

## Physiological Changes in Largemouth Bass Caused by Live-Release Angling Tournaments in Southeastern Ontario

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**Abstract.**—Several largemouth bass *Micropterus salmoides* tournaments in Ontario were visited in the summers of 1999 and 2000 to examine the physiological changes that occur in largemouth bass as a result of tournament procedures. Physiological variables were compared among tournament-caught largemouth bass, resting laboratory controls, and angled controls. The plasma cortisol and glucose concentrations and plasma osmolarity in tournament-caught largemouth bass sampled within 5 min following the weigh-in were significantly greater than those in both control groups. Tournament-caught fish also exhibited ionic disturbances that involved increases in plasma sodium and potassium concentrations, but there were no significant changes in the levels of plasma chloride. Large changes in the metabolic status of largemouth bass sampled following the weigh-in included major reductions in the muscle energy stores phosphocreatine, adenosine triphosphate, and glycogen and large increases in muscle and plasma lactate concentrations. In contrast, no significant changes occurred in the mRNA levels of heat-shock protein 70 in several tissues or in plasma creatine phosphokinase activity. Taken together, these results indicate that live-release angling tournaments cause a significant physiological disturbance in largemouth bass, but there is no evidence that these events normally result in serious cell damage.

The practice of releasing fish following angling (catch and release) has increased over the last several decades due to changes both in regulations and in angler ethics (Barnhart 1989; Quinn 1996). By returning fish to the water following angling, catch rates should be maintained over time, and the abundance of large fish within a system should increase (Noble and Jones 1999). Provided that fish are not deeply hooked (Pelzman 1978) and angling does not occur near the fish's thermal maximum (Wilkie et al. 1996), fish returned to the water immediately following angling typically exhibit low mortality rates (Pelzman 1978; Green et al. 1987; Lee 1987; Gustaveson et al. 1991; Booth et al. 1995).

Live release has also become an integral part of the angling tournament industry in North America during the past few decades (Shupp 1979; Dutweiler 1985; Schramm et al. 1991). The popularity of tournaments escalated in the 1970s (Shupp 1979), and largemouth bass *Micropterus salmoides* have proven to be the most popular target species for these events (Schramm et al. 1991). Tournaments targeting largemouth bass occur in 48 of 50 U.S. states (Schramm et al. 1991) and in many Canadian provinces (Schramm et al. 1991; Kerr 1999). Although live-release angling tournaments

provide an important economic benefit to many rural communities, fisheries managers have expressed concern regarding the potential impacts of angling tournaments on bass populations (Schramm et al. 1991; Grant 1999).

Evidence from previous studies indicates that although many live-release tournaments are successful at releasing a large portion of their fish alive (Schramm et al. 1985, 1987; Kwak and Henry 1995; Wilde 1998), some mortality may occur in these events (Wilde 1998). In an attempt to understand the causes of fish mortality associated with tournaments, previous studies have correlated mortality with variables such as water temperature (Schramm et al. 1987), the water quality in live wells (Hartley and Moring 1993; Kwak and Henry 1995), the size of fish (Meals and Miranda 1994), and organizational procedures (Weathers and Newman 1997). According to Wilde (1998), however, the importance of these variables in influencing mortality rates can vary between studies. Some researchers have also suggested that mortality at tournaments probably results from the cumulative effects of many sublethal stressors (Schramm et al. 1987; Kwak and Henry 1995). Because mortality can vary widely among tournaments, Schramm et al. (1987) proposed that a greater survival of bass following tournaments should be possible.

Although previous studies have attempted to determine the exogenous factors that may contribute

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to fish mortality following live-release tournaments, little information exists about the physiological condition of the fish following these events. Thus, very little is known about (1) the nature of the physiological disturbances in species such as largemouth bass that have experienced a live-release tournament and (2) the potential mechanisms that may contribute to the mortality of some individuals.

The objective of this study was therefore to determine whether live-release angling tournaments cause significant physiological changes in largemouth bass. To accomplish this, we examined a wide range of physiological variables in resting laboratory control bass, angled control bass, and bass that had been involved in live-release angling tournaments. It is expected that live-release angling tournaments will cause significant physiological changes in largemouth bass, and the results of this study should provide important information about the nature of this physiological disturbance.

### Methods

*Sample collection.*—Five live-release bass tournaments in southeastern Ontario (Charleston Lake, Fenelon Falls, Newboro Lake, Lake Simcoe, and Trilakes) were visited in the summers of 1999 and 2000 to obtain muscle and blood samples for analyses. The surface water temperatures at these events were 23–24°C, and the tournaments where fish were sampled were competitions for the greatest combined weight of fish over a 2-d period. The organizational procedures and rules resembled previous tournament descriptions (e.g., Kwak and Henry 1995) in that only artificial baits were used, the anglers were penalized for dead fish, the fish had to comply with a minimum length limit, and the anglers reported to a common weigh-in point at the conclusion of the angling day. The largemouth bass used in the study were randomly selected directly from weigh-in baskets prior to being deposited in the live-release boat (i.e., following weigh-in procedures). The fish were then transported in a cooler filled with lake water to a sampling tent and held until processing. The time between placement in the aerated cooler and sampling was generally less than 5 min.

*Blood sampling.*—Blood samples were taken from nonanesthetized largemouth bass that were wrapped in a damp cloth and placed in a foam-lined holding box filled with aerated lake water. Fish were normally very lethargic after the weigh-in and remained docile during the sampling procedure. Approximately 3 mL of blood, obtained

by caudal puncture using a 21-G needle and heparinized syringe, was withdrawn from the vessel (Houston 1990), transferred to a 1.5-mL microcentrifuge tube, and immediately centrifuged at  $10,000 \times$  gravity for 2 min. The plasma was then separated from the cellular portion of the blood using a pipette. Both plasma and red cells were kept on dry ice until returning to the laboratory, at which time they were stored at  $-80^{\circ}\text{C}$  until processing.

*Tissue sampling.*—White muscle and heart samples were collected from 11 largemouth bass caught during the tournament at Fenelon Falls. To obtain tissue samples, fish were randomly selected prior to being placed into the live-release boat. After being transported to the sampling tent, the fish were anesthetized using a mixture of tricaine methanesulfonate and sodium bicarbonate ( $\text{NaHCO}_3$ ), at concentrations of approximately 250 mg tricaine/L and 500 mg  $\text{NaHCO}_3$ /L (Summerfelt and Smith 1990). The fish were lightly sedated after approximately 30 s and had lost equilibrium after about 1.5 min. Following the complete loss of reactivity, a 5–10-g portion of white muscle was taken from the epaxial musculature behind the operculum and above the lateral line. The muscle samples were immediately freeze-clamped in pre-cooled tongs and then transferred into liquid nitrogen (Booth et al. 1995). This method of tissue preparation is the most appropriate method for preparing tissue samples for metabolite analyses (Wang et al. 1994). Excised hearts were removed from anesthetized fish, and all tissue samples were stored in liquid nitrogen until processing.

*Control group sampling.*—To obtain resting laboratory control values, largemouth bass were angled from lakes in southeastern Ontario and returned to the aquatic holding facility at Queen's University, Kingston, Ontario. Once in the laboratory, the fish were transferred to individual, darkened perspex boxes (within 1–2 d of entering the laboratory) supplied with aerated, dechlorinated municipal water for 24–48 h. Before sampling, the flow of water to the boxes was stopped, and a mixture of tricaine and  $\text{NaHCO}_3$  (see above for concentrations) was added to the individual boxes (Wang et al. 1994; Booth et al. 1995). Once fish had totally lost reactivity, they were sampled for blood and tissue in the identical manner used at tournaments.

A second control group was collected to obtain the blood cortisol levels of largemouth bass that are freely swimming in the wild. To obtain these values, largemouth bass from lakes in southeastern

Ontario were angled and sampled onboard a boat. Once hooked, the fish were quickly reeled into the boat, wrapped in a damp cloth, and submersed in a box containing aerated lake water. Blood was then quickly drawn from the caudal vessel using a syringe without an anesthetic. The samples were collected within 1 min of the fish being hooked, and the fish were subsequently released. Because plasma cortisol levels normally do not increase until at least a few minutes following the onset of a stressor (Wedemeyer et al. 1990), we felt that this sampling approach would enable us to obtain plasma cortisol values very similar to those in free-swimming fish.

*Plasma.*—Cortisol concentrations were determined using a commercially available radioimmunoassay kit (Coat-a-Count, Diagnostic Products Corporation, Los Angeles, California) with the appropriate intra- and interassay standards, and the determination of glucose concentrations in plasma followed the methods of Lowry and Passonneau (1972). Plasma osmolality was determined using a freezing-point depression osmometer (Advanced Instruments, Inc., model 3M0). Plasma sodium and potassium levels were quantified using a flame photometer (Corning Clinical Flame Photometer, model 410C; Wedemeyer and Yasutake 1977; Booth et al. 1995), while plasma chloride levels were assayed using a chloride titrator (Radiometer, Inc., model CMT 10; Wedemeyer and Yasutake 1977). Activity of creatine phosphokinase (creatinase; Enzyme Number 2.7.3.2; IUBMBNC 1992) in plasma was measured according to the methods described by Hørder et al. (1990). Plasma lactate concentrations were determined following the methods of Lowry and Passonneau (1972).

*White muscle tissue.*—Muscle tissue was prepared for metabolite assays according to the method of Booth et al. (1995). Approximately 1 g of frozen tissue was ground under liquid nitrogen using a mortar and pestle. Once ground, the tissue powder was transferred to a tared vial, and 4 mL of an ice-cold solution of 8% perchloric acid and 1 mM EDTA was added. After 10 min of incubation, this mixture was centrifuged, and the supernatant was divided into four, tared microcentrifuge tubes. A volume of neutralizing solution (containing 2 M KOH, 0.4 M KCl, and 0.3 M Imidazole) equal to 55% of the supernatant weight was added to each tube and, after centrifugation, the supernatant was transferred to 1.5-mL microcentrifuge tubes and stored in liquid nitrogen until sampling. Analyses of tissue lactate, phosphocreatine (PCr), and adenosine triphosphate (ATP)

concentrations were performed on the prepared muscle samples based on the enzymatic assay methods of Lowry and Passonneau (1972). An additional portion of muscle tissue was used to measure glycogen according to the method of Hassid and Abraham (1957).

To determine the quantity of water in white muscle, samples of frozen tissue (each weighing between 100 and 800 mg) were placed in a tared, 1.5-mL microcentrifuge tube. The tube was reweighed to determine the initial weight of the sample and was then transferred to an 80°C oven for several days. The tubes were monitored until a constant weight was obtained, and water content (%) was calculated.

*Heat-shock protein.*—The level of mRNA coding for heat shock protein 70 (HSP70) expressed by tournament-caught largemouth bass was compared with two control groups. One control group consisted of fish held in the laboratory in darkened perspex boxes sampled for blood and tissues (as described above) to serve as a negative control. The second control group consisted of largemouth bass that were held in the laboratory and subjected to an acute temperature shock to serve as a positive control. For this, largemouth bass were transferred to a holding tank containing aerated, dechlorinated municipal water at approximately  $18 \pm 2^\circ\text{C}$ . Over a 2-h period, the temperature of the water in the tank was increased to  $30 \pm 2^\circ\text{C}$  and maintained at this elevated temperature for approximately 2 h. Water temperatures were reduced to  $19 \pm 3^\circ\text{C}$ , and fish were left to recover for 1.5-h at  $19 \pm 3^\circ\text{C}$ . The fish were then transferred to anesthetic (at concentrations described above) and sampled for blood and tissues as mentioned earlier.

Total RNA was extracted from powdered white muscle, heart, and packed red blood cells by the acid-phenol method of Chomczynski and Sacchi (1987). Both white muscle and heart were washed in 4 mM LiCl (diethyl-pyrocyanate treated) prior to the final resuspension to reduce glycogen contamination. Total RNA was quantified with a Spectramax Plate Spectrophotometer (Molecular Devices, Sunnyvale, California) in triplicate.

For Northern blots, 10  $\mu\text{g}$  of total RNA was fractionated by glyoxal–dimethyl sulfoxide denaturing electrophoresis on a 1% agarose gel and transferred to a nylon membrane (Stratagene Duralon) using  $20\times$  standard saline citrate (SSC). Membranes were UV-crosslinked (Fisher UV Crosslinker) twice at the optimal setting prior to hybridization.

An HSP70 probe was generated using first-

strand cDNA reverse transcribed from total RNA obtained from largemouth bass blood. The HSP70 probe was a 238-bp fragment amplified at 60°C using forward and reverse primers designed for rainbow trout *Oncorhynchus mykiss*, as previously described (Currie et al. 1999). The probe was labeled using [ $\alpha$ - $^{32}$ P]dCTP (specific activity  $1 \times 10^9$  counts per minute/ $\mu$ g DNA) and the Ready-To-Go labeling system (Pharmacia). Membranes were prehybridized at 68°C for 30 min with QuickHyb Hybridization Solution (Stratagene). Blots were hybridized for 2 h in the same solution at 68°C, with approximately  $10^9$  cpm denatured probe. Blots were then washed twice with a  $1 \times$  SSC-0.1% sodium dodecyl sulfate (SDS) solution (15 min, 26°C), followed by an additional wash in  $0.1 \times$  SSC-0.1% SDS (30 min, 68°C). The blots were exposed to a phosphor screen (Kodak) and then visualized and quantified using a phosphoimager (Molecular Dynamics) driven by ImageQuant software. Band densities were expressed as a ratio of the band density of the sample containing the most HSP70 mRNA. All membranes were also probed with a human 18 S rRNA (Battersby and Moyes 1998) to correct blots for loading differences.

**Statistical analyses.**—The mean concentrations of plasma variables were compared across all sampling groups using a one-way analysis of variance (ANOVA), and the statistical differences in variable concentrations between individual tournaments and both control group means were detected using a Dunnett's test (Zar 1999). For tissue variables, the metabolite concentrations of tournament-caught largemouth bass were compared with those of laboratory control fish using a *t*-test. All statistical tests were performed using JMPIN Version 4.0 (SAS Institute), and the level of significance ( $\alpha$ ) for all tests was 0.05.

## Results

Live-release angling tournaments caused significant changes in almost all the variables monitored in this study. The plasma cortisol levels from the five bass tournaments were approximately 140 times greater than those for fish sampled immediately following angling, but were only about 2.5 times greater than the values in the laboratory controls (Figure 1A). The plasma glucose concentration for tournament-caught fish was also approximately double that measured in the laboratory control group (Figure 1B).

Live-release tournaments also caused a significant increase in the osmotic concentration of the

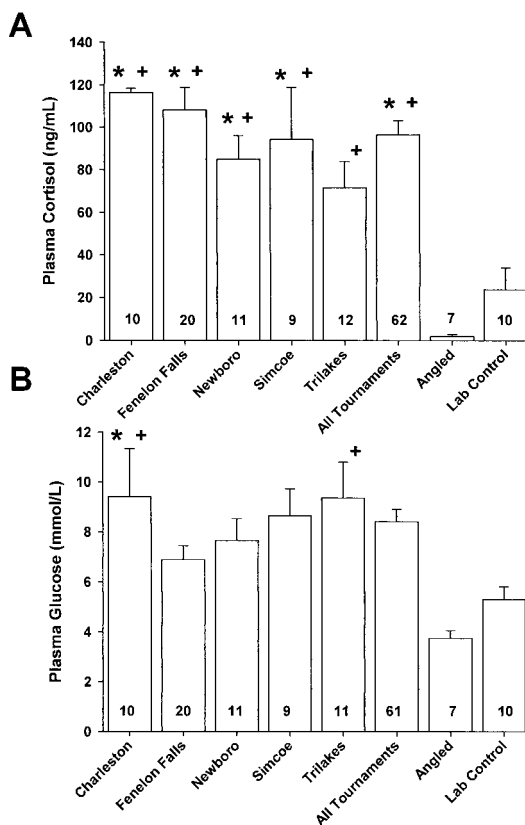


FIGURE 1.—Mean (+SE) (A) plasma cortisol and (B) plasma glucose concentrations for eight populations of largemouth bass. The sample size for each group is shown on its bars. Group means are significantly different (ANOVA,  $P < 0.05$ ). An asterisk indicates a tournament mean that is significantly different from the laboratory control mean (Dunnett's test,  $P < 0.05$ ), and a plus sign indicates a tournament mean that is significantly different from the angled mean (Dunnett's test,  $P < 0.05$ ).

plasma in largemouth bass (Figure 2A). The tournaments caused significant increases in plasma sodium (Figure 2B) and potassium concentrations (Figure 2C) relative to the laboratory control group. However, there were no significant differences in the plasma chloride concentration among the tournament and control groups (Figure 2D).

The metabolic status of largemouth bass sampled after the tournaments was also significantly different than that for bass sampled under laboratory conditions and immediately following angling. The plasma lactate concentrations in tournament-caught largemouth bass were about five times greater than those in both control groups (Figure 3). Similarly, the muscle lactate concentration in tournament bass

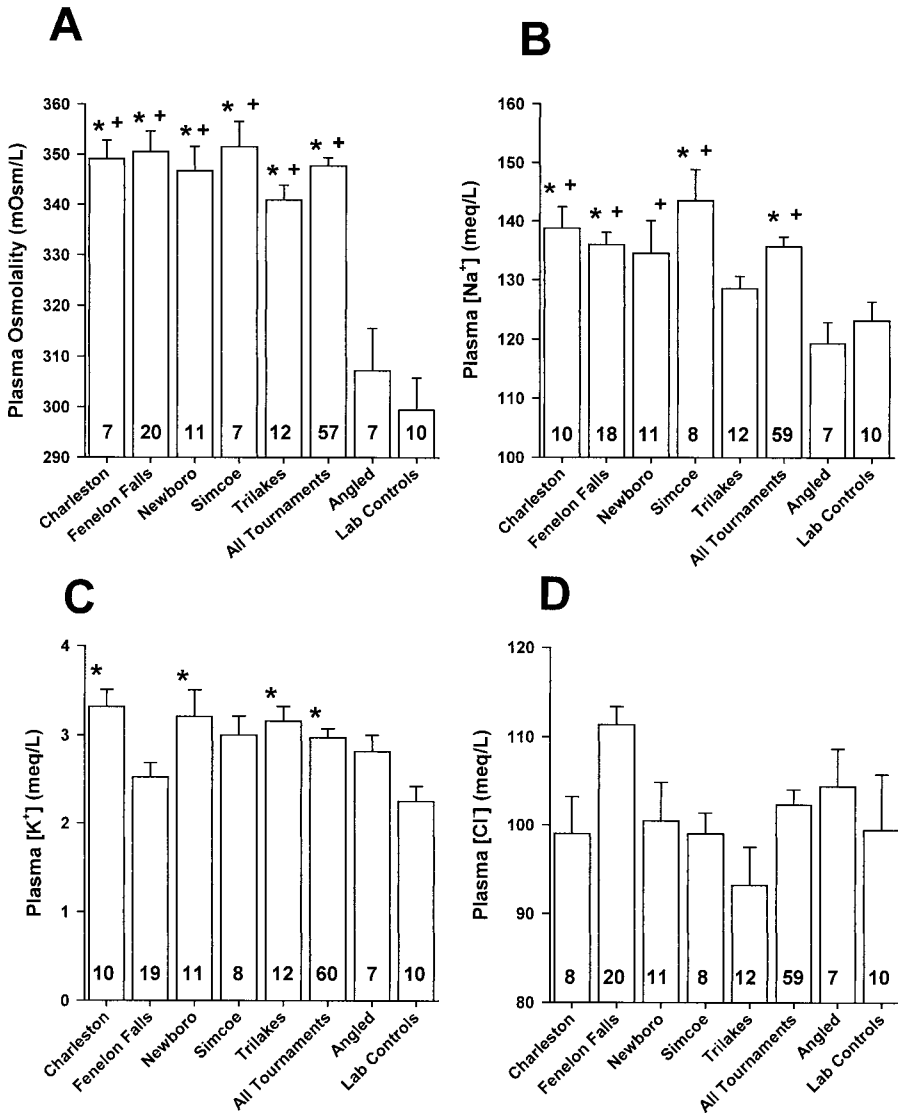


FIGURE 2.—Mean (+SE) (A) plasma osmolality, (B) plasma sodium, (C) plasma potassium, and (D) chloride concentrations for eight populations of largemouth bass. The sample size for each group is shown on its bars. Group means are significantly different (ANOVA,  $P < 0.05$ ). An asterisk indicates a tournament mean that is significantly different from the laboratory control mean (Dunnett's test,  $P < 0.05$ ), and a plus sign indicates a tournament mean that is significantly different from the angled mean (Dunnett's test,  $P < 0.05$ ).

was almost ten times greater than that in laboratory control individuals (Figure 4).

Tournament practices also caused significant reductions in the concentrations of several important muscle energy reserves. Specifically, the concentrations of white muscle PCr, ATP, and glycogen were reduced by 92, 60, and 75%, respectively, following the weigh-in (Figure 4). There was no significant difference, however, between the muscle water content of tournament-caught bass ( $798.0 \pm$

2.6 mL/kg wet weight) and that for the laboratory control bass ( $798.0 \pm 4.6$  mL/kg wet weight).

In contrast to the trends observed for most other variables, the tournaments did not cause a significant increase in the mRNA levels coding for HSP70 within any of the tissues examined (Table 1). The mRNA levels for HSP70 were significantly increased, however, in these tissues from largemouth bass exposed to an acute heat stressor as a positive control (Table 1).

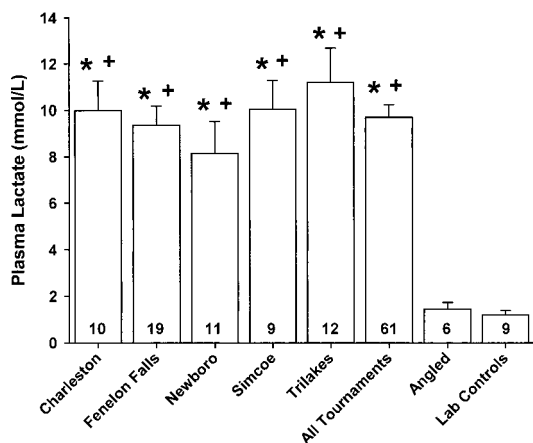


FIGURE 3.—Mean (+SE) plasma lactate concentration for eight populations of largemouth bass. The sample size for each group is shown on its bar. Group means are significantly different (ANOVA,  $P < 0.05$ ). An asterisk indicates a tournament mean that is significantly different from the laboratory control mean (Dunnett's test,  $P < 0.05$ ), and a plus sign indicates a tournament mean that is significantly different from the angled mean (Dunnett's test,  $P < 0.05$ ).

The level of CPK activity measured in the plasma of largemouth bass sampled immediately following angling was almost identical to that measured in fish sampled following the weigh-in of a live-release tournament, although neither value

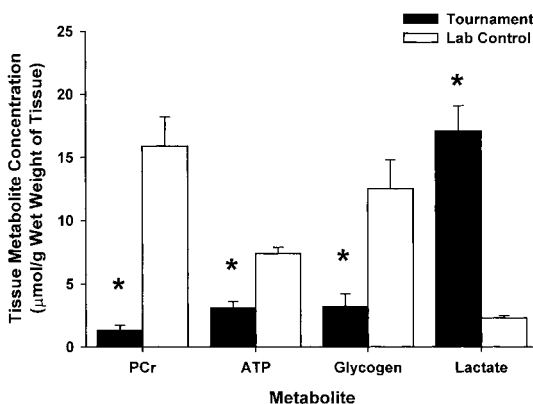


FIGURE 4.—Mean (+SE) white muscle metabolite levels for largemouth bass sampled following the weigh-in at a live-release angling tournament ( $n = 11$ ) and laboratory control (resting) largemouth bass ( $n = 8$ ). The abbreviation PCr stands for phosphocreatine, the abbreviation ATP for adenosine triphosphate. An asterisk indicates a tournament mean that is significantly different from the laboratory control mean (unpaired  $t$ -test,  $P < 0.05$ ).

TABLE 1.—Relative mRNA concentrations  $\pm$  SEs for Northern blots of heat shock protein 70 (HSP70) in blood, heart, and white muscle of largemouth bass sampled following the weigh-in at three live-release tournaments (tournament), following an acute, sublethal heat shock (heat shock), or resting quietly in darkened boxes in the laboratory (lab control). Means without letters in common were significantly different from laboratory controls ( $P < 0.05$ ).

Tissue	Treatment group	Relative mRNA content	Sample size
Blood	Tournament	0.03 $\pm$ 0.002 z	20
	Heat shock	0.4 $\pm$ 0.2 y	5
	Lab control	0.07 $\pm$ 0.012 z	8
Heart	Tournament	0 $\pm$ 0 z	11
	Heat shock	0.6 $\pm$ 0.1 y	4
	Lab control	0 $\pm$ 0 z	8
White muscle	Tournament	0.05 $\pm$ 0.04 z	11
	Heat shock	0.6 $\pm$ 0.2 y	5
	Lab control	0 $\pm$ 0 z	8

was significantly different from that in laboratory control fish (Table 2).

**Discussion**

The elevated levels of plasma cortisol, glucose, and osmolality observed after tournaments in the present study were comparable to those obtained for largemouth bass exposed to other disturbances, such as electroshock and transport (Carmichael et al. 1984; Davis and Parker 1986). In the past, plasma cortisol, glucose, and osmolality were among the commonly measured variables used to assess the general physiological condition of fish, and to provide insight about the degree of stress these animals might be experiencing (Carmichael et al. 1984; Wedemeyer et al. 1990; Gustaveson et al. 1991; Booth et al. 1995; Wendelaar Bonga 1997). The fact that these three plasma constituents were all significantly increased from control (resting) levels following live-release angling tournaments

TABLE 2.—Mean  $\pm$  SE creatine phosphokinase (CPK) activity from plasma of largemouth bass sampled following the weigh-in of a live-release angling tournament (tournament;  $N = 47$ ), resting in darkened boxes in the lab (laboratory control;  $N = 8$ ), and sampled immediately following angling (angled;  $N = 7$ ). Differences across groups were not statistically significant (ANOVA;  $P = 0.34$ ).

Treatment group	CPK activity ( $\mu\text{mol NADH}\cdot\text{min}^{-1}\cdot\text{mL plasma}^{-1}$ )
Tournament	4.8 $\pm$ 1.3
Angled	5.1 $\pm$ 2.2
Laboratory control	0.4 $\pm$ 0.2

indicates that these events cause a significant physiological disturbance in largemouth bass. Although posttournament cortisol values were several orders of magnitude greater than those in free-swimming fish (i.e., angled controls in Figure 1), it is interesting to note that they were only about double the values observed in resting control bass under laboratory conditions. These findings suggest that the latter group may have been experiencing some degree of stress, possibly from the holding and confinement protocol. The cortisol results from our laboratory controls also indicate that plasma cortisol levels in wild largemouth bass are probably very responsive to any type of disturbance.

The increase in plasma osmolality in largemouth bass following tournaments was similar to that which has been previously documented following acute disturbances such as exhaustive exercise and angling (Gustaveson et al. 1991; Wood 1991). In the present study, this increase in plasma osmolality can be attributed to significant increases in a number of plasma variables (including glucose, lactate, sodium, and potassium). In contrast to these variables, however, the plasma chloride levels in largemouth bass following tournaments were not significantly different from those of either control group. The status of plasma electrolytes (such as sodium and chloride) may provide some insight into the nature of the physiological disturbance following tournaments. As explained by McDonald and Milligan (1992), acute stresses such as the exhaustive exercise associated with angling, struggling, or capture have been shown to cause a short-term elevation in plasma sodium and chloride levels in fish, largely due to fluid shifts between the intracellular and extracellular space. In contrast, prolonged stressors will result in net losses of these ions across sites such as the gills and kidneys, and a significant decrease in their concentrations in plasma. For example, Carmichael et al. (1984) documented significant losses of chloride ions from largemouth bass subjected to transport stress. In our study, however, none of the tournaments examined caused any significant reduction in the concentrations of plasma sodium and chloride in largemouth bass. The absence of any significant reductions in the plasma concentrations of these ions may be partially attributed to the fact that the gill permeability of black bass appears to be lower than that of other teleosts (McDonald et al. 1991). It is also possible that the timing of our sampling may have had an important impact on our plasma ion values. Future studies should prob-

ably further investigate this issue by monitoring fish for several hours following tournaments to determine whether significant ion losses are evident once any fluid shifts caused by acute stresses (such as the weigh-in) have subsided. Nonetheless, our results indicate that tournaments probably do not normally cause a level of chronic stress in largemouth bass that is severe enough to cause profound reductions in plasma ions.

The metabolic disturbance in largemouth bass following tournaments was very similar to that in fish that have been exercised to exhaustion (Wang et al. 1994; Kieffer et al. 1995; Milligan 1996). In tournament-caught largemouth bass, muscle energy reserves—PCr, ATP, and glycogen—were greatly reduced, while blood and muscle lactate concentrations increased (Figure 5 and 6). The metabolic status of these fish after tournaments is somewhat surprising because many hours probably passed since the fish were angled. Moreover, fish caught in tournaments are not typically angled to the point of exhaustion. Our results therefore suggest that other factors (such as live-well confinement, handling, and air exposure during weigh-in) may also play an important role in the metabolic disturbance in tournament fish. At this time, however, the relative contribution of the different practices within tournaments towards this posttournament metabolic disturbance is not known.

Although HSP mRNA levels are rapid and sensitive indicators of cell stress (Lis and Wu 1994), HSP70 mRNA failed to change in any of the tissues we examined from the largemouth bass following tournaments (Table 1). HSPs are a highly conserved group of proteins that have been shown to be induced in different organisms following exposure to a wide range of stresses, including heat shock, restraint stress, hypoxia, ischemia, and exposure to heavy metals (Blake et al. 1991; Parsell and Lindquist 1993; Udelsman et al. 1993; Mestrlil and Dillman 1995). In recent years, increases in the mRNA or protein levels of inducible HSPs (such as HSP70) have therefore often been used as an indicator of cellular stress in a wide range of vertebrates, including fish (Iwama et al. 1998, 1999; Feder and Hofmann 1999). Because protein misfolding and aggregation are believed to be the primary initiators of the HSP response, our results indicate that live-release tournaments probably do not commonly expose largemouth bass to stressors that result in significant damage to cellular proteins (Anathan et al. 1986; Baler et al. 1992; McDuffee et al. 1997).

The magnitude of the increase in plasma CPK

activity in largemouth bass following tournaments was much less than that which has been shown to occur in other species following severe disturbances (Powell et al. 1974; Clarkson et al. 1992). CPK is an intracellular enzyme that is present in many different vertebrate tissues, including heart, muscle, and brain. In response to stressors such as severe exercise, the plasma levels of CPK in humans have been found to increase many thousand times above resting levels as a result of cell damage to skeletal muscle (Clarkson et al. 1992). The posttournament increase in plasma CPK activity that we observed in largemouth bass was only about 10-fold higher than that in the laboratory control largemouth bass, and not significantly different from either of the two control groups. The plasma CPK activity in fish following tournaments was also very similar to the plasma activity in largemouth bass immediately sampled after angling (Table 2). Taken together, our CPK results suggest that tournaments may cause some cell damage in largemouth bass, damage which may be comparable to that caused by angling. Because the laboratory control fish used in our study were obtained by angling 24–48 h prior to sampling, our results also indicate that recovery from these disturbances seems to occur quickly.

In summary, our results clearly indicate that live-release angling tournaments caused a significant physiological disturbance in largemouth bass. This disturbance is characterized by increases in established indicators of stress in fish such as plasma cortisol and glucose, short-term increases in osmolality, decreases in muscle energy reserves, and increases in blood and muscle lactate levels. However, we obtained no evidence that serious cell damage in largemouth bass is a normal consequence of tournaments. More specifically, the levels of HSP70 mRNA in several tissues were unchanged after tournaments, and plasma CPK levels were only increased to levels similar to those in quickly angled bass, which are known to fully recover.

The results of this study also have important implications for tournament organizers and fisheries managers. Our findings indicate that the profound metabolic disturbance is probably one of the most important physiological consequences of the current tournament format for largemouth bass. Although angling and other forms of physical activity may contribute to this disturbance, it is likely that hypoxia is also an extremely important factor in this regard. Based on our experience, there are many locations in the current tournament format

where hypoxia can potentially be a significant problem. Strategies to ensure that sufficient oxygenation is constantly provided in areas such as live wells, weigh-in bags, holding tanks, and release vessels and to minimize the periods of air exposure during the weigh-in should be viewed as extremely important, as they would probably reduce the magnitude of this metabolic disturbance. Effective strategies that minimize the magnitude of this disturbance would also probably reduce the overall stress levels in bass, and, in turn, fish mortality and other potentially important sublethal impacts of stress (Barton and Iwama 1991) that may arise from these events.

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