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[Article]

Incidence and Physiological Consequences of Decompression in Smallmouth Bass after Live-Release Angling Tournaments

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Abstract.—Decompression can be an important problem for fish that are rapidly brought to the surface. The main objectives of this study were to (1) examine the incidence of external signs of decompression in smallmouth bass *Micropterus dolomieu* after live-release angling tournaments on lakes with different depths, (2) determine the physiological changes in smallmouth bass that exhibit external decompression signs, and (3) identify the best methodological approaches for evaluating this type of disturbance. Our results indicate that decompression does not cause problems for smallmouth bass when tournaments are held on relatively shallow lakes. However, when tournament anglers have access to deep water (>5 m), significant numbers of smallmouth bass may exhibit decompression signs after these events. Decompression signs include swim bladder overinflation, increased plasma lactate, and increased activity of tissue enzymes in plasma. Tournament-caught smallmouth bass exhibiting external signs of decompression also experience internal physiological changes, including significant elevations in the plasma levels of intracellular enzymes (lactate dehydrogenase [LDH]; enzyme number 1.1.1.27; creatine phosphokinase [CPK]; 2.7.3.2; and aspartate aminotransferase [AST]; 2.6.1.1), red blood cell lysis, and a larger anaerobic disturbance after the weigh-in than observed in nondecompressed smallmouth bass. Additional laboratory experiments indicate that plasma AST activity may be the most useful indicator of the extent of tissue damage in decompressed smallmouth bass because it is highly correlated to plasma LDH and CPK levels but is not influenced by the blood sampling method.

The practice of voluntarily releasing fish after angling has grown considerably over the past several decades (Quinn 1996). Numerous studies have shown that the vast majority of fish released after angling survive and may be caught again (Green et al. 1987; Lee 1987; Quinn 1989; Gustaveson et al. 1991; Muoneke and Childress 1994; Booth et al. 1995). Live release has also become an integral part of the angling tournament industry in North America, and black basses *Micropterus* spp. have proven to be the most popular target for these events (Shupp 1979; Duttweiler 1985; Schramm et al. 1991). Although most fish released after angling tournaments appear to survive (Wilde 1998; Killen et al. 2003; Suski et al. 2003), significant mortality has also been documented after some tournaments (Wilde 1998).

One stressor that has the potential to affect bass during angling tournaments is decompression (i.e., when fish hooked at depth are quickly brought to the surface). Smallmouth bass *M. dolomieu* are more likely to experience decompression than largemouth bass *M. salmoides* because smallmouth bass typically reside in deeper habitats than do largemouth bass (Coble 1975; Heidinger 1975). This difference in susceptibility to decompression may be one reason why smallmouth bass experience greater mortality at angling tournaments than do largemouth bass (Bennett et al. 1989; Hartley and Moring 1995).

The impact of decompression in fish has been the subject of several previous studies (Gotshall 1964; Feathers and Knable 1983; Lee 1992; Keniry et al. 1996; Shasteen and Sheehan 1997). Most of these studies, however, have focused on initial or delayed mortality after decompression in largemouth bass or have examined the effects of swim bladder puncture (i.e., ''fizzing'') on the survival of fish experiencing decompression. At the present time, virtually nothing is known about the sublethal physiological impacts of decompression in any of the black bass species after angling tournaments. There has also been no attempt to determine the incidence of decompression signs in smallmouth bass during angling tournaments, despite the fact that this species is probably much more susceptible than largemouth bass to this type of disturbance.

There is clearly a need for additional information about the incidence and physiological consequences of decompression in fish species that

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are targeted by angling tournaments. The present study had three main objectives. First, we examined the incidence of external signs of decompression (Feathers and Knable 1983) in smallmouth bass in two tournaments: one where the fish were mainly caught from shallow water and another where the fish were caught from deep water. Next, we examined the nature of the sublethal physiological disturbance in smallmouth bass that exhibited external signs of decompression. Finally, we also sought to identify the best methodological approaches for evaluating these disturbances in future studies. Based on information from tournament organizers and participants, we hypothesized that a significant proportion of tournament-caught smallmouth bass would exhibit signs of decompression when these events were held on lakes where anglers have access to water deeper than 5 m. We also hypothesized that there would be significant sublethal physiological changes in tournament-caught smallmouth bass exhibiting external signs of decompression.

Methods

Incidence of decompression at tournaments.—A tournament during the summer of 2002 held on Lakes Erie and St. Clair in southwestern Ontario and northern Michigan was selected for the first part of the study to quantify the incidence of decompression at angling tournaments. In this paper, we consider deep water to be depths in excess of 5 m, and information from tournament organizers indicated that many of the smallmouth bass targeted by anglers in this event would probably be caught from depths greater than 5 m (Lake Erie has a maximum depth of 18.9 m and a mean depth of 7.4 m). In this tournament, anglers competed for the greatest combined weight within a limit of five fish in any combination of smallmouth bass and largemouth bass. Surface water temperature during the tournament in the nearshore area was 21°C.

For this portion of the study, smallmouth bass that had completed the weigh-in were observed prior to being placed in the tournament live-release boat. Each fish was visually assessed for the external signs of decompression trauma described by Feathers and Knable (1983). These signs were bloating (swim bladder overinflation) and hemorrhaging inside of the mouth (gums), on the body surface, or within the dorsal, caudal, anal, pelvic, and pectoral fins. Bloating was scored as none, moderate (moderately enlarged body), or severe (greatly deformed body). Hemorrhaging was also scored as none, moderate (only a light pink color was visible over less than half of the surface area of the body part in question), or severe (a deep red color was observed, or hemorrhaging covered more than half of the body part in question).

For the purposes of comparison, we also recorded signs of decompression in smallmouth bass at a tournament held on Rice Lake in southern Ontario, a relatively shallow lake that had a mean depth of 3 m and a maximum depth of 7.9 m. This tournament was conducted in a manner similar to that described above. Using the same visual assessment procedures outlined above, we recorded the presence or absence of decompression signs in 103 smallmouth bass at this tournament.

Sampling technique experiment.—For the second experiment, smallmouth bass showing no external signs of decompression were collected from a tournament on Rice Lake, where decompression had previously been noted as rare. These fish were transported in a large, aerated container to Queen's University in Kingston, Ontario, where they were allowed to recover and acclimate to a temperature of 20°C for 2 weeks. These fish were then individually removed from their holding tank with a rubber net, and approximately 2 mL of blood was drawn by caudal puncture (Houston 1990) with 22-guage needles rinsed with heparinized physiological saline. A similar volume of blood was then immediately withdrawn from the vessels in the gills of the same fish by means of a new needle and syringe. The blood taken by both sampling techniques was then transferred to 1.5-mL microcentrifuge tubes and was spun at 10,000 × gravity for 2 min. The plasma was removed, and both the plasma and the red blood cell (RBC) pellets were stored immediately on dry ice until they could be transferred to a −80°C freezer. Anesthetic was not used during blood sampling in this experiment so that these samples could be directly compared to samples taken at tournaments, where fish are generally lethargic, making anesthetic unnecessary (Suski et al. 2003). Immediately after sampling, all fish were quickly killed by a sharp blow to the head.

Tournament sampling for physiological variables.—For the third experiment in this study, smallmouth bass were sampled after three different angling tournaments on Lake Simcoe, central Ontario, in September 2002. The tournament format was similar to that described above. Lake Simcoe is another lake where smallmouth bass are commonly caught from significant depth. Information from anglers and tournament organizers also in-

dicated that smallmouth bass often showed external signs of decompression after capture on this lake. Smallmouth bass were selected for sampling on the basis of whether they displayed the external signs of decompression. Fish showing no signs of decompression (i.e., no bloating or hemorrhaging) were assigned to a ''not decompressed'' sampling group, and fish showing several of the signs were assigned to a ''decompressed'' group. The water temperature during these three tournaments ranged from 15°C to 18°C.

Immediately after the weigh-in, tournament-caught smallmouth bass were taken aside and held temporarily in aerated tanks. Both decompressed and nondecompressed fish were then sampled by withdrawing blood from the vessels of the gill arches (as described above); our previous experiment found that this was the most appropriate blood sampling technique for monitoring indicators of cell damage. All of the blood samples were processed as described above and then were transported to Queen's University for storage and analyses.

Some samples that had previously been collected by caudal puncture from decompressed and nondecompressed smallmouth bass were also included in this study. These samples were only analyzed for plasma aspartate aminotransferase (AST; enzyme number 2.6.1.1; IUBMB 1992) activity, plasma osmolality, and concentrations of hemoglobin, lactate, chloride, and cortisol, since these variables were not affected by blood sampling technique (see below). The tournaments where these samples were collected were held on Lakes Ontario, Erie, and St. Clair, and water temperature ranged from 21°C to 23°C.

Samples taken from smallmouth bass by gill puncture in the sampling technique experiment were used as controls in this part of the study for comparison against samples from smallmouth bass that showed external signs of decompression at tournaments. These fish had been acclimated to a temperature (20°C) that was intermediate to the temperatures at the different tournaments we visited.

Red blood cell experiments.—Because the nucleated RBCs of fish contain metabolic enzymes, hemolysis (rupturing of RBCs) could also contribute to the plasma activities of the enzymes we measured. We therefore conducted an additional experiment to determine the relationship between the concentration of hemoglobin and the activities of lactate dehydrogenase (LDH; 1.1.1.27), creatine phosphokinase (CPK; 2.7.3.2), and AST in RBC lysates from smallmouth bass. For this experiment, solutions of RBCs (1% by volume) in physiological saline (Houston 1990) were produced from four different smallmouth bass. These solutions were then briefly sonicated (Virsonic, The Virtis Company, Gardiner, New York), and the activities of LDH, CPK, and AST, as well as the concentrations of hemoglobin, were determined. Ratios of the three enzyme activities to the hemoglobin concentration were then calculated.

Plasma analyses.—Activities of LDH, CPK, and AST in plasma samples were analyzed spectrophotometrically based on standard clinical techniques (Wroblewski and LaDue 1955 for LDH; Hørder et al. 1990 for CPK; Yagi et al. 1985 for AST). Plasma hemoglobin was measured based on the method of Eilers (1967) as a marker of damage to RBCs, because hemolysis was observed during visual inspection of samples taken from decompressed fish in the tournament sampling experiment (see below). Plasma lactate concentration was measured spectrophotometrically by means of the technique of Lowry and Passonneau (1972). Plasma osmolality was determined with a freezing-point depression osmometer (Model 3M0, Advanced Instruments, Inc., Norwood, Massachusetts), and plasma chloride concentration was assayed by use of a chloride titrator (Model CMT 10, Radiometer, Inc., Copenhagen, Denmark). The plasma cortisol concentration was measured by competitive protein binding performed with a commercially available kit (Coat-a-Count, Diagnostic Products Corp., Los Angeles, California).

Statistical analyses.—Data from the investigation quantifying the incidence of decompression signs at tournaments were not analyzed statistically. In the sampling technique experiment, plasma enzyme activity levels collected via gill and caudal puncture from the same fish were compared by use of a paired *t*-test. Treatment groups in the tournament sampling portion of the study were compared by a one-way analysis of variance followed by a Fisher's post-hoc test to detect differences between treatment groups. Plasma activity levels of intracellular enzymes were compared to each other by means of regression analysis. The level of significance (α) for all tests was 0.05.

Results

Incidence of External Signs of Decompression

After the tournament on Rice Lake, which is relatively shallow, 1.9% of 103 smallmouth bass sampled exhibited two or more signs of decom-

TABLE 1.—The effects of two different sampling techniques on plasma enzyme activity level (units/L ± SE) in smallmouth bass ($N = 6$ fish/group). An asterisk denotes a significant difference between sampling techniques (paired *t*-test; $P < 0.05$).

Enzyme	Gill puncture	Caudal puncture
Lactate dehydrogenase	27.3 ± 6.8	428.2 ± 127.1*
Creatine phosphokinase	26.4 ± 3.4	593.7 ± 187.9*
Aspartate aminotransferase	14.4 ± 3.3	16.0 ± 3.4

pression. In contrast, of the 168 smallmouth bass evaluated at the deepwater tournament on Lakes Erie and St. Clair, 56.5% showed two or more signs of decompression. The most common symptom of decompression was hemorrhaging from within the mouth, which occurred in 66.2% of the smallmouth bass. Hemorrhaging from the caudal fin and swim bladder distention were also common, occurring in 58.6% and 29.3% of live fish, respectively. Hemorrhaging from the anal, pectoral, and dorsal fins occurred in 10–50% of the fish. Hemorrhaging from the pelvic fins and body was relatively rare. Some (<20%) instances of bloating and hemorrhaging from the mouth, caudal fin, and pectoral fins were classified as severe.

Evaluation of Sampling Techniques

Plasma activities of LDH and CPK in samples taken by caudal puncture were significantly higher than those of samples drawn from the vessels of the gills (Table 1). However, there was no difference in plasma AST activity between the two sampling techniques.

Physiological Consequences of Decompression

Indicators of tissue damage in decompressed tournament fish were elevated relative to those of nondecompressed tournament fish and controls. Mean plasma LDH, CPK, and AST activities of decompressed smallmouth bass at tournaments were elevated by 3.4-, 2.6-, and 2.0-fold, respectively, over values for nondecompressed tournament fish (Figure 1). Mean plasma LDH, CPK, and AST activities of decompressed smallmouth bass at tournaments were elevated by 15-, 17-, and 4.7-fold, respectively, over control values.

Because the trends in AST activity closely paralleled those in LDH and CPK activities, we also evaluated the utility of measuring only AST activity as a nonlethal indicator of tissue damage. As noted above, acquisition of reliable LDH and CPK activities requires lethal sampling, but reliable AST values can be obtained by nonlethal sampling (caudal puncture). We therefore examined the correlations between plasma AST, LDH, and CPK activities in all of the fish that we sampled by gill puncture in this study (controls, as well as both groups of tournament fish). Plasma LDH and CPK activities were both significantly correlated with plasma AST activity (Figure 2).

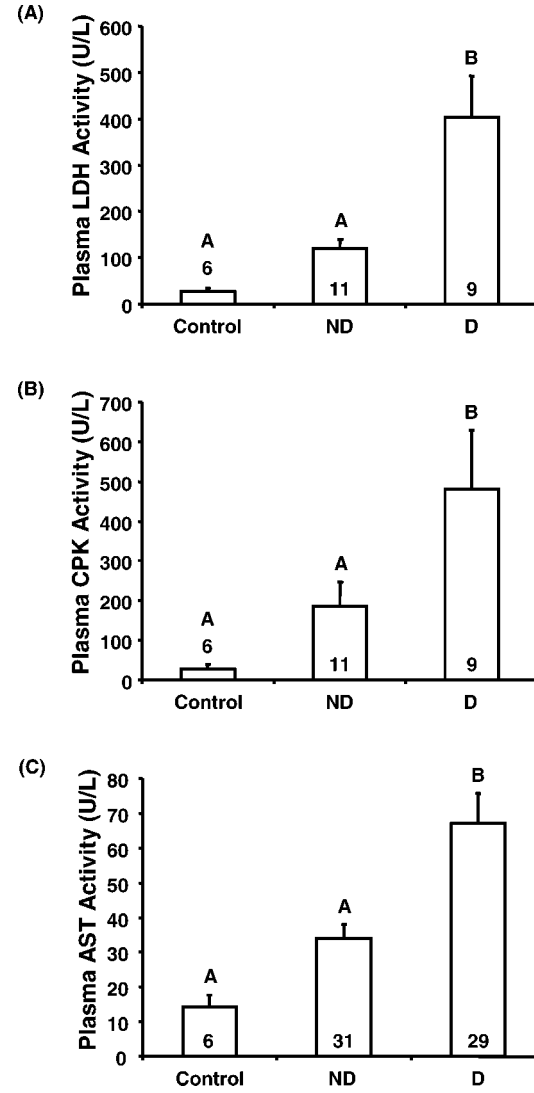

FIGURE 1.—**(A)** Lactate dehydrogenase (LDH), **(B)** creatine phosphokinase (CPK), and **(C)** aspartate aminotransferase (AST) activities (units/L [U/L]) in the plasma of control, nondecompressed (ND), and decompressed (D) smallmouth bass after live-release angling tournaments in southern Ontario. Sample sizes are displayed numerically on each bar. Different letters represent significant differences between groups (Fisher's post hoc test; $P < 0.05$).

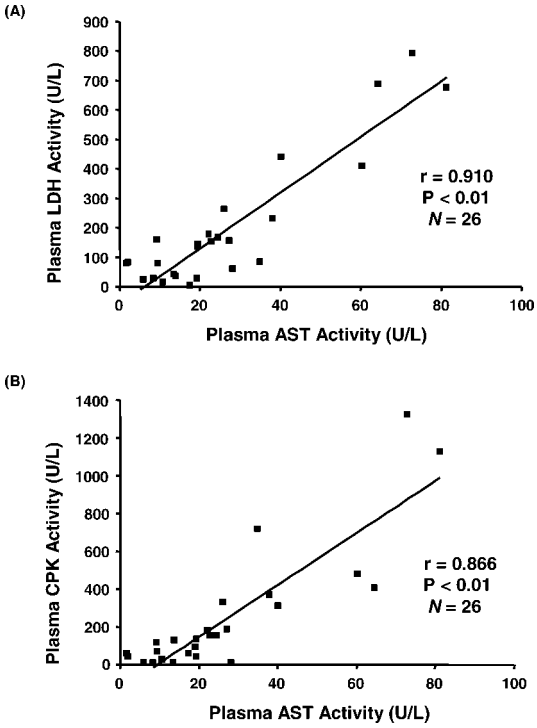

FIGURE 2.—Relationship between aspartate aminotransferase (AST) activity (units/L [U/L]) and **(A)** lactate dehydrogenase (LDH) activity or **(B)** creatine phosphokinase (CPK) activity in the plasma of smallmouth bass caught during live-release angling tournaments in southern Ontario. The *P*-values displayed on the figures denote the significance of the correlations.

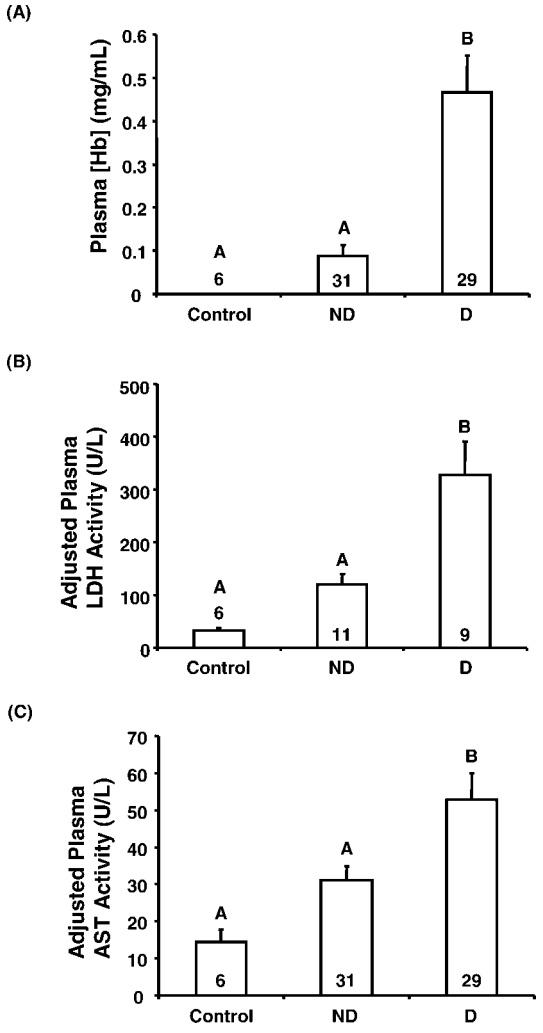

FIGURE 3.—**(A)** Plasma hemoglobin (Hb) concentration, **(B)** plasma lactate dehydrogenase (LDH) activity (units/L [U/L]) adjusted for contributions from lysed red blood cells, and **(C)** adjusted plasma aspartate aminotransferase (AST) activity in control, nondecompressed (ND), and decompressed (D) smallmouth bass after live-release angling tournaments in southern Ontario. Sample sizes are displayed numerically on each bar. Different letters represent significant differences between groups (Fisher's post hoc test; $P < 0.05$).

Plasma hemoglobin concentration in decompressed tournament smallmouth bass was higher than that of control fish and nondecompressed tournament fish (Figure 3A). The results of the experiment investigating the impact of hemolysis on plasma indicators of tissue damage indicated that hemolysis increased the plasma activities of LDH and AST (Table 2). Adjustment of plasma LDH and AST activities for contributions of these enzymes from hemolyzed RBCs significantly reduced their values in most cases (compare Figure 1A, C with Figure 3B, C). Despite these reductions, the overall trends in plasma LDH and AST activities were not changed by the adjustments (Figure 1A, C versus Figure 3B, C).

Plasma cortisol concentration was significantly higher in tournament-caught smallmouth bass than in control fish but was not significantly different between the two groups of tournament-caught fish (Figure 4A). Similarly, plasma osmolality and chloride concentration in tournament-caught smallmouth bass were significantly higher than control values (except for plasma chloride in decompressed fish, where $P = 0.051$; Figure 4B, C). However, there were no significant differences between the two tournament fish groups for either plasma osmolality or plasma chloride.

Plasma lactate concentration was elevated in both tournament groups relative to that of control

TABLE 2.—Activities of lactate dehydrogenase (LDH), creatine phosphokinase (CPK), and aspartate aminotransferase (AST) (units/L [U/L] ± SE) and the ratio of each to hemoglobin (Hb) concentration in the red blood cells of smallmouth bass ($N = 4$). All measurements were performed on 1% (by volume) solutions or lysed red blood cells (ND = not detectable).

Variable	Activity or concentration	Ratio (U/L per mg/L of Hb)
LDH	277.2 ± 34.5 U/L	313.5
CPK	ND	
AST	23.8 ± 1.3 U/L	26.9
Hb	0.884 ± 0.0157 mg/mL	

smallmouth bass (Figure 5). Furthermore, for tournament-caught smallmouth bass, decompressed fish had a significantly higher plasma lactate concentration than did nondecompressed fish (Figure 5).

Discussion

This is the first study to examine the incidence and consequences of decompression in smallmouth bass after live-release angling tournaments. Our results indicate that the vast majority of tournament-caught smallmouth bass do not exhibit any external signs of decompression when these events are held on relatively shallow lakes. In contrast, when tournament anglers have access to deep water, a significant proportion of the smallmouth bass caught may exhibit external decompression signs. These results are consistent with the findings from earlier studies on largemouth bass (Feathers and Knable 1983; Lee 1992; Shasteen and Sheehan 1997). The findings support our general observation based on several years of research at tournaments, that this is only a significant concern on deep water bodies. Unlike largemouth bass, however, smallmouth bass are frequently found in deep water when it is available. It is therefore likely that decompression is a much more important issue for tournament-caught smallmouth bass than for largemouth bass.

The specific external changes in smallmouth bass that we report in this study are consistent with the signs of decompression previously described by Feathers and Knable (1983) for largemouth bass. We also found that in smallmouth bass showing external signs of decompression, the signs did not occur with equal frequency. Hemorrhages from the tail and from inside the mouth were the most common external signs of decompression observed in these fish. In situations where it is not possible to monitor a wide range of variables, these signs might therefore serve as useful indicators of decompression in this species.

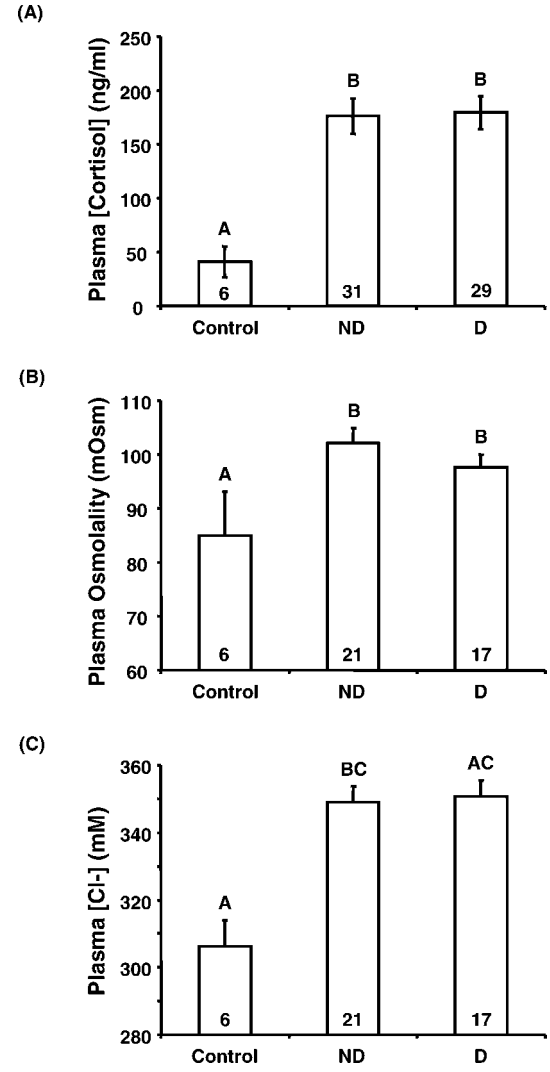

FIGURE 4.—**(A)** Plasma cortisol concentration, **(B)** plasma osmolality (milliosmols [mOsm]), and **(C)** plasma chloride concentration in control, nondecompressed (ND), and decompressed (D) smallmouth bass after live-release angling tournaments in southern Ontario. Sample sizes are displayed numerically on each bar. Different letters represent significant differences between groups (Fisher's post hoc test; $P < 0.05$).

Decompression also appears to cause significant physiological changes in smallmouth bass. Fish exhibiting external signs of decompression had significantly elevated plasma levels of LDH, CPK, and AST (Figure 1). The appearance of these intracellular enzymes in plasma is a commonly used

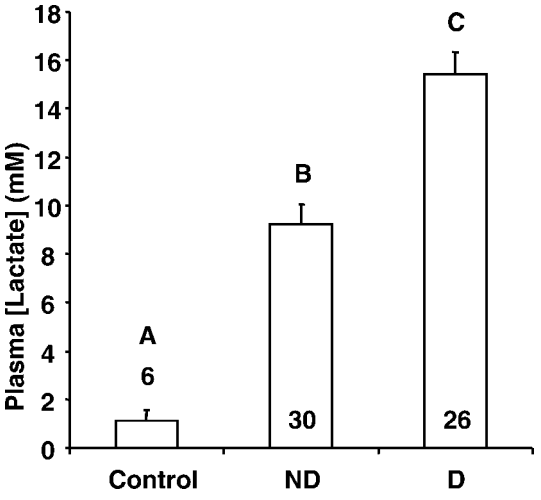

FIGURE 5.—Plasma lactate concentration in control, nondecompressed (ND), and decompressed (D) smallmouth bass after live-release angling tournaments in southern Ontario. Sample sizes are displayed numerically on each bar. Different letters represent significant differences between groups (Fisher's post hoc test; $P < 0.05$).

indicator of tissue damage in vertebrates (Freeman and Philp 1976; Kolesari and Kindwall 1982; Folmar 1993). The changes we observed are similar in magnitude to those produced by decompression in mammals (Powell et al. 1974; Freeman and Philp 1976). These changes are also roughly similar to those observed after other disturbances in fish, such as attack by sea lamprey *Petromyzon marinus* (Edsal and Swink 2001) and exposure to toxins (Folmar 1993; Folmar et al. 1993). The extent to which damage to different tissues contributes to the elevation of these enzymes in the plasma of smallmouth bass is difficult to determine. Lactate dehydrogenase could originate from almost any tissue, whereas CPK probably arises from heart or skeletal muscle and AST is predominantly located in the heart and liver. Since each of these enzymes exists in heart tissue, it is possible that much of the tissue damage observed in decompressed smallmouth bass after tournaments occurs in the heart. Evidence from other vertebrates, however, suggests that multiple tissues are probably affected by decompression (Freeman and Philp 1976). Further study will therefore be required to determine which tissues are most affected by decompression in smallmouth bass after tournaments. Nonetheless, the observed increases in the plasma levels of these intracellular enzymes provide convincing evidence that the smallmouth bass exhibiting external decompression symptoms have also experienced some degree of tissue damage.

The evidence of tissue damage in decompressed smallmouth bass may have important implications for some of the strategies that have been proposed to deal with this issue. In previous studies, for example, it has been suggested that deflation of the overinflated swim bladder may be a useful strategy for resolving some of the problems associated with decompression in fish (Gotshall 1964; Lee 1992; Keniry et al. 1996). Although this practice may provide some benefits to decompressed fish by allowing them to return to depth, it would probably have little impact on any tissue damage that has already occurred. This may be especially relevant in tournament situations, where fish are retained at surface pressures for several hours prior to release.

The levels of LDH and CPK measured in the plasma of smallmouth bass were influenced by the blood sampling technique. A parallel experiment showed that LDH and CPK activities in plasma were about 20 times greater in samples taken by caudal puncture than in samples taken by gill puncture (Table 1). This probably reflects the fact that there is some degree of damage to muscle cells, which contain these enzymes, during caudal puncture. In contrast, additional cell damage is minimized when blood samples are obtained via the gill. In view of these findings, all of the samples taken from tournament fish for analyses of LDH and CPK were obtained by gill puncture (Figures 2, 4). In contrast, plasma AST activity in smallmouth bass was not significantly influenced by blood sampling technique (Table 1). Hence, our analyses of plasma AST levels in tournament-caught smallmouth bass included samples that had been obtained by both sampling techniques (Figures 1C, 3C). Although gill puncture appears to be a superior sampling technique for analyses of all three of these cell damage indicators, it is extremely difficult to obtain blood samples in this manner without killing the fish. It is therefore important to note that the plasma AST activity in smallmouth bass seems to be a relatively good predictor of plasma LDH or CPK activity (Figure 2). Thus, future studies in this area should consider the fact that plasma AST activity may be one of the best indicators of the extent of cell damage caused by decompression in fish, because it can be accurately measured after nonlethal sampling by caudal puncture.

Smallmouth bass showing external symptoms of

decompression also had significantly elevated plasma hemoglobin levels (Figure 3A). This finding was unexpected because there is no previous evidence that decompression causes damage to RBCs. Since the nucleated RBCs of fish contain metabolic enzymes (Phillips et al. 2000), we conducted an additional experiment to determine whether the hemolysis observed in decompressed smallmouth bass could account for the observed increases in plasma levels of LDH, CPK, and AST. This experiment indicated that only LDH and AST were within our limits of detection in the RBCs of smallmouth bass (Table 2). Correction of the observed plasma levels of LDH and AST for the percentages of these enzymes that could result from hemolysis also had little impact on the observed trends for these variables (Table 2; Figure 3B, C). The additional presence of hemolysis in decompressed smallmouth bass therefore does not alter our conclusion that these animals have experienced a significant degree of tissue damage. Indeed, the significant increase in plasma hemoglobin levels provides evidence that RBCs are also damaged by decompression and that the ability of the RBCs to transport respiratory gases may be compromised.

The plasma cortisol levels in both groups of tournament-caught smallmouth bass were significantly greater than that of control smallmouth bass (Figure 4A). These findings are also similar to those recently obtained by Suski et al. (2003). Interestingly, the plasma cortisol levels in tournament-caught smallmouth bass exhibiting symptoms of decompression were not significantly different from those in the tournament-caught smallmouth bass that were categorized as nondecompressed (Figure 4A). These results are somewhat surprising because one might anticipate that the tissue and RBC damage in decompressed smallmouth bass would further increase the stress levels in these fish. One explanation for these findings may be that the cortisol response in tournament fish has reached maximal levels, or perhaps even become attenuated, by the end of these events. Such a response has been observed in other situations where fish have been exposed to multiple stressors (Barton et al. 1980, 1998). Another possibility is that the internal damage caused by decompression may not have been perceived by the fish's central nervous system as a stressor within the time frame of measurement in this study. In this regard, however, it is noteworthy that other traditional indicators of stress, such as plasma osmolality and plasma chloride concentration, were also similar between decompressed and nondecompressed tournament-caught smallmouth bass (Figure 4B, C). Taken together, these findings suggest that the external and internal symptoms of decompression observed in smallmouth bass do not cause additional stimulation of the species' general stress mechanisms.

As was recently observed in other species, such as largemouth bass (Suski et al. 2003) and walleye *Sander vitreus* (Killen et al. 2003), the plasma lactate concentration in smallmouth bass was elevated after the tournament weigh-in (Figure 5). Additional studies from our laboratory show that this is mainly due to the fact that the handling and air exposure within the traditional weigh-in format cause a large anaerobic disturbance in largemouth bass (Suski et al. 2004). Results from the present study also indicate that the anaerobic disturbance is exacerbated in smallmouth bass exhibiting symptoms of decompression. The larger anaerobic disturbance in decompressed smallmouth bass may have multiple causes. As mentioned earlier, decompressed fish have probably experienced some degree of damage to their circulating RBCs (Figure 3). In humans, the formation of bubbles in the circulatory system is also considered an important aspect of decompression (James 1993). According to Beyer et al. (1976), bubbles in the circulatory system can impair circulatory function in fish. Bubble formation in the circulatory system and associated circulatory dysfunction and/or hypoxia may also be contributing to the greater anaerobic disturbance in decompressed smallmouth bass after the weigh-in at tournaments.

In conclusion, the results of this study indicate that significant numbers of smallmouth bass may exhibit external symptoms of decompression at some live-release tournaments. In our experience, this is not a major problem on most southeastern Ontario water bodies where bass tournaments are held. However, on certain water bodies where anglers have access to smallmouth bass at greater depths, this may be an important issue that deserves further attention. Our results indicate that smallmouth bass exhibiting external symptoms of decompression also experience internal physiological changes, including significant tissue and RBC damage. Decompression does not appear to cause a further increase in certain physiological stress indicators (e.g., plasma cortisol) in tournament-caught smallmouth bass, but it seems to elevate the anaerobic disturbance associated with the weigh-in. Although most decompressed smallmouth bass are still alive after the weigh-in, the

ultimate fate of these fish is unknown. Further research is therefore warranted to examine a number of issues in this area, such as the exact time course of the physiological disturbance caused by decompression in smallmouth bass and the impact of decompression on survival.

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