



CSAS

Canadian Stock Assessment Secretariat

SCÉS

Secrétariat canadien pour l'évaluation des stocks

Research Document 2000/050

Document de recherche 2000/050

Not to be cited without
permission of the authors¹

Ne pas citer sans
autorisation des auteurs¹

Effects of low environmental pH on Atlantic salmon (*Salmo salar* L.) in Nova Scotia

G.J. Farmer

Department of Fisheries and Oceans
Science Branch, Maritimes Region
P.O. Box 1006, Dartmouth, NS B2Y 4A2

¹ This series documents the scientific basis for the evaluation of fisheries resources in Canada. As such, it addresses the issues of the day in the time frames required and the documents it contains are not intended as definitive statements on the subjects addressed but rather as progress reports on ongoing investigations.

¹ La présente série documente les bases scientifiques des évaluations des ressources halieutiques du Canada. Elle traite des problèmes courants selon les échéanciers dictés. Les documents qu'elle contient ne doivent pas être considérés comme des énoncés définitifs sur les sujets traités, mais plutôt comme des rapports d'étape sur les études en cours.

Research documents are produced in the official language in which they are provided to the Secretariat.

Les documents de recherche sont publiés dans la langue officielle utilisée dans le manuscrit envoyé au Secrétariat.

This document is available on the Internet at:

Ce document est disponible sur l'Internet à :

<http://www.dfo-mpo.gc.ca/csas/>

Abstract

Acidic rivers in Nova Scotia have low concentrations of Ca^{++} (calcium) and high concentrations of dissolved organic matter and total dissolved aluminum. Dissolved organic matter binds to aluminum to form organic aluminum complexes which are not toxic to Atlantic salmon. Increased H^+ ion concentrations (acidity) coupled with the low concentrations of Ca^{++} have caused the mortality of salmon in Nova Scotia. When $\text{pH} \leq 5.0$, the active uptake of Na^+ (sodium) and Cl^- (chloride) across the gill epithelium is reduced and the passive efflux is increased resulting in the net loss of both ions. The increased passive efflux of ions results from the displacement of Ca^{++} from binding sites on the gill epithelium by H^+ . The loss of ions results in a shift of water from the extracellular fluid volume to the intracellular fluid volume causing a reduction in blood volume. The reduced blood volume and an increase in hematocrit causes an increase in blood viscosity which results in circulatory system failure. Sensitivity of the various salmon stages to low pH can be summarized in the following order of decreasing sensitivity:

fry > smolt > small parr > large parr > alevin > egg

Thus, significant mortality (19 - 71%) of fry occurs at a pH of about 5.0. Mortality of smolts also occurs at a pH of 5.0 but the rate is lower (1-5%). Mortality of parr and smolts is relatively great (72 – 100%) when pH declines to the 4.6 – 4.7 range. Mortality of eggs and alevins does not begin until pH declines below 4.8.

Résumé

En Nouvelle-Écosse, les rivières acidifiées présentent de faibles concentrations de Ca^{++} (calcium) et des concentrations élevées en matière organique dissoute et en aluminium total dissout. La matière organique dissoute se lie à l'aluminium pour former des complexes organiques qui ne sont pas toxiques pour le saumon de l'Atlantique. Des concentrations élevées en ions H^+ (acidité) associées à de faibles concentrations de Ca^{++} ont causé la mort de saumons en Nouvelle-Écosse. À un $\text{pH} \leq 5.0$, l'absorption active du Na^+ (sodium) et du Cl^- (chlore) à travers l'épithélium branchial diminue et la sortie passive augmente, ce qui se traduit par une perte nette des deux types d'ions. La sortie passive accrue des ions résulte du déplacement du Ca^{++} par le H^+ des sites de fixation sur l'épithélium branchial. La perte des ions occasionne un transfert d'eau du fluide extracellulaire vers l'intérieur des cellules, ce qui cause une réduction du volume sanguin. Cette réduction donne lieu à une augmentation de l'hématocrite, donc de la viscosité du sang, qui se traduit par une défaillance du système circulatoire. La sensibilité des saumons à un faible pH au cours des divers stades de leur existence peut être résumée dans l'ordre décroissant suivant :

alevin > saumoneau > petit tacon > gros tacon > alevin vésiculé > oeuf

Une mortalité importante (19 - 71%) des alevins survient à un pH d'environ 5,0. La mort des saumoneaux survient également à un pH de 5,0, mais le taux est plus faible (1-5%). Le taux de mortalité des tacons et des saumoneaux est relativement élevé (72 – 100%) lorsque le pH diminue aux environs de 4,6 – 4,7 . La mort des œufs et des alevins vésiculés ne commence que lorsque le pH descend en deçà de 4,8.

Introduction

The Atlantic salmon resource in the Southern Upland (Atlantic Coast) area of Nova Scotia has been impacted by acid rain (Watt 1997). Salmon stocks are extinct in 14 rivers where mean annual pH is < 4.7 and severely impacted where mean pH is $4.7 - 5.0$. The impact of acid rain on stocks in 16 rivers where mean pH is $5.1 - 5.4$ has not been as great. Most rivers on the Atlantic coast show a seasonal variation in pH whereby pH declines in October and remains low (minimum pH is typically 0.5 units below the mean) until March when pH begins to increase reaching a peak in September (Watt 1997).

Why Salmon Die

Acidic rivers in Nova Scotia have low concentrations of Ca^{++} ($\leq 1 \text{ mg. L}^{-1}$) and high concentrations of dissolved organic carbon ($5 - 30 \text{ mg. L}^{-1}$) and total dissolved aluminum ($100 - 350 \text{ } \mu\text{g. L}^{-1}$) (Lacroix and Kan 1986). Dissolved organic matter, which is reflected by measurements of total dissolved organic carbon (DOC), is important because of its ability to chelate or bind to ionic forms of aluminum and form organic aluminum complexes (Lacroix and Kan 1986). Organic aluminum is the dominant form of aluminum in Nova Scotia rivers (mean 88%) and inorganic aluminum concentrations are usually $< 50 \text{ } \mu\text{g. L}^{-1}$ (Lacroix and Kan 1986). It is the inorganic form of aluminum which can be toxic to fish (Rosseland and Skogheim 1984; Fivelstad and Leivestad 1984; Lacroix and Townsend 1987; Lacroix et al. 1990). Lacroix and Townsend (1987) and Lacroix et al. (1990) have demonstrated that Al is not responsible for the mortality of salmon associated with the acidification of rivers in Nova Scotia. Increased H^+ ion concentrations coupled with the low Ca^{++} concentrations have caused the mortality of salmon in Nova Scotia.

The gill epithelium is the primary site of ionic regulation. In fresh water, the osmotic gradient of about 350 mosm. L^{-1} results in the passive diffusion of water into the blood and of ions out of the blood (Wood and McDonald 1982). Passive losses of ions are countered by active uptake of Na^+ (sodium) and Cl^- (chloride) from the water to maintain homeostasis. At or below pH 5.0, active influx of Na^+ and Cl^- is reduced and passive efflux is increased resulting in a net loss of both ions (Wood and McDonald 1982). The increased passive efflux of ions results from the displacement of Ca^{++} (calcium) from anionic sites on the paracellular channels of the gill epithelium by H^+ (acidity) (McDonald et al. 1989). An increase in the Ca^{++} content of water reduces the passive efflux of ions because the excess Ca^{++} replaces H^+ on the binding sites restoring membrane integrity. Under acidic, low Ca^{++} conditions, the net loss of Na^+ and K^+ (potassium) is approximately equal to the loss of Cl^- (Wood 1989). When losses of Na^+ or Cl^- or both exceed 30%, fish normally die within hours (Wood 1989). Under acidic conditions, there is also a loss of Ca^{++} , K^+ and $\text{PO}_4^{=}$ (phosphate) ions in the urine (Wood and McDonald 1982). The loss of ions results in a shift of water from the extracellular fluid volume to the intracellular fluid volume causing a reduction in blood volume (Wood and McDonald 1982). Red blood cells swell because of the reduced plasma osmotic pressure and additional red blood cells are released from the spleen. The reduced blood volume and increased hematocrit causes a doubling of blood viscosity and arterial pressure and death is a result of failure of the circulatory system (Wood and McDonald 1982; Wood 1989).

Acid Toxicity Levels for Salmon

Measurements of salmon mortality attributable to acidic brooks and rivers found in Nova Scotia are listed in Table 1. Also shown are measurements of DOC, total Al (aluminum) and Ca^{++} because of their importance in determining the toxic response of salmon to low pH. Atlantic salmon fry are the most sensitive stage to low environmental pH. Cumulative mortality of fry was 70.8% at a pH of 4.96 (Westfield River) and only 4.0% at a pH of 6.11 over a 53 day period (Lacroix et al. 1985). Decreases in the ionic content (Na^+ , K^+ , Cl^-) of the fry at pH 4.96 occurred during the third week and were coincident with death. Similarly, mortalities of fry ranging from 18.9 to 37.6% were observed in four consecutive years 14 to 28 days after swim-up when pH was about 5.0 (Mersey River) (Farmer et al. 1980). Mortality of fry was only 4.6 to 4.9% during the following two years when pH was increased to 6.5 (Farmer et al.

1980). Environmental pH must be increased to about 5.4 to reduce mortality during the fry stage (Goff, pers. comm.)¹.

Smolting salmon are also relatively sensitive to low environmental pH as low-levels of mortality are apparent at a pH of about 5.0 (Goff et al. 1993; Farmer et al. 1989). Farmer et al. (1989) exposed smolting salmon to pH levels of 4.6, 5.0 and 5.5 (Mersey River) over a 112-day period and observed mortalities of 72%, 2% and 0%, respectively. Plasma osmolarity and plasma Na^+ , Cl^- and Ca^{++} concentrations of the salmon at pH 4.6 were significantly lower than for salmon exposed to pH 5.0 or 5.5. A significant increase in the hematocrit and plasma protein concentration of the smolting salmon at pH 4.6 indicated that plasma volume was reduced and that death was attributable to failure of the circulatory system. Neither branchial Na^+ , K^+ ATPase activity nor tolerance to a salinity of 37.5‰ (parts per thousand) increased during the spring among salmon exposed to a pH of 4.6 as was observed for salmon exposed to pH 5.0 or 5.5 indicating that the process of smoltification is inhibited at low pH. Branchial Na^+ , K^+ ATPase activity in smolts, which peaks in the spring, is involved in ionic regulation and is indicative of greater salinity tolerance (Saunders and Henderson 1978). Goff et al. (1993) also observed mortality (1.7 to 5.3%) among smolting salmon during the overwinter period (about 105 days) when pH was 5.0 (Mersey River). Treatment of the water with CaCO_3 to increase the pH to 5.3 reduced the overwinter loss of smolting salmon to 0.6 to 0.7%.

Lacroix and Townsend (1987) exposed parr held in floating pens to different pH levels over a 54 day period during the autumnal episode of increasing acidity (Medway and Westfield rivers; Halfway and Moose Pit brooks). All parr died in the brooks where mean pH was 4.6 or 4.7 whereas no parr died where pH was 5.0 or 5.2. Small parr (< 10 cm) died more rapidly at low pH than large parr (≥ 10 cm). Plasma Na^+ and Cl^- decreased in the parr at low pH and the rate of ion loss was related to rate of pH change. Hematocrit increased and was also related to decreasing pH levels. This information indicates that parr exposed to pH levels ≤ 4.7 were unable to regulate ion loss and that death was attributable to circulatory failure.

More information is required on the effects of low environmental pH on alevins in Nova Scotia rivers. Mortality of alevins during two consecutive years ranged from 5.0 to 8.5% when pH of the Mersey River was about 5.0 (Farmer et al. 1980). Mortality of this magnitude is considered normal and not related to low environmental pH. Similarly, Peterson et al. (1989) and Peterson and Martin-Robichaud (1986) observed mortality rates of only 0 to 5% for alevins at pH levels of 5.0 and 5.1. The latter authors observed a 30% mortality of alevins at pH 4.5 while Haines (1981) indicates that mortality of alevins occurs within the pH range of 4.0 to 4.5. Accumulations of Ca^{++} , K^+ and Na^+ by alevins at a pH of 4.5 was reduced as was their dry weight at terminal yolk resorption compared with alevins at pH 5.1 or 6.8 (Peterson and Martin-Robichaud 1986). They concluded that high acidity affects ion uptake from the ambient water more than the yolk-to-larvae transfer of ions.

Hatching success of eggs planted after fertilization in the natural substrate of four acidic rivers (Westfield and Medway rivers; Halfway and Fifteen Mile brooks) was correlated with pH of the interstitial water (Lacroix 1985). The LL50 (lethal limits at which 50% die) for the eggs was calculated to be at a pH of 4.7. Peterson et al. (1980) observed that the median hatching date of salmon eggs exposed to different pH levels from the eyed stage was delayed by about six days at pH levels of 4.5 to 5.0 and that hatching did not occur when pH was 4.0 to 4.2. Perivitelline pH falls to ambient levels when eyed eggs are exposed to low pH. Peterson et al. (1980) suggested that these effects on hatching were due to inhibition of the hatching enzyme, chorionase. In contrast, eggs reared from fertilization at pH levels of 4.5 to 5.5 were not delayed in hatching (Peterson et al. 1980). Eggs reared from fertilization at a pH of 4.0 died in the early stages of development.

Sensitivity of the various Atlantic salmon stages to the acidic, dark coloured water of low calcium content found in Nova Scotia can be summarized as follows (decreasing order of sensitivity):

fry > smolt > small parr > large parr > alevin > egg

¹ Goff, T.R., Manager, Mactaquac Fish Culture Station.

Thus, significant mortality (19 - 71%) of fry occurs at a pH of about 5.0. Ambient pH must be increased to about 5.4 to reduce mortality at the fry stage. Mortality of smolts also occurs at a pH of 5.0 but the rate is lower (1.3 - 5.3%). Mortality of parr and smolts is relatively great (72 - 100%) when pH declines to the 4.6 - 4.7 range. Small parr (< 10 cm) die earlier during exposure to pH levels of 4.6 - 4.7 than do large parr (\geq 10 cm). Mortality of eggs and alevins does not begin until pH declines below 4.8 although mortality information for alevins exposed to low pH (4.0 - 4.8) in Nova Scotia rivers is lacking. Low environmental pH has been shown to interfere with ionic regulation at the alevin, fry, parr and smolt stages. The perivitelline pH of eyed eggs exposed to acidic water declines and hatching is delayed (pH 4.5 - 5.0) or prevented (pH 4.0 - 4.2) because the hatching enzyme, chorionase, is inhibited. In contrast, eggs exposed to various levels of pH (4.5 - 6.8) from the time of fertilization are not delayed in hatching.

References

- Farmer, G.J., T.R. Goff, D. Ashfield and H.S. Samant. 1980. Some effects of the acidification of Atlantic salmon rivers in Nova Scotia. Can. Tech. Rep. Fish. Aquat. Sci. No. 972. viii + 13p.
- Farmer, G.J., R.L. Saunders, T.R. Goff, C.E. Johnston and E.B. Henderson. 1989. Some physiological responses of Atlantic salmon (*Salmo salar*) exposed to soft, acidic water during smolting. Aquaculture 82: 229-244.
- Fivelstad, S. and H. Leivestad. 1984. Aluminum toxicity to Atlantic salmon (*Salmo salar* L.) and brown trout (*Salmo trutta* L.): Mortality and physiological response. Rep. Inst. Freshwater Res. Drottningholm 61: 69-77
- Goff, T.R., F.S. Baker, and D.L. MacDonald. 1993. Improved survival of Atlantic salmon parr by limestone filtration of an acidic hatchery water supply, p. 185-191. In: C.A. Staicer, M.J. Duggan and J.J. Kerekes [ed.] Kejimkujik watershed studies: monitoring and research five years after "Kejimkujik '88". Environment Canada Occasional Report No. 3.
- Haines, T.A. 1981. Effects of acid rain on Atlantic salmon rivers and restoration efforts in the United States, p. 57-63. In: L. Sochasky [ed.] Acid rain and the Atlantic salmon. ISAF Special Publication Series No. 10.
- Lacroix, G.L. 1985. Survival of eggs and alevins of Atlantic salmon (*Salmo salar*) in relation to the chemistry of interstitial water in redds in some acidic streams of Atlantic Canada. Can. J. Fish. Aquat. Sci. 42: 292-299.
- Lacroix, G.L., D.J. Gordon, and D.J. Johnston. 1985. Effects of low environmental pH on the survival growth and ionic composition of postemergent Atlantic salmon (*Salmo salar*). Can. J. Fish. Aquat. Sci. 42: 768-775.
- Lacroix, G.L., and K.T. Kan. 1986. Speciation of aluminum in acidic rivers of Nova Scotia supporting Atlantic salmon: a methodological evaluation. Can. Tech. Rep. Fish. Aquat. Sci. 1501: iii + 12p.
- Lacroix, G.L., and D.R. Townsend. 1987. Responses of juvenile Atlantic salmon (*Salmo salar*) to episodic increases in acidity of Nova Scotia rivers. Can. J. Fish. Aquat. Sci. 44: 1475 - 1484.
- Lacroix, G.L., D.J. Hood, C.S. Belfry, and T.G. Rand. 1990. Plasma electrolytes, gill aluminum content, and gill morphology of juvenile Atlantic salmon (*Salmo salar*) and brook trout (*Salvelinus fontinalis*) indigenous to acidic streams of Nova Scotia. Can. J. Zool. 68: 1270 - 1280.

- McDonald, D.G., J.P. Reader, and T.R.K. Dalziel. 1989. The combined effects of pH and trace metals on fish ion regulation, p. 221-242. In: R. Morris, E. Taylor, D.J.A. Brown and J.A. Brown [ed.] Acid toxicity and aquatic animals. Cambridge University Press, Cambridge.
- Peterson, R.H., P.G. Daye, and J.L. Metcalfe. 1980. Inhibition of Atlantic salmon (*Salmo salar*) hatching at low pH. Can. J. Fish. Aquat. Sci. 37: 770-774.
- Peterson, R.H., and D.J. Martin-Robichaud. 1986. Growth and major inorganic cation budgets of Atlantic salmon alevins at three ambient acidities. Trans. Am. Fish. Soc. 115: 220-226.
- Peterson, R.H., R.A. Bourbonnière, G.L. Lacroix, D.J. Martin-Robichaud, P. Takats and G. Brun. 1989. Responses of Atlantic salmon (*Salmo salar*) alevins to dissolved organic carbon and dissolved aluminum at low pH. Water, Air, and Soil Pollution 46: 399-413.
- Rosseland, B.O., and O.K. Skogheim. 1984. A comparative study on salmonid fish species in acid aluminum-rich water. II Physiological stress and mortality of one- and two-year-old fish. Rep. Inst. Freshwater Res. Drottningholm. 61: 186-194
- Saunders, R.L., and E.B. Henderson. 1978. Changes in gill ATPase activity and smolt status of Atlantic salmon (*Salmo salar*). J. Fish. Res. Board Can. 35: 1542-1546.
- Watt, W.D. 1997. The Atlantic Region acid rain monitoring program in acidified Atlantic salmon rivers; Trends and present status. DFO Can. Stock Assess. Sec. Res. Doc. 97/28.
- Wood, C.M., and D.G. McDonald. 1982. Physiological mechanisms of acid toxicity to fish. P. 197-226. In: R.E. Johnson [ed.] Acid Rain / Fisheries. Proceedings of an International Symposium on Acidic Precipitation and Fishery Impacts in Northeastern North America. American Fisheries Society, 5410 Grosvenor Lane, Bethesda, Maryland, 20814.
- Wood, C. 1989. The physiological problems of fish in acid waters. P. 125-152. In: R. Morris, E. Taylor, D. Brown, and J. Brown [ed.] Acid toxicity and aquatic animals. Cambridge University Press, Cambridge.

Table 1. Mortality of different stages of Atlantic salmon in acidic Nova Scotia rivers.

Stage	pH	DOC	Total Al Mg. L ⁻¹	Ca ⁺⁺	Exposure, days	Mortality, %
egg	4.64	>10.0	0.24	0.67	152-164	54.5 ^a
egg	4.72	>10.0	0.11	0.64	152-164	46.7 ^a
egg	4.77	>10.0	0.21	0.71	152-164	46.5 ^a
egg	4.92	>10.0	0.15	0.85	152-164	22.2 ^a
alevin	4.50	--	--	3.0	45	30.0 ^b
alevin	5.10	--	--	3.0	45	2.0-5.0 ^b
alevin	6.80	--	--	3.0	45	2.0-5.0 ^b
alevin	5.00	6.9	0.16	0.70	29-59	5.0-8.5 ^c
alevin	5.00	6.8	0.15	0.84	30-40	0 ^d
alevin	5.00	24.5	0.25	0.72	30-40	5.0 ^d
fry	4.96	>10.0	0.20	0.68	53	70.8 ^e
fry	6.11	>10.0	0.20	1.68	53	4.0 ^e
fry	5.00	6.9	0.16	0.70	14	18.9-37.6 ^c
fry	6.50	6.9	0.16	2.10	14	4.6-4.9 ^c
parr	4.60	18.4	0.25	1.02	20	100 ^f
parr	4.70	16.7	0.33	0.83	40	100 ^f
parr	5.00	11.7	0.26	0.79	54	0 ^f
parr	5.20	12.5	0.23	1.06	54	0 ^f
smolt	4.58	6.9	0.08	0.58	112	72.0 ^g
smolt	5.03	6.9	0.08	0.58	112	2.0 ^g
smolt	5.46	6.9	0.08	1.00	112	0 ^g
smolt	5.00	6.9	0.08	0.58	105	1.3-5.3 ^h
smolt	5.30	6.9	0.08	1.00	105	0.6-0.7 ^h

^aLacroix 1985.

^bPeterson and Martin-Robichaud 1986. Laboratory experiment performed with water of greater Ca⁺⁺ concentration.

^cFarmer et al. 1980.

^dPeterson et al. 1989.

^eLacroix et al. 1985.

^fLacroix and Townsend 1987.

^gFarmer et al. 1989.

^hGoff et al. 1993.