

Effect of Calcium and Aluminum Concentrations on the Survival of Brown Trout (*Salmo trutta*) at Low pH

D. J. A. Brown

*C.E.R.L. Freshwater Biology Unit, Midlands Region Scientific Services
Department, Ratcliffe-on-Soar, Nottingham, NG11 0EE, United Kingdom*

Experimental evidence suggests that the two most important ions with respect to survival of fish at low pHs are calcium (BROWN 1981, 1982a and b; BROWN and LYNAM 1981) and aluminium (SCHOFIELD and TROJNAR 1980; BAKER and SCHOFIELD 1980, 1982). A minimal concentration of the former ion is necessary for survival, and in general, elevated concentrations of aluminium reduce survival. These same two ions are also clearly important in the field because acid lakes in Europe and North America have typically low concentrations of calcium and high concentrations of aluminium (WRIGHT et al. 1980).

The only published experimental study of the combined effects of low calcium and elevated aluminium concentrations on fish at low pH shows that the rate of loss of plasma chloride at pH 5.3 and 5.5 in solutions containing 0.9 mg l^{-1} of aluminium is higher when they contain 0.8 mg l^{-1} of calcium compared with solutions containing $3.0\text{--}4.2 \text{ mg l}^{-1}$ of calcium (MUNIZ AND LEIVESTAD 1980). In this paper, the results of experiments to determine the survival times of yolk sac fry of brown trout (*Salmo trutta*) in a range of pHs, calcium and aluminium solutions are described, and the relevance of the results to the field situation is also discussed.

METHODS AND MATERIALS

The bioassay apparatus used has been previously described by BROWN and LYNAM (1981). All the experiments were carried out in a cold room at $10 \pm 1^\circ\text{C}$, and sulphuric acid was used to control pH. Sodium, calcium and aluminium were all added as chloride. All test solutions contained 1 mg l^{-1} of sodium. Four pH controlled (± 0.07) reservoirs were used (pH 4.5, 4.8, 5.1 and 5.4). Two 16-day bioassay experiments were carried out on groups of twenty yolk sac fry which had been reared at the hatchery (at Mercaston in Derbyshire). In both experiments, three aluminium concentrations (0, 0.25 and 0.5 mg l^{-1}) were used. In the first, the two calcium concentrations used were 1.0 and 2.0 mg l^{-1} and in the second, 0.25 and 0.5 mg l^{-1} . One combination of pH, calcium and aluminium (5.4, 1.0 and 0.5 mg l^{-1} respectively) was repeated during both sets of bioassays and on four subsequent occasions including a group of 10 one year old fish, to ascertain how toxicity of aluminium/calcium solutions changed with time after hatching.

The concentrations of ions in the bioassay chambers were measured at 3-4 day intervals throughout the experiments and values obtained were within 20% of the nominal concentration. Observations were made daily throughout the experiments, and the number of dead fry were recorded and removed. The criterion for death was when opercular activity had ceased and there was no response to gentle prodding. Median periods of survival and their 95% confidence intervals were determined by the graphical method of LITCHFIELD (1950).

RESULTS

The results of the fry bioassay at pH 5.4, 1 mg Ca l⁻¹, 0.5 mg Al l⁻¹, repeated at various times after hatching are shown in Fig. 1 together with the median period of survival of the group of one year old fish. The median period of survival is significantly shorter after swim-up than before, but during the period when the two major 16-day bioassays were conducted (up to day 36 after hatching), survival is relatively constant and data from the two bioassays are comparable.

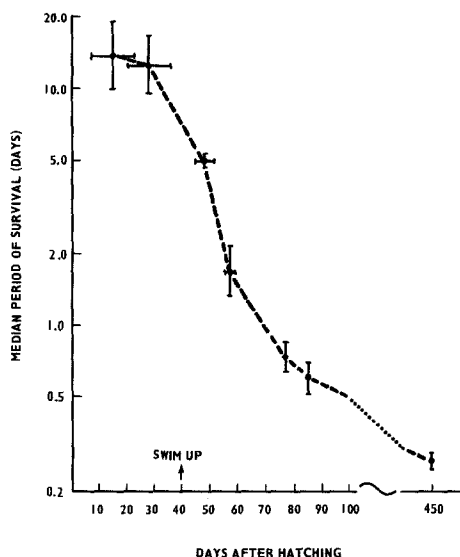


Figure 1: The median period of survival of brown trout fry and fingerlings at various times after hatching, when kept in a solution at pH 5.4 containing 1 mg l⁻¹ of sodium and calcium and 0.5 mg l⁻¹ of aluminium. (Horizontal bars represent time period of experiment and vertical bars are 95% confidence intervals).

The results of these two experiments, both in terms of the percentage survival after 16 days and the median period of survival, are combined in Fig. 2. It can be seen that, in the absence of aluminium, mortalities occur at pH 4.5 only with 0.25

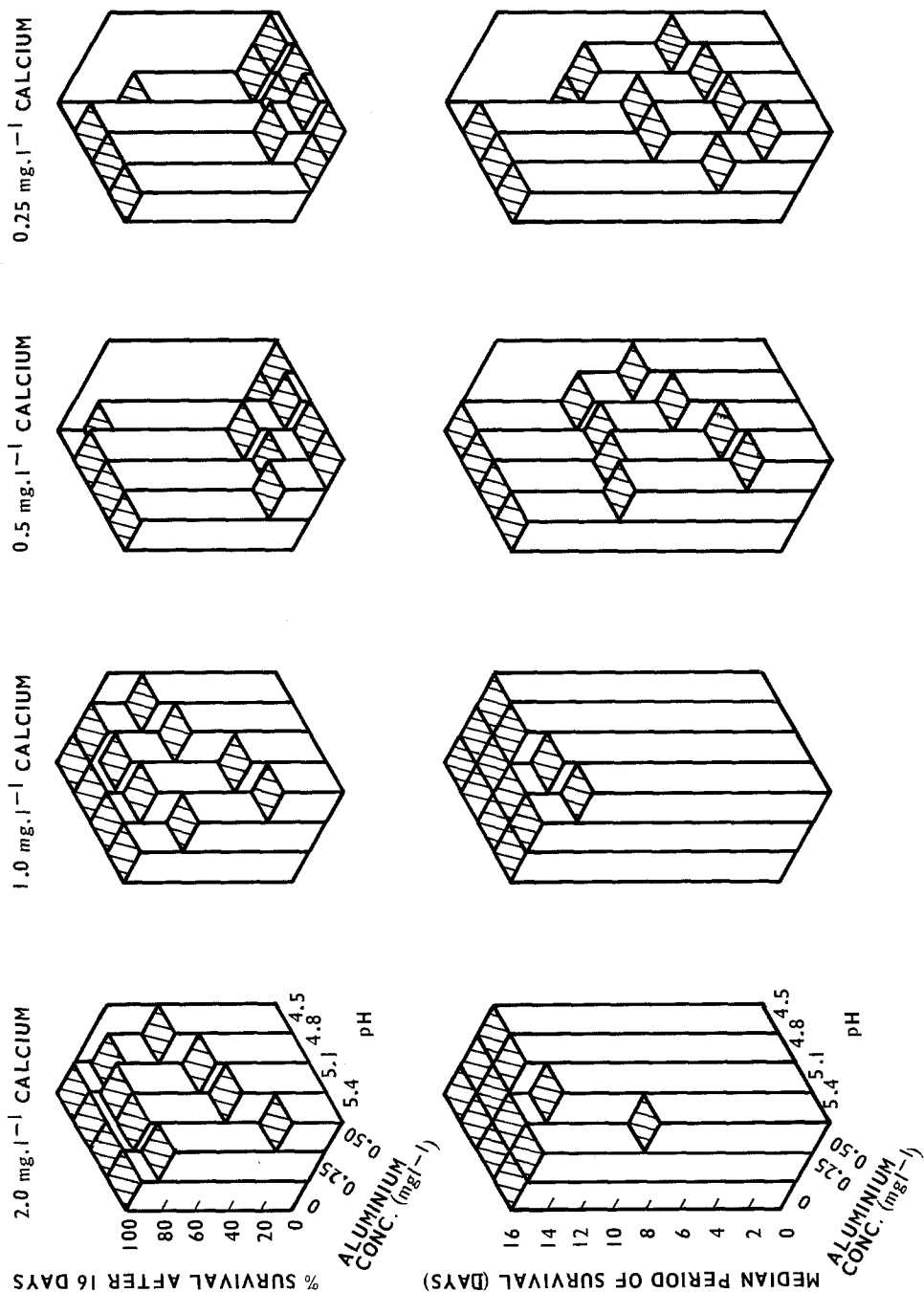


Figure 2: The percentage survival after 16 days, and the median period of survival of brown trout fry at various pHs, calcium and aluminium concentrations.

and 0.5 mg l^{-1} of calcium. In solutions containing 0.25 mg l^{-1} of aluminium, however, a complete range of responses is seen, from no or very low survival with 0.25 mg l^{-1} of calcium to almost complete survival with 2.0 mg l^{-1} of calcium. Solutions containing 0.5 mg l^{-1} of aluminium lead to almost complete mortalities with calcium concentrations of 0.25 and 0.5 mg l^{-1} and still significant mortalities at higher calcium concentrations. In general, the effect of pH throughout the range tested is not so marked, but there is a tendency for higher pHs to be more toxic especially in solutions containing 0.5 mg l^{-1} of aluminium. Overall one of the most striking results is the difference between the numbers surviving in solutions containing 0.5 mg l^{-1} of calcium and less when compared with those in 1.0 mg l^{-1} of calcium or more.

DISCUSSION

The toxicity and chemistry of aluminium in aquatic systems have been reviewed by BURROWS (1977). The chemistry of aqueous solutions is complex, with solubility minimal around pH 5.5. Below pH 5.5 aluminium exists as the cation although ionic speciation in solutions changes with time. At pH 5.0 Al^{3+} , $\text{Al}(\text{OH})^{++}$ and $\text{Al}(\text{OH})_2^+$ are approximately equal but Al^{3+} tends to hydrolyse and polymerize forming colloidal $\text{Al}(\text{OH})_3$ which may eventually precipitate. Large complexes of aluminium and other organic and inorganic ligands seem to occur readily and the toxicity of dissolved aluminium can be reduced or even eliminated by this complexation (BAKER and SCHOFIELD 1980; DRISCOLL et al. 1980).

The fact that swim up fry are more sensitive to pH/aluminium toxicity than yolk sac fry has previously been noted by BAKER and SCHOFIELD (1980), but the dramatic increase in sensitivity that occurs between days 30 and 80 after hatching (Fig. 1), and the fact that year-old fish retain this increased sensitivity have not been previously recorded.

The trend in the bioassays towards increasing toxicity of total aluminium with increasing pH over the range used in these experiments, has been previously noted by SCHOFIELD and TROJNAR (1980) and BAKER and SCHOFIELD (1982) who suggested that changes in speciation involving hydroxy-complexing alters the toxicity of the aluminium cation.

One of the most striking results is the difference between the % survivals found in aluminium solutions containing 0.5 mg l^{-1} of calcium or less when compared with those containing 1.0 mg l^{-1} or more. The physiological effect on fish of increasing calcium concentration in waters of low pH has been shown to be to change the gill permeability to sodium, chloride and hydrogen ions (McWILLIAMS and POTTS 1978). The effect of low pH on the trans-epithelial gill potential is thus moderated, and the rate of passive efflux of sodium from the fish is thereby reduced (McWILLIAMS 1982). The fact that survival in aluminium solutions

in these experiments is very dependent on calcium concentration would therefore suggest that the physiological effect of aluminium under the conditions of these experiments is also mediated via the ion-regulatory system, as suggested by MUNIZ and LEIVESTAD (1980).

The relationship of these experimental findings to conditions in acid lakes is of interest. In southern Norwegian lakes, over the same range of pH and calcium concentrations used in the experiments, average aluminium concentrations range from 0.1 mg l^{-1} at pH 5.1-5.4 and calcium $0.25\text{-}0.5 \text{ mg l}^{-1}$, to 0.25 mg l^{-1} at pH 4.5-4.8 and calcium $1.0\text{-}2.0 \text{ mg l}^{-1}$ (Fig. 3, data of WRIGHT and SNEKVIK, 1978).

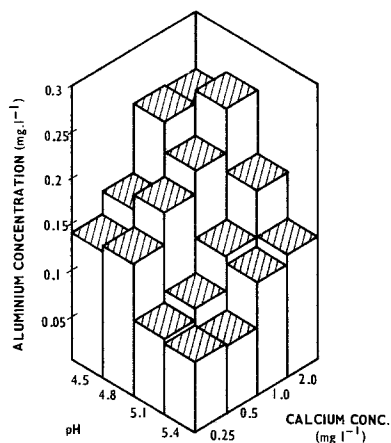


Figure 3: Total aluminium concentrations at various pH and calcium concentrations in lakes in southernmost Norway - data of WRIGHT and SNEKVIK (1978). (pH intervals used are 4.33-4.62, 4.63-4.92, 4.93-5.22 5.23-5.52, and calcium concentration intervals used are up to 0.35, 0.35-0.70, 0.71-1.40, 1.41-2.81 mg l^{-1} .)

WRIGHT and SNEKVIK (1978) found no evidence from multiple regression analysis that aluminium was a factor determining fishery status of these lakes, yet the experiments reported here suggest that aluminium concentrations in the range observed could be a significant factor alongside pH and calcium. Since the field measurements are of total aluminium, much of it possibly present in non toxic complexes, and the bioassay aluminium is presumably in toxic inorganic form, this may in part explain the anomaly. Nevertheless, it would appear that although the relationship between calcium and aluminium in determining the fishery status of low pH lakes is complex, both ions will need to be considered in any attempt to explain the field observations. Further bioassay experiments are needed using lower concentrations of aluminium, and a knowledge of its speciation would be an advantage.

ACKNOWLEDGEMENTS

The invaluable assistance given by Mrs S. Lynam with the bio-assays is gratefully acknowledged, as are the helpful criticisms of an early manuscript by Dr G.D. Howells and Dr K. Sadler.

This paper is published by permission of the Central Electricity Generating Board.

REFERENCES

- BAKER, J.P. and C.L. SCHOFIELD: In DRABLOS, D. and A. TOLLAN (eds), Proc. Int. Conf. Ecol. Impact Acid Precip., Norway SNSF project, pp. 292-293 (1980).
- BAKER, J.P. and C.L. SCHOFIELD: Water, Air and Soil Pollut. 18, 289 (1982).
- BROWN, D.J.A.: J. Fish Biol. 18, 31 (1981).
- BROWN, D.J.A.: Water, Air and Soil Pollut. 18, 343 (1982 a).
- BROWN, D.J.A.: Bull. Environm. Contam. Toxicol. 28, 664 (1982 b).
- BROWN, D.J.A. and S. LYNAM: J. Fish Biol. 19, 205 (1981).
- BURROWS, W.D.: C.R.C. Critical Reviews in Env. Control. 7, 167 (1977)
- DRISCOLL, C.D., J.P. Baker, J.J. BISOGNI and C.L. SCHOFIELD: Nature, 284, 161 (1980).
- LITCHFIELD, J.T.: J. Pharmacol. Exp. Ther. 97, 399 (1950).
- McWILLIAMS, P.G.: J. Exp. Biol. 96, 439 (1982).
- McWILLIAMS, P.G. and W.T.W. POTTS: J. Comp. Physiol. 126, 277 (1978).
- MUNIZ, I.P. and H. LEIVESTAD: In DRABLOS, D. and A. TOLLAN (eds), Proc. Int. Conf. Ecol. Impact Acid Precip., Norway, SNSF project, pp. 84-92 (1980).
- SCHOFIELD, C.L. and J.R. TROJNAR: In TORIBARA, T.Y., M.W. MILLER and P.E. MORROW (eds), 'Polluted Rain', Plenum Press, New York, pp. 341-366 (1980).
- WRIGHT, R.F., N. CONROY, W.T. DICKSON, R. HARRIMAN, A. HENRIKSEN and C.L. SCHOFIELD: In DRABLOS, D. and A. TOLLAN (eds), Proc. Int. Conf. Ecol. Impact Acid Precip., Norway, SNSF project, pp. 377-379 (1980).
- WRIGHT, R.F. and E. SEKVIK: Verh. Internat. Verein. Limnol. 20, 765. (Raw data available in SNSF project TN 37/77.) (1978).

Accepted February 20, 1983