Acid–base balance, ionic status, and renal function in resting and acid-exposed white suckers (*Catostomus commersoni*)

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Received October 6, 1982


Renal function was investigated in the stenohaline *Catostomus commersoni* held at water pH 7.3 for 5 days. Urine displayed remarkably low levels of Na\(^+\), Cl\(^-\), K\(^+\), Ca\(^{2+}\), and Mg\(^{2+}\) and was very acidic (mean pH 6.5). Renal electrolyte conservation was characterized by almost complete reabsorption of NaCl and to a lesser degree, K\(^+\), Ca\(^{2+}\), and Mg\(^{2+}\). Net H\(^+\) excretion was +11.2 μeqiv·kg\(^{-1}\)·h\(^{-1}\), resulting primarily from a high titratable acid content. The physiological consequences of exposure to ambient pH 4.3 for 4 days included disturbances in plasma acid–base status with relatively minor changes in both plasma ion levels and renal output. A mixed metabolic and respiratory acidosis developed, which was not compensated for by increased renal H\(^+\) excretion. Both plasma NaCl levels and \(E_{Na^+}\) decreased but \(E_{Cl^-}\) remained unchanged. \(E_{K^+}\) was correlated with plasma hyperkalemia. No changes in plasma levels or excretion of Ca\(^{2+}\) or Mg\(^{2+}\) occurred. These findings provide some physiological evidence supporting the contention that suckers are relatively acid tolerant. It is suggested that the copious amounts of mucus covering their gills may have served a protective role by retarding proton entry and limiting branchial ion loss.

**Introduction**

The occurrence of acid rain and the resultant acidification of many freshwater lakes and rivers has been recognized globally as an anthropogenic environmental problem. Studies of the physiological responses of adult fish to low ambient pH have demonstrated that disturbances in acid–base balance, ionoregulation, and respiratory function all occur, but their relative importance appears to be modulated by several factors: the severity of acid stress (i.e., pH < 4 versus pH > 4) and type of acid (HCl vs. \(H_2SO_4\)) as well as ambient calcium and carbon dioxide levels (see reviews by Fromm 1980; Spry et al. 1981; Haines 1981). However, few studies have monitored or accounted for these factors; notable exceptions have mainly focussed on an active, pelagic, euryhaline fish species, the hatchery-bred rainbow trout (*Salmo gairdneri*) (Neville 1979; McDonald et al. 1980; McDonald and Wood 1981; Milligan and Wood 1982). Since ecological and toxicological studies have established interspecies differences in tolerance to acid stress (Harvey 1979), it was of interest to determine whether this phenomenon also had a physiological basis.

While it is well established that fish regulate internal acid–base homeostasis by modulation of the strong ion difference (Heisler 1980), renal participation in acid–base regulation, relative to the gill, has just recently become appreciated (Wood and Caldwell 1978), and thus few species have been examined. The general pattern which has emerged is that the kidney appears to be more important in freshwater species which produce a copious urine (*Salmo gairdneri*, Wood and Caldwell 1978; *Ictalurus punctatus*, Cameron and Kormanik 1982) relative to marine forms whose urine output is low (*Parophrys vetulus*, McDonald et al. 1982).
Thus, the present study concentrated on the effects of low ambient pH on acid–base balance, ionic status, and renal function in the North American white sucker (Catostomus commersoni). This species was of interest because of its benthic habitat and stenohalinity, features which contrast markedly with those of the commonly renal physiology of suckers (Hickman 1965), a secondary aim was to characterize normal patterns of renal ion and acid–base output.

Materials and methods

Experimental animals and test conditions

Catostomus commersoni Lacépède (200–800 g; both sexes, mature and immature) were collected from Lake Bonavista, Calgary, Alberta, using baited cylindrical wire-mesh traps. Fish were maintained in running dechlorinated tap water (12–15°C) for 2 weeks to 2 months prior to use in experiments. While in holding tanks, they were fed commercial dry pellets and kept but starved 7 days prior to and during experimentation to remove the influence of diet on renal acid excretion (Wood and Caldwell 1978).

In preparation for experiments, fish were surgically fitted with caudal arterial or venous catheters for repetitive blood sampling (Watters and Smith 1973; Wilkes et al. 1981) and with bladder catheters for continual urine collection (Wood and Caldwell 1978). During surgery, the gills were irrigated with aerated, buffered, anaesthetic solution (MS 222, tricaine methane sulfonate; 1 : 10 000 dilution). Immediately after cannulation, fish were placed in individual, aerated, darkened incubation chambers, each receiving a continuous flow (500–1000 mL min⁻¹) of either dechlorinated tap water (control group) or recirculated water (acid group), both maintained at 12.5 ± 1°C. At least 36–48 h of recovery was allowed before beginning experimentation (Wood and Caldwell 1978).

Ion levels (Na⁺, K⁺, Cl⁻, Ca²⁺, Mg²⁺) in each water system were measured daily over the experimental period (Table 1). In the recirculated system (45 L/kg fish), water was deaerated prior to use, as outlined in McDonald et al. (1980). Water was acidified with sulfuric acid and maintained within pH range 4.0 to 4.5 by manual addition of 1.0 N H₂SO₄ or 1.0 N NaOH as required (Table 1); a Fisher Accumet 140A pH meter and combination electrode, calibrated with Fisher buffers (pH 4.0, pH 7.0) at the experimental temperature, were used.

Experimental protocol

Plasma acid–base and ionic status and renal output were monitored in suckers exposed to pH 7–7.5 (control group, mean weight = 371 ± 42 g; for blood data n = 11, for urine data n = 5) or pH 4–4.5 (acid group, mean weight = 316 ± 19 g; for blood data n = 13, for urine data n = 9). Following recovery from surgery, “day 0” values were measured in both experimental groups at water pH 7.3. Fish were subsequently either maintained at pH 7.3 (control group) or exposed to pH 4.3 (acid group) for 4 days (days 1 to 4 in figures).

Blood samples (600 µL) were withdrawn at 24-h intervals, usually at the end of two urine collection periods, and immediately analyzed for hematocrit, hemoglobin, plasma pH, total carbon dioxide content, and osmolality. Remaining plasma was stored (−10°C) for later ion analysis (Na⁺, Cl⁻, K⁺, Ca²⁺, Mg²⁺). Ion measurements on aliquots of fresh and frozen plasma obtained from the same fish exhibited no significant differences (p > 0.05). The bladder was drained continually by a siphon into covered flasks. At 12-h intervals, urine was collected and immediately analyzed for volume, pH, and titratable acid minus bicarbonate content. Remaining urine was stored (−10°C) for later analysis of total ammonia and ionic content.

Analytical procedures

Hemoglobin (Hb) was determined using the cyanmethemoglobin technique (Blaxhall and Daisey 1973) and Hycel reagents. Mean corpuscular hemoglobin concentration (MCHC) was calculated as the ratio of Hb/hematocrit (Ht).

Plasma pH and total carbon dioxide levels (PCO₂) were measured using Radiometer microelectrodes and methodology outlined in McDonald et al. (1980). Plasma bicarbonate levels (HCO₃⁻) and carbon dioxide tensions (PCO₂) were then estimated using the Henderson–Hasselbalch equation with pH and temperature-corrected pK’ values (Severinghaus et al. 1956) and temperature-corrected αCO₂ values (Severinghaus 1965).

ΔH⁺, representing the change in concentration of H⁺ ions (milliequivalents per litre) added to blood plasma by nonvolatile acids over time, was estimated from daily values (subscripts 1 and 2) of HCO₃⁻ and pH according to the formula:

\[ \Delta H_+ = (HCO_3^- - HCO_3^-) - \beta(pH_1 - pH_2) \]

where β represents the nonbicarbonate buffer capacity of true plasma (McDonald et al. 1980). The β value was calculated from the regression relationship, β = −23.44 (Ht) − 2.28, derived by Wilkes et al. (1981) for true plasma and the mean Ht over the time interval in question.

Urine titratable acid minus bicarbonate content (TA – HCO₃⁻) was determined as a single value in a double titration procedure (Hills 1973) using the control blood pH as the endpoint of the titration (i.e., absolute TA – HCO₃⁻; see McDonald and Wood 1981). In fish with no blood catheter, urine samples were titrated to the average blood pH of cannulated fish on day 0. Urine ammonia levels were assessed colorimetrically using the phenol–hypochlorite method (Solorzano 1969) and considered virtually equivalent to [NH₄⁺] (Cameron and Wood 1978). Net renal acid excretion (E_{H⁺}) was calculated as urine [NH₄⁺] plus [TA – HCO₃⁻] times urine flow rate (U_{FR}) (Hills 1973). Plasma osmolality (Osm) was determined with a Wescor Vapor Pressure Osmometer (Model 5100B). Urine ([Cl⁻]_u), plasma ([Cl⁻]_p), and water ([Cl⁻]_w) levels of chloride were measured by coulometric titration using a Digital Chloridometer (Buchler Model 4-2500); cation levels (Na⁺, K⁺, Ca²⁺, Mg²⁺) were measured, after appropriate dilution and swamping, by atomic absorption spectrophotometry (Jarrell Ash 850). Urine excretion rates ([E_{Na⁺}]_u) were calculated using the equation:

\[ E_{Na⁺} = ([Na⁺]_u × U_{FR}) \]

Renal ion clearances (C_{Na⁺}) were estimated by the equation:

\[ C_{Na⁺} = ([Na⁺]_u × U_{FR})/([Na⁺]_p) \]

Urine to plasma ratios (U/P) were also computed.

Statistical data analyses

The data presented in this study represent pooled values for two replicate control series and three replicate acid-exposure
series; data for fish with blocked catheters or ruptured bladders (determined at the end of experiments) have not been included. Values throughout have been expressed as means ± SEM, unless otherwise stated. Differences between mean day values throughout have been expressed as means (determined at the end of experiments) have not been included. Repeated measures values of control and acid groups at water pH 7.3 were tested using a Student’s two-tailed t-test (unpaired design); significance determined from this analysis has only been reported in the text. Possible relationships between some variables were evaluated by computing simple Pearson’s correlation coefficients.

Control group data were analyzed according to the multiple comparison procedure of Dunnett (1955) which dictated the use of the residual mean square, derived from a single-factor repeated measures ANOVA, as an estimate of the error variance, for computation of a Dunnett’s t-statistic (critical values in Winer 1971). Acid group data were analyzed in an identical manner in cases where sampling effects in the control group were not significant (p > 0.05). In figures, one asterisk (*) represents significance (p < 0.05) demonstrated with this within-group comparison of means. A two-factor repeated measures ANOVA (Winer 1971) was employed on both control and acid group data (days 1 to 4 only) for any variables where significant sampling effects were demonstrated. Time-dependent acid responses were then tested using an unpaired Student’s two-tailed t-test. In figures, two asterisks (**) represent significance (p < 0.05) shown with this between-group comparison of means. Data computations were performed using BMDP (Dixon and Brown 1979) and SPSS (Hull and Nie 1979) computer program packages.

**Results**

Ion levels and pH of the control and acid water systems are listed in Table 1. The marked difference in [Na⁺] between the two systems was attributed to the gradual addition of 1.0 N NaOH for water pH control. The higher levels of other ions in the acid relative to the control water further suggested that some net body ion loss probably ensued during acid exposure.

Cannulation and repetitive blood sampling had some effect on blood data; minor but statistically significant changes occurred in Ht, Hb (data not included), pH, pAO₂, [HCO₃⁻], [Cl⁻], [Ca²⁺] (Fig. 1), Osm, [Cl⁻], and [Ca²⁺] (Fig. 2). There were no significant differences between any of the repeated samples taken over 5 days for any of the urinary variables (Figs. 3, 4) with the exception of [H⁺] (Fig. 3B).

The regression equation relating urine flow rate and inulin clearance, Cᵢᵣᵣ = (Uᵢᵣ − 0.010)/0.64 (r = 0.92), derived by MacKay and Beatty (1968), was used to estimate Cᵢᵣᵣ; a mean value of 4.2 ± 0.29 was calculated from data for fish with both blood and urinary catheters (day 0 values, n = 8). U/P ratios and renal ion clearances were estimated (Table 2) and the magnitude of the latter compared with Cᵢᵣᵣ (Pitts 1974), to gain some information on renal tubular processes (i.e., reabsorption or secretion; see Discussion). To test for relationships between urinary variables, the control group data (10 samples per fish, n = 5, 50 complete data sets) were subjected to simple correlation analysis and results are shown in Table 3 (see Discussion).

**Table 1. Ion concentrations (milliequivalents per litre) and pH of the control and acid water systems.** Water samples were collected daily over the experimental period. Values are means ± SEM, (n = 20)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control system</th>
<th>Acid system</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.30±0.04</td>
<td>4.30±0.04</td>
</tr>
<tr>
<td>[Na⁺]</td>
<td>0.051±0.001</td>
<td>0.640±0.040*</td>
</tr>
<tr>
<td>[K⁺]</td>
<td>0.013±0.001</td>
<td>0.041±0.010</td>
</tr>
<tr>
<td>[Cl⁻]</td>
<td>0.040±0.008</td>
<td>0.215±0.010</td>
</tr>
<tr>
<td>[Ca²⁺]</td>
<td>1.29±0.05</td>
<td>1.78±0.04</td>
</tr>
<tr>
<td>[Mg²⁺]</td>
<td>0.86±0.10</td>
<td>0.92±0.012</td>
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</table>

*1.0 N NaOH added for pH control

Exposure to ambient pH 4.3 resulted in a slight transitory elevation of both Ht (70%) and Hb (44%), from initial control values of 17.0 ± 1.5% and 4.23 ± 0.04 g%, respectively, and thus, MCHC (0.249 ± 0.001 g·mL⁻¹) remained unchanged; over days 2 to 4, Ht and Hb gradually declined, probably as a consequence of repetitive blood sampling, as seen in the control group, rather than acid stress (data not included).

Plasma acid-base responses were progressive, and hence, by day 4, significant reductions had occurred in pH, (0.24 units), pAO₂, (0.26 units), [HCO₃⁻], (25%), and [HCO₃⁻], (28%) (Figs. 1B, 1C). Plasma PSCO₂, (35%) and PVCO₂, (21%) increased concomitantly (Fig. 1A). ΔH⁺ was relatively constant over the 4 days with a total accumulation of 4.3 mequiv·L⁻¹.

Changes in plasma ion levels were minor (Fig. 2) in comparison with acid-base responses (Fig. 1). [Na⁺] declined on day 2 with a net change of 10.3 mequiv·L⁻¹ by day 4 (Fig. 2B). This pattern reflected Osm which decreased by 6% over the 4 days (Fig. 2A). The depression in [Cl⁻] (Fig. 4A) in the acid system was not significant as similar changes occurred in the control group (Fig. 2C). [K⁺] increased on day 2, stabilizing thereafter, with an overall rise of 0.36 mequiv·L⁻¹ (Fig. 2D). No significant decrease in either [Ca²⁺] or [Mg²⁺] occurred (Figs. 2E, 2F), suggesting that the drop in Osm was mainly due to loss of other ions (i.e., Na⁺, Cl⁻, HCO₃⁻) rather than to water gain.

Renal output varied markedly with time (Figs. 3, 4). Uᵢᵣᵣ decreased by 10% on day 2, returning to control rates by day 4 (Fig. 3A). The transitory elevation in Eᵢᵣᵣ (Fig. 3C) and sustained rise in Eᵢᵣᵣ (Fig. 3D) were not significant and hence, Eᵢᵣᵣ was unchanged (Fig. 3E). [H⁺] (measured as pH) exhibited a gradual increase which was significant on day 4 (Fig. 3B). Eᵢᵣᵣ increased by 45% on day 2 (Fig. 4A) in concert with Uᵢᵣᵣ (Fig. 3A). Eᵢᵣᵣ displayed a peak increase of 120% over days 2 and 3 but returned to con-
TABLE 2. Urine concentrations, \(U/P\) ratios, and renal clearance rates of ions (\(x\)) in *Catostomus commersoni* maintained in water pH 7.3 at 12.5 ± 1°C. Values are means ± SEM with \(n\) in parentheses for day 0 measurements of control and acid groups combined (two 12-h urine collection periods averaged; single blood sample).

<table>
<thead>
<tr>
<th>(\left[x\right]_u, \text{ mequiv. L}^{-1})</th>
<th>(\text{Na}^+)</th>
<th>(\text{Cl}^-)</th>
<th>(\text{K}^+)</th>
<th>(\text{Ca}^{2+})</th>
<th>(\text{Mg}^{2+})</th>
<th>(\text{H}^+)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(14)</td>
<td>2.15 ± 0.29</td>
<td>2.09 ± 0.32</td>
<td>1.67 ± 0.23</td>
<td>1.47 ± 0.23</td>
<td>1.21 ± 0.10</td>
<td>29.33 ± 4.54*</td>
</tr>
<tr>
<td>(U/P)</td>
<td>(8)</td>
<td>0.018 ± 0.002</td>
<td>0.022 ± 0.005</td>
<td>0.920 ± 0.117</td>
<td>0.381 ± 0.089</td>
<td>0.726 ± 0.059</td>
</tr>
<tr>
<td>(C_r, \mu\text{equiv. kg}^{-1} \cdot \text{h}^{-1})</td>
<td>(8)</td>
<td>0.048 ± 0.007</td>
<td>0.056 ± 0.011</td>
<td>2.423 ± 0.358</td>
<td>1.016 ± 0.217</td>
<td>1.947 ± 0.162</td>
</tr>
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</table>

\(\left[H^+\right]_u = 1 \times 10^{-4} \text{M}.\)

TABLE 3. Correlations between several urine variables in *Catostomus commersoni*. The analysis was performed on the control group data (10 samples per fish, \(n = 5\); 50 complete data sets). Only those correlation coefficients significant at the 5% level are shown. (*, \(p < 0.01\)). With the exception of urine flow, concentrations of each of the variables (e.g., [\(\text{Na}^+\]) were used in the analysis.

<table>
<thead>
<tr>
<th>Flow</th>
<th>(\text{Na}^+)</th>
<th>(\text{Cl}^-)</th>
<th>(\text{K}^+)</th>
<th>(\text{Ca}^{2+})</th>
<th>(\text{Mg}^{2+})</th>
<th>(\text{H}^+)</th>
<th>(\text{NH}_4^+)</th>
<th>(\text{TA} - \text{HCO}_3^-)</th>
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<tbody>
<tr>
<td>(\text{Na}^+)</td>
<td>—</td>
<td>0.543*</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<tr>
<td>(\text{Cl}^-)</td>
<td>—</td>
<td>—</td>
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<td>—</td>
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<tr>
<td>(\text{K}^+)</td>
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<tr>
<td>(\text{Ca}^{2+})</td>
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<td>(\text{Mg}^{2+})</td>
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<tr>
<td>(\text{H}^+)</td>
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<td>(\text{NH}_4^+)</td>
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<tr>
<td>(\text{TA} - \text{HCO}_3^-)</td>
<td>—</td>
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Control values by day 4 (Fig. 4C). \(E_{\text{Cl}^-}\) (Fig. 4B), \(E_{\text{Ca}^{2+}}\) (Fig. 4D), and \(E_{\text{Mg}^{2+}}\) (Fig. 4E) were all not significantly affected by acid exposure.

These physiological responses were further accompanied by a progressive accumulation of mucus on sucker gills (visual observation). All fish survived ambient pH 4.3 for 4 days.

**Discussion**

**Normal blood and renal physiology**

Plasma acid–base and ion levels in cannulated suckers are in agreement with those reported by Wilkes et al. (1981); Hb, Osm, and [\(\text{Mg}^{2+}\)]\(_p\) have not been previously measured but lie within the range found in other freshwater teleosts (Hickman and Trump 1969). The marked difference in [\(\text{Ca}^{2+}\)]\(_p\) between the control and acid groups on day 0 (Fig. 2E) was attributed to the greater proportion of mature females in the control group; mature females had significantly higher [\(\text{Ca}^{2+}\)]\(_p\) than males \((p < 0.05)\), as found by Beamish et al. (1975).

The nephron of a freshwater teleost kidney is typified by a glomerulus, ciliated neck segment, first and second proximal tubular segments, a distal segment, and a collecting tubule and duct, but lacks a loop of Henle (Hickman and Trump 1969). The bladder has been shown to function as part of this complex, at least in euryhaline fish species (e.g., Renfro 1975). Since the catheterization technique employed in the present study rendered the urinary sphincters incompetent so that fluid drained from the bladder as soon as it was produced, any modification in urine volume or composition by transport mechanisms in the bladder wall (e.g., Renfro 1975; Beyenbach and Kirschner 1975) would therefore not be seen. Thus, the following discussion pertains to the features of urethral urine in stenohaline white suckers.

Urine flow was quite variable with time (Fig. 3A) and probably reflected intermittent glomerular filtration (Hickman 1965; MacKay and Beatty 1968). The inverse relationship between urine flow and electrolyte levels (Table 3) implied that in suckers, renal ion absorption and water movement were probably dissociated processes. Urine [\(\text{Na}^+\)] and [\(\text{Cl}^-\)] were comparatively low (cf. euryhaline species, Kobayashi and Wood 1980; Cameron 1980) and even lower than those reported by Hickman (1965) for this species (larger specimens). Movements of \(\text{Na}^+\) and \(\text{Cl}^-\) were probably coupled to some extent, as shown by the significant correlation...
Fig. 1. Blood acid-base status (means ± SEM) in white suckers held at pH 7.3 (day 0) followed by 4 days of exposure to pH 4.3 (-) or maintained at pH 7.3 throughout (--), for arterial and venous samples (acid group arterial samples: ●, n = 8; control group arterial samples: ○--○, n = 6) (acid group venous samples: ■, n = 5; control group venous samples: □--□, n = 5). Asterisks represent significance (p < 0.05) obtained either by a "within group" (*) or "between group" (**) comparison of means (see text for details). For the sake of clarity, significant changes in PCO2 were not illustrated but occurred in arterial (days 2, 3, 4) and venous samples (day 4) in the acid group only (*).

between respective concentrations (Table 3) and equimolar excretion rates (Figs. 4A, 4B). Since U/P ratios were well below 1, and CNA+ and CCl much less than Cin (Table 2), almost complete reabsorption of both ions probably occurred from the filtrate (cf. Hickman 1965). Patterns of renal K+ output were at variance with those noted by Hickman (1965); [K+]u, the U/P ratio, and CK+ were all lower (Table 2), findings which favored net tubular reabsorption of K+ (Ck+ < Cin), although tubular secretion cannot be entirely discounted. The inverse relationship between [K+] and [Na+] found by Hickman which implicated countertransport of these ions via an Na+ /K+ exchanger, was also not observed (Table 3). No such correlation has been reported in either Salmo gairdneri (Wood and Randall 1973) or Ictalurus punctatus (Cameron 1980). While the reasons for these discrepancies are uncertain, they may be related to the shorter sampling regime (i.e., 24 h) and (or) small number of fish employed in Hickman's study.

Urinary divalent cation levels (Table 2) were quantitatively similar to those reported in Salmo gairdneri (Wood and Randall 1973) but twofold less than in Salvelinus fontinalis (Hammond 1969). The coupled movement of Ca2+ and Mg2+ implicated in studies of euryhaline species (Miles 1971; Wood and Randall 1973), was not observed (Table 3), suggesting that in suckers, independent renal processes probably regulate movement of these ions. Since U/P ratios were less than one and renal clearances less than Cin (Table 2), some tubular reabsorption of both Ca2+ and Mg2+ probably occurred. Calcium is strongly reabsorbed against its concentration gradient in the euryhaline Channa argus (Oguri 1968) but apparently follows tubular volume reabsorption in Anguilla rostrata, either by simple diffusion or across an electrochemical gradient (Fenwick 1981).

The sucker kidney further demonstrated a definite
capacity to acidify urine (mean pH 6.5, Table 2), at variable rates (Fig. 3). Renal excretion of H⁺ apparently depends upon the availability of urinary buffers such as titratable acids (TA – HCO₃⁻; mainly dibasic phosphates) and ammonia (NH₃ + H⁺ → NH₄⁺; Heisler 1980). Since TA – HCO₃⁻ largely accounted for the observed pattern of net acid rather than base excretion (i.e., 77%; Fig. 3), its role in defending the normal acid–base balance of suckers probably superseded that of NH₄⁺. Moreover, the level of urine pH appeared to have contributed more substantially to the generation of TA – HCO₃⁻ than to NH₄⁺, as shown by the strong positive correlation between urine [H⁺] and [TA – HCO₃⁻] (Table 3). Few interrelationships between renal acid and electrolyte levels were apparent; in fact, the positive correlation between either [Na⁺] or [K⁺] and various components of urine acidity (Table 3) was exactly opposite to that expected if Na⁺/H⁺ or K⁺/H⁺ exchange processes regulated urine pH (mammalian kidney, Pitts 1974). Relationships of this nature have also not been reported in euryhaline species such as Salmo gairdneri (Kobayashi and Wood 1980) and Ictalurus punctatus (Cameron 1980). In contrast with suckers, however, these species normally excrete a more alkaline urine (pH > 7.0) with NH₄⁺ as the predominant component of $E_{\text{H}^+}$.

The mechanisms of urinary acidification and ion transport in teleosts clearly deserve further investigation.

Effects of acid exposure

The physiological consequences of exposure to ambient pH 4.3 included disturbances in both plasma acid–base status (Fig. 1) and to a lesser degree plasma ion levels (Fig. 2), findings which qualitatively agree with those found in other fish species maintained under comparable ambient pH and [Ca²⁺] (Salmo gairdneri, McDonald and Wood 1981; Cyprinus carpio, Ultsch et
al. 1981); however, the nature of the plasma pH depression was different. The observed respiratory acidosis (i.e., CO₂ excess; Fig. 1A) was atypical and implicated some limitation in CO₂ diffusion and (or) catalyzed HCO₃⁻ dehydration (Cameron 1979) at the gill. Endogenous (i.e., lactic acid; Krishna Murthy et al. 1981) and exogenous factors (i.e., H⁺ influx or base efflux, McDonald and Wood 1981; Ultsch et al. 1981) as well as changes in the relative intensities of the counterion exchanges across the gill (i.e., Na⁺/H⁺ or NH₄⁺ and Cl⁻/HCO₃⁻ or OH⁻, Maetz 1973), may all have contributed to the observed metabolic acidosis (i.e., bicarbonate depletion; Fig. 1B). Since an increase in gill H⁺ permeability has been documented at low ambient pH (McWilliams and Potts 1978), enhanced branchial H⁺ influx would be the most plausible explanation for the observed acidosis. However, without measurements of ion flux rates and indices of anaerobiosis, other causal factors (as above) cannot be entirely discounted.

The role of the sucker kidney in compensating for the plasma acidosis was negligible since the rate of net H⁺ excretion was virtually unchanged by acid exposure (Fig. 3E). By contrast, at least 50% of the incoming proton load was excreted by the kidney in acid-exposed Salmo gairdneri (McDonald and Wood 1981). The extent of renal participation in acid–base homeostasis in other fish species, however, appears to be dictated not only by the prevailing environmental conditions but also by the nature (metabolic or respiratory) of the acid–base disorder. For example, Cameron and Kormanik (1982) found that in Ictalurus punctatus, the kidney clearly adjusted for the blood pH disturbance resulting from infusions of both NH₄Cl and NH₄HCO₃, yet its response to either mineral acid infusion (HCl) or environmental hypercapnia was relatively minor (Cameron 1980). Thus, it is conceivable that the contribution of the sucker kidney may become significant, perhaps under conditions of more severe external acid stress.

Some compensatory adjustments in renal output were notable (Figs. 3A, 4A, 4C). Firstly, a reduction in the total permeability of the body surface to water was suggested from the drop in Uᵦᵣₑᵦₑᵦₑᵦₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑطور هر beast it must have provided some compensatory adjustments in renal output were notable (Figs. 3A, 4A, 4C). Firstly, a reduction in the total permeability of the body surface to water was suggested from the drop in Uₑᵦᵦₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑₑelectron charge and acid–base regulation in the freshwater catfish, Ictalurus punctatus. J. Exp. Biol. 86: 171–183.


Acknowledgements

We wish to thank L. Linton and B. Smith for advice on data analysis and computer programming. The staff of Lake Bonavista Recreation Centre helped greatly with the collection of fish. Financial support was provided by an operating grant (A5762) to B. R. McMahon from the Natural Sciences and Engineering Research Council of Canada and a Research Allowance to H. Höbe from the Alberta Heritage Fund for Medical Research. C. M. Wood’s travel was supported by an NSERC strategic operating grant in environmental toxicology. H. Höbe was supported by an NSERC postgraduate scholarship.


