological and antecedent conditions, and ongoing land development.

Acidification of River Reaches Downstream of Flood Mitigation Structures

The acidity produced in drained floodplains is not necessarily retained in the artificial drainage systems. At Tuckean Swamp, the acidified water can discharge through the Bagotville Barrage as an acidic plume, which typically has a pH less than 4.5, but which has been recorded as low as pH 2.7. The plume can completely displace pH-neutral water in the Tuckean Broadwater on the outgoing tide during periods of high discharge. During periods of low discharge, acidity in the Broadwater is neutralised by brackish-water mixing on the incoming tide. Seawater has a neutralising capacity of about 2 moles m^{-3} (Dent and Bowman, 1993), much greater than that of fresh and brackish water. Because the barrage gates close on the high tide, reaches upstream of the barrage do not benefit from tidal neutralisation except when trapped debris enables leakage through the gates.

After heavy rainfall, sections of Tuckean Broadwater may remain acidified regardless of tidal fluctuation. This is illustrated in Figure 5, where prior to a flood-front on 13 March 1991, pH in the Broadwater fluctuated with the tide at the monitoring site because of alternating acid discharges (low tide) and acid-neutralisation (high tide). After the flood-front passed in the Richmond River, the neutralising effect of the incoming tidal water decreased and the acidic discharge from Tuckean Swamp increased. The mean pre-flood electrical conductivity measured at the peak of each high tide was 10.3 ± 0.74 dS m^{-1} (n=9), whereas after the flood-front, the mean value was 0.49 ± 0.18 dS m^{-1} (n=11). Consequently, pH remained low irrespective of tidal influence. Our recent sampling has shown that such acidification can persist for more than six weeks if river salinities are depressed by high rainfall and associated flooding.

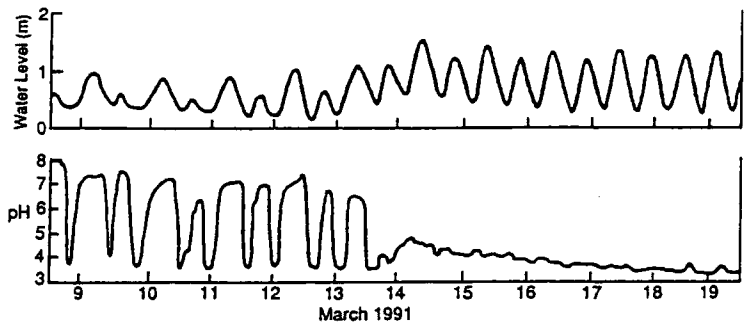

Fig. 5 Tidal and floodwater controls on pH in the Tuckean Broadwater, 50 m downstream of the Bagotville Barrage from 9/3/91 to 19/3/91. Data collected by submersible data logger, 4 samples per hour. Water level is at an arbitrary datum. *Source*: C. Peak and G. McLennan, NSW Agriculture, Wollongbar

Acidic pulses in Tuckean Broadwater can occur intermittently for months during low tides and they can be moved backwards and forwards as a slug by the tide when mixing is limited. Stratification occasionally occurs in the Tuckean Broadwater and the Richmond River junction because of density variations between the acidic and pH-neutral waters that are unrelated to temperature differences. Rather, the electrical conductivity of the Richmond River can be lowered by minor flooding with the electrical conductivity of the acidified water in Tuckean Broadwater being higher because of the transport of dissolved pyrite-oxidation products. The acidified water, therefore, is denser, and can flow beneath the pH-neutral water of the Richmond River during low tide. By contrast, when the salinity of the Richmond River increases, the tidal water becomes many times denser than the acidified water and tends to flow beneath it. A wedge of brackish water can migrate up the Tuckean Broadwater causing physico-chemical stratification. The interaction between the acidic and neutral water is variable and dependent on a complex of factors including the ionic strength of the acidic and neutral water, and the hydrological conditions.

Several times during 1993, the tidal reaches of the main tributaries of the lower Richmond River were simultaneously acidified (Sammut *et al.*, 1993). These tributaries have all been modified by flood-control structures and artificial drainage systems. An aerial survey of the lower Richmond River and its tributaries in July 1993 demonstrated the extent of the acidification problem in the main tributaries. More than 70 per cent of drains and tributaries showed evidence of iron precipitation and clear or green water associated with aluminium-induced flocculation. Acidic plumes were evident below barrages and floodgates and there was evidence of acidification in the main channel of the Richmond River. The acidified water contrasted markedly with the pH-neutral reaches, which were characteristically turbid. These data and observations indicate that acidification of estuarine tributaries, in both tidal and non-tidal reaches, can be spatially extensive with serious implications for aquatic biota.

Chemistry of acidified water

Although low pH is the primary chemical feature of water drained from oxidised pyritic material, there are associated chemical changes which may also degrade ecosystems. Lin and Melville (1992) showed that both monomeric aluminium and exchangeable aluminium concentrations, in acid sulphate soil, increase exponentially as pH decreases. Inorganic monomeric aluminium species (Al^{3+}, $Al[OH]^{2+}$ and $Al[OH]_2^{+}$) are especially toxic to aquatic life (>0.1 mg L^{-1}) and are usually present in acidified water (Driscoll *et al.*, 1980; Driscoll, 1989).

During 1993 water samples were collected from the lower Richmond River at widely located sites (Fig. 1) and were analysed for inorganic monomeric aluminium using a modification of the catechol violet method of Dougan and Wilson (1974). The purpose of the sampling was to determine if toxic concentrations of inorganic monomeric aluminium (> 0.1 mg L^{-1}) were present at various river sites where recent acid outflows had lowered pH. Inorganic monomeric aluminium was measured at concentrations up to 4.2 mg L^{-1} at sites where pH was less than or equal to 5.4; it was not detected by the method at sites where pH was greater than 5.4 (Table I). Flocs of aluminium hydroxides were frequently observed at sites where pH was greater than 6. Variation in inorganic monomeric aluminium concentration between sites of similar pH may be due to the stage of acidification, or the presence of ligands such as humic substances (Witters *et al.*, 1990), and sulphate and hydroxides (Driscoll, 1989). More detailed chemical analyses to assess and measure the influence of ligands on inorganic monomeric aluminium concentration are being undertaken by the authors.

Soluble ferrous iron is present at pH<4 in acidified drainage water, but when pH increases above 4, and oxygen is present, iron oxyhydrox-

Site No.	Date	pH	Inorganic monomeric aluminium (mg L^{-1})
1	19 July	5.4	0.5
2	19 July	3.0	1.8
3	19 July	3.3	2.4
4	19 July	6.2	ND
5	9 June	3.9	4.2
5	19 July	3.6	1.7
5	20 August	3.6	3.4
6	19 July	3.3	1.8
6	20 August	5.4	ND
7	20 July	5.8	ND
7	21 July	5.8	ND
8	20 July	3.9	2.7
8	21 July	3.8	1.7
8	20 August	3.7	2.7
9	20 August	5.8	ND
10	20 August	3.5	3.9
11	20 August	6.3	ND
12	9 June	2.9	0.7
12	19 July	3.4	1.4
13	19 July	3.3	1.7

Table I: Simultaneous pH and Inorganic Monomeric Aluminium Concentration Measured in the Richmond River in 1993

ND = not detected

ides may be formed (Simpson and Pedini, 1985). The oxidation of ferrous iron (an initial product of pyrite oxidation) to iron hydroxide, consumes oxygen and releases hydronium ions (H_3O^+), thereby decreasing dissolved oxygen concentration and pH.

Impacts on aquatic biota

Impacts of Inorganic Monomeric Aluminium

Although exposure to low pH alone is directly injurious to fish (McDonald, 1983), elevated concentrations of inorganic monomeric aluminium species are considered to be the primary cause of injury and death in fish exposed to acidified water (Driscoll *et al.*, 1980). Norrgren *et al.* (1991) identified the gills as the main site for aluminium accumulation during experimental exposure of fish to low pH and high dissolved aluminium concentrations. Accumulation was also reported in the olfactory mucosa.

Fish maintain an ionic balance with their environment through differential permeability of their gills and the excretion of ions in urine (Ferguson, 1989). The impermeability of the gills is thought to be dependent on calcium and other divalent ions (Freda and McDonald, 1988). When dissolved, aluminium binds to negatively charged gill surfaces, displaces calcium and gill permeability is increased (Playle and Wood, 1991). This results in a net efflux of sodium and chloride from the bloodstream under freshwater conditions causing an ionic imbalance and physiological stress (Freda and McDonald, 1988). The toxicity of inorganic monomeric aluminium to fish may be moderated by high concentrations of calcium, which protect the epithelium of gills and skin (Ingersoll *et al.*, 1990).

In addition to impaired ion regulation, damaged gills may become swollen and fused, thereby impairing gas exchange (Norrgren *et al.*, 1991). Excessive mucous production may occur on the gills due to irritation caused by aluminium precipitation, further impairing oxygen uptake (Evans *et al.*, 1988). Gill damage has been suggested as a cause of fish mortalities in Australia where acidified water and high concentrations of aluminium have been recorded (eg. Brown *et al.*, 1983). However, we have been unable to find published reports on the pathology of gill damage in Australian fish caused by high concentrations of aluminium at low pH.

Seasonally recurrent fish kills are a common feature of acidification (Callinan *et al.*, 1993). Clinical signs, such as gulping at the water surface and abnormal opercular movement, are usually observed in fish exposed to low dissolved oxygen concentration. Such behaviour was observed in fish exposed to acidified water (pH 5) at a fish kill upstream of Bagotville Barrage, Tuckean Swamp, in March 1993. Dissolved oxygen concentrations (4.8 to 5.8 mg L^{-1}) recorded at the kill site (M. Digby, Southern Cross University, unpublished data) were not at lethal levels. Although inorganic monomeric aluminium was not measured at this site, it is likely to have been present, as aluminium-induced flocculation was observed, and inorganic monomeric aluminium has been measured at and below pH 5.4 elsewhere in the Richmond River system (Table I). Histological examination of gills from moribund yellowfin bream (*Acanthopagrus australis)* and mullet (*Mugil cephalus*), showed severe inflammation, epithelial cell necrosis and extensive fusion of the gill filaments. Mortalities were attributed to impaired oxygen uptake and osmoregulatory stress. Given that gill damage from acid exposure may also trigger gulping at the surface and respiratory distress, it is likely that, in the absence of appropriate water quality data, some coastal fish kills have been blamed erroneously on low dissolved oxygen rather than acidification. Nevertheless, low dissolved oxygen concentrations are still a common cause of coastal fish kills.

Experimental exposure of fish to acidified water with high aluminium concentrations (Tandjung *et al.*, 1982) resulted in degenerative changes to epidermis, and in extreme cases, epidermal necrosis. In Tuckean Swamp, moribund fish (*Hypseleotris compressa*), exposed to a change in pH from 6 to 3.9 in less than six days, and to elevated concentrations of inorganic monomeric aluminium (1.5 to > 2.0 mg L^{-1}), had macroscopic evidence of skin damage similar to that reported in experimental exposure of fish to acidified water. Histological examination showed epidermal proliferation, with scattered foci of severe dermatitis. Because the skin, along with the gills, plays a role in oxygen uptake (Ferguson, 1989), such pathological changes in the skin will interfere with this process.

The impacts of acidified water on fish are most severe behind floodgates and barrages, as these structures limit escape from acidified water and prolong the duration of acid exposure. Where possible, fish avoid acidified water (Sammut *et al.*, 1993).

Impacts of Iron

Iron may also have deleterious effects on aquatic fauna. Simpson and Pedini (1985) reported that iron precipitated as iron hydroxide onto the gills of crustaceans and fish, limited gas exchange and caused suffocation. They also reported that eggs and larvae may be affected by iron precipitates and decreases in dissolved oxygen caused by the iron oxidation process. Cruz (1969) reported that iron could be absorbed in the digestive tracts of fish in large quantities, causing lethal degenerative changes in various internal organs.

Mass mortalities of worms and bivalves were observed at pH-neutral sites in the lower Richmond River in 1993 and 1994. Dead and dying specimens were coated with iron oxyhydroxides which may have caused suffocation.

The compounds had been transported from acidified areas and deposited at pH-neutral sites or had formed *in situ*. The transport and precipitation of iron oxyhydroxides persists for months after initial acidification.

Extensive seagrass beds, once present in the Tuckean Broadwater, are now absent. Iron precipitation and acidified water may have contributed to their disappearance as the system is now frequently affected by acidic discharge. Photosynthesis in seagrasses may be limited by iron oxyhydroxide coatings but we do not discount other influences unrelated to acidification.

Grise *et al.* (1986) found that aluminium and iron concentrations were high in stunted specimens of *Vallisneria americana* (a freshwater plant) and suggested that metal toxicity causes growth reduction at low pH. In the March 1993 acidification event, beds of *Vallisneria gigantea* in Tuckean Swamp drains were killed. The dying plants were heavily coated with iron oxyhydroxides and exposed to toxic concentrations of inorganic monomeric aluminium (> 4.0 mg L^{-1}). By contrast, waterlilies (*Nymphaea gigantea* Hook and *N. caerulea* ssp. *zanzibarensis* [synonym *N. capensis*]) and spike rushes (*Eleocharis* spp.) have successfully completed all life stages in the acid drains of Tuckean Swamp where they can occur in high densities from pH 4 down to 1.8. They are, therefore, also aluminium-tolerant. The photosynthetic surfaces on the leaves of water lilies are usually free of iron oxyhydroxide when pH levels rise and iron precipitates. Spike rushes are semi-emergent so that photosynthesis is unaffected by iron deposits. The pH-neutral drains in Tuckean Swamp support a greater variety of waterplants, most of which are not present in the acidified drains (eg. *Nymphoides indica*, *Marsilea mutica*, *Najas tenuifolia*, *Potamogeton* spp. and *Vallisneria gigantea*).

Epizootic ulcerative syndrome and acidified water

Epizootic ulcerative syndrome (EUS), also known in Australia as red spot disease, is an ulcerative skin disease affecting estuarine and freshwater fish. First reported in Australia from the Burnett River in 1972 (McKenzie and Hall, 1976), EUS has since shown a pattern of seasonal recurrence in many east coast river systems (Callinan *et al.*, 1993). Outbreaks have been recorded from northern Queensland (Rodgers and Burke, 1981) to southern NSW (Callinan, unpublished data), in southwest Western Australia since 1983 (Pass, Langdon and Kabay, unpublished data) and in the Northern Territory since 1986 (Pearce, 1990). The disease has also been recorded in Papua New Guinea and throughout southeast and southern Asia, where it is considered one of the most destructive fish diseases to have occurred in that region (Lilley *et al.*, 1992).

Callinan *et al.* (1989) showed that massive invasion of the skin by fungi plays a central role in the induction of ulcers. Fraser *et al.* (1992) recovered the oomycete fungus *Aphanomyces* sp., from 27 out of 28 ulcers on three estuarine fish species from three widely separated river systems in Australia. Morphologically and culturally similar fungi are also consistently present in lesions of fish affected with EUS in Asia (Roberts *et al.*, 1993; Callinan, unpublished data). Callinan *et al.* (1993) suggest that an *Aphanomyces* sp. is the causative infectious agent of EUS. In recent research, attempts to induce typical lesions by exposing fish experimentally to fungal spores at concentrations of up to 100 spores ml^{-1} were successful only when skin was abraded prior to exposure (Callinan and Fraser, unpublished data). EUS has also been artificially induced by hyphae experimentally inserted into the muscle of fish (Roberts *et al.*, 1993). These findings suggest that factors other than simple exposure to the fungus are necessary for EUS outbreaks to occur in wild fish populations. This fungus can survive and produce motile zoospores (the probable infectious stages) at pH 5, which is acidic enough to cause epidermal degeneration and necrosis in fish (Tandjung *et al.*, 1982; Linnenbach *et al.*, 1987).

The putative association of acid sulphate soils with outbreaks of EUS is currently the subject of a major study by the authors. EUS outbreaks typically occur several weeks after heavy rainfall (Callinan *et al.*, 1989; Virgona, 1992) when acidification affects many kilometres of fish habitat. Our research is exploring the hypothesis that acidified water may damage the epidermis and/or overlying protective mucus on sub-lethally exposed fish. Acid-induced skin damage, like experimental abrasion, may allow the invasion of the skin by *Aphanomyces* sp. propagules, such as zoospores, leading to the development of EUS lesions. In support of this hypothesis, EUS affected yellowfin bream (*A. australis*) collected in water with pH 5, had branchitis and areas of epidermal degeneration and necrosis consistent with acid-induced damage (Callinan *et al.*, 1995).

Another hypothesis for EUS induction, beyond the scope of the current research programme, also involves acidified water. It is possible that water quality changes following heavy lower-catchment rainfall induce *Aphanomyces* sporulation in low-flow, freshwater sections of the estuary. Stressed fish, displaced from acidified habitat, may congregate in these low-flow areas where they are exposed to very high concentrations of spores; EUS lesions may result.

Conclusion

In an unmodified system, aquatic biota can adapt and recover from the adverse effects of acidified water. However, recovery cannot occur if the aquatic biota is unable to modify physiological and behavioural processes to accommodate an increased rate of environmental change. The rate of environmental change in acid sulphate soil landscapes, as a consequence of coastal development and agriculture, appears unlikely to allow this gradual adaptation. A new equilibrium, characterised by simpler assemblages of acid-tolerant species or species with exceptional recruitment capabilities, may result.

Our observations suggest that acidification results in the loss of significant habitat due to spatially and temporally extensive water quality changes, the destruction of aquatic plant communities, reduced food resources and the precipitation of iron onto submerged surfaces. Our water quality monitoring has shown that habitat loss upstream of floodgates can occur for up to 21 months because of reduced acid-neutralisation due to diminished tidal action. Reaches downstream can be affected continuously for several weeks and episodically for many months. It is likely that this loss of habitat displaces fish into other river locations, which interferes with recruitment and dispersion processes, and increases pressure on the remaining food resources and shelter. Reproduction may be adversely affected through the acidification of spawning sites. Similarly, downstream and return migration of estuary-spawning, freshwater species, such as the Australian bass (*Macquaria novemaculeata*), may be affected by barriers of acidified water. These impacts could lead to reductions in estuary-spawning freshwater fish populations in the river reaches upstream of acid sulphate soil areas. Slugs of acid water also affect fish movement as fish generally avoid acidified water. Fish trapped behind acidified water can be isolated from other habitats for prolonged periods.

Fish kills caused by acidification also have implications for recruitment because there may be fewer breeding fish in future spawnings. Fish kills also reduce the availability of food resources to predators. The effects of fish kills are likely to be higher in lentic environments such as floodplain lakes, where the total fish population can be destroyed. In lentic systems, recruitment would be limited to flood periods.

Until recently, only limited research had been conducted into the effects on aquatic biota of acidified water derived from acid sulphate soils. Consequently, understanding of the impacts is limited. In this paper, we have described linkages between acid sulphate soil formation, estuarine floodplain drainage, estuarine water quality and a variety of biological

impacts. We identify habitat loss as the most serious consequence of acidification, especially upstream of flood mitigation structures. The significance of long-term habitat loss caused by acidification has yet to be recognised.

Management of fisheries depends on the management of land and water resources. Integrated research and management, as well as education and awareness, are essential to ameliorate existing, and to limit future, acid sulphate soil-related problems. Our ongoing research is detailing the environmental impacts of estuarine acidification with an emphasis on describing acidification processes and fish health issues.

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