

Environmental effects of aluminium

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Abstract

Aluminium (Al), when present in high concentrations, has for long been recognised as a toxic agent to aquatic freshwater organisms, *i.e.* downstream industrial point sources of Al-rich process water. Today the environmental effects of aluminium are mainly a result of acidic precipitation; acidification of catchments leads to increased Al- concentrations in soil solution and freshwaters. Large parts of both the aquatic and terrestrial ecosystems are affected.

In the aquatic environment, aluminium acts as a toxic agent on gill-breathing animals such as fish and invertebrates, by causing loss of plasma- and haemolymph ions leading to osmoregulatory failure. In fish, the inorganic (labile) monomeric species of aluminium reduce the activities of gill enzymes important in the active uptake of ions. Aluminium seems also to accumulate in freshwater invertebrates. Dietary organically complexed aluminium, maybe in synergistic effects with other contaminants, may easily be absorbed and interfere with important metabolic processes in mammals and birds.

The mycorrhiza and fine root systems of terrestrial plants are adversely affected by high levels of inorganic monomeric aluminium. As in the animals, aluminium seems to have its primary effect on enzyme systems important for the uptake of nutrients. Aluminium can accumulate in plants. Aluminium contaminated invertebrates and plants might thus be a link for aluminium to enter into terrestrial food chains.

Introduction

Acidic waters have been recognised as a problem for freshwater fisheries in certain regions of Norway since the 1920s (Dahl, 1927). Forty years later a link between acidic waters and pH of precipitation was hypothesised (Dannevig, 1959). Aluminium (Al) as a toxic element in acidic waters was recognised nearly 20 years later (Schofield, 1977, Dickson, 1979). Today the two elements, H⁺ (pH) and Al, are still considered to be most important causes of toxicity in freshwater biota (Wood and McDonald, 1987).

The biological significance of Al-speciation and the toxicity of the monomeric labile Al-species on fish was demonstrated by Driscoll *et al.* (1982). Heriksen *et al.* (1984) demonstrated their relevance during episodic changes in water quality occurring in streams and rivers.

Freshwater invertebrates disappear in acidic waters as a response to low pH and aluminium (Herrmann, 1987a). In addition, bioaccumulation of aluminium in invertebrate prey organisms has been suggested as a possible explanation to the impaired hatching success observed among birds (Nyholm, 1981), as dietary fed Al accumulates in both birds and mammals.

In the late 1970s, another part of the terrestrial ecosystem, forest trees, was suspected to be threatened by acid rain (Ulrich, 1980). Today, in many of the forested areas of central Europe, trees are dying. Aluminium concentration in the soil solution increases due to soil

acidification. Aluminium has been shown to have a negative effect on the root systems of herbaceous plants and trees, and might thus be one of the factors involved in the forest decline.

This paper reviews the role of aluminium in terrestrial and aquatic ecosystems affected by acid rain.

Effects of Aluminium on Vascular Plants at Low pH

Aluminium is the third most abundant element in the soil, constituting on the average 8% of the minerals. On weathered mineral surfaces Al exists as oxides and polymeric hydroxides. Under acidic conditions these compounds dissolve to form the hydrated ion Al (H₂O)₆³⁺ (written Al³⁺ for simplicity) or hydrolysis products of this ion. The Al ions bind to cation- exchange sites on soil particles and are thus accessible for plant roots.

Al³⁺ is the dominant Al ion at pH < 4.5. Since the beginning of this century, Al³⁺ has been regarded as an important growth limiting factor in acid soils, together with low pH and lack of macronutrients in such soils. Experiments to elucidate the effects of Al have been done mainly with herbaceous species (for review, see *e.g.* Foy, 1974). During the last 40 years several studies have also been done with tree species, especially after Ulrich *et al.* (1980) claimed that Al ions might be an important factor in the forest decline in central Europe.

Aluminium taken up by roots is mainly found in the mucilage layer on the root tip surface (Horst *et al.*, 1982)

and in the walls of the epidermis and cortex cells (Huett and Menary, 1980). In the cell wall pectins, Al ions compete with Ca^{2+} for the same absorption sites (Wagatsuma, 1983). Some Al is taken up in the cytoplasm and bound to nucleic acids and acid soluble phosphates (Wagatsuma, 1983). Aluminium is translocated only to a small extent to shoots.

All studies of growth and mineral nutrition in the presence of Al^{3+} (e.g. Göransson and Eldhuset, 1987) confirm that after Al addition, symptoms are first seen in roots by a reduction in length growth, root thickening and root tip dieback. New side roots are short, thick and brittle, and sometimes loss of geotropism occurs. With prolonged Al treatment, symptoms are also seen in shoots as growth reduction, yellowing and purpling, wilting, and sometimes loss of apical dominance. Ca and Mg concentrations are also greatly reduced in roots and shoots of Al-treated plants.

Al^{3+} is also toxic to many fungi. Thus, Al may negatively affect the symbiosis between fungi and roots, the so-called mycorrhiza, where the ecto-mycorrhiza seems most affected (Rolf A. Olsen, personal communication). Mycorrhiza is important for the water and nutrient uptake in plants.

Al^{3+} affects several physiological processes. One important effect is reduced membrane potential in root cells, probably due to Al interference with the Ca-binding protein, calmodulin (Siegel and Haug, 1983a). Al reduces the activity of several enzymes, including ATPases important in cation uptake (Siegel and Haug, 1983a, Suhayda and Haug, 1986). The ATPases are activated by Ca-calmodulin and are involved in cation uptake. Al ions and nutrient cations compete for the same uptake sites on cell surface (Kinraide and Parker, 1987). Increased permeability of non-electrolytes and decreased water permeability in cortex cell membranes, probably due to changes in membrane lipids (Zhao *et al.* 1987), are also a significant response to Al. Aluminium phosphate complexes in mucilage and cell walls reduce P availability (McCormick and Borden, 1974). Inhibition of DNA synthesis in cell nuclei is observed, probably due to Al binding to phosphate groups in DNA (Matsumoto and Morimura, 1980). This may be one reason for inhibited cell division and root growth (Horst *et al.*, 1983).

Plants growing on acid soil may have developed mechanisms for tolerating high Al^{3+} concentrations in the soil. Such mechanisms might include excretion of organic acids which act as complexing agents for Al^{3+} (Lee and Foy, 1986), increase of pH outside roots so that Al is polymerised or precipitated (Taylor and Foy, 1985), and active exclusion of Al outside the plasmalemma of root cells (Wagatsuma and Yamasaku, 1985).

Not only the absolute concentration of Al^{3+} , but also the molar ratio of Al^{3+} to other ions in soil or nutrient solution is important for the extent of Al toxicity. This is especially true for Ca^{2+} . Increasing Ca concentration decreases Al toxicity (Rost-Siebert, 1983; Abrahamsen, 1984; Alva *et al.*, 1986a). The activity of other monomeric forms of Al (Alva *et al.*, 1986b) or total monomeric Al (Wright and Wright, 1987) may in some cases be a better measure of Al-toxicity than is the Al^{3+} concentration.

Experiments to determine toxicity of Al are most often done on young plants growing in nutrient solutions in a growth chamber or glasshouse. Such experiments generally indicate that growth reductions start at external Al concentrations below 5.4 mg/L in herbaceous species and above 20 mg/L in tree species adapted to growth on acid soils. There are great variations among species. Also, the tolerance threshold for a species is to some extent dependent on which growth parameter is measured. The growth systems mentioned are simple and verifiable, but the results cannot be directly transferred to the field situation.

Most tree roots are situated in the upper soil horizons, i.e. the O, A, and the upper B horizon. The total Al concentration increases with soil depth, and in the O and A horizon the organic Al complexes dominate (Nilsson and Bergkvist, 1983). These are regarded as non-toxic. In the B horizon from acid podzolic forest soils in Scandinavia, total Al concentrations of maximum 5.4 mg/L (typically less than 2.7 mg/L) have been measured (Abrahamsen, 1983; Nilsson and Lundmark, 1986).

On the other hand, in some forest soils in southern Sweden there has been a long term decrease in pH (Hallbäck and Tamm, 1986), a decrease in Ca/Al ratio (Tyler, 1987), and an increase in inorganic Al concentration (Bergkvist, 1987). The same tendency has been seen in West Germany (Ulrich, 1980, 1981).

Among the common forest tree species in Scandinavia, Scots pine (*Pinus sylvestris*) seems to be more tolerant to Al than white birch (*Betula pendula*), which is again more tolerant than Norway spruce (*Picea abies*). The latter shows growth reductions at Al^{3+} concentrations of 20-27 mg/L (Abrahamsen, 1984; Eldhuset and Göransson, 1989). These results indicate that Norway spruce may respond adversely to a further increase in exchangeable Al in the soil.

Effects of Aluminium on Aquatic Biota

Although high Al concentrations in waters polluted by industrial sources are toxic to invertebrates and fish (Hunter *et al.*, 1980; Lamb and Bailey, 1981), it is the acidification caused by acid rain that has caused the ecological significance of Al toxicity.

Aquatic plants have so far not been demonstrated to be affected by elevated levels of Al in acidified freshwaters (B. Rørslett, personal communication). pH and Al affect both invertebrates and fish, and the effects are dependent not only on species but also on life history stage of the animals. In the field, the effects of Al alone is difficult to isolate from a variety of potentially interrelated adverse factors. Especially during episodes, large variations in pH, Al-species distribution and calcium can occur (Henriksen *et al.*, 1984; Gagen and Sharpe, 1987; Lacroix and Townsend, 1987); all influence the biological response. In the laboratory, the effects of Al *per se* can be studied at given levels of pH, Ca *etc.* The results from such studies will be used in the following, presenting only the specific Al-response.

In experiments, Al has been added either as AlCl_3 or $\text{Al}_2(\text{SO}_4)_3$. The chemical used might be of importance as

to the specific animal response, as many experiments using $\text{Al}_2(\text{SO}_4)_3$ have induced an increased mucus production at the fish gills, whereas use of AlCl_3 or natural acidic waters have not. In the following, however, no distinction is made between experiments using the different chemicals.

Whenever a specific response is related to certain species of aluminium, *i.e.* the monomeric inorganic species termed "labile Al" (LAl), the separation technique of Driscoll (1984) with or without minor modifications, has been used.

Effects of Aluminium on Invertebrates

Acidification has generally been accompanied by declining numbers of both planktonic and benthic invertebrates (Leivestad *et al.*, 1976; Haines, 1981; Økland and Økland, 1986). However, the mechanisms by which Al *per se* can act as a harmful agent on these organisms are largely unknown (Herrmann, 1987a).

Hörnström *et al.* (1984) indicated that Al could affect the zoo-plankton community in acidified surface waters. On adding Al to an acid stream, Hall *et al.* (1985; 1987), Raddum and Fjellheim (1987) and Ormerod *et al.* (1987) observed an increased drift of mayfly nymphs, chironomids and dipterid midges. Many of the animals related to the surface film were found dead, presumably caused by reduction of surface tension; this was indicated by a pronounced foam production (Hall *et al.* 1985; Ormerod *et al.*, 1987). Al-induced mortality on stoneflies, the isopod *Asellus* and caddis larvae was reported by Burton and Allan (1986), who also demonstrated a reduced mortality whenever the organic content of the water was high. The counteracting effect of humic acids relative to Al-toxicity was also demonstrated by Petersen *et al.* (1986) on blackfly larvae.

Not all invertebrate species tested have shown high sensitivity to Al. No additional mortality due to Al was observed on bivalves and gastropods (Mackie, 1986) or crayfish (Berrill *et al.*, 1985) in acidic waters. Appelberg (1985), however, demonstrated reduced haemolymph Na^+ content in crayfish exposed to acidic Al-rich waters.

At very low pH, high concentrations of Al can have an ameliorating effect, on for example mayfly nymphs (*Heptogenia sulphurea*) (Herrmann, 1987a) and small planktonic crustacean (*Daphnia magna*) (Havas, 1985; Havas and Likens, 1985). The mechanisms involved might be the same as found for fish (see below), but in both cases the actual concentrations of Al are much higher than normally found in acidic waters containing these organisms.

Raddum and Steigen (1981) found that the caloric content of stone flies and caddis flies from acidic rivers was lower than from more neutral rivers; this implies an increased energy consumption (metabolism) in acidic waters. Increased respiration was later demonstrated to be a response to Al, most pronounced for the most sensitive mayfly species (Herrmann and Andersson, 1986). As is the case with fish (Rosseland, 1980), pH alone seemed less important for the respiratory response.

Aluminium can also impair reproduction, shown on *Daphnia magna* (Beisinger and Christensen, 1972).

Otto and Svensson (1983) suggested that as in fish, Al affects invertebrates by disturbing osmoregulation. Appelberg (1985) demonstrated a reduced haemolymph Na^+ content in crayfish in response to Al, and Malley and Chang (1985) showed a reduced Ca^{2+} uptake. In *Daphnia magna*, Al reduced the Na^+ influx and to a lesser extent increased the outflux, thus impairing osmoregulation (Havas and Likens, 1985). The temporal reduced outflux at low pH might explain the reported beneficial effects of Al at low pH. Witters *et al.* (1984) demonstrated reduced haemolymph Na^+ content in *Corixa* exposed to high Al-concentrations, and Herrmann (1987b) found that Al caused a reduced Na^+ content of mayfly nymphs at low pH.

As in fish, Al acts on the respiratory organs of invertebrates, for example, the anal papillae of the phantom midge (Havas, 1986). This might explain why air-breathing invertebrates like the waterboatmen (*Corixa*) are very tolerant to acidic waters (Vangenechten *et al.*, 1979; Witters *et al.*, 1984).

Aluminium can accumulate in the bodies of invertebrates living in acidic waters (Hall and Likens, 1981; Herrmann, 1987a). Insects with aquatic larvae and nymph stages leave their "metal content" in their excuviae on emerging, thus only the water stage in their life history is metal-contaminated (Otto and Svensson, 1983). Birds such as the pied flycatcher, which lives on insects in or close to acidic lakes, have been reported to have high Al-concentrations in bone marrow and eggs indicating a food-chain transport (Nyholm, 1981). The impairment of egg hatching of these birds might therefore link the environmental catastrophe appearing in the acidic aquatic ecosystem to the terrestrial.

Effects of Aluminium on Fish

Until recently, pH alone was considered to be toxic at the egg stage with an increasing influence of Al with age after hatch (Baker and Schofield, 1980; 1982; Wood and McDonald, 1982). Leivestad *et al.* (1987), however, demonstrated that Al reduced both the ion uptake at the eyed-egg stage and the activity of the Na-K-ATPase in the embryo.

After hatch, the main target organ for the Al-effects is the gill where the ion and gas exchange takes place. In addition to H^+ , Al causes loss of plasma ions (Na^+ , Cl^-), reduced osmolality, and increased hematocrit (Muniz and Leivestad, 1980; Rosseland, 1980; Rosseland and Skogheim, 1982; 1984; 1987; Rosseland *et al.*, 1986a, b; Fivelstad and Leivestad, 1984; Neville, 1985; Witters, 1986; Leivestad *et al.* 1987; Wood and McDonald, 1987).

In acidic waters (pH 4.6-5.3) with low levels of calcium (0.5-1.5 mg Ca/L), labile Al between 25-75 $\mu\text{g/L}$ is toxic (Henriksen *et al.*, 1984; Rosseland *et al.*, 1986a; Rosseland and Skogheim, 1987; Skogheim and Rosseland, 1986). The Al-induced ion loss reflects both an increased outflux and a decreased influx of ions (Dalziel *et al.*, 1986; 1987; Wood and McDonald, 1987). The effect on influx is probably caused by a reduced activity of enzymes as Na-K-ATPase, Mg-ATPase and carbonic anhydrase (Staurnes *et al.*, 1984; Kjartansson, 1984; Leivestad *et al.*, 1987; Reite and Staurnes, 1987). Aluminium acts specifically on

the enzymes of the gill, as neither the ATPase systems in the pseudobranch or the kidney was affected (Kjartansson, 1984). The Al-induced efflux is considered to reflect modifications on opening of the tight junctions of the paracellular channels (Wood and McDonald, 1987). The ameliorating effect of Ca on Al- and pH response (Leivestad *et al.*, 1980; Brown, 1983) is by tightening of the junctions, thereby preventing the passive loss of ions (Wood and McDonald, 1987).

Fish exposed to acidic Al-rich waters will accumulate Al on the gill surface (Schofield, 1977; Schofield and Trojnar, 1980; Muramoto, 1981; Buerger and Soltero, 1983; Pagenkoff, 1983; Skogheim *et al.*, 1984; Neville, 1985; Karlsson-Norrgrén *et al.*, 1986a, b; Harvey and McArdle, 1986; Witters *et al.*, 1987; Wood and McDonald, 1987; Jagoe *et al.*, 1987). The precipitation and accumulation is due to the negative charge of the mucus caused by sialic acid residues (McDonald, 1983). The gill also serves as an excretion organ for ammonia (NH_4^+) (Masoni and Payan, 1974). At low pH and high Al, the reduced blood pH (acidosis) and increased CO_2 (hypercapnia) will interfere with the formation from ammonium (NH_3) to ammonia, thus more is excreted as NH_3 . At the interface between mucus and water, the ammonium will be transformed to ammonia, changing the pH and thus enhancing precipitation of Al at the gill surface (Wood and McDonald, 1987).

There are species and strain differences in sensitivity to low pH (Grande *et al.* 1978; Gjedrem, 1980) and Al (Rosseland and Skogheim, 1984; 1987; Rosseland *et al.*, 1986b; Wood and McDonald, 1987). These species differences are also reflected in the accumulation rate of Al on the gills, as well as the whole body (wb) ion concentration, *i.e.* fish with the lowest wb Na (greatest loss) have the highest Al-concentration on the gill surface (Wood and McDonald, 1987). Precipitated Al-complexes can irritate the gill and cause inflammation, oedema, swelling and sometimes irradiation of the secondary lamella (Schofield, 1977; Schofield and Trojnar, 1980; Karlsson-Norrgrén *et al.*, 1986a, b; Jagoe *et al.*, 1987). Also an increased number of mucus cells (Linnenbach *et al.*, 1987) and chloride cells (Jagoe *et al.*, 1987) have been observed relative to Al-accumulation onto the gills. In spite of high Al-concentration on the gill these histopathological changes are not observed when the humus content in the water is high, indicating a labile Al-dependent irritation (Karlsson-Norrgrén *et al.*, 1986b).

Although increased levels of Al in blood plasma have not been found (Neville, 1985; Wood and McDonald, 1987), Al-accumulation in body tissue does occur (Hunter *et al.*, 1980; Muramoto, 1981; Buerger and Soltero, 1983; Haines *et al.*, 1987). In the field, such accumulation might reflect both a direct gill-dependent uptake and a food chain dependent uptake (Haines *et al.*, 1987). It was suggested by Muramoto (1981) that Al could pass through the gill as metal-complexes in the presence of complexing ligands.

Sometimes extensive mucus-clogging of the secondary lamella has been observed (Muniz and Leivestad, 1980; Rosseland, 1980; Rosseland and Skogheim, 1984). This response is not universal, as fish dying in field in natural acidic waters at labile Al-

concentrations of 59-110 $\mu\text{g/L}$ have not shown excess mucus despite an Al-accumulation on the gills (Skogheim *et al.*, 1984; Rosseland *et al.*, 1986a). Adding excess aluminium as $\text{Al}_2(\text{SO}_4)_3$ to such waters (LA1 130 $\mu\text{g/L}$) rapidly induced mucus clogging (Muniz and Leivestad, 1980; Rosseland, 1980; Rosseland and Skogheim, 1984). The relevance of the mucus clogging might therefore be questioned with respect to natural conditions.

Both histopathological changes and increased mucus layer will serve to increase the diffusion distance for O_2 and CO_2 between the water and blood. This can lead to a decreased oxygen tension in the arterial blood, reduced hemoglobin oxygenation and pH, and an increased blood CO_2 and blood lactate (Neville, 1985; Malte, 1986; Wood and McDonald, 1987). At low pH, the increased mucus layer will reduce the rate of ion loss, thereby temporarily increasing the resistance, as observed by Baker and Schofield (1982), Hutchinson *et al.* (1987) and Wood and McDonald (1987). At such high concentrations of H^+ and Al, the primary cause of mortality might thus be respiratory rather than osmoregulatory failure (Rosseland, 1980; Muniz and Leivestad, 1980; Neville, 1985; Wood and McDonald, 1987).

Metabolic activity, measured as oxygen uptake, is not affected by H^+ alone, but increases as a response to Al in the water (Rosseland, 1980; Neville, 1985; Malte, 1986; Wood and McDonald, 1987). The increased respiratory and heart rate observed in acidic waters (Rosseland, 1980; Muniz and Leivestad, 1980; Ogilvie and Stechey, 1983; Giles *et al.*, 1984; Fivelstad and Leivestad, 1984; Neville, 1985; Malte, 1986; Leivestad *et al.*, 1987; Wood and McDonald, 1987) are not believed to cause the increased energy expenditure *per se*, as the increased metabolism rather reflects the increased activity of the intrinsic compensatory mechanism trying to restore homeostasis (Rosseland, 1980). In long-term experiments, even low concentrations of Al have reduced growth (Sadler and Turnpenny, 1986). Hyperventilation in acidic waters is a specific response to the labile Al-concentration, as the addition of chelator such as citrate depresses hyperventilation (Leivestad *et al.*, 1987).

Prolactin and cortisol are important hormones related to osmoregulation (Potts and Flemming, 1970; Johnson, 1973); prolactin reduces ion-permeability and increases mucus production, while cortisol stimulates the onset of cellular proliferation and differentiation in the primary gill epithelium, and increases the specific activity of Na-K-ATPase. Both hormones are affected by acidic waters (Wendelaar Bonga and Balm, 1987). Plasma cortisol increases in fish exposed to low pH and high Al concentration, presumably as a response to compensate the H^+/Al -response (Kjartansson, 1984). Prolactin production increases in acidic waters mainly as a response to a drop in plasma electrolytes (Wendelaar Bonga *et al.*, 1987).

Avoidance reactions to low pH waters have been observed when plasma cation concentrations have been reduced by acidic waters (Pedder and Maly, 1987). Olfaction is an important part of behavioural response and can lead to both positive and negative chemotaxes, including avoidance. Low pH alone reduces the olfactory response to aminoacids and increases the mucus layer in

the olfactory organ (Thommesen, 1975; Klaprat *et al.*, 1988). Adding Al to the water depresses olfactory response even more and causes histopathological changes such as irradiation of the microvillie, swelling and disformation of the olfactory epithelium (Klaprat *et al.*, 1988). During episodic changes in water quality related to snowmelt or heavy rain, fish are often observed gathered at the outlet of, or having migrated into, a less acid brook or stream (Muniz *et al.*, 1978; Rosseland, 1986). Chronic exposure to sublethal acidic conditions causing disturbances of the olfactory sense prior to a toxic episode, might thus reduce the chance for a fish population to find refuges and survive in their environment.

Effects of Aluminium on Terrestrial Animals

When related to other metals, the toxicity of dietary Al has been regarded to be low. The intestinal absorption of orally-ingested Al salts is poor, and the small amount absorbed is almost completely excreted in urine under conditions with normal kidney function (Ganrot, 1986; Sørensen *et al.*, 1974). However, from medical research Al is known under certain conditions to cause severe disturbances, especially in the mineral balance and nervous system, mostly in connection with renal failure. In the last decade an increasing number of human as well as animal studies have been conducted, both *in vivo* and *in vitro*, and these reveal several physiological and biochemical implications of Al (reviewed by Haug, 1984; Siegel, 1985; Ganrot, 1986; Trapp, 1986).

Environmental impacts of Al on terrestrial wildlife, however, are poorly known. The only experimental evidence for such a connection is that of Nyholm (1981), who proposed a possible etiological role of Al in breeding impairment observed in wild passerines at some lakes in Swedish Lapland. The impairment was caused by severe eggshell defects as well as reduced clutch sizes and high incidences of mortality. Since these findings were restricted to birds nesting by the shore of a lake suspected to be acid-stressed, some kind of poisoning associated with the lake was proposed (Nyholm and Myhrberg, 1977). Extensive analyses excluded DDT, PCB and metals such as Cd, Cr, Cu, Pb and Hg to be the causal factors (Nyholm and Myhrberg, 1977). In 1981, however, the specific occurrence of Al in birds producing defective egg-shells was demonstrated semi-quantitatively by X-ray multielement microanalysis in bone marrow tissue of humeri of pied flycatchers (*Ficedula hypoleuca*), one of the most affected species (Nyholm, 1981). A possible route of transport of Al to the birds was presented, in which insects swarming from the acidic lakes was the proximate link. Emerging insects collected from the lake contained on dry weight basis 70-1,230 mg Al/kg (Nyholm 1982). In comparison, Hall and Likens (1981) reported 840 ± 140 mg Al/kg in aquatic insects in an artificially acidified stream of pH 4.

Several findings, such as successively increasing severity of the shell defects with the egg-laying sequence, very porous structure of the defective shells, and less apatite depositions in marrow cavities of limb bones (a

necessary Ca storage for formation of eggshell, Simkiss (1967)), indicated Ca depletion (Nyholm, 1981).

The complex mechanism by which Al induces osteomalecia is not yet fully understood. The presence of Al in bone marrow associated with the reduced apatite deposition (Nyholm, 1981) seems, however, to be in agreement with the principal results from human studies as well as animal experiments. Both from *in vivo* and *in vitro* studies Al is reported to accumulate in the calcification front of bones, inhibiting calcium phosphate precipitation by some physical chemical action. Al may also interfere with biochemical reactions as well as cell functions important in the mineralisation processes (reviewed by Kanis, 1981; Parkinson *et al.*, 1981; Goodman, 1985; Kreuger *et al.*, 1985; Ganrot, 1986; Starkey, 1987).

From the study area of Nyholm (1981), two to three times higher blood plasma Ca have been found in affected pied flycatchers relative to birds producing normal eggshells several kilometres from the lake shore. Also the Ca concentration was similar or even somewhat higher in the shell gland tissue of affected birds compared to non-affected birds (Staurnes, M. and Nyholm, N.E., in preparation). Both these findings may also indicate some direct dysfunctions in the shell formation processes.

Calmodulin is a multifunctional, Ca-dependent protein that regulates a variety of cellular reactions (Cheung, 1980). This Ca-binding protein seems to be of crucial importance in stimulation of the shell gland secretion (mediation of Ca-dependent formation of prostaglandins, and Ca-dependent binding of progesterone to its receptors), as well as the ATP-dependent transport of Ca into the shell gland lumen (Lundholm, 1987). This process involves the enzymes Ca-Mg- and Na-K-ATPase. Another enzyme important in the egg-shell formation is carbonic anhydrase (Simkiss, 1967; Simkiss and Taylor, 1971). In fish gills, Al inhibits these enzymes (Kjartansson, 1984, Staurnes *et al.*, 1984; Leivestad *et al.*, 1987). Al binds to calmodulin (Siegel and Haug, 1983b). As the eggshell-thinning effect of the biocide DDE mostly seems to be mediated by its inhibition of calmodulin (Lundholm, 1987), a similar role of Al might be hypothesised.

A four-month feeding period with an experimental diet supplemented with 1,000 mg Al/L as Al sulphate, however, did not result in any effect on egg production, fertility, hatchability, or fledging success in ringed turtle-doves, *Streptopelia risoria* (Carrière *et al.*, 1986). Compared to controls given the same diet without supplemental Al, egg permeability was decreased initially but subsequently recovered to a normal level. Plasma Ca, P, and Mg of adults were not affected. Dietary Al (1,500 mg/L, 63 days) did not affect growth rate of juveniles although Al tended to accumulate in the bones of sternum, but not in leg and wing bones. In treated adults, however, there was a significant accumulation also in leg and wing bones. The overall conclusion, however, was that Al had no significant influence which could support Nyholm's (1981) hypothesis of an adverse effect of Al on the Ca-metabolism.

Japanese quail (*Coturnix coturnix japonica*) has been shown to be especially sensitive to reproductive effects of Pb (Edens and Garlich, 1983). Al-citrate in drinking water

(250 and 750 mg Al/L, 37 days), however, did not cause reproductive failure in spite of elevated levels of Al in bones as well as liver, kidney and brain (Nyholm, N.E., Gohenson, A. and Paulsson, J., in preparation).

Both in the experiments of Carrière *et al.* (1986) and Nyholm *et al.* (in preparation), the diet Ca content was about 1% and P content 0.5-0.9%. Carrière *et al.* (1986) stated that the lack of effect in their study could be due to higher concentrations of both Ca and P in the experimental diets compared to the feed levels which may occur in acidified areas. In rabbits, Al ingestion lowered the inorganic P in plasma and bone (Cox *et al.* 1931). In humans, extensive use of Al-containing antacids can result in a similar P-depletion which may cause osteomalacia (Spencer *et al.*, 1975; Spencer and Lender, 1979). Dietary Al has also been reported to lower plasma P levels in poultry species (Deobald and Elvehjem, 1935; Storer and Nelson, 1968; Lipstein and Hurwitz, 1981), but Street (1942) concluded from experiments with rats that such lowering is likely to occur only when the concentration of Al equals the concentration of P. Experiments with lower P level than Al (dose range 2,000-25,000 mg/L as sulphate and hydroxide) in the diet of young chicks has shown to cause decreased growth and increased mortality (Deobald and Elvehjem, 1935; Storer and Nelson, 1963) as well as wing and leg weakness (Steinborn *et al.*, 1957). These effects, which Deobald and Elvehjem (1935) found most marked when dietary Al levels attained more than 50% of the P dietary level, were ameliorated by supplemental dietary P (Steinborn *et al.*, 1957; Lipstein and Hurwitz, 1981).

Thus, with respect to potential environmental effects of Al, the risk for a similar P-depletion due to restricted availability and intestine retention is obvious in both mammals and birds. In most acidified areas, Ca concentration is low, at least in the freshwaters. In association with Pb intoxication, low dietary Ca is known to increase strongly the intestinal absorption of Pb in mammals (Six and Goyer, 1970; Mahaffey *et al.*, 1973; Stowe *et al.*, 1973; Van Barneveld and Van den Hamer, 1985), as well as in birds (Carlson and Nielson, 1985). Pb can occupy the Ca-binding sites of an intestinal Ca-binding protein (Barton *et al.*, 1978), thus representing an efficient route of intestinal absorption. The basis for the increased uptake may be induction of synthesis of this protein in response to the low dietary Ca (Scheuhammer, 1987). As a result the Pb accumulation in egg-laying birds is several times that of non-laying females (Finley and Dieter, 1978) and in males (Kendall and Scanlon, 1981). In view of the binding of Al to calmodulin (Siegel and Haug, 1983b), the possibility for a similar effect of Al should be elucidated. Moreover, both Carrière *et al.* (1986) in their experimental administration of Al-sulphate to ringed turtle-doves, and Nyholm *et al.* (in preparation) feeding Al-citrate to Japanese quail, reported much higher Al absorption and accumulation in laying females than in males. This might indicate a similar Ca-deficiency induced increase in Al-absorption as was demonstrated for Pb.

Increased parathyroid activity and vitamin D level found in egg-laying birds is known to enhance the absorption of several metals in chicks (Worker and

Migicovsky, 1986a, b). In pregnant women there is an elevated level of PTH (Cushard *et al.*, 1972). The role of parathyroids and PTH in relation to Al accumulation has been a matter of dispute, some claiming that PTH might increase the Al absorption (Mayor *et al.*, 1977; 1980), while this has been questioned by others (Alfrey *et al.*, 1979; Alfrey, 1980).

Nevertheless, in view of the effects of PTH on absorption of other metals and the findings of increased Al accumulation in laying birds, especial attention should be paid to the possibility of Al intoxication during periods of high demands of Ca as in rapidly growing young animals. This is particularly relevant during reproduction where increased levels of PTH may mediate Al absorption and accumulation. Naturally, the risk for such an intoxication would be highest in areas with a high load of bioavailable Al accompanied with low availability of Ca. Both conditions normally hold for acidified areas. Interestingly, secondary hyperparathyroidism provoked by chronic deficiency of Ca, Mg and causing increased absorption of toxic metals including Al, has been postulated by Yase (1977) to be the most plausible explanation to the high incidence of amyotrophic lateral sclerosis (ALS) and Parkinsonism-dementia (PD) in some indigenous populations in western Pacific. Red Al- and Fe-rich soil with very low Ca content is common to these areas. Al has been suggested to be important in the etiology since the reported Al content in spinal cord and brain as well as the pathologic changes are comparable to those from human and animal studies where Al has been associated with disorders in the central nervous system (discussed by Ganrot, 1986). The neurotoxicity of Al, characterised by degeneration of neurons with Al accumulation in dense tangles (neurofibrillary degeneration) and interactions with several biochemical reactions, is well documented from both human and animal studies (reviewed by Alfrey, 1978; Crapper *et al.*, 1978; 1981; Liss, 1980; Parkinson *et al.*, 1981; Kreuger *et al.*, 1985; Perl, 1985; Ganrot, 1986; Starkey, 1987).

In nature even minor behavioural abnormalities may be critical for reproduction success and survival. Chronic exposure to neurotoxic contaminants might cause changes in behavioural pattern at levels insufficient to produce increased mortality or other acute effects (Heinz *et al.*, 1983; Peakall, 1985; Donald *et al.*, 1986). Animal experiments have shown that Al administration may be accompanied by behavioural and motor disturbances (Crapper and Dalton, 1973; Crapper *et al.*, 1973; Petit *et al.*, 1980; 1985; Commissaris *et al.*, 1982; Rabe *et al.*, 1982; Yokel, 1984; 1985; 1987; Bernuzzi *et al.*, 1986) even at Al levels that do not cause overt signs of ill health (Commissaris *et al.*, 1982). Since neurotoxicity of Al normally has been associated with slow-operation, low-dose effects, often related to ageing processes of the individual neurons as well as the whole organism (reviewed and discussed by Ganrot, 1986), it is reasonable to anticipate that longliving animals would be most vulnerable. These are normally at the top of the food chain and also most vulnerable to accumulation of other pollutants. In acidified areas the increased Al load in freshwaters is also accompanied with higher bioavailable

concentrations of other metals (Haines *et al.*, 1987), and elevated levels of several heavy metals are demonstrated in terrestrial animals from areas exposed to acid rain compared to uncontaminated areas (Frøslie *et al.* 1984; 1985). However, nothing is known about potential synergistic effects of Al with other metals, but contaminants interfering with the renal functions as Cd (White *et al.* 1978; Goyer *et al.*, 1984) and Hg (Ware *et al.*, 1975), would be suspected to strengthen potential deleterious effects of Al due to the risk for decreased excretion and thus increased accumulation.

Routes for entry into and degree of accumulation of Al along food chains have not been investigated. The findings of Nyholm (1981; 1982) indicate that increased Al concentrations in streams and waters may cause Al accumulation in terrestrial animals eating prey originated from contaminated water.

The forest vole (*Clethrionomus glareolus*) is one of the small rodents which are correlated to be key organisms in the terrestrial food chain. Feeding on blueberries from an acidified area (containing 156 mg Al/L) resulted in a significant higher Al content in bones than feeding on blueberries from an uncontaminated area (57 mg Al/L) (E. Nyholm, P. Mattson and P. Slanina, in preparation). Thus, the increased concentrations of labile Al in acidified soil may enter the food chain directly through plants. Human and animal studies have shown that complexes as Al-citrate are much more readily absorbed and accumulated than the inorganic salts of Al (Slanina *et al.*, 1984; 1986; Yokel and McNamara, 1987). The short period of exposure of the mice (one week), along with the relatively moderately elevated levels of Al in the blueberries, may indicate that Al in plants is bound to similar easily-absorbed complexes which strongly enhance the bioavailability of Al. The potential toxicity of such easily-absorbed complexes is seen from an experiment with one week old chicks of wood grouse (*Tetrao urogallus*), where addition of Al-citrate to commercial bird feed to give 4,000 mg Al/L strongly increased the mortality compared to those given uncontaminated feed; none survived two weeks exposure (M. Staurnes and T.K. Spidso, in preparation). The same dose of Al caused accumulation in kidney and liver of five week old chickens, but no mortality. Similarly, Nyholm observed especially high mortality just after hatching in the populations of passerines with impaired breeding in Swedish Lapland (N.E. Nyholm, personal communication). Thus, at least in birds, the greatest risk for acute intoxication seems to be during the earliest life stages.

Future studies, however, will show if the increased concentration of Al associated with acidification, either as labile Al or organic-complexed represents a potential risk for birds and mammals as well as other terrestrial animals. The degree of and routes for bioaccumulation, differences in species and life-stage sensitivity, occurrence and toxicity of different Al compounds, potential synergistic effects with other contaminants, and potential interactions because of restricted availability of substances as Ca and P will all be key questions in future studies. A comparative study of wildlife in regions with a naturally high concentration of Al, as those mentioned areas in western Pacific, where occurrence of ALS and PD are high, could possibly

contribute to a better understanding of potential environmental hazards of Al as well as adaptation mechanisms.

References

- Abrahamsen, G. 1983. Sulphur pollution: Ca, Mg and Al in soil and soil water and possible effects on forest trees. In: Ulrich, B. and Pankrath, J. (eds.), *Effects of Accumulation of Air Pollutants in Forest Ecosystems*, pp.207-218. D. Reidel Publishing Company, Dordrecht. ISBN 90-277-1476-2.
- Abrahamsen, G. 1984. Effects of acidic deposition on forest soil and vegetation. *Phil. Trans. R. Soc. Lond. B*, **305**, 369-382.
- Alfrey, A.C. 1978. Dialyse encephalopathy syndrome. *Ann. Rev. Med.*, **29**, 93-98.
- Alfrey, A.C. 1980. Aluminum neurotoxicology in uremia. *Neurotoxicology*, **1**, 43-53.
- Alfrey, A.C. Hegg, A., Miller, N., Berl, T. and Berns, A. 1979. Inter-relationship between calcium and aluminum metabolism in dialyzed uremic patients. *Miner. Electrolyte Metab.*, **2**, 81-87.
- Alva, A.K., Asher, C.J. and Edwards, D.G. 1986a. The role of calcium in alleviating aluminum toxicity. *Aust. J. Agric. Res.*, **37**, 375-382.
- Alva, A.K., Edwards, D.G., Asher, C.J. and Blamey, F.P.C. 1986b. Relationships between root length of soybean and calculated activities of aluminum monomers in nutrient solution. *Soil Sci. Soc. Am. J.*, **50**, 959-962.
- Appelberg, M. 1985. Changes in haemolymph ion concentration of *Astacus astacus* L. and *Pacifastacus leniusculus* (DANA) after exposure to low pH and aluminium. *Hydrologia*, **121**, 19-25.
- Baker, J.P. and Schofield, C.L. 1980. Aluminum toxicity to fish as related to acid precipitation and Adirondack surface water quality. In: Drabløs, D. and Tollan, A. (eds.), *Ecological impact of acid precipitation*, pp.292-293. SNSF-project.
- Baker, J.P. and Schofield, C.L. 1982. Aluminum toxicity to fish in acidic waters. *Water, Air, Soil Pollut.*, **18**, 289-309.
- Barton, J.C., Conra, M.E., Harrison, L. and Nuby, S. 1978. Effects of calcium on the absorption and retention of lead. *J. Lab. Clin. Med.*, **91**, 366-376.
- Beisinger, K.E. and Christensen, G.M. 1972. Effects of various metals on survival, growth, reproduction and metabolism of *Daphnia magna*. *J. Fish. Res. Bd. Canada*, **29**, 1691-1700.
- Bergkvist, B. 1987. Leaching of metals from forest soils as influenced by tree species and management. *For. Ecol. Manag.*, **22**, 29-56.
- Bernuzzi, V., Desor, D. and Lehr, P. 1986. Development and learning abilities of rats prenatally intoxicated with aluminum. *Neurosci. Lett. Suppl.*, **0(26)**, 32.
- Berrill, M., Hollett, L., Margosian, A. and Hudson, J. 1985. Variation in tolerance to low environmental pH by the crayfish *Orconectes rusticus*, *O. propinquus* and *Cambarus robustus*. *Can. J. Zool.*, **63**, 2586-2589.
- Brown, D.J.A. 1983. Effects of calcium and aluminium concentration on the survival of brown trout (*Salmo trutta*) at low pH. *Bull. Environ. Contam. Toxicol.*, **30**, 382-387.
- Buergel, P.M. and Soltero, R.A. 1983. The distribution and accumulation of aluminium in rainbow trout following whole-lake alum treatment. *J. Freshw. Ecol.*, **2**, 37-44.
- Burton, T.M. and Allan, J.W. 1986. Influence of pH, aluminium, and organic matter on stream invertebrates. *Can. J. Fish. Aquat. Sci.*, **43**, 1285-1289.
- Carlson, B.L. and Nielson, S.W. 1985. Influence of dietary calcium on lead poisoning in mallard ducks (*Anas platyrhynchos*). *Am. J. Vet. Res.*, **46**, 276-282.
- Carrière, D., Fischer, K.L., Peakall, D.B. and Anghem, P. 1986. Effects of dietary aluminum sulphate on reproductive success and growth on ringed turtle-doves (*Streptopelia risora*). *Can. J. Zool.*, **64**, 1500-1505.
- Cheung, W.Y. (ed.) 1980. *Calcium and Cell Function*, Volume 1. Calmodulin. Academic Press, New York.
- Commissaris, R.L., Cordon, J.J., Srague, S., Keiser, J., Mayor, G.H. and Rech, R.H. 1982. Behavioural changes in rats after chronic aluminum and parathyroid hormone administration. *Neurobeh. Toxicol. Teratol.*, **4**, 403-410.

- Cox, G.J., Dodds, M.L., Wigman, H.B. and Murphy, F.J. 1931. The effects of high doses of aluminum and iron on phosphorus metabolism. *J. Biol. Chem.*, **92**, 11-12.
- Crapper, D.R. and Dalton, A.J. 1973. Aluminum induced neurofibrillary degeneration, brain electrical activity and alternations in acquisition and retention. *Physiol. Behav.*, **10**, 935-945.
- Crapper, D.R., Krishnan, S.S. and Dalton, A.J. 1973. Brain aluminium distribution in Alzheimer's disease and experimental neurofibrillary degeneration. *Science*, **180**, 511-513.
- Crapper, D.R., Karlik, S.J. and DeBoni, U. 1978. Aluminum and other metals in senile (Alzheimer) dementia. In: Katzman, R., Terry, R.D. and Bick, K.L. (eds.), *Disease: Senile Dementia and Related Disorders: Aging*, Volume 7, pp.471-485. Raven Press, New York.
- Crapper, D.R., Dalton, A.J., Karlik, S.E. and DeBoni, U. 1981. Role of aluminum in Alzheimer's disease. In: Alexander, P.E. (ed.), *Electrolytes and neuropsychiatric disorders*, pp.89-111. MTP Press Limited, Lanchaster.
- Cushard, W.G.Jr., Creditor, M.A., Cantebury, J.M. and Reiss, E. 1972. Physiologic hyperparathyroidism in pregnancy. *J. Clin. Endocrinol. Metabol.*, **34**, 767-771.
- Dahl, K. 1927. The effect of acid water on trout fry. *Salmon and Trout Magazine*, **46**, 35-43.
- Dalziel, T.R.K., Morris, R. and Brown, D.J.A. 1986. The effects of low pH, low calcium concentration and elevated aluminium concentrations on sodium fluxes in brown trout, *Salmo trutta* L. *Water, Air, Soil Pollut.*, **30**, 569-578.
- Dalziel, T.R.K., Morris, R. and Brown, D.J.A. 1987. Sodium uptake inhibition in brown trout, *Salmo trutta*, exposed to elevated aluminium concentrations at low pH. *Annls. Soc. R. Zool. Belg.*, **117**, Suppl.1, 421-434.
- Dannevig, A. 1959. Nedbørens innflytelse på vassdragenes surhet og på fiskebestanden. (Influence of precipitation on river acidity and fish population). *Jeger og Fisker*, **3**, 116-118. (In Norwegian).
- Deobald, H.J. and Elvehjem, C.A. 1935. The effect of feeding high amounts of soluble iron and aluminium salts. *Am. J. Physiol.*, **111**, 118-123.
- Dickson, W. 1979. Exempel på metalltoxicitet vid försurning och kalkning. (Examples of metaltoxicity related to acidification and liming). *Aquannalen*, **1**, 2-7. (In Swedish).
- Donald, J.M., Cutler, M.G., Moeer, M.R. and Bradley, M. 1986. Effects of lead in the laboratory mouse - 2. Development and social behaviour after lifelong administration of a small dose of lead acetate in drinking fluid. *Neuropharmacol.*, **25**, 151-160.
- Driscoll, C.T. 1984. A procedure for the fractionation of aqueous aluminum in dilute acidic waters. *Int. J. Envir. Analyt. Chem.*, **16**, 267-283.
- Driscoll, C.T., Baker, J.P., Bisogni, J.J.Jr., and Schofield, C.L. 1982. Effects of aluminum speciation on fish in dilute acidified waters. *Nature*, **284**, 161-164.
- Edens, F.W. and Garlich, J.D. 1983. Lead induced egg production decrease in leghorn and Japanese quail. *Poul. Sci.*, **62**, 1757-1763.
- Eldhuset, T.D. and Göransson, A. 1989. Effects of aluminium on growth and nutrient uptake of *Pinus sylvestris* and *Picea abies* seedlings. (In preparation).
- Finley, M.T. and Dieter, M.P. 1978. Influence of laying on lead accumulation in bone of mallard ducks. *J. Toxicol. Envir. Health*, **4**, 123-129.
- Fivelstad, S. and Leivestad, H. 1984. Aluminium toxicity to Atlantic salmon (*Salmo salar* L.) and brown trout (*Salmo trutta* L.): Mortality and physiological response. *Rep. Inst. Freshw. Res. Drottningholm*, **61**, 69-77.
- Foy, C.D. 1974. Effects of aluminium on plant growth. In: Carson, E.W. (ed.), *The Plant Root and Its Environment*, pp.601-642. University Press of Virginia, Charlottesville. ISBN 0-8139-0411-0.
- Frøslie, A., Norheim, G. and Steinnes, E. 1984. Levels of trace elements in liver from Norwegian moose, reindeer and red deer in relation to atmospheric deposition. *Acta Vet. Scand.*, **25**, 333-345.
- Frøslie, A., Norheim, G. and Steinnes, E. 1985. Heavy metals in lamb liver: contribution from atmospheric fallout. *Bull. Envir. Contam. Toxicol.*, **34**, 175-182.
- Gagen, C. and Sharpe, W. 1987. Influence of acid runoff episodes on survival and net sodium balance of brook trout (*Salvelinus fontinalis*) confined in a mountain stream. *Annls. Soc. R. Zool. Belg.*, **117**, Suppl.1, 219-230.
- Ganrot, P.-O. 1986. Biochemistry and metabolism of Al³⁺ and similar ions. A review. *Envir. Health Persp.*, **65**, 363-441.
- Giles, M.A., Majewski, H.S. and Holden, B. 1984. Osmoregulatory and hematological responses of rainbow trout (*Salmo gairdneri*) to extended environmental acidification. *Can. J. Fish. Aquat. Sci.*, **41**, 1686-1694.
- Gjedrem, T. 1980. Genetic variation in acid tolerance in brown trout. In: Drablos, D. and Tollan, A. (eds.), *Ecological impact of acid precipitation*, pp.308. SNSF-project.
- Goodman, W.G. 1985. Bone disease and aluminum: pathogenetic considerations. *Am. J. Kidney Dis.*, **6**, 330-335.
- Goyer, R.A., Cherian, M.G. and Delaquerriere-Richardson, L. 1984. Correlation of parameters of cadmium exposure with onset of cadmium-induced nephropathy in rats. *JEPTO*, **5**, 89-100.
- Göransson, A. and Eldhuset, T.D. 1987. Effects of aluminium on growth and nutrient uptake of *Betula pendula* seedlings. *Physiol. Plantarum*, **69**, 193-199.
- Grande, M., Muniz, I.P. and Andersen, S. 1978. Relative tolerance of some salmonids to acid water. *Verh. Intern. Verein. Limnol.*, **20**, 2076-2084.
- Haines, T. 1981. Acidic precipitation and its consequences for aquatic ecosystems: a review. *Trans. Am. Fish. Soc.*, **110**, 669-707.
- Haines, T., Pauwels, S.J., Jagoe, C.H. and Norton, S.A. 1987. Effects of acidity-related water and sediment chemistry variables on trace metal burdens in brook trout (*Salvelinus fontinalis*). *Annls. Soc. R. Zool. Belg.*, **117**, Suppl.1, 45-55.
- Hall, R.J. and Likens, G.E. 1981. Chemical flux in an acid-stressed stream. *Nature*, **292**, 329-331.
- Hall, R.J., Driscoll, C.T., Likens, G.E. and Pratt, J.M. 1985. Physical, chemical and biological consequences of episodic aluminum additions to a stream. *Limnol. Oceanogr.*, **30**, 212-220.
- Hall, R.J., Driscoll, C.T. and Likens, G.E. 1987. Importance of hydrogen ion and aluminum in regulating the structure and function of stream ecosystems: an experimental test. *Freshw. Biol.*, **18**, 17-43.
- Hallbäck, L. and Tamm, C.O. 1986. Changes in soil acidity from 1927 to 1982-84 in a forest area of south-west Sweden. *Scand. J. For. Res.*, **1**, 219-232.
- Harvey, H.H. and McArdle, J.M. 1986. Physiological responses of rainbow trout *Salmo gairdneri* exposed to Plastic Lake inlet and outlet stream water. *Water, Air, Soil Pollut.*, **30**, 687-694.
- Haug, A. 1984. Molecular aspect of aluminum toxicity. *CRC Crit. Rev. Plant Sci.*, **1**, 345-373.
- Havas, M. 1985. Aluminum bioaccumulation and toxicity to *Daphnia magna* in soft water at low pH. *Can. J. Fish. Aquat. Sci.*, **42**, 1741-1748.
- Havas, M. 1986. A hematoxylin staining technique to locate sites of aluminum binding in aquatic plants and animals. *Water, Air, Soil Pollut.*, **30**, 735-741.
- Havas, M. and Likens, G.E. 1985. Changes in ²²Na influx and outflux in *Daphnia magna* (Straus) as a function of elevated Al concentrations in soft water at low pH. *Proc. Natl. Acad. Sci. USA*, **82**, 7345-7349.
- Heinz, G.H., Haseltine, S.D. and Sileo, L. 1983. Altered avoidance behaviour of young black ducks fed cadmium. *Envir. Toxicol. Chem.*, **2**, 419-421.
- Henriksen, A., Skogheim, O.K. and Rosseland, B.O. 1984. Episodic changes in pH and aluminium-speciation kill fish in a Norwegian salmon river. *Vatten*, **40**, 255-260.
- Herrmann, J. 1987a. Aluminium impact on freshwater invertebrates at low pH: A review. In: Landner, L. (ed.), *Speciation of metals in water, sediments and soil systems. Lecture Notes in Earth Sciences*. Volume 11, 157-175. Springer-Verlag, Berlin.
- Herrmann, J. 1987b. Sodium levels of lotic mayfly nymphs being exposed to aluminium at low pH - a preliminary report. *Annls. Soc. R. Zool. Belg.*, **117**, Suppl.1, 181-188.
- Herrmann, J. and Andersson, K.G. 1986. Aluminium impact on respiration of lotic mayflies at low pH. *Water, Air, Soil Pollut.*, **30**, 703-709.
- Horst, W.J., Wagner, A. and Marschner, H. 1982. Mucilage protects

- root meristems from aluminium injury. *Z. Pflanzenphysiol.*, **105**, 435-444.
- Horst, W.J., Wagner, A. and Marschner, H. 1983. Effect of aluminium on root growth, cell-division rate and mineral element contents in roots of *Vigna unguiculata* genotypes. *Z. Pflanzenphysiol.*, **109**, 95-103.
- Hörnström, E., Ekström, C. and Duraini, M.O. 1984. Effects of pH and different levels of aluminium on lake plankton in the Swedish west coast area. *Rep. Inst. Freshw. Res. Drottningholm*, **61**, 115-127.
- Huett, D.O. and Menary, R.C. 1980. Aluminum distribution in freeze-dried roots of cabbage, lettuce, and Kikuyu grass by energy-dispersive X-ray analysis. *Aust. J. Plant Physiol.*, **7**, 101-111.
- Hunter, J.B., Ross, S.L. and Tannahill, J. 1980. Aluminium pollution and fish toxicity. *Water Pollut. Contr.*, **79**, 413-420.
- Hutchinson, N., Holtze, K., Munro, J. and Pawson, T. 1987. Lethal responses of salmonid early life stages to H⁺ and Al in dilute waters. *Annls. Soc. R. Zool. Belg.*, **117**, Suppl.1, 201-217.
- Jagoe, C., Haines, T. and Buckler, D. 1987. Abnormal gill development in Atlantic salmon (*Salmo salar*) fry exposed to aluminium of low pH. *Annls. Soc. R. Zool. Belg.*, **117**, Suppl.1, 375-386.
- Johnson, D.W. 1973. Endocrine control of hydromineral balance in teleosts. *Am. Zool.*, **13**, 799-818.
- Kanis, J.A. 1981. Osteomalacia and chronic renal failure. *J. Clin. Pathol.*, **34**, 1295-1307.
- Karlsson-Noorgren, L., Dickson, W., Ljungberg, O. and Runn, P. 1986a. Acid water and aluminium exposure: gill lesions and aluminium accumulation in farmed brown trout, *Salmo salar* L. *J. Fish Dis.*, **9**, 1-9.
- Karlsson-Noorgren, L., Björklund, I., Ljungberg, O. and Runn, P. 1986b. Acid water and aluminium exposure; experimentally induced gill lesions in brown trout (*Salmo trutta*). *J. Fish Disease*, **9**, 11-25.
- Kendall, R.J. and Scanlon, P.F. 1981. Effects of chronic lead ingestion on reproductive characteristics of ringed turtle doves (*Streptopelia risoria*) and on tissue lead concentrations of adults and their progeny. *Envir. Pollut. Ser. A.*, **26**, 203-213.
- Kinraide, T.B. and Parker, D.R. 1987. Cation amelioration of aluminium toxicity in wheat. *Plant Physiol.*, **83**, 546-551.
- Kjartansson, H. 1984. Na-K-ATPase and residual Mg-ATPase activity of gill, pseudobranch and kidney tissues from salmonids before and after exposure to aqueous aluminium at low pH. Cand. Scient. Thesis, University of Bergen, Norway.
- Klaprat, D.A., Brown, S.B. and Hara, T.J. 1988. The effect of low pH and aluminium on the olfactory organ of rainbow trout (*Salmo gairdneri*). *Envir. Biol. Fishes*, **22**, 69-77.
- Kreuger, G.L., Morris, T.K., Suskind, R.R. and Widner, E.M. 1985. The health effects of aluminium compounds in mammals. *CRC Critical Rev. Toxicol.*, **13**, 1-24.
- Lacroix, G. and Townsend, D. 1987. Responses of juvenile Atlantic salmon to episodic increases in the acidity of some rivers of Nova Scotia, Canada. *Annls. Soc. R. Zool. Belg.*, **117**, Suppl.1, 297-307.
- Lamb, D.S. and Bailey, G.G. 1981. Acute and chronic effects of alum to midge larva (*Dipteria: Chronomidae*). *Bull. Envir. Contam. Toxicol.*, **27**, 59-67.
- Lee, E.H. and Foy, C.D. 1986. Aluminum tolerances of two snapbean cultivars related to organic acid content evaluated by high-performance liquid chromatography. *J. Plant Nutr.*, **9**, 1481-1498.
- Leivestad, H., Hendrey, G., Muniz, I.P. and Snekvik, E. 1976. Effect of acid precipitation on freshwater organisms. In: Braekke, F.H. (ed.), *Impact of acid precipitation on forest and freshwater ecosystems in Norway*, pp.87-111. SNSF-project, FR 6/76.
- Leivestad, H., Muniz, I.P. and Rosseland, B.O. 1980. Acid stress in trout from a dilute mountain stream. In: Drablos, D. and Tollan, A. (eds.), *Ecological impact of acid precipitation*, pp.318-319. SNSF-project.
- Leivestad, H., Jensen, E., Kjartansson, H. and Xingfu, L. 1987. Aqueous speciation of aluminium and toxic effects on Atlantic salmon. *Annls. Soc. R. Zool. Belg.*, **117**, Suppl.1, 387-398.
- Linnenback, M., Marthaler, R. and Gebhardt, H. 1987. Effects of acid water on gill and epidermis in brown trout (*Salmo trutta*) and in tadpoles of the common frog (*Rana temporaria* L.). *Annls. Soc. R. Zool. Belg.*, **1**, Suppl.1, 365-374.
- Lipstein, B. and Hurwitz, S. 1981. The nutritional value of sewage-grown, alum-flocculated microactinium algae in broiler and layer diets. *Poult. Sci.*, **60**, 2628-2638.
- Liss, L. (ed.) 1980. *Aluminum Neurotoxicity*. Pathox, Chicago.
- Lundholm, E. 1987. Review. Thinning of egg-shells in birds by DDE: mode of action on the egg-shell gland. *Comp. Biochem. Physiol.*, **88C**, 1-22.
- Mackie, G.L. 1986. *Am. Malac. Bull.* (referred to in Herrmann 1987a).
- Mahaffey, K.R., Goyer, R. and Haseman, J.K. 1973. Dose-response to lead ingestion in rats fed low dietary calcium. *J. Lab. Clin. Med.*, **82**, 92-100.
- Malley, D.F. and Chang, P.S.S. 1985. Effects of aluminium and acid on calcium uptake by the crayfish *Oronectes virilis*. *Arch. Envir. Contam. Toxicol.*, **14**, 739-747.
- Malte, H. 1986. Effects of aluminium in hard, acid water on metabolic rate, blood gas tensions and ionic status in rainbow trout. *J. Fish Biol.*, **29**, 187-198.
- Masoni, A. and Payan, P. 1974. Urea, inulin and para-aminohippuric acid (PAH) excretion by the gills of the eel, *Anguilla anguilla* L. *Comp. Biochem. Physiol.*, **47A**, 1241-1244.
- Matsumoto, H. and Morimura, S. 1980. Repressed template activity of chromatin of pea roots treated by aluminium. *Plant Cell Physiol.*, **21**, 951-959.
- Mayor, G.H., Keiser, J.A., Makdani, D. and Ku, P.K. 1977. Aluminum absorption and distribution: effect of parathyroid hormone. *Science*, **197**, 1187-1189.
- Mayor, G.H., Sprague, S.M., Hourani, R.M. and Sanchez, T.V. 1980. Parathyroid hormone mediated aluminum deposition and egress. *Kidney Int.*, **17**, 40-44.
- McConnick, L.H. and Borden, F.Y. 1974. The occurrence of aluminumphosphate precipitate in plant roots. *Soil Sci. Soc. Am. Proc.*, **38**, 931-934.
- McDonald, D.G. 1983. The effects of H⁺ upon the fish gills of fresh water fish. *Can. J. Zool.*, **61**, 691-703.
- Muniz, I.P., Leivestad, H. and Rosseland, B.O. 1978. Stressmalingar pa fisk i sure vassdrag. (Stress measurements on fish in acidic rivers). *Nordforsk*, publ. 1978 **2**, 233-247. (In Norwegian).
- Muniz, I.P. and Leivestad, H. 1980. Toxic effects of aluminium on the brown trout (*Salmo trutta* L.). In: Drablos, D. and Tollan, A. (eds.), *Ecological impact of acid precipitation*, pp.320-321. SNSF-project.
- Muramoto, S. 1981. Influence of complexants (NTA, EDTA) on the toxicity of aluminium chloride and sulfate to fish at high concentrations. *Bull. Envir. Contam. Toxicol.*, **27**, 221-225.
- Neville, C.M. 1985. Physiological response of juvenile rainbow trout, *Salmo gairdneri*, to acid and aluminium - prediction of field responses from laboratory data. *Can. J. Fish. Aquat. Sci.*, **42**, 2004-2019.
- Nilsson, S.I. and Bergkvist, B. 1983. Aluminium chemistry and acidification processes in a shallow podzol on the Swedish west coast. *Water, Air, Soil Pollut.*, **20**, 311-329.
- Nilsson, S.I. and Lundmark, J.-E. 1986. Exchangeable aluminium as a basis for lime requirement determinations in acid forest soils. *Acta Agric. Scand.*, **36**, 173-185.
- Nyholm, E. 1981. Evidence of involvement of aluminium in causation of defective formation of eggshells and of impaired breeding in wild passerine birds. *Environ. Res.*, **26**, 363-371.
- Nyholm, N.E. 1982. Aluminium concentrations of stoneflies from lakes Tjülträsk and Torneträsk in northern Sweden. Internal report to the Swedish Environmental Institute, B05207, S-40224, Gothenburg, Sweden.
- Nyholm, N.E. and Myhrberg, H.E. 1977. Severe eggshell defects and impaired reproductive capacity in small passerines in Swedish Lapland. *Oikos*, **29**, 336-341.
- Ogilvie, D.M. and Stechey, D.M. 1983. Effects of aluminium on respiratory responses and spontaneous activity of rainbow trout, *Salmo gairdneri*. *Envir. Toxicol. Chem.*, **2**, 43-48.
- Økland, J. and Økland, K.A. 1986. The effects of acid deposition on benthic animals in lakes and streams. *Experientia*, **42**, 471-486.
- Ormerod, S., Weatherley, N., French, P., Blake, S. and Jones, W. 1987. The physiological response of brown trout, *Salmo trutta* to induced episodes of low pH and elevated aluminium in a Welsh hill stream. *Annls. Soc. R. Zool. Belg.*, **117**, Suppl.1,

- 435-447.
- Otto, C. and Svensson, B.S. 1983. Properties of acid brown water streams in South Sweden. *Arch. Hydrobiol.*, **99**, 15-36.
- Pagenkopf, G.K. 1983. Gill surface interaction model for trace-metal toxicity to fishes, role of complexation, pH and water hardness. *Envir. Sci. Technol.*, **17**, 342-347.
- Parkinson, I.S., Ward, M.K. and Kerr, D.N.S. 1981. Dialysis encephalopathy, bone disease and anaemia: the aluminum intoxication syndrome during regular haemodialysis. *J. Clin. Pathol.*, **34**, 1285-1294.
- Peakall, D.B. 1985. Behavioral responses of birds to pesticides and other contaminants. *Resid. Rev.*, **96**, 46-77.
- Pedder, S.C.J. and Maly, E.J. 1987. The effect of avoidance on the plasmacation concentration of brook trout (*Salvelinus fontinalis*) subjected to varying levels of acidity. *Aquat. Toxicol.*, **10**, 29-39.
- Perl, D.P. 1985. Relationship of aluminum to Alzheimer's disease. *Envir. Hlth. Persp.*, **63**, 149-153.
- Petersen, R.C.Jr., Petersen, L.B.-M., Persson, U., Kullberg, A., Hargeby, A. and Paarlberg, A. 1986. Health aspects of humic compounds in acid environments. *Water Qual. Bull.*, **11**, 44-50.
- Petit, T.L., Biederman, G.B. and McMullen, P.A. 1980. Neurofibrillary degeneration, dendritic dying back, and learning-memory deficits after aluminum administration: implications for brain aging. *Expl. Neurol.*, **67**, 152-162.
- Petit, T.L., Beiderman, B.G., Jonas, P. and Leboutillier, J.C. 1985. Neurobehavioral development following aluminum administration in infant rabbits. *Expl. Neurol.*, **88**, 640-651.
- Potts, W.T.W. and Flemming, W.R. 1970. The effect of prolactin and divalent ions on the permeability to water of *Fundulus kanasae*. *J. Expl. Biol.*, **53**, 317-327.
- Rabe, A., Lee, M.H., Shek, J. and Wisniewski, H.M. 1982. Learning deficit in immature rabbits with aluminium-induced neurofibrillary changes. *Expl. Neurol.*, **76**, 441-446.
- Raddum, G.G. and Steigen, A.L. 1981. Reduced survival and caloric content of stoneflies and caddisflies in acid water. In: Singer, R. (ed.), *Effects of acidic precipitation on Benthos. Proc. Symp. Acidic Precipitation on Benthos*, 1980, pp.97-101. North American Benthological Society. Hamilton, New York.
- Raddum, G.G. and Fjellheim, A. 1987. Effects of pH and aluminium on mortality, drift and moulting of the mayfly *Baetis rhodani*. *Annls. Soc. R. Zool. Belg.*, **117**, Suppl.1, 77-87.
- Reite, O.B. and Staumes, M. 1987. Acidified water: effects on physiological mechanisms in the gills of salmonids. Surface Water Acidification Program, Midterm Review Conference, Bergen 22-26 June 1987, pp.298-304.
- Rosseland, B.O. 1980. Effects of acid water on metabolism and gill ventilation in brown trout, *Salmo trutta* L., and brook trout, *Salvelinus fontinalis* Mitchell. In: Drablos, D. and Tollan, A. (eds.), *Ecological Impacts of Acid Precipitation*, pp.348-349. SNSF-project.
- Rosseland, B.O. 1986. Ecological effects of acidification on tertiary consumers. Fish population responses. *Water, Air, Soil Pollut.*, **30**, 451-460.
- Rosseland, B.O. and Skogheim, O.K. 1982. Physiological stress and mortality of Atlantic salmon, *Salmo salar* L. in acid water with high levels of aluminium. Intern. Council for Exploration of the Sea. C.M. 1982/M:29, 15 pp.
- Rosseland, B.O. and Skogheim, O.K. 1984. A comparative study on salmonid fish species in acid aluminium-rich water. II. Physiological stress and mortality of one and two year old fish. *Rep. Inst. Freshw. Res. Drottningholm*, **61**, 186-194.
- Rosseland, B.O. and Skogheim, O.K. 1987. Differences in sensitivity to acidic soft water among strains of brown trout (*Salmo trutta* L.). *Annls. Soc. R. Zool. Belg.*, **117**, Suppl.1, 258-265.
- Rosseland, B.O., Skogheim, O.K., Abrahamsen, H. and Matzow, D. 1986a. Limestones slurry reduces physiological stress and increases survival of Atlantic salmon (*Salmo salar*) in an acidic Norwegian river. *Can. J. Fish. Aquat. Sci.*, **43**, 1888-1893.
- Rosseland, B.O., Skogheim, O.K., Kroglund, F. and Hoell, E. 1986b. Mortality and physiological stress of year-classes of landlocked and migratory Atlantic salmon, brown trout and brook trout in acid aluminium-rich soft water. *Water, Air, Soil Pollut.*, **30**, 751-756.
- Rost-Siebert, K. 1983. Aluminium-Toxizität und -Toleranz an Keimpflanzen von Fichte (*Picea abies* Karst.) und Buche (*Fagus sylvatica* L.). *Allg. Forstz.*, **38**, 686-689.
- Sadler, K. and Tumpenny, A.W.H. 1986. Field and laboratory studies of exposure of brown trout to acid waters. *Water, Air, Soil Pollut.*, **30**, 593-599.
- Scheuhammer, A.M. 1987. The chronic toxicity of aluminum, cadmium, mercury and lead in birds: a review. *Envir. Pollut.*, **46**, 263-295.
- Schofield, C.L. 1977. Acid snow-melt effects on water quality and fish survival in the Adirondack Mountains of New York State. Res. Proj. Tech. com. rep. Project A-072-NY. Cornell University, Ithca, New York. 27 pp.
- Schofield, C.L. and Trojnar, R.J. 1980. Aluminum toxicity to brook trout (*Salvelinus fontinalis*) in acidified waters. In: Toribara, T.Y., Miller, M.W. and Morrow, P.E. (eds.), *Polluted Rain*, pp.341-366. Plenum Press, New York.
- Siegel, N. 1985. Aluminum interaction with biomolecules: The molecular basis for aluminium toxicity. *Am. J. Kidney Dis.*, **6**, 353-357.
- Siegel, N. and Haug, A. 1983a. Calmodulin-dependent formation of membrane potential in barley root plasma membrane vesicles: A biochemical model of aluminium toxicity in plants. *Physiol. Plant.*, **59**, 285-291.
- Siegel, N. and Haug, A. 1983b. Aluminum interaction with calmodulin. Evidence for altered structure and function from optical and enzymatic studies. *Biochem. Biophys. Acta*, **744**, 36-45.
- Simkiss, K. (ed.) 1967. *Calcium in Reproductive Physiology*. Chapman and Hall, London.
- Simkiss, K. and Taylor, T.G. 1971. Shell formation. In: Bell, D.J. and Freeman, B.M. (eds.), *Physiology and Biochemistry of the Domestic Fowl*, Volume 3, pp.1331-1343. Academic Press, New York.
- Six, K.M. and Goyer, R.A. 1970. Experimental enhancement of lead toxicity by low dietary Ca. *J. Lab. Clin. Med.*, **76**, 933-942.
- Skogheim, O.K., Rosseland, B.O. and Sevaldud, I.H. 1984. Deaths of spawners of Atlantic salmon in River Ognå, SW Norway, caused by acidified aluminium-rich water. *Rep. Inst. Freshw. Res. Drottningholm*, **61**, 195-202.
- Skogheim, O.K. and Rosseland, B.O. 1986. Mortality of smolts of Atlantic salmon, *Salmo salar* L., at low levels of aluminium in acidic soft water. *Bull. Envir. Contam. Toxicol.*, **37**, 258-265.
- Slanina, P., Falkeborn, Y., Frech, W. and Cedergren, A. 1984. Aluminum concentrations in the brain and bone of rats fed citric acid, aluminum citrate or aluminum hydroxide. *Fd. Chem. Toxicol.*, **22**, 391-397.
- Slanina, P., Frech, W., Ekstrøm, L., Loof, L., Storch, S. and Cedergren, A. 1986. Dietary citric acid enhances absorption of aluminum in antacids. *Clin. Chem.*, **32**, 539-541.
- Sørensen, J.R.J., Campbell, I.R., Tepper, L.B. and Lingg, R.D. 1974. Aluminum in the environment and human health. *Envir. Hlth. Persp.*, **8**, 3-95.
- Spencer, H. and Lenger, M. 1979. Adverse effect of aluminum-containing antacids on the mineral metabolism. *Gastroenterology*, **76**, 603-606.
- Spencer, H., Norris, C., Coffey, J. and Wiatrowski, E. 1975. Effect of small amounts of antacids on calcium, phosphorus and fluoride metabolism in man. *Gastroenterology*, **68**, 990.
- Starkey, B.J. 1987. Aluminum in renal disease: current knowledge and future developments. *Ann. Clin. Biochem.*, **24**, 337-344.
- Staumes, M., Sigholt, T. and Reite, O.B. 1984. Reduced carbonic anhydrase and Na-K-ATPase activity in gills of salmonids exposed to aluminium containing acid water. *Experientia*, **40**, 226-227.
- Steinbohm, K., Robbards, S. and Williams, C. 1957. Phosphate treatment of alumina gel weakness in chicks. *J. Appl. Physiol.*, **11**, 435-438.
- Storer, N.L. and Nelson, T.S. 1968. The effect of various aluminum compounds on chick performance. *Poul. Sci.*, **47**, 244-247.
- Stowe, H.D., Goyer, R.A., Krigman, M.M., Wilson, M. and Cates, M. 1973. Experimental oral lead toxicity in young dogs. Clinical and morphological effects. *Arch. Pathol.*, **95**, 106-116.
- Street, H.R. 1942. The influence of aluminum sulfate and aluminum hydroxide upon the absorption of dietary phosphorus by the rat. *J. Nutr.*, **24**, 111-119.
- Suhayda, C.G. and Haug, A. 1986. Organic acids reduce Al toxicity in

- maize root membranes. *Physiol. Plant.*, **68**, 189-195.
- Taylor, G.J. and Foy, C.D. 1985. Mechanisms of aluminium tolerance in *Triticum aestivum* (wheat). IV. The role of ammonium and nitrate nutrition. *Can. J. Bot.*, **63**, 2181-2186.
- Thommessen, G. 1975. Effekter på ørretens luktesans av kortvarige eksponeringer for surt miljø. (Effects on the olfactory sense of brown trout to short time exposure to acidic environment). IR 9/75, 17 pp. SNSF-Project. (In Norwegian).
- Trapp, G.A. 1986. Interactions of aluminum with co-factors, enzymes, and other proteins. *Kidney Int.*, **29**, Suppl.18, S12-S16.
- Tyler, G. 1987. Acidification and chemical properties of south Swedish beech (*Fagus sylvatica* L.) forest soils. *Scand. J. For. Res.*, **2**, 263-271.
- Ulrich, B. 1980. Die Wälder in Mitteleuropa: Messergebnisse ihrer Umweltbelastung, Theorie ihrer Gefährdung, Prognose ihrer Entwicklung. *Allg. Forstz.*, **35**, 1198-1202.
- Ulrich, B. 1981. Destabilisierung von Waldökosystemen durch Akkumulation von Luftverunreinigungen. *Forst- und Holzwirt.*, **36**, 525-532.
- Ulrich, B., Mayer, R. and Khanna, P.K. 1980. Chemical changes due to acid precipitation in a loess derived soil in Central Europe. *Soil Sci.*, **130**, 193-199.
- Van Bameveld, A.A. and Van den Hamer, C.J.A. 1985. Influence of Ca and Mg on the uptake and deposition of Pb and Cd in mice. *Toxicol. Appl. Pharmacol.*, **79**, 1-10.
- Vangenechten, J.H.D., Van Puymbroeck, S. and Vanderborght, O.L.J. 1979. Effect of pH on the uptake of sodium in the Waterbugs *Corixa dentipes* (Thoms.) and *Corixa punctata* (Illig) (Hemiptera, Heteroptera). *Comp. Biochem. Physiol.*, **64A**, 509-521.
- Wagatsuma, T. 1983. Characterization of absorption sites for aluminum in the roots. *Soil Sci. Plant Nutr.*, **29**, 499-515.
- Wagatsuma, T. and Yamasaku, K. 1985. Relationship between differential aluminum tolerance and plant-induced pH-change of medium among barley cultivars. *Soil Sci. Plant Nutr.*, **31**, 521-535.
- Ware, R.A., Burkholder, P.M. and Chang, L.W. 1975. Ultrastructural changes in renal proximal tubules after chronic organic and inorganic mercury intoxication. *Envir. Res.*, **10**, 121-140.
- Wendelaar Bonga, S.E. and Balm P.H.M. 1987. Endocrine responses to acid stress in fish. *Mem. Soc. Exp. Biol.*
- Wendelaar Bonga, S., Flik, G. and Balm, P.H.M. 1987. Physiological adaptation to acidic stress in fish. *Anns. Soc. R. Zool. Belg.*, **117**, Suppl.1, 243-254.
- White, D.H., Finley, M.T. and Ferrell, J.F. 1978. Histopathologic effects of dietary cadmium on kidneys and testes of mallard ducks. *J. Toxicol. Envir. Hlth.*, **4**, 551-558.
- Witters, H.E. 1986. Acute acid exposure of rainbow trout *Salmo gairdneri* Richardson; effects of aluminium and calcium on ion balance and haematology. *Aquat. Toxicol.*, **8**, 197-210.
- Witters, H., Vangenechten, J.H.D., Van Puymbroeck, S. and Vanderborght, O.L.J. 1984. Interference of aluminium and pH on the Na-influx in an aquatic insect *Corixa punctata* (Illig.). *Bull. Envir. Contam. Toxicol.*, **32**, 575-579.
- Witters, H., Vangenechten, J., Van Puymbroeck, S. and Vanderborght, O.L.J. 1987. Ionoregulatory and haematological responses of rainbow trout, *Salmo gairdneri* to chronic acid and aluminium stress. *Anns. Soc. R. Zool. Belg.*, **117**, Suppl.1, 411-420.
- Wood, C.M. and McDonald, D.G. 1982. Physiological mechanisms of acid toxicity to fish. In: Johnson, R.E. (ed.), *Acid Rain/Fisheries*, pp.197-226. American Fisheries Society, Bethesda.
- Wood, C.M. and McDonald, D. 1987. The physiology of acid/aluminium stress in trout. *Anns. Soc. R. Zool. Belg.*, **117**, Suppl.1, 399-410.
- Worker, N.A. and Magicovsky, B.B. 1961a. Effect of vitamin D on the utilization of beryllium, magnesium, calcium, strontium and barium in the chick. *J. Nutr.*, **74**, 490-494.
- Worker, N.A. and Magicovsky, B.B. 1961b. Effect of vitamin D on the utilization of zinc, cadmium and mercury in the chicks. *J. Nutr.*, **75**, 222-224.
- Wright, R.J. and Wright, S.F. 1987. Effects of aluminum and calcium on the growth of subterranean clover in Appalachian soils. *Soil Sci.*, **143**, 341-348.
- Yase, Y. 1977. The basic process of amyotrophic lateral sclerosis as reflected in Kii Peninsula and Guam. In: Den Hartog Jager, W.A., Bruyn, G.W. and Hejstee, A.P.J. (eds.), *Proc. 11th World Congr. Neurology*, Amsterdam, pp. 413-427.
- Yokel, R.A. 1984. Toxicity of aluminum exposure during lactation to the maternal and suckling rabbit. *Toxicol. Appl. Pharmacol.*, **75**, 35-43.
- Yokel, R.A. 1985. Toxicity of gestational aluminum exposure to the maternal rabbit and its offspring. *Toxicol. Appl. Pharmacol.*, **79**, 121-133.
- Yokel, R.A. 1987. Toxicity of aluminum exposure to the neonatal and immature rabbit. *Fund. Appl. Toxicol.*, **9**, 795-806.
- Yokel, R.A. and McNamara, P.J. 1987. Oral absorption of 8 representative aluminum (Al) compounds and kinetics of intravenous Al in renally-intact and impaired rabbits. *Fed. Proc.*, **46**, 1147.
- Zhao, X.J., Sucoff, E. and Stadelmann, E.J. 1987. Al³⁺ and Ca²⁺ alteration of membrane permeability of *Quercus rubra* root and cortex cells. *Plant Physiol.*, **83**, 159-162.