Table 1
Measured water chemistry parameters in the laboratory study examining the impacts of acid/Al on the physiology of Atlantic salmon parr and smolts

Exposure	pH	Al_{tot} ($\mu g\,l^{-1}$)	Al_i ($\mu g\,l^{-1}$)	Ca^{2+} ($mg\,l^{-1}$)	Na^+ ($mg\,l^{-1}$)
Control	6.40 ± 0.03 (20) (6.29–6.56)	33 ± 5 (6) (18–49)	20 ± 5 (5) (11–37)	1.4 ± 0.1 (12) (0.9–1.8)	2.2 ± 0.1 (12) (1.7–2.6)
Acid/Al	5.23 ± 0.04 (26) (4.99–5.42)	72 ± 3 (6) (61–83)	53 ± 4 (6) (43–68)	1.3 ± 0.1 (12) (1.0–1.8)	2.2 ± 0.1 (12) (1.7–2.8)

Values are mean ± S.E. of all measurements made throughout the 6-day study in both replicate tanks. Number of measurements made for each parameter is given in parentheses to the right. Range is given in parentheses below.

9 ± 2 and $26 \pm 10\,\mu g\,g^{-1}$ throughout the study and did not differ from $T = 0$ fish ($P > 0.25$, one-way ANOVA; Fig. 3). Gill Al of treated parr was significantly greater (6.5–19-fold) than control parr after 2 and 6 days (Fig. 3). Gill Al of treated parr increased by 69% between days 2 and 6 ($P < 0.05$; Fig. 3). Gill Al of treated smolts was significantly greater (12–15-fold) than control smolts after 2 and 6 days (Fig. 3). Gill Al of treated parr was significantly greater (two-fold) than treated smolts after 6 days.

3.2. Field exposure

Over the course of the study, pH ranged from 7.44 to 7.55 at the reference site (RR) and from 5.59 to 5.85 at the acid/Al-impacted site (BMB) (Table 2). Mean Al_{tot} concentrations were 36 ± 12 and $186 \pm 9\,\mu g\,l^{-1}$ and mean Al_i concentrations were 7 ± 4 and $53 \pm 7\,\mu g\,l^{-1}$ at RR and BMB, respectively (Table 2). Mean Ca^{2+} concentrations were 2.5 ± 0.1 and $0.7 \pm 0.1\,mg\,l^{-1}$, and mean Na^+ concentrations were 5.9 ± 0.3 and $8.8 \pm 0.4\,mg\,l^{-1}$ at RR and BMB, respectively (Table 2).

Plasma Cl^- levels were 136 ± 0.5 and 136 ± 0.8 mM for $T = 0$ parr and smolts, respectively (Fig. 4A). Plasma Cl^- levels of RR parr and smolts did not differ from $T = 0$ fish after either time-point ($P > 0.05$, one-way ANOVA; Fig. 4A). Parr and smolts held at BMB experienced losses in plasma Cl^- levels compared to RR fish, but impacts were greater in smolts after 2 days. Plasma Cl^- levels of BMB parr were significantly lower (9–30 mM) than RR parr after 2 and 6 days (Fig. 4A). Plasma

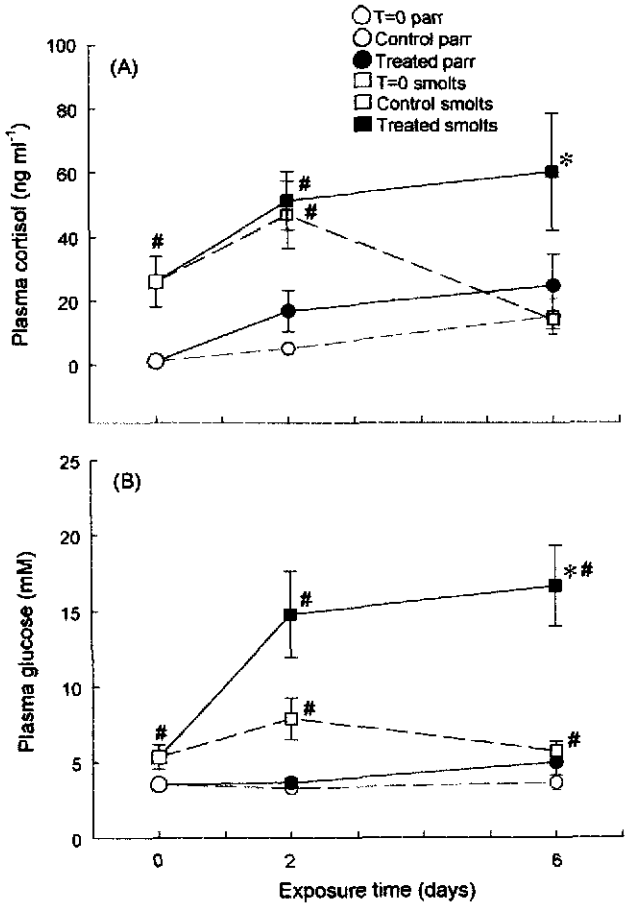

Fig. 2. Impacts of short-term laboratory exposure to acid/Al on the stress response of Atlantic salmon parr and smolts. Plasma cortisol (A) and plasma glucose (B) levels, of control and treated parr and smolts after 2 and 6 days. Values are mean ± S.E. ($n = 7$–10). An * indicates a significant difference between control and treatment within exposure time and life-stage (Duncan's; $P < 0.05$). An # indicates a significant difference between parr and smolt within a treatment and an exposure time (Duncan's; $P < 0.05$). Values at day 0 represent parr and smolts sampled prior to the start of the study ($T = 0$). Three-way ANOVA for plasma cortisol levels determined significant effects of treatment ($P = 0.002$) and lifestage ($P < 0.001$), and a significant timing/life-stage interaction ($P = 0.002$). Three-way ANOVA for plasma glucose levels determined significant effects of treatment ($P < 0.001$) and life-stage ($P < 0.001$).

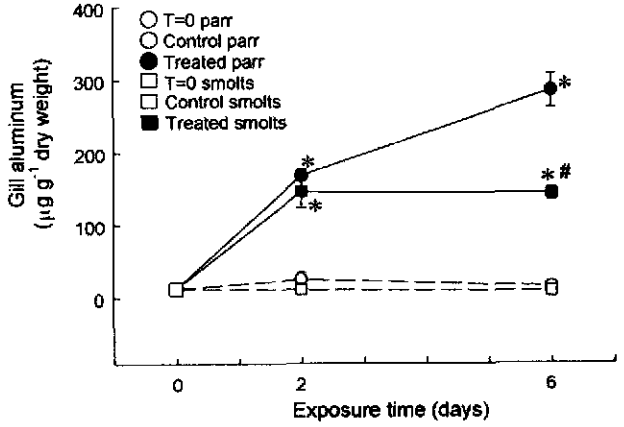

Fig. 3. Impacts of short-term laboratory exposure to acid/Al on gill Al accumulation of Atlantic salmon parr and smolts. Gill Al levels of control and treated parr and smolts after 2 and 6 days. Values are mean ± S.E. ($n = 8$–10). An * indicates a significant difference between control and treatment within exposure time and life-stage (Duncan's; $P < 0.05$). An # indicates a significant difference between parr and smolt within a treatment and an exposure time (Duncan's; $P < 0.05$). Values at day 0 represent parr and smolts sampled prior to the start of the study ($T = 0$). Three-way ANOVA for gill Al determined significant effects of treatment ($P < 0.001$) and life-stage ($P < 0.001$), and a significant treatment/time ($P = 0.002$) interaction.

Table 2
Measured water chemistry parameters of 2 tributaries of the West River during the 6-day cage study

Stream	pH	Al_{tot} ($\mu g\,l^{-1}$)	Al_i ($\mu g\,l^{-1}$)	Ca^{2+} ($mg\,l^{-1}$)	Na^+ ($mg\,l^{-1}$)
RR	7.47 ± 0.03 (4) (7.44–7.55)	36 ± 12 (4) (16–69)	7 ± 4 (3) (2–15)	2.5 ± 0.1 (4) (2.1–2.7)	5.9 ± 0.3 (4) (4.9–6.4)
BMB	5.75 ± 0.06 (4) (5.59–5.85)	186 ± 9 (4) (164–207)	53 ± 7 (3) (42–66)	0.7 ± 0.1 (4) (0.7–0.8)	8.8 ± 0.4 (4) (8.1–9.8)

Values are mean ± S.E. of all measurements made throughout the 6-day study. Number of measurements made for each parameter is given in parentheses to the right. Range is given in parentheses below.

Cl^- levels of BMB smolts were significantly lower (20–43 mM) than RR smolts after 2 and 6 days (Fig. 4A). Plasma Cl^- levels of BMB smolts were significantly lower (32 mM) than BMB parr after 2 days (Fig. 4A).

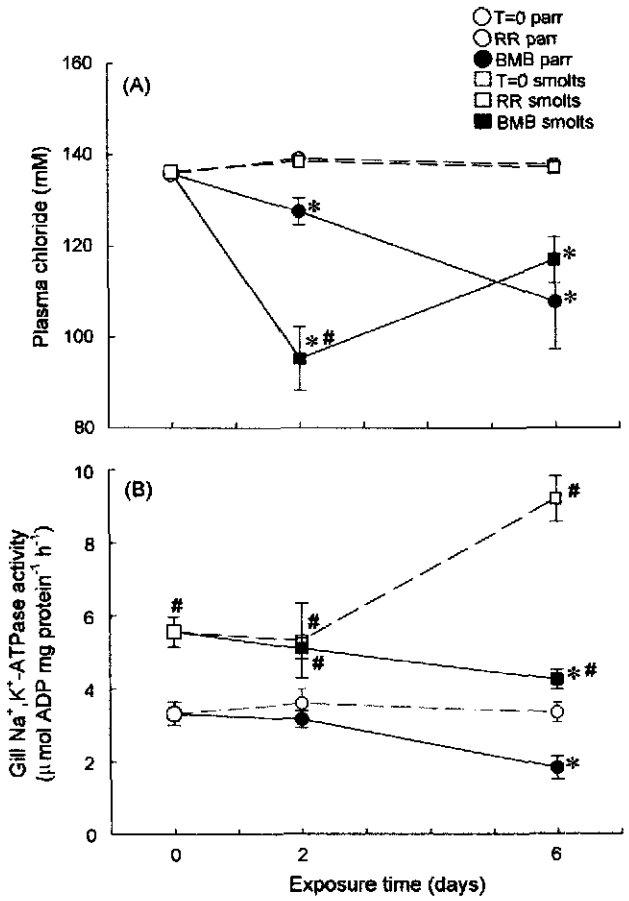

Fig. 4. Impacts of short-term field exposure to acid/Al on the ion regulatory ability of Atlantic salmon parr and smolts. Plasma Cl^- (A) and gill NKA activity (B) levels of parr and smolts held at a reference site (RR) and an acid/Al-impacted site (BMB) for 2 and 6 days. Values are mean ± S.E. (n = 4–16). An * indicates a significant difference between stream within an exposure time and life-stage (Duncan's; $P < 0.05$). An # indicates a significant difference between parr and smolt within a stream and an exposure time (Duncan's; $P < 0.05$). Values at day 0 represent parr and smolts sampled prior to the start of the study ($T = 0$). A significant cage effect was found for RR smolts at both time-points of exposure. For this group only, an outlier cage (one out of four replicate cages) was removed from the analysis, as it differed by ≥6 standard deviations. Three-way ANOVA for plasma Cl^- levels determined a significant stream effect ($P < 0.001$). Three-way ANOVA for gill NKA activity determined significant effects of stream ($P < 0.001$) and life-stage ($P < 0.001$), and significant stream/time ($P < 0.001$) and life-stage/time ($P = 0.008$) interactions.

Gill NKA activity of $T = 0$ parr and smolts was 3.3 ± 0.3 and 5.6 ± 0.4 μmol ADP mg protein $^{-1}$ h^{-1}, respectively (Fig. 4B). Gill NKA activity of RR parr was not significantly different from $T = 0$ parr after either time-point ($P > 0.55$, one-way ANOVA), whereas gill NKA activity of RR smolts was significantly greater (65%) than $T = 0$ smolts after 6 days (Fig. 4B). Gill NKA activity of parr was not affected by stream after 2 days, whereas after 6 days, gill NKA of BMB parr was significantly lower (45%) than RR parr (Fig. 4B). Gill NKA activity of smolts was not affected by stream after 2 days, whereas after 6 days, gill NKA activity of BMB smolts was significantly lower (54%) than RR smolts (Fig. 4B). Gill NKA activity of smolts was significantly greater (47%-2.8-fold) than parr in all groups throughout the study (Fig. 4B). As in the laboratory, the observed life-stage differences in NKA activity confirm the status of parr and smolts used in the field.

Plasma cortisol levels of parr and smolts were 8.1 ± 3.6 and 25 ± 4.6 ng ml^{-1}, respectively (Fig. 5A). Plasma cortisol levels of RR parr were significantly greater (6.9-fold) than $T = 0$ parr after 2 days, whereas plasma cortisol levels of RR smolts were not significantly different from $T = 0$ smolts after either time-point ($P = 0.19$, one-way ANOVA; Fig. 5A). Plasma cortisol levels of BMB parr were not affected by stream after 2 days, but were significantly greater (9.6-fold) than RR parr after 6 days (Fig. 5A). Plasma cortisol levels of BMB smolts were significantly greater (3.5–15-fold) than RR smolts after 2 and 6 days (Fig. 5A). Plasma cortisol levels were not significantly different between parr and smolts in any group throughout the study.

Plasma glucose levels of $T = 0$ parr and smolts were 4.2 ± 0.4 and 5.2 ± 0.4 mM, respectively (Fig. 5B). Plasma glucose levels of RR parr and smolts were not significantly different from $T = 0$ fish after either time-point ($P > 0.01$, one-way ANOVA; Fig. 5B). Plasma glucose levels of BMB parr were significantly greater (2.7–7.0-fold) than RR parr after 2 and 6 days (Fig. 5B). Plasma glucose levels of BMB smolts were significantly greater (3.7–4.4-fold) than RR smolts after 2 and 6 days (Fig. 5B). Plasma glucose levels of RR smolts were significantly greater (46–54%) than RR parr throughout the study (Fig. 5B).

Gill Al levels of $T = 0$ parr and smolts were 8.7 ± 3.2 and 13 ± 3.1 μg g^{-1}, respectively (Fig. 6). Gill Al levels of all RR fish were between 52 ± 10 and 92 ± 16 μg g^{-1} and were significantly greater (4.2–11-fold) than $T = 0$ fish after both time-points (Fig. 6). Gill Al of BMB parr was significantly greater (8.7–16-fold) than RR parr after 2 and 6 days (Fig. 6). Gill Al of BMB smolts was significantly greater (7.2–11-fold) than RR smolts after 2 and 6 days (Fig. 6). Gill Al of BMB parr was significantly greater (2.3-fold) than BMB smolts after 6 days (Fig. 6).

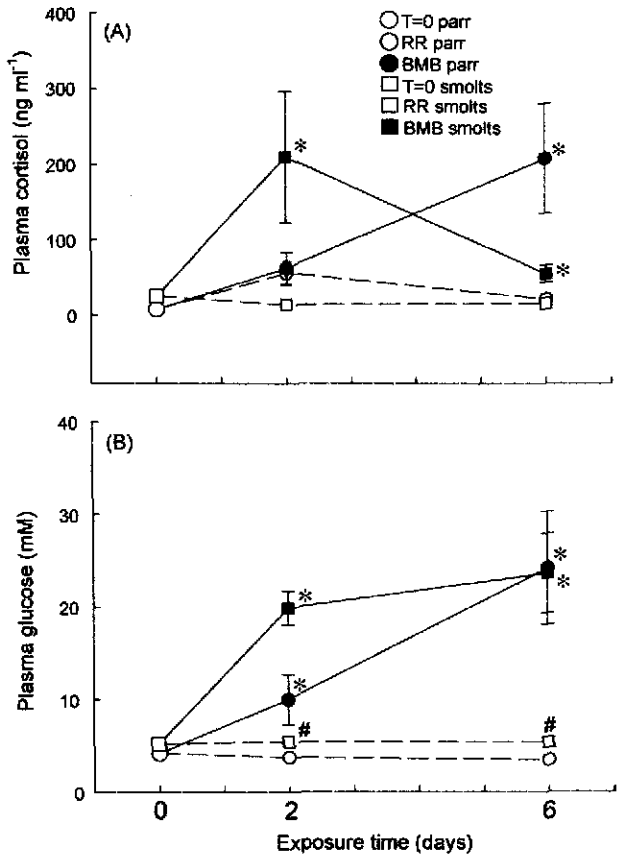

Fig. 5. Impacts of short-term field exposure to acid/Al on the stress response of Atlantic salmon parr and smolts. Plasma cortisol (A) and plasma glucose (B) levels of parr and smolts held at a reference site (RR) and an acid/Al-impacted site (BMB) for 2 and 6 days. Values are mean ± S.E. (n = 4–16). An * indicates a significant difference between stream within an exposure time and life-stage (Duncan's; $P < 0.05$). An # indicates a significant difference between parr and smolts within a stream and an exposure time (Duncan's; $P < 0.05$). Values at day 0 represent parr and smolts sampled prior to the start of the study ($T = 0$). A significant cage effect was found for RR smolts at both time-points of exposure. For this group only, an outlier cage (one out of four replicate cages) was removed from the analysis, as it differed by ≥6 standard deviations. Three-way ANOVA for plasma cortisol levels determined a significant stream effect ($P < 0.001$). Three-way ANOVA for plasma glucose levels determined significant effects of stream ($P < 0.001$) and life-stage ($P < 0.001$), and a significant stream/lifestage interaction ($P < 0.05$).

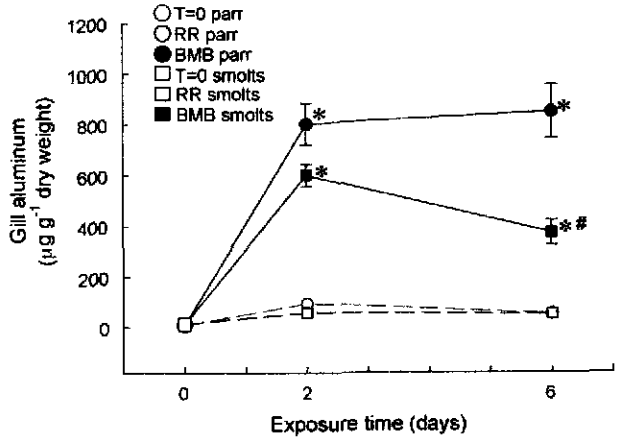

Fig. 6. Impacts of short-term field exposure to acid/Al on gill Al accumulation of Atlantic salmon parr and smolts. Gill Al levels of parr and smolts held at a reference site (RR) and an acid/Al-impacted site (BMB) for 2 and 6 days. Values are mean ± S.E. (n = 4–16). An * indicates a significant difference between stream within an exposure time and life-stage (Duncan's; $P < 0.05$). An # indicates a significant difference between parr and smolt within a stream and an exposure time (Duncan's; $P < 0.05$). Values at day 0 represent parr and smolts sampled prior to the start of the study ($T = 0$). A significant cage effect was found for RR smolts at both time-points of exposure. For this group only, an outlier cage (one out of four replicate cages) was removed from the analysis, as it differed by ≥6 standard deviations. Three-way ANOVA for gill Al determined significant effects of stream ($P < 0.001$) and life-stage ($P = 0.002$).

4. Discussion

To maintain ion homeostasis in freshwater, fish must combat the passive influx of water and efflux of plasma ions, which they accomplish by excreting a dilute urine and by taking up ions across the gill (Evans et al., 2005). Consequently, when the integrity or function of the gill is disturbed, loss of plasma Cl^- is observed. Laboratory exposure to short-term acid/Al impaired ion regulatory ability in smolts as indicated by reduced plasma Cl^- levels, but had no detectable impact on parr. In the field where conditions were potentially more severe (physical transfer to streams, lower ambient calcium concentrations), both life-stages held in an acid/Al-impacted stream (BMB) exhibited ion regulatory disturbance, but the pattern of ion loss differed between parr and smolts. Smolts held at BMB experienced large and rapid declines in plasma Cl^- levels after 2 days, with levels dropping to near the lethal threshold (<95–100 mM) reported for Atlantic salmon smolts (Staurnes et al., 1993). This was followed by partial recovery of plasma Cl^- levels after 6 days, suggesting that if smolts survive the initial toxic effects of acid/Al they may be able to recover ion homeostasis. However, acclimation to acid/Al would likely come as a cost to multiple aspects of physiology and behavior as has been shown in other salmonids (Wilson et al., 1994a, b, 1996). In contrast to smolts, parr held at BMB experienced only minor declines in plasma Cl^- levels after 2 days, clearly indicating lower sensitivity relative to smolts under these conditions. Parr at BMB continued to lose plasma Cl^- throughout the 6-day study, although levels did not approach the lethal threshold (<60 mM) reported for this life-stage (Lacroix and Townsend, 1987). The greater susceptibility of smolts to ion perturbations caused by acid/Al exposure is consistent with previous studies that have used either long-term or indirect approaches (Rosseland and Skogheim, 1984; Leivestad et al., 1987; Staurnes et al., 1993; Rosseland et al., 2001). The difference in short-term sensitivity demonstrated here has important implications, as the magnitude of initial ion losses may be closely related to fish survival during acid/Al exposure (Booth et al., 1988).

This study reports the effects of a single dose of acid and Al on Atlantic salmon parr and smolts. Based on previously published research and unpublished research from our own laboratory, it is likely that exposure to lower pH or higher Al would have resulted in more severe physiological consequences. Booth et al. (1988) exposed adult brook trout to three different pH levels (pH 5.2, 4.8, 4.4) with increasing total Al concentrations (0, 111, 333 μg l^{-1}) for up to 11 days and found that both mortality

and net ion losses increased with decreasing pH and increasing Al, with almost 100% mortality at the highest Al concentration at each pH level. We have found that Atlantic salmon smolts exposed to pH 5.2 with increasing Al_i concentrations (10, 41, 88, 140 $\mu g\,l^{-1}$) for 2 days suffer large losses in plasma ions when Al_i is 88 $\mu g\,l^{-1}$ ($Al_t = 107\ \mu g\,l^{-1}$) and 100% mortality when Al_i is 140 $\mu g\,l^{-1}$ ($Al_t = 179\ \mu g\,l^{-1}$) (Monette and McCormick, unpublished). However, in the same study, mortality and plasma ion losses of smolts exposed to 92 $\mu g\,l^{-1}$ of Al_t are decreased when pH is 5.6 and no impacts are observed when pH is >6.0. Together these results clearly demonstrate that the magnitude of physiological response (i.e. loss of ion regulatory ability) depends on the interaction of pH and Al levels.

In this study, plasma cortisol and glucose concentrations were measured as indicators of the stress response. Previous studies have shown that both parameters are affected by acid/Al exposure in salmonids (Brown et al., 1990; Waring et al., 1996; Kroglund et al., 2001). In our laboratory study, acid/Al-treated smolts experienced significant increases in plasma cortisol and glucose levels after 6 days, whereas acid/Al had no statistically significant effect in parr. In the field, acid/Al exposure caused elevations in plasma cortisol and glucose levels in both life-stages, but the time-course of impact differed between life-stages. Smolts held at BMB experienced large and rapid increases in both plasma cortisol and glucose levels, whereas increases in parr occurred more slowly. These observed life-stage differences reflect the patterns of plasma Cl^- losses in parr and smolts. Together, these results demonstrate that during short-term acid/Al exposure the stress response of smolts is more rapid than that of parr, suggesting a heightened sensitivity of the hypothalamic-pituitary-interrenal (HPI) axis in smolts. Smolts have been shown to have a heightened stress response during an acute handling stress, and it is thought that this may be important to survival during downstream migration and SW entry (Barton et al., 1985; Carey and McCormick, 1998). However, in this study, increased stress sensitivity of smolts may also be related to greater ion regulatory disturbance and/or other aspects of acid/Al exposure. There may be several advantages to a heightened HPI response including rapid mobilization of energy stores for damage repair and/or acclimation processes and increased respiratory capacity. However, negative consequences include decreased energy resources for other energetic demands such as downstream migration and predator avoidance, as well as negative long-term effects on growth and immunity.

We sought to examine the mechanisms(s) of increased smolt sensitivity by testing the hypothesis that a greater loss in gill NKA activity underlies increased sensitivity. Na^+,K^+-ATPase, an enzyme located in the basolateral membrane of the gill epithelium, plays a major role in teleost ion regulation in both FW and SW (Evans et al., 2005). In Atlantic salmon, gill NKA activity increases during the parr–smolt transformation and is directly related to the ability to maintain plasma ion homeostasis in SW (McCormick et al., 1998). In the present study, gill NKA activity levels of parr and smolts held at BMB for 6 days were lower than reference fish held at RR. In these fish declines in plasma Cl^- levels are likely due, in part, to an inhibition of ion uptake via reductions in gill NKA activity. Negative impacts on gill NKA activity are consistent with previous studies examining effects of long-term (weeks–months) acid/Al exposure on Atlantic salmon smolts (Staurnes et al., 1993; Magee et al., 2003). Decreased gill NKA activity may be attributed to increased chloride cell death via apoptosis and necrosis (Verbost et al., 1995), to the direct inhibition of enzyme activity by Al ions (Silva and Goncalves, 2003), or to the increased appearance of immature gill chloride cells with low levels of NKA protein (Wendelaar Bonga et al., 1990). Interestingly, gill NKA activity of smolts held at RR was significantly greater than $T=0$ smolts after 6 days which may reflect the seasonal rise in gill NKA activity that occurs in smolts during the spring. This was not observed in smolts held at BMB, indicating that exposure to acid/Al may inhibit this aspect of smolt development.

In both the lab and the field, we observed declines in plasma Cl^- levels, despite no detectable impact on gill NKA activity. Also, when negative impacts on gill NKA activity were observed (field study, 6 days), the magnitude of activity loss was similar for parr and smolts. This suggests that under the conditions present in this study, impaired ion regulatory ability and thus increased smolt sensitivity may not be explained by reductions in gill NKA activity (i.e. ion uptake) alone. Instead, it is likely that ion losses are due to the stimulation of passive ion efflux resulting from increases in paracellular permeability as has been found for other salmonids (Booth et al., 1988; Freda et al., 1991). However, we cannot rule out the possibility that there were significant impacts of acid/Al on the *in vivo* activity of gill NKA as the assay employed in this study is a measure of total NKA protein present in the gill epithelium, and is not a measure of how much of the enzyme is working *in vivo*. Also, recent work has indicated that the alpha 1a and 1b isoforms of NKA are differentially regulated in the gill during acclimation to seawater in salmonids (Bystriansky et al., 2006). Thus, it is possible that acid/Al has significantly altered NKA isoform expression and subsequent ion transporting capacity without affecting total NKA protein in the gill.

We hypothesized that another mechanism underlying increased smolt sensitivity during acid/Al exposure may be greater gill Al accumulation. We observed elevated gill Al in parr and smolts during both laboratory and field exposures to acid/Al. Previous studies have found that elevations in gill Al occur in salmonids during both short- and long-term acid/Al exposure (Neville, 1985; Lacroix and Townsend, 1987; Kroglund et al., 2001; Teien et al., 2004, 2006; Winter et al., 2005). It is also known that gill Al is directly related to water Al_i concentration (Booth et al., 1988; Kroglund et al., 2001; Teien et al., 2006). Interestingly, in both the lab and the field, gill Al accumulation was two-fold greater in parr compared to smolts after 6 days. This is consistent with previous findings that smaller fish accumulate more gill copper and may be due to the greater surface area to volume ratio present in smaller fish (Kamunde et al., 2001; Taylor et al., 2002). Alternatively, smolts may have a lower capacity for gill Al accumulation or are better able to eliminate Al from the gill (i.e. sloughing of mucus-bound Al). Finally, exposure to acid/Al has been shown to increase degeneration of chloride cells in the gills of fish (Wendelaar Bonga et al., 1990; Verbost et al., 1995; Jagoe and Haines, 1997). Since gill

chloride cells can accumulate Al (Youson and Neville, 1987), increased chloride cell death may represent a mechanism to eliminate Al from the gill. Given the greater chloride cell abundance in the gill epithelium of smolts, increased chloride cell death may explain lower gill Al levels of smolts. Regardless of the mechanism, smolts exhibited impaired ion regulatory ability at lower concentrations of gill Al than parr, indicating a lower threshold of gill Al to cause damage and/or elicit a physiological response. It is likely that this is a result of the reorganization of the gill to increase salt secretory capacity that occurs during the parr–smolt transformation. Together, these results provide further evidence for the increased sensitivity of smolts however greater gill Al accumulation does not appear to play a role in increased sensitivity.

The present study clearly demonstrates that smolts are more sensitive than parr to impacts of short-term acid/Al however this appears to be independent of gill NKA activity and gill Al accumulation. Instead, it may be speculated that increased smolt sensitivity results from morphological changes in the gill epithelium during smolting, including an increase in the number and size of chloride cells (McCormick et al., 1998), as well as ultrastructural changes in chloride cell associations with other cells in the gill (Pisam et al., 1988; Mizuno et al., 2000). In particular, Pisam et al. (1988) demonstrated that accessory cells linked to apical portions of chloride cells by shallow junctions were present in Atlantic salmon smolts but not in parr. This change in chloride cell morphology is thought to play a role in the paracellular pathway of Na^+ excretion in teleosts, and may be necessary for SW adaptation. It is likely that this ultrastructural change renders smolt gills more permeable and therefore more vulnerable to rapid ion efflux during episodic acid/Al exposure. Increased smolt sensitivity may also occur from changes in the presence of other ion transporters/channels in the gill epithelium (i.e. Na^+, K^+,$2Cl^-$ cotransporter and apical Cl^- channel) in preparation for SW entry and residence.

In the laboratory study, plasma Cl^- levels of control parr and smolts were significantly lower than fish sampled prior to the start of the study ($T=0$) indicating that there was an effect on plasma Cl^- independent of acid/Al treatment. This effect may be due to fish handling and/or transfer to smaller experimental tanks, as previous work has shown reductions in plasma Cl^- levels (15 mM) after an acute handling and confinement stress in Atlantic salmon (Carey and McCormick, 1998), and this response is well known to be part of the general stress response in fish (Wendelaar Bonga, 1997). Loss of plasma Cl^- may also be part of a physiological response to an acute reduction in ambient ion concentrations (including calcium, sodium and chloride) that was part of our experimental design. Hard water acclimated rainbow trout gills exhibit a greater increase in permeability than soft water acclimated gills during exposure to Al *in vitro* (Gundersen and Curtis, 1995). Increased membrane permeability might then allow for greater metal accumulation and this has been shown by Taylor et al. (2002) who found that rainbow trout previously acclimated to hard water exhibited greater gill copper accumulation than soft water acclimated fish. These effects are most likely due to changes in chloride cell size and density shown to occur during soft-water acclimation in salmonids (Greco et al., 1996; Uchida et al., 2002). Acute reductions in ionic strength in this study may thus have exacerbated observed impacts on physiological indices and gill Al concentrations. However, reductions in ambient ion concentrations are known to occur during increased discharge events in several rivers in both Maine and Nova Scotia, where acid/Al impacts are believed to be present (Lacroix and Townsend, 1987; Haines et al., 1990). Furthermore, we have found that prior acclimation to low ion water for 10 days does not prevent loss of ions or elevations in plasma cortisol and glucose levels in response to similar levels of acid/Al as used in the present study (Monette and McCormick, unpublished).

In conclusion, we have demonstrated by direct comparison that smolts are more sensitive than parr to impacts of short-term exposure to low pH and moderate Al_i in soft water. This is indicated by greater and more rapid losses in plasma Cl^- levels, heightened stress responsiveness, and a lower level of gill Al resulting in impaired ion regulation. We also provide evidence that under the conditions present in this study, increased smolt sensitivity appears to be independent of a reduction in gill NKA activity and greater gill Al accumulation. We suggest that smolts are more vulnerable to rapid ion losses as a result of the reorganization of the gill that has occurred during the parr–smolt transformation in preparation for seawater entry and residence. The heightened sensitivity of the smolt life-stage has substantial implications for salmon populations in regions affected by acid precipitation, as this critical developmental period occurs in the spring when episodic acidification due to seasonal rainfall and snowmelt may be greatest. Furthermore, compromised ion regulatory ability of smolts may have significant impacts on downstream migration and marine survival, which could in turn have population level effects.

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