

---

# Failure of Low-Velocity Swimming to Enhance Recovery from Exhaustive Exercise in Largemouth Bass (*Micropterus salmoides*)

Cory D. Suski<sup>1,\*</sup>

Steven J. Cooke<sup>2,3</sup>

Bruce L. Tufts<sup>1</sup>

<sup>1</sup>Department of Biology, Queen's University, Kingston, Ontario K7L 3N6, Canada; <sup>2</sup>Institute of Environmental Science and Department of Biology, Carleton University, 1125 Colonel By Drive, Ottawa, Ontario K1S 5B6, Canada; <sup>3</sup>Center for Aquatic Ecology and Conservation, Illinois Natural History Survey, Champaign, Illinois 61820

Accepted 8/21/2006; Electronically Published 11/29/2006

---

## ABSTRACT

This study was intended to discover whether forcing largemouth bass (*Micropterus salmoides*) to swim at 0.5 body lengths/second following exercise would expedite recovery relative to fish recovered in static water. Exercise resulted in a suite of physiological disturbances for largemouth bass that included a depletion of anaerobic energy stores, an accumulation of lactate, and increased cardiac output. At 1 h following exercise, exhaustively exercised largemouth bass forced to swim exhibited expedited recovery relative to fish in static water, evidenced by lower concentrations of lactate in white muscle, elevated concentrations of phosphocreatine in white muscle, and reduced concentrations of glucose in plasma. By 4 h post-exercise, largemouth bass forced to swim during recovery exhibited signs of physiological disturbance that were absent in fish recovered in static water. These signs of disturbance included a loss of osmotically active particles from plasma, elevated lactate in plasma, reductions of phosphocreatine in white muscle, and increased cardiac output. These results are discussed in relation to the body of work with salmonid fishes showing physiological benefits to recovering fish in flowing water.

## Introduction

Exhaustive exercise is a common occurrence for wild fish and is important for activities such as migration, food acquisition, and predator avoidance. Exhaustive exercise is primarily driven by white muscle and results in the anaerobic consumption of energy stores such as phosphocreatine (PCr) and adenosine triphosphate (ATP), the production of lactate, and numerous other ionic, osmotic, and cardiac disturbances and typically can only be maintained for short periods of time (Wood 1991; Wang et al. 1994; Kieffer 2000). In contrast, sustained (i.e., low velocity) exercise is characterized by aerobic respiration and little physiological disturbance and is generally fueled by glucose oxidation in red muscle (Randall 1982; Wood 1991; West et al. 1993). Recovery from exhaustive exercise and the return of physiological parameters to a normal resting state is a lengthy process for fish, often taking in excess of 12 h to complete (Wood 1991; Wang et al. 1994; Milligan 1996). The duration of recovery is important because it may dictate the next time that high-intensity anaerobic swimming activity can occur.

Recent work has suggested that forcing exhausted fish to swim at low velocities (i.e., using anaerobic respiration) may reduce the time required to recover from exhaustive exercise (Meyer and Cook 1996; Milligan et al. 2000; Farrell et al. 2001a). Milligan et al. (2000), for example, demonstrated that slow swimming expedited the recovery of certain physiological parameters in rainbow trout (*Oncorhynchus mykiss*, Walbaum) relative to fish recovered in static water. Similarly, Farrell et al. (2001b) used net pens adjacent to commercial fishing vessels that forced exhausted coho salmon (*Oncorhynchus kisutch*, Walbaum) to swim during recovery and showed decreased recovery times relative to still water. Although the exact mechanism responsible for this enhanced recovery is not known, it is hypothesized that swimming prevents production of the stress hormone cortisol, and the presence of cortisol may impede recovery (Milligan 2003). This practice, however, has not yet been tested on nonsalmonid fishes.

Largemouth bass (*Micropterus salmoides*, Lacepède) are top predators in many aquatic ecosystems and feed by ambushing prey. Overall, these fish are generally considered to be quiescent relative to salmonids and are not regarded for their high-intensity activity or endurance. In fact, estimates of largemouth bass swimming speeds are roughly half those of salmonids (e.g., Beamish 1978; Cooke et al. 2001), emphasizing the differences in lifestyles between the two groups of fishes. From an applied perspective, largemouth bass are the most popular target for

---

\* Corresponding author. Present address: Queen's University Biological Station, Queen's University, Department of Biology, Kingston, Ontario K7L 3N6, Canada; e-mail: suskic@biology.queensu.ca.

recreational anglers during competitive angling events (Kerr and Kamke 2003). Studies have shown that the primary source of physiological disturbance for largemouth bass angled (Gustaveson et al. 1991) and caught during live-release angling tournaments is anaerobic respiration—similar to disturbances that result from exhaustive burst-type exercise (Wood 1991; Suski et al. 2003, 2004). Following angling or live-release tournaments, fish are returned to the wild, and it is assumed that they behave normally despite the anaerobic disturbances experienced as part of the angling tournament. Thus, live-release angling tournaments may be an ideal setting for examining whether low-velocity swimming can enhance recovery from exercise in largemouth bass. If slow swimming can facilitate recovery from angling tournaments, less time will be required before largemouth bass can undertake anaerobic swimming associated with prey capture and predator avoidance.

With this background, the objective of this study was to test the hypothesis that low-velocity swimming would enhance the recovery of largemouth bass following exercise relative to fish recovered in static water. On the basis of our knowledge of largemouth bass ecology and in particular their sedentary lifestyle, which contrasts with salmonid fishes, we predict that low-speed swimming will provide less benefit for largemouth bass, as compared with the benefits observed in more active salmonids (e.g., Milligan et al. 2000; Farrell et al. 2001a).

## Material and Methods

Experiments were performed between April 8 and April 16, 2002, at the Sam Parr Biological Research Station near Kinmundy, Illinois. We collected largemouth bass from outdoor earthen ponds (either by angling, electroshocking, or pond draining) and moved them to an indoor laboratory with recirculated, biofiltered water. Fish were allowed at least 48 h to acclimate to the laboratory before experimentation; water temperatures during all experiments were 20°C ( $\pm 1.5^\circ\text{C}$  SEM), and fish were not fed while in the laboratory. Previous work has shown that after 24 h of recovery time, fish have fully recovered from the physiological disturbances (i.e., depletion of energy stores and accumulation of lactate) that arise from angling and exercise (Wood 1991; Wang et al. 1994; Milligan 1996; Kieffer 2000; Suski et al. 2006).

For this study, largemouth bass were sampled as part of two series of experiments. In the first series, we terminally sampled largemouth bass for blood and muscle samples so we could compare the recovery from exercise in static water or in water flowing at 0.5 body lengths per second (BL/s). In the second series, we performed real-time monitoring of cardiac variables in largemouth bass recovering from exercise in either static water or water flowing at 0.5 BL/s. In previous studies (i.e., Milligan et al. 2000), higher swimming speeds (0.9 BL/s) have been used for the recovery of salmonid fishes, reflecting their swimming performance capabilities; we chose 0.5 BL/s for our

study because previous work has shown that estimates of largemouth bass swimming speeds are roughly half those of salmonids (e.g., Beamish 1978; Cooke et al. 2001).

### *Series 1: Blood and Muscle Sampling*

To obtain control (undisturbed) blood and muscle samples, we netted fish from a common holding tank in the laboratory and transferred them to individual black Perspex chambers continuously supplied with aerated water. Care was taken to ensure that the supply of water to the chambers was low enough that fish were not forced to swim against a current when confined. Following 24 h in the darkened chambers, we terminated the flow of water and added buffered anesthetic (250 mg/L 3-aminobenzoic acid ethyl ester methanesulfonate [MS222] and 500 mg NaCO<sub>3</sub>/L) through a hole in the lid. Once fish had lost equilibrium and ceased ventilation (approximately 1 min), we collected blood and muscle samples (Suski et al. 2004).

For sampling, blood was obtained by caudal vessel puncture using an 18-gauge needle and syringe heparinized with sodium heparine, transferred to a 1.5-mL microcentrifuge tube, and immediately centrifuged for 1 min (Summerfelt and Smith 1990). We then drew the plasma (supernatant) from the corpuscular portion of the blood using a pipette. We stored both plasma and erythrocytes in liquid nitrogen until we returned to the laboratory, at which time samples were stored at  $-80^\circ\text{C}$  (Suski et al. 2003). For muscle sampling, we removed a portion of white muscle (ranging in size from about 5 to 10 g) from the epaxial musculature behind the operculum and above the lateral line. Muscle samples were immediately freeze-clamped in aluminum tongs precooled in liquid nitrogen, wrapped in aluminum foil, and then transferred to a dewar containing liquid nitrogen until samples were returned to the laboratory. The time between the collection of samples and processing was less than 6 mo (Suski et al. 2003).

To exercise largemouth bass, we netted fish from a common holding tank, transferred them to a circular tank, and chased them by tail grabbing for 5 min. Following 5 min of exercise, fish were randomly assigned to one of two groups. One group was immediately transferred to anesthetic and sampled for blood and muscle as described above (exercise treatment). The second group of fish was recovered in either static or flowing water. Largemouth bass that were recovered in static water were placed in black Perspex chambers immediately after exercise. As with control fish, the boxes were supplied with aerated water, and care was taken to ensure that the supply of water to the boxes was low enough to prevent any swimming by the fish. After either 1, 2, or 4 h of recovery in static water, anesthetic was added to the boxes, and fish were sampled for blood and muscle (Milligan et al. 2000; Farrell et al. 2001a).

To sample largemouth bass recovered in flowing water, we constructed modified recovery chambers that were also opaque. The modified chambers were virtually identical in dimensions

to the static recovery chambers (10 cm × 13 cm × 50 cm) but had wire mesh on the front and back of the chamber (near the head and caudal fin of the fish) to permit a current of water to flow through each chamber. The relative sizes of the chambers and fish in our experiment were such that fish had sufficient room to swim without contacting the side of the chambers. We placed these modified chambers in a fiberglass raceway filled with water at a height in which the lower 90% of the chamber was under water and the top 10% was left above water. A pump connected to a section of plastic pipe was used to create a laminar flow of water aimed into each chamber. Before the addition of all fish, we used a flowmeter to quantify the flow in each chamber and to ensure that each fish was recovered in water that was indeed flowing at 0.5 BL/s. Following 1, 2, or 4 h of recovery in flowing water, we placed a sheet of clear acrylic over the front and back of each chamber to seal it from the surrounding water in the raceway, added buffered anesthetic to each chamber through the lid, and sampled fish for blood and muscle. The mean size of control fish was  $251 \pm 8$  mm ( $\pm$ SE) and  $185 \pm 30$  g, and the mean size of fish from the exercise treatment was  $270 \pm 21$  mm and  $252 \pm 68$  g. The mean size of fish from the swimming recovery group was  $257 \pm 8$  mm and  $217 \pm 33$  g, while the mean size of fish from the static recovery group was  $264 \pm 10$  mm and  $227 \pm 26$  g.

#### *Series 2: Cardiac Monitoring*

For the cardiac monitoring series of experiments, we continuously recorded cardiac data from largemouth bass during the entire exercise and recovery periods. For this, we first surgically fitted fish with a silicone cuff-type Doppler flow probe as described in Cooke et al. (2003) and then left them to recover for 24 h. As with the blood and muscle sampling protocol, resting control values were obtained by placing fish in an individual black plastic chamber continuously supplied with aerated water. Control fish were left to acclimate in individual containers for 24 h, and we then collected resting cardiac output data from each fish for a 5-min period. Next, we exercised these fish for 5 min by tail grabbing in a circular tank and then randomly assigned them to either a static recovery group or a swimming recovery group. Static recovery of exercised fish occurred in individual plastic chambers, and recovery in flowing water occurred in the modified flow-through chambers. We collected 5 min of cardiac output data from each fish immediately following exercise, and at 1, 2, and 4 h of recovery time. The mean size of fish in the static recovery group was  $248 \pm 9$  mm and  $164 \pm 23$  g, while the mean size of fish in the swimming recovery group was  $233 \pm 8$  mm and  $137 \pm 14$  g.

#### *Analyses*

Analyses for plasma and white muscle variables are described in detail in Suski et al. (2003). We quantified plasma cortisol

using a commercially available radioimmunoassay kit (Coat-a-Count, Diagnostic Products Corporation, Los Angeles, CA), plasma chloride concentrations using a chloride titrator (Radiometer, model CMT 10), and plasma osmolarity with a freezing-point depression osmometer (Advanced Instruments, model 3M0). We followed the methods of Lowry and Passonneau (1972) to determine plasma lactate concentrations. Muscle samples were prepared according to the methods of Suski et al. (2004), and quantification of muscle lactate, PCr, and ATP concentrations followed the enzymatic methods of Lowry and Passonneau (1972). Water content was determined by placing samples of frozen muscle, each weighing between 100 and 800 mg, in a tared 1.5-mL microcentrifuge tube. The initial weight of the sample was recorded, and the tube was then transferred to an 80°C oven for several days. Samples were monitored until a constant weight was obtained and percent water content was calculated.

Following experimentation, we killed all fish outfitted with Doppler flow cuffs using an overdose of anesthetic (180 ppm clove oil), and a postmortem calibration was conducted to convert Doppler shift (in V) to actual blood flow (mL/min; see Cooke et al. 2003 for details). Cow blood perfused through the aorta was used to calibrate the probes over the range of flow rates observed during experiments, and we analyzed reference flow rates with linear least squares regression. All experiments were conducted in accordance with the guidelines of the Canadian Council on Animal Care (Queen's University approval number Tufts-1999-088-R2).

#### *Statistical Methods*

Comparisons of blood and muscle variables within each recovery group were made using a one-way ANOVA followed by a Dunnett's test to compare each sampling time to the control group (Zar 1999). Comparisons at a particular sampling time for blood and muscle variables were made between the two recovery groups using a *t*-test (Zar 1999). For the cardiac variables, comparisons both within and among the two treatment groups were made using a two-way repeated-measures ANOVA (main effects: time and recovery group) followed by a Tukey-Kramer HSD test to detect differences between and within treatments (Sokal and Rohlf 1995). Statistical tests were performed using JMPIN Version 4.0 (SAS Institute, Cary, NC), all values are shown as means  $\pm$  SE, and the level of significance ( $\alpha$ ) for all tests was 0.05.

#### **Results**

Five minutes of exhaustive exercise did not result in a significant change in the concentration of cortisol in the plasma of largemouth bass. By 1 h following exercise, the plasma cortisol concentration of fish recovered in static water had increased almost sixfold relative to resting control individuals (Fig. 1A).

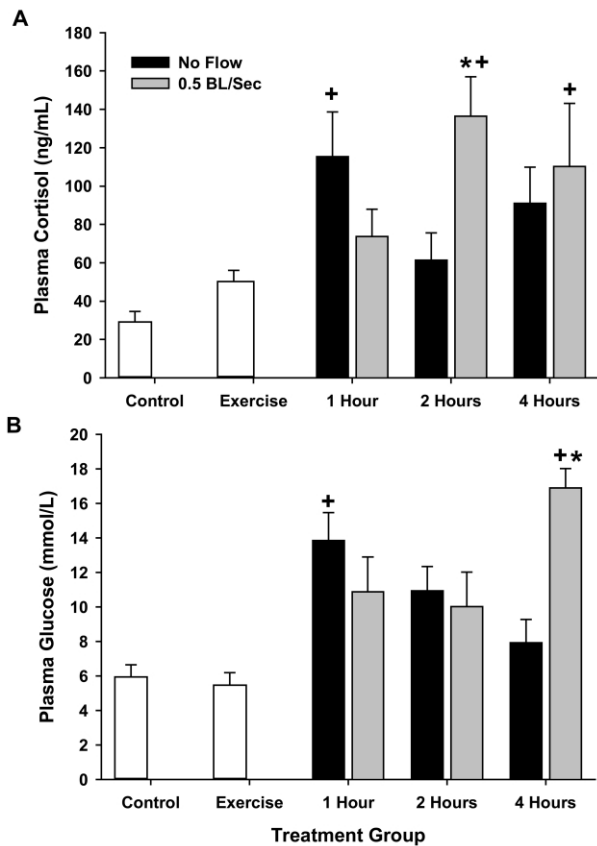


Figure 1. Plasma cortisol and glucose. Changes in plasma cortisol concentration (A) and plasma glucose concentration (B) for largemouth bass exercised and then recovered in either static water (solid bars) or water flowing at 0.5 BL/s (shaded bars). Sample sizes are  $N = 9$  for the control group and  $N = 6$  for all other treatment groups. A plus sign denotes a significant difference between a sampling time and the control group (ANOVA, Dunnett's test,  $P < 0.05$ ), and an asterisk denotes a significant difference between treatment groups at a sampling time ( $t$ -test,  $P < 0.05$ ).

By 2 h after exercise, however, the plasma cortisol concentration of fish recovering in static water had returned to control values (Fig. 1A) and remained at that level for the remainder of the study. In contrast, the plasma cortisol concentrations of largemouth bass recovered in flowing water was not significantly different than resting control fish 1 h after exercise (Fig. 1A). The plasma cortisol concentrations of the swimming fish, however, continued to climb during recovery and were approximately six times greater than control values by 4 h following exercise (Fig. 1A). One hour after exercise, the plasma glucose concentration of largemouth bass recovered in static water had more than doubled relative to control fish but returned to control values after 2 h (Fig. 1B). The plasma glucose concentration of fish recovered at 0.5 BL/s did not differ relative to the control group until 4 h after exercise, at which time concentrations were almost three times greater than control fish

and were 113% greater than fish recovered in static water (Fig. 1B).

The plasma chloride concentration of largemouth bass did not differ from resting control values either after exercise or following 4 h of recovery in either flowing or static water (Fig. 2A). Plasma osmolarity of largemouth bass during this experiment did not show any significant change relative to control fish either following exercise or during recovery in static water (Fig. 2B). Largemouth bass that were recovered in flowing water, however, showed plasma osmolarity values that were significantly lower than fish recovered in static water 1 h after exercise, 2 h after exercise, and 4 h after exercise. By 4 h after exercise, the plasma osmolarity of largemouth bass recovered at 0.5 BL/s was significantly lower than control values and those for fish recovered in static water (Fig. 2B).

Five minutes of exercise caused muscle PCr concentrations

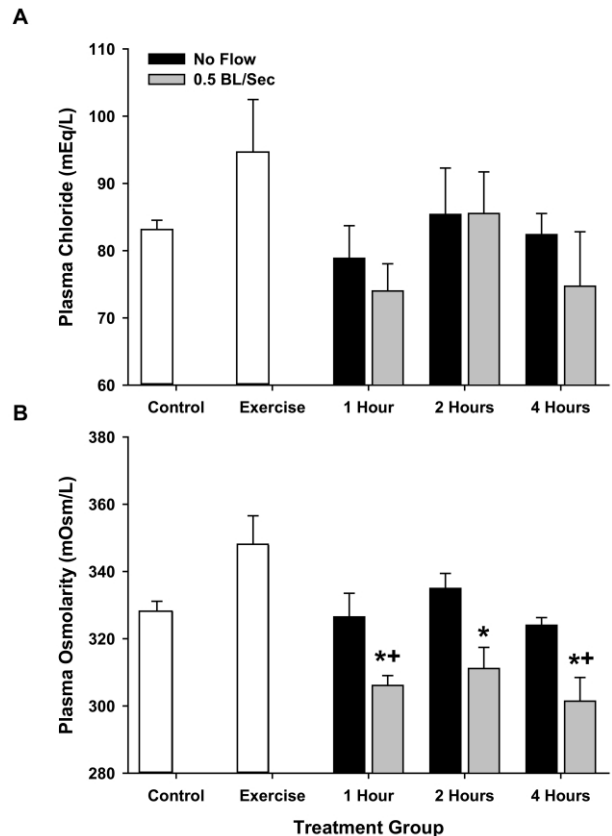


Figure 2. Plasma chloride and osmolarity. Changes in plasma chloride concentration (A) and plasma osmolarity (B) for largemouth bass exercised and then recovered in either static water (solid bars) or water flowing at 0.5 BL/s (shaded bars). Sample sizes are  $N = 9$  for the control group and  $N = 6$  for all other treatment groups. A plus sign denotes a significant difference between a sampling time and the control group (ANOVA, Dunnett's test,  $P < 0.05$ ), and an asterisk denotes a significant difference between treatment groups at a sampling time ( $t$ -test,  $P < 0.05$ ).

to fall by almost 90% (Table 1). In largemouth bass recovered in flowing water, muscle PCr concentrations returned to resting control values by 1 h after exercise (Table 1). In fish recovered in static water, however, muscle PCr values were significantly below control values 1 h after exercise but had returned to normal by 2 h postexercise (Table 1). By 4 h after exercise, the muscle PCr of fish recovered in static water had fallen below resting values (Table 1). Muscle ATP concentrations fell by two-thirds relative to control fish following 5 min of exercise (Table 1). For both swim recovery fish and static-water recovery fish, muscle ATP concentrations returned to control values by 1 h after exercise, and there were no significant differences between the ATP concentrations of fish recovered in static water and fish recovered in flowing water at the two other sampling periods (Table 1). Throughout the experiment, muscle water content did not differ significantly from resting control values for either of the recovery treatments (Table 1).

Exercise resulted in a sixfold increase in plasma lactate relative to control largemouth bass, and plasma lactate concentrations remained significantly elevated relative to control individuals until 4 h postexercise for all recovery treatments (Fig. 3A). Largemouth bass that swam at 0.5 BL/s during recovery, however, exhibited plasma lactate concentrations approximately one-third lower than the static recovery group at 1 h postexercise (Fig. 3A) and 57% lower than the static recovery group at 2 h postexercise (Fig. 3A). Following 4 h of recovery, however, the plasma lactate concentration of fish recovered in flowing water was significantly greater than fish recovered in static water (Fig. 3A). Muscle lactate concentrations increased 13 times relative to control largemouth bass following 5 min of exercise (Fig. 3B). By 1 h after exercise, muscle lactate concentrations had returned to control levels in both largemouth bass recovered in flowing water and in largemouth bass recovered in static water. At 1 h postexercise, however, the muscle lactate concentration of largemouth bass recovered at a swim-

ming speed of 0.5 BL/s was 85% lower than fish recovered in static water at that sampling time (Fig. 3B).

#### Cardiac Variables

Five minutes of exercise resulted in a 63% increase in cardiac output for largemouth bass (Fig. 4A), a change primarily driven by an increase in heart rate (Fig. 4B) as stroke volume decreased by approximately 80% (Fig. 4C). By 2 h following exercise, the stroke volume of fish recovered in static water had returned to control values (Fig. 4C), and the heart rate of fish recovered in static water was significantly lower than swimming fish (Fig. 4B). By 4 h following exercise, all cardiac parameters for fish recovering in static water had returned to control values (Fig. 4). Fish recovered in flowing water exhibited significant elevations in cardiac output (Fig. 4A), significant increases in heart rate (Fig. 4B), and significant reductions in stroke volume (Fig. 4C) relative to control values and to fish recovered in static water.

#### Discussion

Manual chasing of largemouth bass to exhaustion resulted in a suite of physiological changes. Specifically, exercise caused significant depletions of white muscle energy stores and significant productions of lactate in white muscle. In addition, exercise caused an increase in cardiac output and heart rate and a decline in stroke volume. Despite these physiological changes, no fish mortality occurred during this study. Previous results of this nature and magnitude have been described for largemouth bass (Gustavson et al. 1991; Cooke et al. 2003; Suski et al. 2006) as well as salmonid fishes (Jones and Randall 1978; Randall 1982; Wood 1991; Wang et al. 1994; Milligan 1996) that were exercised. During exhaustive exercise, the energy requirements of fish cannot be met aerobically, and they

Table 1: White muscle energy stores and water content

	Control	Exercise	1 h	2 h	4 h
PCr ( $\mu\text{mol/g}$ wet weight)	16.7 $\pm$ 1.3	1.8 $\pm$ .4*			
No flow			9.4 $\pm$ 3.1*	13.4 $\pm$ 6.8	10.2 $\pm$ 1.8*
.5 BL/s			14.0 $\pm$ 4.7	17.3 $\pm$ 2.8	15.4 $\pm$ 1.8
ATP ( $\mu\text{mol/g}$ wet weight)	6.1 $\pm$ .4	2.4 $\pm$ .8*			
No flow			4.8 $\pm$ 1.8	5.8 $\pm$ 1.8	5.5 $\pm$ 1.8
.5 BL/s			4.2 $\pm$ .8	5.9 $\pm$ 1.1	4.3 $\pm$ 1.1
Water content (%)	79.8 $\pm$ .2	79.4 $\pm$ .5			
No flow			80.2 $\pm$ .6	79.7 $\pm$ .8	79.8 $\pm$ .5
.5 BL/s			79.7 $\pm$ 1.1	79.6 $\pm$ .7	79.2 $\pm$ 1.7

Note. White muscle energy stores and white muscle water content for largemouth bass exercised and then recovered in either static (no flow) or flowing water following exercise. PCr = phosphocreatine; BL/s = body lengths per second;  $N = 6$  for all treatments, except control, where  $N = 9$ .

\* Significant difference from control value (Dunnnett's test,  $P < 0.05$ ).

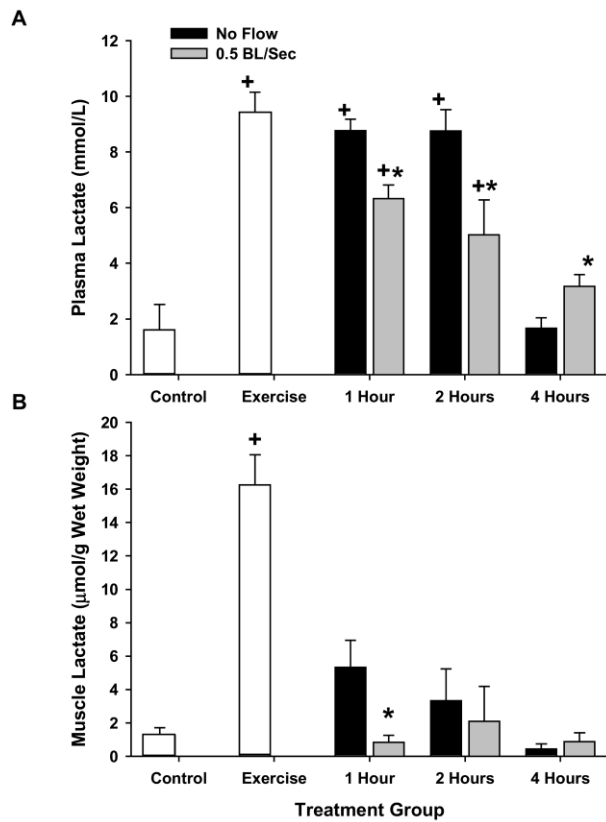


Figure 3. Plasma and muscle lactate. Changes in plasma lactate concentration (A) and muscle lactate concentration (B) for largemouth bass exercised and then recovered in either static water (solid bars) or water flowing at 0.5 BL/s (shaded bars). Sample sizes are  $N = 9$  for the control group and  $N = 6$  for all other treatment groups. A plus sign denotes a significant difference between a sampling time and the control group (ANOVA, Dunnett's test,  $P < 0.05$ ), and an asterisk denotes a significant difference between treatment groups at a sampling time ( $t$ -test,  $P < 0.05$ ).

are forced to meet their energetic demands though anaerobic respiration (Hochachka 1991). As a result of this anaerobic metabolism, energy stores in white muscle are consumed, lactate is produced, and cardiac output is increased (Wood 1991). The 5-min exercise regime used in this study clearly resulted in exhaustion for largemouth bass and physiological disturbances consistent with previous work.

In this study, largemouth bass forced to swim following a bout of exercise showed expedited recovery relative to fish recovered in static water 1 h following exercise. Specifically, swimming largemouth bass showed reduced concentrations of lactate in white muscle and plasma relative to fish from static water at 1 h postexercise. Concentrations of plasma glucose, plasma cortisol, and muscle PCr were also significantly different from controls in the static treatment fish at 1 h postexercise, whereas these parameters were not significantly different from control

values in swimming fish 1 h after exercise. Following exercise, fish begin the process of recovery and attempt to correct the physiological disturbances induced by burst exercise. As such, energy stores within white muscle are replenished, lactate accumulations are cleared, and osmotic imbalances are corrected (Wood 1991; Milligan 1996). Research using rainbow trout has shown this process to be fueled largely by lipid metabolism (Richards et al. 2002), requiring an aerobic environment (Moyes et al. 1992; Richards et al. 2002) and taking up to 8–12 h to complete (Milligan 1996; Richards et al. 2002). Recent work involving two salmonid species (rainbow trout and coho salmon) has shown that forcing fish to swim at slow speeds

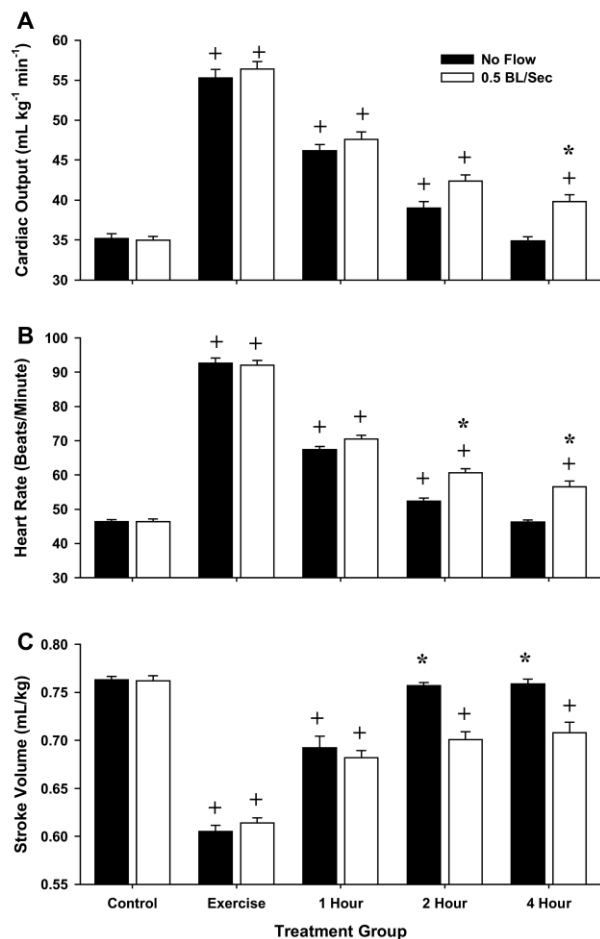


Figure 4. Cardiac activity. Changes in cardiac output (A), heart rate (B), and stroke volume (C) for largemouth bass exercised and then recovered in either static water (solid bars) or water flowing at 0.5 BL/s (unfilled bars). Sample sizes are  $N = 8$  for both treatment groups. A plus sign denotes a significant difference between a sampling time and the control group (repeated-measures two-way ANOVA, Tukey-Kramer HSD test,  $P < 0.05$ ), and an asterisk denotes a significant difference between treatment groups at a sampling time (repeated-measures two-way ANOVA, Tukey-Kramer HSD test,  $P < 0.05$ ).

(0.5–1.5 BL/s) rather than leaving them in static water will reduce the time required for recovery from exercise (Milligan et al. 2000; Farrell et al. 2001a). The exact mechanism responsible for this expedited recovery is not known but is believed to be associated with reduced cortisol levels in swimming fish (Milligan 2003). Results from our study indicated that, similar to rainbow trout and coho salmon, largemouth bass show expedited recovery 1 h postexercise when forced to swim at slow velocities. Similar to studies with salmonid fishes, this expedited recovery in largemouth bass may be linked to reduced concentrations of circulating cortisol; concentrations of plasma cortisol had returned to control levels in swimming largemouth bass at 1 h postexercise but were significantly higher than control levels in fish from the static treatment at 1 h postexercise. Studies involving rainbow trout, however, have shown an absence of a postexercise cortisol spike in swimming fish (Milligan et al. 2000), which was not the case for largemouth bass.

While recovering largemouth bass in flowing water showed benefits at 1 h postexercise, by 4 h after exercise, recovery in flowing water resulted in additional physiological disturbances—contrary to previous work with salmonid fishes (Milligan et al. 2000; Farrell et al. 2001a). Specifically, largemouth bass swimming at 0.5 BL/s showed concentrations of plasma glucose and plasma lactate that were significantly elevated relative to nonswimming fish. In addition, largemouth bass from the swimming treatment showed increased cardiac output, elevated heart rate, and reduced stroke volume compared with nonswimming fish 4 h postexercise. Both exercise and stress have been shown to increase the cardiac output of fish and raise plasma glucose concentrations to fuel aerobic tissues such as red muscle and heart (Randall 1982; West et al. 1993, 1994a, 1994b; Wendelaar Bonga 1997). Swimming of largemouth bass at 0.5 BL/s, therefore, resulted in an increased energetic demand that resulted in elevated cardiac output and glucose concentrations as energy and oxygen needed to be delivered to muscle to sustain swimming. By 4 h following exercise, the plasma lactate concentrations of swimming fish were significantly greater than nonswimming fish, indicating that swimming fish were either using anaerobic metabolism to meet their energy demands (Jones and Randall 1978; Hochachka 1991) or had an impaired ability to clear lactate accumulations. Taken together, these results indicate that forcing largemouth bass to swim for 4 h causes significant metabolic disturbances.

In addition to metabolic disturbances, swimming largemouth bass showed ionic disturbances that were absent in fish recovered in static water. Both stress and exercise in fish result in secretions of catecholamines as part of the primary stress response designed to readjust biological activities and help fish cope with perceived stressors (Mazeaud et al. 1977; Wendelaar Bonga 1997). Secretions of catecholamines work to stimulate branchial blood flow and improve oxygen uptake and delivery (Wendelaar Bonga 1997). In addition, however, chronic stressors can alter both the structure and function of gills and result

in a loss of ions from plasma (McDonald and Milligan 1992; Wendelaar Bonga 1997). Therefore, a common symptom of freshwater fish subjected to chronic or prolonged stressors is a loss of ions from plasma (McDonald and Milligan 1992; Wendelaar Bonga 1997). In our study, swimming largemouth bass exhibited a significant reduction in plasma osmolarity (relative to both nonswimming fish and control fish) at 4 h postexercise, indicating a loss of ionically active plasma constituents (e.g.,  $\text{Na}^+$ ,  $\text{K}^+$ ,  $\text{H}^+$ ,  $\text{HCO}_3^-$ ). Because the water content of white muscle in this study did not change during either exercise or recovery, this drop in the concentration of plasma constituents was likely a result of ion losses rather than fluid shifts to or from muscle often seen following exhaustive exercise (Wood 1991). Ion losses from plasma have been previously noted in largemouth bass subjected to prolonged stressors such as hauling (Carmichael et al. 1984), even though previous work has indicated that the gills of *Micropterus* species are relatively impermeable to ion loss relative to other freshwater fishes (McDonald et al. 1991).

The failure of largemouth bass to respond favorably to swimming recovery, unlike the two salmonid species previously examined (Milligan et al. 2000; Farrell et al. 2001a), can likely be explained by one of two different mechanisms. First, both rainbow trout and coho salmon are migratory, active fishes that spend a large portion of their lifetime swimming and foraging in the pelagic zone (Brett 1995; Hinch et al. 2006). In contrast, largemouth bass are “sit-and-wait” ambush-type predators and do not routinely engage in long-distance swimming (Demers et al. 1996). To emphasize these ecological differences among fishes in the field, we computed the daily and instantaneous swimming rates for species previously recovered in static and flowing water (i.e., coho salmon, rainbow trout, and largemouth bass) and for which comparable literature accounts using mobile telemetry tracking exist. Results showed that largemouth bass are much less active than salmonids with empirical swimming speeds estimated between 3.2 and 8.0 cm/s (Rice 1981; Savitz et al. 1983), averaging approximately 5 cm/s (Rice and Cochran 1984). Assuming an average size of 35 cm, this would produce speeds of 0.14 BL/s and daily travel of 4.3 km/d. In contrast, adult salmon similar to those used by Farrell et al. (2001a) typically travel up to 40 km/d when in the ocean, or approximately 0.77 BL/s (e.g., Hinch et al. 2006; S. J. Cooke and A. P. Farrell, unpublished data). Rainbow trout similar in size to those used by Milligan et al. (2000) and Meyer and Cook (1996) travel between 0.25 and 0.3 BL/s in lentic environments (e.g., Warner and Quinn 1995) and probably faster in lotic systems. The swimming speeds used in previous recovery studies have varied across species but were generally higher (i.e., >0.5 BL/s) for salmonids (Table 2) than for largemouth bass in this study. In addition, the recovery speed when expressed as a percentage of both  $U_{\text{crit}}$  and  $U_{\text{max}}$  was always higher (by as much as 3.5 times) for salmonids than for the largemouth bass in our study (Table 2). Forcing largemouth bass to swim for

Table 2: Characteristics of recovery experiments, including all examples from the literature and this study

Species (Life Stage/Source)	Recovery Temperature (°C)	$U_{crit}$ (BL/s)	$U_{max}$ (BL/s)	Recovery Speed (BL/s)	Recovery Speed (% $U_{crit}$ )	Recovery Speed (% $U_{max}$ )	Recovery Study Citation
Atlantic salmon (smolts, hatchery)	11.5	8.30 <sup>a</sup>	11.28	.50	6.02	4.43	Boesgaard et al. 1993
Coho salmon (adult, wild)	12–16	2.30 <sup>b</sup>	2.96	.75–1.50	32.61–65.22	25.34–50.68	Farrell et al. 2001b
Rainbow trout (juvenile, hatchery)	12–16	3.50 <sup>c</sup>	4.76	.90	25.71	18.91	Milligan et al. 2000
Rainbow trout (juvenile, hatchery)	15	3.50 <sup>c</sup>	4.76	1.50	42.86	31.51	Meyer and Cook 1996
Largemouth bass (sub-adult, pond raised)	20	2.46 <sup>d</sup>	3.34	.50	20.30	16.70	This study

Note. Critical swimming speeds (i.e.,  $U_{crit}$ ) for each species were chosen from the literature to best match the size, life stage, source, and water temperature of this recovery experiment (see footnotes for specific details). Maximum  $U_{crit}$  (i.e.,  $U_{max}$ ) as defined by Farrell et al. (2001b) was set at  $1.36 \times U_{crit}$ , except for coho salmon, where empirical data were available (Farrell et al. 2001b). Recovery speeds used in experiments were reported in absolute relative speeds (BL/s) and are also presented as a percentage of  $U_{crit}$  and as a percentage of  $U_{max}$ .

<sup>a</sup> Data from Peake et al. (1997).  $U_{crit}$  for hatchery Atlantic salmon smolts at 12°C.

<sup>b</sup> Data from Farrell et al. (2001b).  $U_{crit}$  for wild adult coho salmon at 12°–16°C.

<sup>c</sup> Data from Jones (1971).  $U_{crit}$  for juvenile hatchery rainbow trout at 14.1°C.

<sup>d</sup> Data from Cooke et al. (2001).  $U_{crit}$  for subadult pond raised largemouth bass at 18°C.

prolonged periods, even at the low absolute and proportional swimming speed in this study (20% of  $U_{crit}$  and 16.7% of  $U_{max}$ ), presented them with a challenge that prevented expedited recovery and induced physiological disturbances such as increased energy consumption, elevated cardiac output, and ion losses from plasma. Indeed, following an exhaustive exercise event, largemouth bass are generally sedentary (Cooke et al. 2000, 2004) relative to salmonids that continue to swim actively postexercise, albeit sometimes downstream (personal communications cited in Milligan et al. 2000; Mäkinen et al. 2000; Thorstad et al. 2003; Meka and McCormick 2005).

A second reason that may explain why largemouth bass did not benefit from swimming following exercise (as opposed to recent work with salmonid fishes) may be related to pedigree. Studies by Milligan and coworkers (Milligan et al. 2000; Milligan 2003) credited enhanced recovery in swimming rainbow trout to the absence of a cortisol response following exercise. The fish used by Milligan et al. (2000), however, were hatchery-reared rainbow trout raised in tanks in captivity, possibly for generations. These fish were likely comfortable in a hatchery/laboratory environment and consequently may have even exhibited a reduced cortisol response that was influenced by artificial selection (Pottinger and Carrick 1999). In contrast, the largemouth bass in our study were wild individuals that exhibited elevated cortisol concentrations when brought into a foreign and artificial environment such as the laboratory. Previous studies involving wild largemouth bass have shown that cortisol concentrations of fish sampled in the laboratory are roughly 20 times greater than free-swimming fish (Suski et al.

2003, 2004). Simply bringing wild largemouth bass into the laboratory, therefore, results in an increase in plasma cortisol concentrations that may prohibit the expedited recovery from exercise noted by Milligan et al. (2000), and this cortisol response was further exacerbated by swimming during recovery. Either one or a combination of both of these mechanisms, therefore, may explain why largemouth bass exhibited elevated plasma cortisol concentrations following prolonged swimming that may have impaired recovery from exercise.

From an applied perspective, this study has important implications for fisheries management. Clearly, previous work with salmonids has shown a recovery benefit to forced swimming following anaerobic disturbances (Milligan et al. 2000; Farrell et al. 2001a, 2001b). Results from this study with largemouth bass show that while 1 h of low-velocity swimming may expedite recovery from exercise, forcing largemouth bass to swim for 4 h at low velocity results in significant physiological disturbances and ion losses. This finding, disparate to previous work with salmonids, emphasizes the importance of examining the species-specific responses of fishes to various stressors before broadscale management recommendations (Cooke and Suski 2005). For this reason, we do not recommend the use of low-velocity swimming to enhance recovery from exhaustive exercise for largemouth bass. However, additional work is needed that compares recovery performance under different flow conditions to determine whether an optimal recovery speed can be determined. Additional empirical work from the field that couples behavioral and physiological consequences of exhaustive exercise is also encouraged.



## Acknowledgments

We thank Ken Ostrand, Mike Siepker, Curt Ostradka, Liz Osier, B. J. Bauer, and David H. Wahl. Financial support was provided by the Natural Science and Engineering Research Council of Canada, the University of Illinois Research Board, and the Illinois Natural History Survey. Additional support was provided by a Sigma Xi Grant in Aid of Research to S.J.C.

## Literature Cited

- Beamish F.W.H. 1978. Swimming capacity. Pp. 101–187 in W.S. Hoar and D.J. Randall, eds. *Fish Physiology*. Vol. 7. Academic Press, New York.
- Boesgaard L., M.E. Nielsen, and P. Rosenkilde. 1993. Moderate exercise decreases plasma cortisol levels in Atlantic salmon (*Salmo salar*). *Comp Biochem Physiol* 106A:641–643.
- Brett J.R. 1995. Energetics. Pp. 3–68 in C. Groot, L. Margolis, and W.C. Clarke, eds. *Physiological Ecology of Pacific Salmon*. University of British Columbia Press, Vancouver.
- Carmichael G.J., J.R. Tomasso, B.A. Simco, and K.B. Davis. 1984. Confinement and water-quality induced stress in largemouth bass. *Trans Am Fish Soc* 113:767–777.
- Cooke S.J., C.M. Bunt, K.G. Ostrand, D.P. Philipp, and D.H. Wahl. 2004. Angling-induced cardiac disturbance of free-swimming largemouth bass (*Micropterus salmoides*) monitored with heart rate telemetry. *J Appl Ichthyol* 20:28–36.
- Cooke S.J., T.A. Kassler, and D.P. Philipp. 2001. Physiological performance of largemouth bass related to local adaptation and interstock hybridization: implications for conservation and management. *J Fish Biol* 59(suppl. A):248–268.
- Cooke S.J., K.G. Ostrand, C.M. Bunt, J.F. Schreer, D.H. Wahl, and D.P. Philipp. 2003. Cardiovascular responses of largemouth bass to exhaustive exercise and brief air exposure over a range of water temperatures. *Trans Am Fish Soc* 132:1154–1165.
- Cooke S.J., D.P. Philipp, J.F. Schreer, and R.S. McKinley. 2000. Locomotory impairment of nesting male largemouth bass following catch-and-release angling. *N Am J Fish Manag* 20:968–977.
- Cooke S.J. and C.D. Suski. 2005. Do we need species-specific guidelines for catch-and-release recreational angling to effectively conserve diverse fisheries resources? *Biodiv Conserv* 14:1195–1209.
- Demers E., R.S. McKinley, A.H. Weatherley, and D.J. McQueen. 1996. Activity patterns of largemouth and smallmouth bass determined with electromyogram biotelemetry. *Trans Am Fish Soc* 125:434–439.
- Farrell A.P., P.E. Gallagher, J. Fraser, D. Pike, P. Bowering, A.K.M. Hadwin, W. Parkhouse, and R. Routledge. 2001a. Successful recovery of the physiological status of coho salmon on board a commercial gillnet vessel by means of a newly designed revival box. *Can J Fish Aquat Sci* 58:1932–1946.
- Farrell A.P., P.E. Gallagher, and R. Routledge. 2001b. Rapid recovery of exhausted adult coho salmon after commercial capture by troll fishing. *Can J Fish Aquat Sci* 58:2319–2324.
- Gustavson A.W., R.S. Wydoski, and G.A. Wedemeyer. 1991. Physiological response of largemouth bass to angling stress. *Trans Am Fish Soc* 120:629–636.
- Hinch S.G., S.J. Cooke, M.C. Healey, and A.P. Farrell. 2006. Behavioural physiology of fish migrations: salmon as a model approach. Pp. 239–295 in K.A. Sloman, R.W. Wilson, and S. Balshine, eds. *Fish Physiology*. Vol. 24. Behaviour and Physiology of Fish. Elsevier, Amsterdam.
- Hochachka P.W. 1991. Design of energy metabolism. Pp. 325–351 in C.L. Prosser, ed. *Environmental and Metabolic Animal Physiology*. Wiley-Liss, New York.
- Jones D.R. 1971. The effects of hypoxia and anaemia on the swimming performance of rainbow trout (*Salmo gairdneri*). *J Exp Biol* 55:541–551.
- Jones D.R. and D.J. Randall. 1978. The respiratory and circulatory systems during exercise. Pp. 425–501 in W.S. Hoar and D.J. Randall, eds. *Fish Physiology*. Vol. 7. Academic Press, New York.
- Kerr S.J. and K.K. Kamke. 2003. Competitive fishing in freshwaters of North America: a survey of Canadian and U.S. jurisdictions. *Fisheries* 28:26–31.
- Kieffer J.D. 2000. Limits to exhaustive exercise in fish. *Comp Biochem Physiol* 126A:161–179.
- Lowry O.H. and J.V. Passonneau. 1972. *A Flexible System of Enzymatic Analysis*. Academic Press, New York.
- Mäkinen T.S., E. Niemelä, K. Moen, and R. Lindström. 2000. Behaviour of gill-net and rod-captured Atlantic salmon (*Salmo salar* L.) during upstream migration and following radio tagging. *Fish Res* 45:117–127.
- Mazeaud M.M., F. Mazeaud, and E.M. Donaldson. 1977. Primary and secondary effects of stress in fish: some new data with a general review. *Trans Am Fish Soc* 106:201–212.
- McDonald D.G., V. Cavdek, and R. Ellis. 1991. Gill design in freshwater fishes: interrelationships among gas exchange, ion regulation, and acid-base regulation. *Physiol Zool* 64:103–123.
- McDonald D.G. and C.L. Milligan. 1992. Chemical properties of the blood. Pp. 55–133 in W.S. Hoar, D.J. Randall, and A.P. Farrell, eds. *Fish Physiology*. Vol. 11. Pt. B. The Cardiovascular System. Academic Press, New York.
- Meka J.M. and S.D. McCormick. 2005. Physiological response of wild rainbow trout to angling: impact of angling duration, fish size, body condition and temperature. *Fish Res* 72:311–322.
- Meyer W.F. and P.A. Cook. 1996. An assessment of the use of low-level aerobic swimming in promoting recovery from handling stress in rainbow trout. *Aquacult Int* 4:169–174.

- Milligan C.L. 1996. Metabolic recovery from exhaustive exercise in rainbow trout. *Comp Biochem Physiol* 113A:51–60.
- . 2003. A regulatory role for cortisol in muscle glycogen metabolism in rainbow trout *Oncorhynchus mykiss* Walbaum. *J Exp Biol* 206:3167–3173.
- Milligan L.C., G.B. Hooke, and C. Johnson. 2000. Sustained swimming at low velocity following a bout of exhaustive exercise enhances metabolic recovery in rainbow trout. *J Exp Biol* 203:921–926.
- Moyes C.D., P.M. Schulte, and P.W. Hochachka. 1992. Recovery metabolism of trout white muscle: role of mitochondria. *Am J Physiol* 262:R295–R304.
- Peake S., R.S. McKinley, D.A. Scruton, and R. Moccia. 1997. Influence of transmitter attachment procedures on swimming performance of wild and hatchery-reared Atlantic salmon, *Salmo salar*, smolts. *Trans Am Fish Soc* 125:707–714.
- Pottinger T.G. and T.R. Carrick. 1999. Modification of the plasma cortisol response to stress in rainbow trout by selective breeding. *Gen Comp Endocrinol* 116:122–132.
- Randall D. 1982. The control of respiration and circulation in fish during exercise and hypoxia. *J Exp Biol* 100:275–288.
- Rice J.A. 1981. Derivation and Application of a Bioenergetics Model for Largemouth Bass (*Micropterus salmoides*). PhD diss. University of Wisconsin, Madison.
- Rice J.A. and P.A. Cochran. 1984. Independent evaluation of a bioenergetics model for largemouth bass. *Ecology* 65:732–739.
- Richards J.G., G.J.F. Heigenhauser, and C.M. Wood. 2002. Lipid oxidation fuels recovery from exhaustive exercise in white muscle of rainbow trout. *Am J Physiol* 282:R89–R99.
- Savitz J., P.A. Fish, and R. Weszely. 1983. Habitat utilization and movement of fish as determined by radio telemetry. *J Freshw Ecol* 2:165–174.
- Sokal R.R. and F.J. Rohlf. 1995. *Biometry*. 3rd ed. W.H. Freeman, New York.
- Summerfelt R.C. and L.S. Smith. 1990. Anesthesia, surgery, and related techniques. Pp. 213–272 in C.B. Schreck and P.B. Moyle, eds. *Methods for Fish Biology*. American Fisheries Society, Bethesda, MD.
- Suski C.D., S.S. Killen, S.J. Cooke, J.D. Kieffer, D.P. Philipp, and B.L. Tufts. 2004. Physiological significance of the weigh-in during live-release angling tournaments for largemouth bass. *Trans Am Fish Soc* 133:1291–1303.
- Suski C.D., S.S. Killen, J.D. Kieffer, and B.L. Tufts. 2006. The influence of environmental temperature and oxygen concentration on the recovery of largemouth bass from exercise: implications for live-release angling tournaments. *J Fish Biol* 68:120–136.
- Suski C.D., S.S. Killen, M.B. Morrissey, S.G. Lund, and B.L. Tufts. 2003. Physiological changes in largemouth bass caused by live-release angling tournaments in southeastern Ontario. *N Am J Fish Manag* 23:760–769.
- Thorstad E.B., T.F. Næsje, F. Fiske, and B. Finstad. 2003. Effects of hook and release on Atlantic salmon in the River Alta, northern Norway. *Fish Res* 60:293–307.
- Wang Y., G.J.F. Heigenhauser, and C.M. Wood. 1994. Integrated responses to exhaustive exercise and recovery in rainbow trout white muscle: acid-base, phosphogen, carbohydrate, lipid, ammonia, fluid volume and electrolyte metabolism. *J Exp Biol* 195:227–258.
- Warner E.J. and T.P. Quinn. 1995. Horizontal and vertical movements of telemetered rainbow trout (*Oncorhynchus mykiss*) in Lake Washington. *Can J Zool* 73:146–153.
- Wendelaar Bonga S.E. 1997. The stress response in fish. *Physiol Rev* 77:591–625.
- West T.G., P.G. Arthur, R.K. Suarez, C.J. Doll, and P.W. Hochachka. 1993. *In vivo* utilization of glucose by heart and locomotory muscles of exercising rainbow trout (*Oncorhynchus mykiss*). *J Exp Biol* 177:63–79.
- West T.G., C.J. Brauner, and P.W. Hochachka. 1994a. Muscle glucose utilization during sustained swimming in the carp (*Cyprinus carpio*). *Am J Physiol* 267:R1226–R1234.
- West T.G., P.M. Schulte, and P.W. Hochachka. 1994b. Implications of hyperglycemia for post-exercise resynthesis of glycogen in trout skeletal muscle. *J Exp Biol* 189:69–84.
- Wood C.M. 1991. Acid-base and ion balance, metabolism, and their interactions, after exhaustive exercise in fish. *J Exp Biol* 160:285–308.
- Zar J.H. 1999. *Biostatistical Analysis*. 4th ed. Prentice Hall, Upper Saddle River, NJ.