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[Article]

Severity of Barotrauma Influences the Physiological Status, Postrelease Behavior, and Fate of Tournament-Caught Smallmouth Bass

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Abstract.—Much research on the fish physiological consequences of tournaments has been conducted to date and has provided anglers and tournament organizers with strategies for reducing stress and mortality. However, one aspect of tournaments that has received little attention is barotrauma. At a fall competitive angling event on Rainy Lake in northwestern Ontario, we evaluated the incidence of barotrauma among tournament-caught smallmouth bass *Micropterus dolomieu*; we then tagged and released a subset of fish that had severe barotrauma indicators and compared physiology, postrelease behavior, and fate between these fish and those with negligible signs of barotrauma. Overall, 76% of fish had at least one sign of barotrauma (either hemorrhaging or swim bladder distention), but only 32% of fish had two or more indicators and were thus deemed to have severe barotrauma. When telemetered fish were released at a common site, we determined that fish with negligible signs of barotrauma evacuated the release site more rapidly than fish with severe barotrauma did. Some fish with barotrauma floundered at the surface when released, and one of these fish was subsequently hit and killed by a boat. At the end of the monitoring period, 20% of fish with severe barotrauma had died; two additional individuals (20%) that were still at the release site were moribund (failed to respond to diver stimuli). Conversely, we failed to observe any mortality in fish with negligible signs of barotrauma. All tournament fish had elevated levels of blood glucose and lactate. However, stress indices were higher in fish with barotrauma and tended to be highest among fish with barotrauma that died after release. This study revealed that the incidence of barotrauma in tournaments can be high; moreover, outside of a laboratory environment, a significant proportion of fish with severe barotrauma may die after release. Additional research is needed to determine the seasonal variation in incidence and consequences of barotrauma as well as the effectiveness of different depressurization techniques in the field that could be used during fishing tournaments.

In North America, live-release tournaments involving black basses *Micropterus* spp. are becoming increasingly popular with recreational anglers (Schramm et al. 1991; Kerr and Kamke 2003). Not surprisingly, many researchers have examined the biological effects of tournaments on black basses (reviewed in Cooke et al. 2002; Siepker et al. 2007) and have found that several factors, including water temperature (Wilde 1998), live-well conditions (i.e., where the fish are held at ambient atmospheric pressure for extended periods; Cooke et al. 2002; Suski et al. 2004, 2005, 2006), and weigh-in procedures (Suski et al. 2003, 2004), can influence stress and mortality. By adopting simple strategies such as refraining from holding tournaments during the warmest times of the year, providing fish with adequate live-well water quality, and improving weigh-in procedures, anglers and tournament organizers have the potential to reduce stress and mortality. Other factors that can influence stress and mortality are depth of capture and associated barotrauma. Unfortunately, the incidence and consequences of barotrauma are poorly understood in relation to bass tournaments, especially those in which live wells are used.

Barotrauma has been documented in many freshwater and marine game fish and is increasingly being recognized as a serious conservation and management issue in catch-and-release fisheries (Bartholomew and Bohnsack 2005; Arlinghaus et al. 2007). Barotrauma results from a process called decompression, where fish are brought from depth to the surface quickly, leading to rapid changes in ambient pressure. The decline in ambient pressure can have profound physiological (Morrissey et al. 2005) and physical (Feathers and Knable 1983) consequences, especially in physoclistous fishes (including black basses), in which the swim bladder does not directly connect to the digestive tract (Fänge 1966). Beyond problems with swim bladder distention (which, in some species, includes stomach or anal eversion or swim bladder bursting; Feathers and Knable 1983), fish that are exposed to decompression can experience internal (peritoneum, kidneys, dorsal

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aorta; Feathers and Knable 1983) and external (fins, gums, body surface; Feathers and Knable 1983; Morrissey et al. 2005) hemorrhaging; ocular pressure; formation of gas bubbles within the circulatory system, gills, heart, and brain (Philp 1974; Casillas et al. 1975); and general tissue damage (Morrissey et al. 2005; Rummer and Bennett 2005). In fact, one study documented over 70 injuries that could arise from severe decompression (Rummer and Bennett 2005).

It is believed that some decompressed fish, if released quickly, will be able to descend enough to recompress the gases and minimize the negative consequences of barotrauma. However, in bass tournaments, fish are retained in live wells at atmospheric pressure, which allows gases to expand; when bloating is severe, fish are unable to equilibrate. When released, fish that are unable to return to depth immediately because of the added buoyancy could face predation (e.g., Keniry et al. 1996; St. John 2003); solar radiation or thermal stress; involuntary transport to shore or undesirable habitats via waves, currents, tides, or wind; injury from impact with boats; or additional physiological disturbances as they struggle to return to depth (Morrissey et al. 2005). The magnitude of the decompression and the potential for mortality appear to increase with depth of capture (Feathers and Knable 1983; Gitschlag and Renaud 1994; St. John and Syers 2005) beyond a minimum of 3.5 m (Shasteen and Sheehan 1997). Previous research has occurred primarily in the laboratory environment (Shasteen and Sheehan 1997) or in field cages (Gitschlag and Renaud 1994; St. John and Syers 2005) and thus has not approximated the actual conditions of live-release tournaments. Only Morrissey et al. (2005) quantitatively assessed incidence and physiological consequences of barotrauma in smallmouth bass *Micropterus dolomieu* at a tournament; however, they did not assess survival of the fish.

The purpose of this study was to evaluate the incidence of barotrauma at a live-release smallmouth bass tournament and the consequences of barotrauma for physiology, postrelease behavior, and fate of affected individuals. Because the focus was on barotrauma rather than other variables, we selected a tournament conducted in the fall, when water (~14°C) and air (~10°C) temperatures were low and thus when environmental conditions during live-well retention and weigh-in were expected to be reasonably benign (Wilde 1998; Schreer et al. 2001). In addition, unlike previous studies, we used biotelemetry to monitor the behavior and fate of released fish. By taking nonlethal biopsies before the fish were released (Cooke et al. 2005), we were able to conduct the first examination of the relationship between individual physiological status and fate in a recreational fishery.

Methods

Study site and tournament.—This study was conducted at a live-release angling tournament (September 30 and October 1, 2006) in northwestern Ontario on Rainy Lake at LaBelle's Camp (48°50′20″N, 93°37′20″W; mean depth = 9.8 m; maximum depth = 49.1 m). This annual tournament involves 75 boats and 150 tournament anglers. Each team may retain and weigh in five smallmouth bass per competition day (minimum tournament size = 30 cm), competing for the greatest combined weight over a 2-d period. Surface water temperatures in the nearshore areas were between 13.6°C and 14.7°C during the tournament. Fish captured during the event were kept in circulating live wells of various sizes for up to 9 h. For the weigh-in, fish were brought to shore in plastic bags filled with water, transferred to water basins, and transported to the weigh-in site in all-terrain vehicles (ATVs; trip lasted ~1 min). At the weigh-in site, fish were held in static-water-filled basins, dry weighed in air, and then ATV-transported in water basins back to the dock region. For our experiments, we intercepted fish as they were delivered to the dock but before they were placed in a pontoon boat, where they were held for up to 3 h until release. During the 2-d period, tournament anglers weighed in 592 smallmouth bass.

Assessment of barotrauma.—To assess incidence of barotrauma at the tournament, we randomly intercepted fish ($N = 64$) after weigh-in over the 2-d period. Fish were processed one at a time and were placed in a 30-L container for an observation period of about 1 min. Because barotrauma causes pressure changes that can be visualized by expanded swim bladders (bloating) and broken blood vessels (hemorrhaging; Feathers and Knable 1983), we evaluated these indicators during the observation period. We emulated the techniques of Morrissey et al. (2005) to enable direct comparison of barotrauma incidence and severity. A fish was categorized as moderately bloated if the body was slightly distended or severely bloated if the body was greatly deformed (Morrissey et al. 2005). Fish of the latter group were unable to maintain equilibrium and some also showed signs of eye bulging. Fish were then placed in a water trough, where gills were submerged in fresh lake water. We then looked for signs of hemorrhaging on the fins, gums, and body surface, where broken blood vessels would be visible as described by Morrissey et al. (2005). Hemorrhaging was categorized as severe if it occurred in two or more areas or moderate if it occurred in only one area.

To assess physiological condition, we placed the fish in supine position in the trough and obtained a 1.5-mL nonlethal blood sample (Cooke et al. 2005) by caudal venipuncture with a Vacutainer (3-mL tube containing lithium heparin anticoagulant; Becton Dickinson, Inc., Franklin Lakes, New Jersey); samples were immediately placed in a water–ice slurry. The fish were then transferred to the live-release pontoon boat. Within 2 min of blood sample collection, we measured lactate and glucose levels in whole blood by adding 10 μL of blood to a handheld glucose meter (Roche Diagnostics Corp., Indianapolis, Indiana; Accu-Chek) and lactate meter (Arkray, Inc., Kyoto, Japan; Lactate Pro LT-1710 Analyzer). Appropriate standards and calibrations were used with the meters before analysis according to the manufacturers' guidelines. These field meters have been shown to produce results comparable with laboratory values for fish and other animals (e.g., Morgan and Iwama 1997; Wells and Pankhurst 1999; Pyne et al. 2000; Venn Beecham et al. 2006); even if the values show minor deviations from results of laboratory assays, the relative differences among treatments are useful (Morgan and Iwama 1997; Mizock 2002; Venn Beecham et al. 2006). Within 2 h, the Vacutainers were gently inverted several times and some of the remaining blood was placed in microhematocrit tubes for 6 min of centrifugation at 10,000 revolutions/min to assess packed cell volume (i.e., hematocrit). The above procedures were performed again during two posttournament dates (October 4and 5, 2006) on a pseudo-control group ($N = 11$) containing fish that were angled from Rainy Lake (maximum capture depth = 8 m), landed within 30 s, and immediately sampled for blood while being held in a water-filled trough to provide context for other physiological values. The pseudo-control fish were subsequently released.

Assessing posttournament behavior and survival.—In addition to the randomly chosen fish described above, we also nonrandomly selected tournament-caught fish that had either negligible (one or no signs of) barotrauma ($N = 12$ fish) or severe (two or more signs of) barotrauma ($N = 10$; Morrissey et al. 2005) toward the end of each sampling period on each day. All of the fish with severe barotrauma exhibited loss of equilibrium in addition to bloating and hemorrhaging. The nonrandomly selected additional fish were used to assess posttournament behavior and survival in relation to barotrauma severity. The blood collection protocol was identical to that described above, but fish were also equipped with small, flattened, external radio transmitters (2.4 g in air; <1 g in water; ~4 × 15 × 18 mm; 20-cm trailing antennas; frequency = 148–151 MHz) according to the techniques described by Cooke (2003). Stainless steel wires were passed through the fish by using paired hypodermic needles. The wires were connected to the transmitters and formed a harness when the wires were twisted and flattened against a backing plate. The transmitters were placed near the dorsal surface (~5 mm ventral to the dorsal midline), at the interface of the soft and spiny dorsal fins. Transmitters had a life expectancy of 10 d. Fish were also affixed with anchor tags (Floy Manufacturing, Inc.) to identify individual fish externally.

Telemetered fish were transported to a common release site in a 200-L transport tank to simulate normal tournament release procedures. The release site was a rocky point (depth ~4 m) in a distinct bay that was off-limits to anglers participating in the tournament. After release on day 1, fish had to swim roughly 1 km before moving into a region where they could be targeted by anglers on day 2. Using three-element Yagi antennas and several radiotelemetry receivers, we tracked the fish by radiotelemetry for 5–6 d or until they left the study site (>2 km). To track fish from shore and by boat (with a trolling motor), we used a combination of triangulation and successive gain reductions (i.e., zero-point tracking). At night, fish were tracked from shore from several high vantage points, enabling general assessment of the presence or absence of fish within different spatial scales from the release point. This was particularly necessary during the day of release so that we could assess immediate postrelease behavior. Tracking calibrations were conducted to assess the reception range of the receiver with different gain settings from the release point. During the day, when the boat could be used, fish positions were marked on scale maps and the positions were noted by using a handheld Global Positioning System unit. At night, observations focused on using maps to determine fish position relative to the release site. When a transmitter had not moved (<5 m), possibly indicating mortality, a snorkeler was deployed to look for the fish or its transmitter.

Statistical analysis.—The mean concentrations of metabolites (i.e., blood lactate and glucose) and hematocrit were compared using one-way analysis of variance (ANOVA) and Tukey–Kramer post hoc tests. For telemetered fish, differences in the probability of survival between fish with negligible barotrauma and those with severe barotrauma were assessed by using a univariate survival analysis with censoring. The same analysis method was used to compare the probability that fish of each group were within a specified distance (25 and 250 m) of the release site at the termination of the monitoring period. Only fish that survived during the entire study period were included in the distance

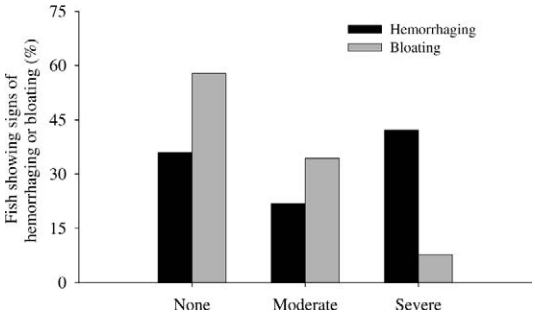

FIGURE 1.—Percent of smallmouth bass ($N = 64$ fish) showing signs of hemorrhaging (none = no visible broken blood vessels; moderate = broken blood vessels visible in only one area of the body; severe = broken blood vessels in more than one area) or bloating (none = no distension of the body; moderate = slight distention; severe = great distention) after a fall tournament on Rainy Lake, Ontario, in 2006.

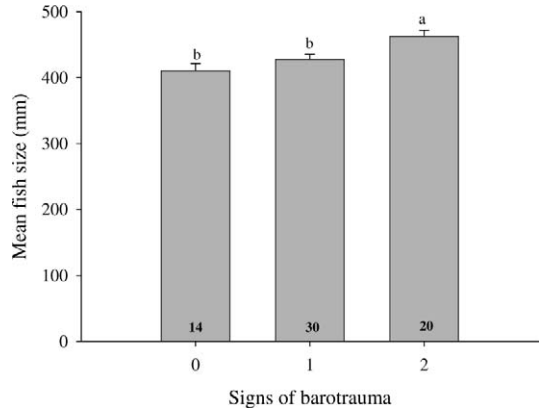

FIGURE 2.—Size-specific patterns (mean total length ± SE) in number of barotrauma signs observed in smallmouth bass caught during a fall tournament on Rainy Lake, Ontario, 2006. Sample sizes are given inside bars. Letter assignments denote significant differences in means between categories (Tukey–Kramer post hoc test: $P < 0.05$).

analysis. All statistical tests were performed in JMPIN version 5.1 (SAS Institute), and α for all tests was 0.05.

Results

Of the 64 randomly selected tournament fish (mean total length ± SE = 435 ± 5.7 mm), 64% showed signs of hemorrhaging (gums, body, or fins) and 42% showed signs of bloating (Figure 1). All fish that showed signs of bloating also had problems with equilibrium. We conservatively defined fish as experiencing barotrauma if they showed signs of bloating and one or more signs of hemorrhaging (Morrissey et al. 2005) or if they showed signs of severe bloating (hereafter, severe barotrauma group). This represented 31% of the randomly selected fish. Fish of the severe barotrauma group were also significantly larger than the other groups (ANOVA: $P = 0.01$; Figure 2). The severe and negligible barotrauma groups had elevated blood lactate and glucose levels relative to those of angled controls (ANOVA: $P < 0.001$; Figure 3A). Hematocrit also varied across treatments, and there was evidence of hemoconcentration in the tournament fish (severe and negligible barotrauma groups) relative to controls (ANOVA: $P < 0.0001$; Figure 4A). Despite clear physiological disturbance and signs of barotrauma, all handled fish were alive when placed into the live-release pontoon boat.

The mean length of radio-tagged fish with negligible barotrauma ($N = 12$; 463 ± 7 mm) did not differ significantly from that of radio-tagged fish with severe barotrauma ($N = 10$; 447 ± 11 mm; t-test: $P = 0.25$). The survival probability for radio-tagged fish with severe barotrauma was significantly lower than that for fish with negligible barotrauma ($\chi^2 = 5.66$, df = 1, $P = 0.017$). Specifically, we observed no mortality in fish with negligible barotrauma and 40% mortality in fish with severe barotrauma. In calculating mortality, we included two moribund fish that were still within 25 m of the release site at the end of monitoring (i.e., after 5 or 6 d) and that did not respond to diver stimulation. One of the dead fish was found floating on the surface 14 h after release; impact with a boat had severed the pelvic fins, scarred the body externally, and ruptured the stomach and liver internally. The other fish was found dead on the bottom of the lake after 24 h. All mortalities were located in close proximity (25 m) to the release site.

By 16 h postrelease, no floating fish were observed, although most of the severely barotraumatized fish were indeed floundering at the surface until low light levels prevented further observation (~3 h postrelease). Telemetered fish with severe barotrauma and those with negligible barotrauma differed in the time required to disperse 25 m from the common release site ($\chi^2 = 4.78$, df = 1, $P = 0.029$). After 15 h, all fish with negligible barotrauma had left the release site, whereas live fish with severe barotrauma took as much as 70 h to leave the site (Figure 5A). We observed the same trend in the time taken by fish to travel more than 250 m from the release site ($\chi^2 = 4.62$, df = 1, $P = 0.032$; Figure 5B). The probability of a fish being within 250 m of the release site at 115 h postrelease was 8% for fish with negligible barotrauma but 57% for fish with severe barotrauma. At the end of the study period, 70% of fish with negligible barotrauma were able to exit the bay of release (>2 km), but only 30% of fish with severe barotrauma were able to do so. Furthermore, the

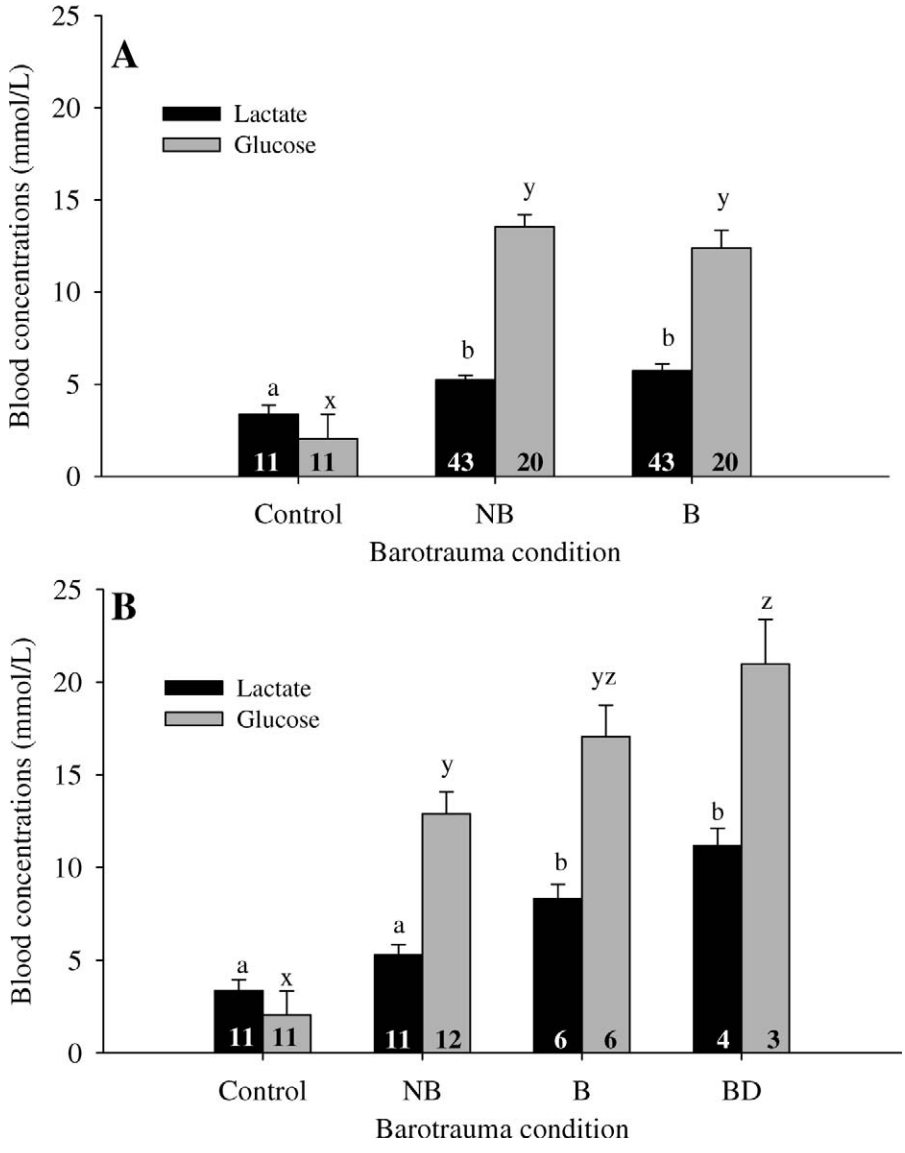

FIGURE 3.—Blood lactate and glucose levels (mean ± SE) in smallmouth bass of varying fates and varying barotrauma severity caught during a fall tournament on Rainy Lake, Ontario, in 2006: **(A)** fish with unknown fates from three severity groups (control; NB = negligible barotrauma; B = severe barotrauma); and **(B)** radio-tagged fish with known fates (control; NB; B = severely barotraumatized fish that lived; BD = severely barotraumatized fish that died. Letter assignments (a and b for lactate; x–z for glucose) denote significant differences in means between groups (Tukey–Kramer post hoc test: $P < 0.05$). Sample size is indicated within each bar.

remaining fish with severe barotrauma (70%) had not traveled further than 1 km from the release site.

Stress indicators varied according to barotrauma severity and fate. Similar to the broader tournament assessment (above), blood glucose and lactate values varied significantly ($P < 0.0001$ for both; Figure 3B); the tournament fish had significantly higher values than the angled controls. Fish with negligible barotrauma had blood glucose levels that were significantly lower than those of severely barotraumatized fish that died during the study period ($P < 0.05$); fish with severe barotrauma that survived had intermediate levels. Fish with negligible barotrauma had blood lactate levels that were significantly lower ($P < 0.05$) than those of fish with severe barotrauma irrespective of fate. Similarly, hematocrit levels also varied by treatment ($P = 0.0002$;

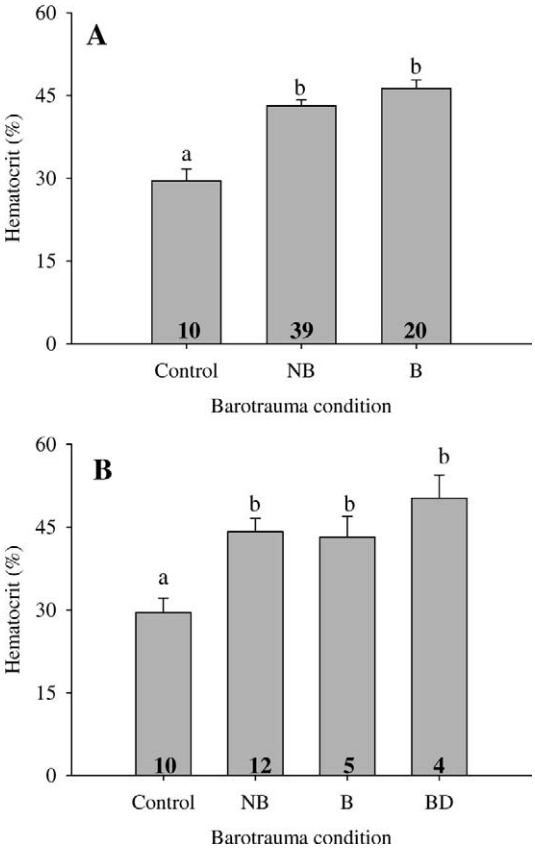

FIGURE 4.—Hematocrit levels (mean ± SE) in smallmouth bass of varying fates and varying barotrauma severity caught during a fall tournament on Rainy Lake, Ontario, in 2006: **(A)** fish with unknown fates from three severity groups (control; NB = negligible barotrauma; B = severe barotrauma); and **(B)** radio-tagged fish with known fates (control; NB; B = severely barotraumatized fish that lived; BD = severely barotraumatized fish that died). Letter assignments denote significant differences in means between groups (Tukey–Kramer post hoc test: $P < 0.05$). Sample size is indicated within each bar.

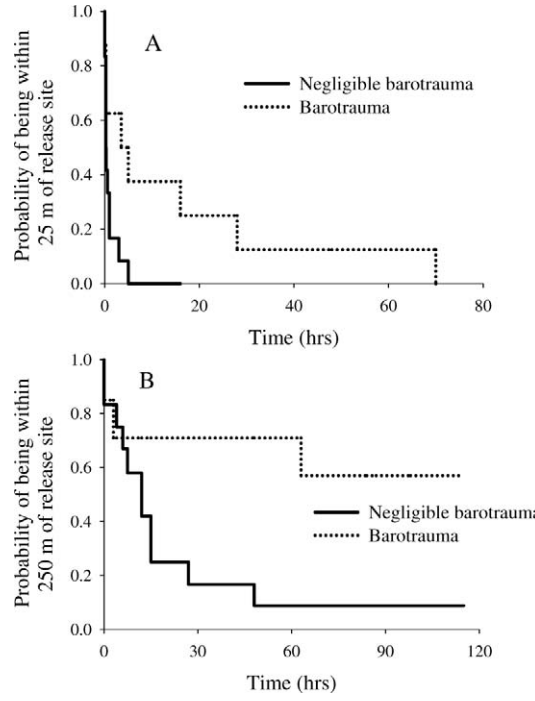

FIGURE 5.—Probability of being located **(A)** within 25 m or **(B)** within 250 m of the release site for radio-tagged smallmouth bass that exhibited severe or negligible barotrauma after capture during a fall tournament on Rainy Lake, Ontario, in 2006.

Figure 4B); the lowest levels were observed in angled controls, and consistently higher values were recorded in all tournament fish irrespective of barotrauma status or fate.

Discussion

Barotrauma is increasingly being recognized as a factor that can affect the condition and survival of fish captured from depth and released (reviewed by Arlinghaus et al. 2007). In a recent meta-analysis, Bartholomew and Bohnsack (2005) reported that capture depth was a highly significant factor in mortality of angled fish, such that mortality was higher in deepwater captures than in shallow-water captures. To our knowledge, however, all of the studies they analyzed were based on experiments conducted in the laboratory, where fish are artificially decompressed in barochambers (e.g., Shasteen and Sheehan 1997), captured in the field and then monitored for survival in tanks or pens (e.g., Keniry et al. 1996), or held in cages at various depths (e.g., St. John and Syers 2005). Several mark–recapture tagging studies have been conducted that evaluate barotrauma issues (e.g., Lee 1992; Burns and Restrepo 2002), but these studies provide little information on fish behavior and are subject to several limitations. In addition, only a limited amount of work has assessed barotrauma at fishing tournaments (see Morrissey et al. 2005), where fish are subjected to multiple stressors (see Cooke et al. 2002; Suski et al. 2004). Our study revealed that at a smallmouth bass tournament in northwestern Ontario, 32% of fish had clear indications of severe barotrauma after weigh-in. In addition, using telemetry coupled with nonlethal physiological sampling, we found that fish with severe barotrauma were slower to disperse from the release site than those with negligible barotrauma. Mortality rates were significant (40%) in fish with severe barotrauma, and those that died were

clearly stressed, as indicated by blood chemistry (i.e., extremely elevated blood glucose). The concept of linking behavior, physiology, and fate with nonlethal physiological sampling and biotelemetry is novel among catch-and-release assessments but has been done previously to assess migration failure in salmonids (Cooke et al. 2006; Young et al. 2006) and bycatch fate in marine pelagic species (Moyes et al. 2006). We believe that this approach has much value for catch-and-release studies, particularly those concerning barotrauma; cage and laboratory experiments fail to expose fish to the suite of predators, environmental conditions, boating traffic, and other variables that exist in actual live-release angling scenarios.

Of our examined fish, 64% showed signs of hemorrhaging and 42% showed signs of extreme bloating. Our analyses also showed that fish with severe barotrauma tended to be larger than other fish. However, the effect of fish size on barotrauma was not the focus of this study because tournament anglers tend to target the largest individuals. All fish with extreme bloating also had problems maintaining equilibrium and were floating on the water surface during observations. In addition, when placed in the live-release boats, many of these same fish floated upside down but continued to ventilate their gills, similar to the tournament-caught largemouth bass *M. salmoides* observed by Lee (1992). A previous study (Morrissey et al. 2005) investigated posttournament barotrauma in smallmouth bass in several lakes in southern Ontario and used the same criteria used here to classify fish with severe barotrauma (i.e., two or more signs). In shallow Rice Lake (mean depth = 3 m; maximum depth = 7.9 m), they observed a low incidence of severe barotrauma (1.9%); however, they found a much higher incidence (56.5%) in a deeper lake (i.e., western basin of Lake Erie; mean depth = 7.4 m; maximum depth = 18.9 m). In contrast, Rainy Lake has a mean depth of 9.8 m and a maximum depth of 49.1 m, and 32% of observed fish had severe barotrauma. Because Rainy Lake is deeper than Rice Lake or Lake Erie, we would expect barotrauma to be more prevalent in Rainy Lake. The fact that it was not suggests that mean or maximum depth of a water body cannot alone explain the extent of barotrauma observed in tournament-caught fish; other factors, such as fishing techniques, tournament protocol, available habitat, depth of fish, water temperature, and season, probably also play a role. Anecdotal discussions with anglers at the tournament on Rainy Lake revealed that a substantial range of depths was targeted (1–10 m) by anglers. In some cases, anglers reported fishing shallowly (~3 m) in deep water (~7 m) and having fish come from depth to strike the lure. Future work is needed to elucidate the various factors that influence variation in barotrauma incidence at tournaments.

Unlike previous assessments of postrelease mortality of fish with barotrauma, we used telemetry to study fish fate in the wild. This also enabled us to assess the sublethal behavioral consequences of barotrauma for the first time. For this aspect of the study, we exploited previous knowledge that smallmouth bass disperse from a release site after being displaced from the site of capture (information obtained either from experiments or after tournaments; e.g., Stang et al. 1996; Bunt et al. 2002; Wilde 2003). Given that no angling was permitted within 1 km of the release site, we expected that all fish would evacuate the release site. Indeed, all fish with negligible barotrauma evacuated the immediate release site (i.e., 25 m) within 15 h and most (92%) evacuated the 250-m zone within 27 h. Fish with severe barotrauma took substantially longer to leave the release site; at the end of the monitoring period (5–6 d), two moribund individuals were still within 25 m of the site. Although no other studies provide data appropriate for comparison, fish with severe barotrauma were clearly impaired in some way that delayed their departure, perhaps reflecting energetic exhaustion or physiological disturbance. In fact, most fish with severe barotrauma were floating on the surface at the time of release. In general, behaviors (including loss of equilibrium and slow dispersal) tend to be sensitive indicators of animal condition and stress (Schreck et al. 1997). Although potential effects of transmitters or attachment procedures on fish behavior cannot be excluded, the small size of the transmitters and the recapture of one tagged fish by angling within several days indicate that any effects were probably minimal.

At the tournament, all 592 fish that were weighed in were deemed to be alive; based on clinical indicators, such as gill ventilation, this assessment is correct. Given that most tournaments monitor only initial mortality (e.g., Wilde et al. 2002), the presumption is that tournament mortality rates are low. In our study, it was apparent that groups of the live-released fish did die within hours to days of release. No evidence of mortality was seen in fish with negligible barotrauma, and one of these fish was actually recaptured by an angler several days after release. Conversely, 40% of smallmouth bass with severe barotrauma died after release. Two fish died within the first 48 h postrelease. Although we did not observe any predation, in some systems or in certain species, fish with signs of barotrauma would be extremely susceptible to predators (e.g., Keniry et al. 1996).

Given that mortality was 40% for telemetered fish with severe barotrauma and that 32% of all tournament-sampled fish exhibited severe barotrauma, we

calculate an overall tournament mortality rate of 12.8% (i.e., 76 of the 592 smallmouth bass that were weighed in). Relative to contemporary studies of catch-and-release mortality at tournaments (summarized by Cooke et al. 2002), this value is not remarkable. However, relatively few studies have used telemetry to assess delayed postrelease survival in black bass tournaments. Usually, studies quantify initial mortality (e.g., Wilde et al. 2002) or hold fish in pens or cages to assess delayed mortality (see Wilde 1998); such procedures may not be representative of conditions in the wild (see Cooke and Schramm 2007). Considering that water temperatures during the study period were moderate for smallmouth bass (i.e., 14°C; Schreer et al. 2001) and given the strong relationship between water temperature and tournament mortality (Wilde 1998), the level of mortality we documented would probably have been higher at a summer tournament. However, whether the incidence and consequences of barotrauma vary seasonally is currently unclear. Interestingly, evidence to date suggests that tournament-related mortality in black bass fisheries has negligible effects at the population level (Hayes et al. 1995; Kwak and Henry 1995; Edwards et al. 2004).

Blood samples obtained from smallmouth bass at the tournament were used to characterize the physiological condition of fish with negligible or severe barotrauma and that of angled controls. The angled controls were not subjected to tournament conditions, but some were captured from depth. However, control fish, sampled within seconds of capture, were intended to provide a benchmark for background physiological condition. Even tournament-caught fish with negligible barotrauma had blood lactate and glucose levels that were greater than those of controls. This is not surprising given the immediate sampling of fish after a dry weigh-in procedure that included exposure to air. Dry weigh-in procedures have been shown to mobilize glucose, deplete tissue energy stores (e.g., glycogen, ATP, phosphocreatine), and lead to accumulation of muscle and blood lactate in largemouth bass (Suski et al. 2004). In the analysis focused on telemetered fish with known fates, severely barotraumatized fish that died clearly had the highest blood glucose concentrations. The fish that died also tended to have the highest levels of blood lactate and the most elevated hematocrit; however, these results were not significant. We suggest that continued efforts by fish with severe barotrauma to right themselves and maintain equilibrium and their continued upside-down swimming led to mobilization of glucose (hyperglycemia) and accumulation of the anaerobic metabolite, lactate. Elevated blood lactate could also be a result of hypoxia, which is produced by gas bubbles that impair circulation (Beyer et al. 1976; Morrissey et al. 2005). Cooke et al. (2004) noted similar problems in largemouth bass exposed to low levels of clove oil for transportation: fish that lost equilibrium had greater cardiac disturbance as they worked continually in an attempt to regain equilibrium. The actual levels of glucose and lactate in the blood of smallmouth bass at this tournament were often twice those values recorded for smallmouth and largemouth bass after exposure to a range of thermal, exercise, and hypoxic stressors (e.g., Furimsky et al. 2003; Suski et al. 2003, 2004, 2006) suggesting that the relative or combined effects of tournament practices and barotrauma are significant.

Our study appears to be the first documentation of elevated hematocrit associated with tournaments and barotrauma. Although Morrissey et al. (2005) did not measure hematocrit, they did measure plasma hemoglobin and found evidence (i.e., elevated hemoglobin) that red blood cells (RBCs) may be damaged by decompression, potentially compromising their ability to transport respiratory gases. The elevated hematocrit may also reflect the fact that teleost fish regulate RBC pH in the presence of a stress-induced acidosis (e.g., tournament- and barotrauma-induced lactate levels) by activating RBC Na^+–H^+ exchange to minimize impairment to oxygen transport (Nikinmaa et al. 1984; Perry and Kinkead 1989). Given the concentrations of lactate observed in this study, release of RBCs may also be a compensatory mechanism to provide more capacity for oxygen transport in the face of acidotic conditions. Our work tends to support these conclusions, in that elevated hematocrit could arise from the secretion of splenic red blood cells in an effort to compensate for damaged RBCs (which themselves can swell and increase hematocrit) or the acidotic state of RBCs. Unfortunately, we did not measure ions or osmolality, so we cannot explore alternative explanations associated with hydromineral balance. Notably, however, Morrissey et al. (2005) did not find plasma Cl^- or osmolality differences between decompressed and nondecompressed fish, which suggests that the most parsimonious explanation for the elevated hematocrit was release of additional RBCs. This indication of tissue damage builds on the ideas presented by Morrissey et al. (2005) and Rummer and Bennett (2005) that decompression can have catastrophic physiological and anatomical consequences. Our study, however, is the first to provide evidence that the physiological disturbances in severely barotraumatized fish that die are greater than those in barotraumatized fish that survive.

Barotrauma has been identified as an important issue in catch-and-release fisheries, including live-release tournaments. We provide the first documentation of

behavioral impairments associated with barotrauma; specifically, we demonstrated that not all fish with barotrauma will survive when released in the wild. The observation that one fish with severe barotrauma was hit and killed by a boat has implications for live-release tournament organizers. There is a need to consider the release location and ensure that the site is proximal to deep water and appropriate habitat and that it contains minimal boat traffic. Tournament organizers should be able to rapidly assess the magnitude of the barotrauma issues by (1) examining the incidence of severe bloating and loss of equilibrium during fish holding in the live-release boat and (2) making decisions about where to release the fish. Not all competitive angling events for black basses result in barotrauma, and although our focus is on a specific fishery, the findings and research approach may have relevance to other fisheries (e.g., for reef fishes).

This work also points to the need to develop strategies for recompressing fish. Despite several assessments of fizzing (i.e., use of hypodermic needles to deflate distended swim bladders; reviewed in Kerr 2001), including several focused on black basses (Shasteen and Sheehan 1997), few field assessments of the long-term survival effects of fizzing have been conducted aside from some mark–recapture studies (e.g., Lee 1992). Telemetry studies are required to compare the behavior and fate of fizzed fish with those of nonfizzed individuals, including appropriate controls and shams. In addition, other techniques that do not require use of needles are needed, because needles can puncture vital organs (Kerr 2001). Despite the fact that other options exist (e.g., using milk crates or cages to lower fish to depth or attaching weighted clips to fish; see Kerr 2001; Theberge and Parker 2005), few rigorous comparative assessments of these different decompression techniques have been made (but see study of rockfishes *Sebastes* spp. by Hannah and Matteson 2007). In terms of live-release tournaments, any solution must consider the realities of tournaments. For example, as noted here and by Morrissey et al. (2005), a substantial number of fish at tournaments can suffer from barotrauma. Is it realistic to try to individually recompress up to several hundred fish? Some tournaments targeting walleyes *Sander vitreus* penalize or prohibit fish with distended swim bladders at weigh-in (Kerr 2001). This means that anglers must either fish in shallow waters or must fizz their fish before weigh-in. Again, without knowing the consequences of fizzing on long-term survival, this regulatory approach contains inherent risk. In tournaments, culling is a common practice; if fizzing is to be encouraged, it should probably be done for culled fish prior to release. In instances when tournament anglers recognize that they are consistently landing barotraumatized fish, they should consider focusing their angling efforts on shallower waters to maintain fish welfare. Although placing the burden on the angler, this approach is the most prudent given the lack of current options. In addition, efforts to reduce all stressors associated with competitive angling events should continue (see Suski et al. 2004); such efforts should focus on minimizing mortality, reducing sublethal disturbances, and maintaining fish welfare during the capture event, retention, weigh-in, and release. Because stress is additive in fish, stress preventative efforts are particularly important in systems where barotrauma is common. Although the focus of the paper was on a black bass competitive angling event, the techniques used in this study (i.e., using telemetry and nonlethal physiological biopsy to assess fate of angled fish) are relevant to other fisheries and environments (e.g., coastal marine fisheries).

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References

Arlinghaus, R., S. J. Cooke, J. Lyman, D. Policansky, A. Schwab, C. D. Suski, S. G. Sutton, and E. B. Thorstad. 2007. Understanding the complexity of catch-and-release in recreational fishing: an integrative synthesis of global knowledge from historical, philosophical, social, and biological perspectives. Reviews in Fisheries Science 15:75–167.

Bartholomew, A., and J. A. Bohnsack. 2005. A review of catch-and-release angling mortality with implications for no-take reserves. Reviews in Fish Biology and Fisheries 15:129–154.

Beyer, D. L., B. G. Dóust, and L. S. Smith. 1976. Decompression-induced bubble formation in salmonids: comparison to gas bubble disease. Undersea Biomedical Research 3:321–338.

Bunt, C. M., S. J. Cooke, and D. P. Philipp. 2002. Mobility of riverine smallmouth bass related to derby displacement and seasonal habitat use. Pages 545–552 *in* D. P. Philipp

and M. S. Ridgway, editors. Black bass: ecology, conservation, and management. American Fisheries Society, Symposium 31, Bethesda, Maryland.

Burns, K. M., and V. Restrepo. 2002. Survival of reef fish after rapid depressurization: field and laboratory studies. Pages 148–151 *in* J. A. Lucy and A. L. Studholme, editors. Catch and release in marine recreational fisheries. American Fisheries Society, Symposium 30, Bethesda, Maryland.

Casillas, E., S. E. Miller, L. S. Smith, and B. G. D'Aoust. 1975. Changes in hemostatic parameters in fish following rapid decompression. Undersea Biomedical Research 2:267–276.

Cooke, S. J. 2003. Externally attached radio transmitters do not affect the parental care behaviour of rock bass. Journal of Fish Biology 62:965–970.

Cooke, S. J., G. T. Crossin, D. Patterson, K. English, S. G. Hinch, J. L. Young, R. Alexander, M. C. Healey, G. Van Der Kraak, and A. P. Farrell. 2005. Coupling non-invasive physiological and energetic assessments with telemetry to understand inter-individual variation in behaviour and survivorship of sockeye salmon: development and validation of a technique. Journal of Fish Biology 67:1342–1358.

Cooke, S. J., S. G. Hinch, G. T. Crossin, D. A. Patterson, K. K. English, M. C. Healey, J. M. Shrimpton, G. Van Der Kraak, and A. P. Farrell. 2006. Mechanistic basis of individual mortality in Pacific salmon during spawning migrations. Ecology 87:1575–1586.

Cooke, S. J., and H. L. Schramm, Jr. 2007. Catch-and-release science and its application to conservation and management of recreational fisheries. Fisheries Management and Ecology 14:73–79.

Cooke, S. J., J. F. Schreer, D. H. Wahl, and D. P. Philipp. 2002. Physiological impacts of catch-and-release angling practices on largemouth bass and smallmouth bass. Pages 489–512 *in* D. P. Philipp and M. S. Ridgway, editors. Black bass: ecology, conservation, and management. American Fisheries Society, Symposium 31, Bethesda, Maryland.

Cooke, S. J., C. D. Suski, K. G. Ostrand, B. L. Tufts, and D. H. Wahl. 2004. Behavioral and physiological assessment of low concentrations of clove oil anesthetic for handling and transporting largemouth bass (*Micropterus salmoides*). Aquaculture 339:509–529.

Edwards, G. P., R. M. Neumann, R. P. Jacobs, and E. B. O'Donnell. 2004. Impacts of small club tournaments on black bass populations in Connecticut and the effects of regulation exemptions. North American Journal of Fisheries Management 24:811–821.

Fänge, R. 1966. Physiology of the swim bladder. Physiological Reviews 46:299–322.

Feathers, M. G., and A. E. Knable. 1983. Effects of depressurization upon largemouth bass. North American Journal of Fisheries Management 3:86–90.

Furimsky, M., S. J. Cooke, C. D. Suski, X. Wang, and B. L. Tufts. 2003. Respiratory and circulatory effects of hypoxia in largemouth bass and smallmouth bass: an application to "live-release" angling tournaments. Transactions of the American Fisheries Society 132:1065–1075.

Gitschlag, G. R., and M. L. Renaud. 1994. Field experiments on survival rates of caged and released red snapper. North American Journal of Fisheries Management 14:131–136.

Hannah, R. W., and K. M. Matteson. 2007. Behavior of nine species of Pacific rockfish after hook-and-line capture, recompression, and release. Transactions of the American Fisheries Society 36:24–33.

Hayes, D. B., W. W. Taylor, and H. L. Schramm, Jr. 1995. Predicting the biological impact of competitive fishing. North American Journal of Fisheries Management 15:457–472.

Keniry, M. J., W. A. Brofka, W. H. Horns, and J. E. Marsden. 1996. Effects of decompression and puncturing the gas bladder on survival of tagged yellow perch. North American Journal of Fisheries Management 16:201–206.

Kerr, S. J. 2001. A review of "fizzing"—a technique for swim bladder deflation. Ontario Ministry of Natural Resources, Fish and Wildlife Section, Peterborough, Ontario, Canada.

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(3):26–31.

Kwak, T. J., and M. G. Henry. 1995. Largemouth bass mortality and related causal factors during live-release fishing tournaments on a large Minnesota Lake. North American Journal of Fisheries Management 15:621–630.

Lee, D. P. 1992. Gas bladder deflation of depressurized largemouth bass. North American Journal of Fisheries Management 12:662–664.

Mizock, B. A. 2002. Point-of-care testing of blood lactate. Laboratoriums Medizin 26:77–81.

Morgan, J. D., and G. K. Iwama. 1997. Measurements of stressed states in the field. Society for Experimental Biology Seminar Series 62:247–268.

Morrissey, M. B., C. D. Suski, K. R. Esseltine, and B. L. Tufts. 2005. Incidence and physiological consequences of decompression in smallmouth bass (*Micropterus dolomieu*) after live-release angling tournaments. Transactions of the American Fisheries Society 134:1038–1047.

Moyes, C. D., N. Fragoso, M. K. Musyl, and R. W. Brill. 2006. Predicting postrelease survival in large pelagic fish. Transactions of the American Fisheries Society 135:1389–1397.

Nikinmaa, M., J. J. Cech, and M. McEnroe. 1984. Blood-oxygen transport in stressed striped bass (*Morone saxatilis*)—role of beta-adrenergic responses. Journal of Comparative Physiology A 154:365–369.

Perry, S. F., and R. Kinkead. 1989. The role of cathecholamine in regulating arterial oxygen-content during hypercapnic acidosis in rainbow trout (*Salmo gairdneri*). Respiration Physiology 77:365–377.

Philp, R. B. 1974. A review of blood changes associated with compression–decompression: relationship to decompression sickness. Undersea Biomedical Research 1:117–150.

Pyne, D. B., T. Boston, D. T. Martin, and A. Logan. 2000. Evaluation of the Lactate Pro blood lactate analyser. European Journal of Applied Physiology 82:112–116.

Rummer, J. L., and W. A. Bennett. 2005. Physiological effects of swim bladder overexpansion and catastrophic decom-

pression on red snapper. Transactions of the American Fisheries Society 134:1457–1470.

Schramm, H. L., M. L. Armstrong, N. A. Funicelli, D. M. Green, D. P. Lee, R. E. Manns, B. D. Taubert, and S. J. Waters. 1991. The status of competitive sport fishing in North America. Fisheries 16(3):4–12.

Schreck, C. B., B. L. Olla, and M. W. Davis. 1997. Behavioural responses to stress. Society for Experimental Biology Seminar Series 62:145–170.

Schreer, J. F., S. J. Cooke, and R. S. McKinley. 2001. Cardiac response to variable forced exercise at different temperatures: an angling simulation for smallmouth bass. Transactions of the American Fisheries Society 130:783–795.

Shasteen, S. P., and R. J. Sheehan. 1997. Laboratory evaluation of artificial swim bladder deflation in largemouth bass: potential benefits for catch-and-release fisheries. North American Journal of Fisheries Management 17:32–37.

Siepker, M. J., K. G. Ostrand, S. J. Cooke, D. H. Wahl, and D. P. Philipp. 2007. A review of the effects of catch-and-release angling on black bass, *Micropterus* spp.: implications for conservation and management of populations. Fisheries Management and Ecology 14:91–101.

Stang, D. L., D. M. Green, R. M. Kindt, T. L. Chiotti, and W. W. Miller. 1996. Black bass movements after release from fishing tournaments in four New York waters. Pages 163–171 *in* L. E. Miranda and D. R. DeVries, editors. Multidimensional approaches to reservoir fisheries management. American Fisheries Society, Symposium 16, Bethesda, Maryland.

St. John, J. 2003. Is your fish 'bent' and will it survive? SPC (Secretariat of the Pacific Community) Live Reef Fish Information Bulletin 11(April):31–35.

St. John, J., and C. J. Syers. 2005. Mortality of the demersal west Australian dhufish, G*laucosoma hebraicum* (Richardson 1845) following catch-and-release: the influence of capture depth, venting and hook type. Fisheries Research 76:106–116.

Suski, C. D., S. J. Cooke, S. S. Killen, D. H. Wahl, D. P. Philipp, and B. L. Tufts. 2005. Behaviour of walleye (*Sander vitreus* L.) and largemouth bass (*Micropterus salmoides* L.) exposed to different wave intensities and boat operating conditions during livewell confinement. Fisheries Management and Ecology 12:19–26.

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. Transactions of the American Fisheries Society 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. Journal of Fish Biology 68:120–136.

Suski, C. D., S. S. Killen, M. Morrissey, S. G. Lund, and B. L. Tufts. 2003. Physiological changes in largemouth bass caused by live-release angling tournaments in southeastern Ontario. North American Journal of Fisheries Management 23:760–769.

Theberge, S., and S. Parker. 2005. Release methods for rockfish. Oregon Sea Grant Coastal and Ocean Issues Workshop, ORESU-G-05-001. Oregon Sea Grant, Corvallis.

Venn Beecham, R., B. C. Small, and C. D. Minchew. 2006. Using portable lactate and glucose meters for catfish research: acceptable alternatives to established laboratory methods? North American Journal of Aquaculture 68:291–295.

Wells, R. M. G., and N. W. Pankhurst. 1999. Evaluation of simple instruments for the measurement of blood glucose and lactate, and plasma protein as stress indicators in fish. Journal of the World Aquaculture Society 30:276–284.

Wilde, G. R. 1998. Tournament-associated mortality in black bass. Fisheries 23(10):12–22.

Wilde, G. R. 2003. Dispersal of tournament-caught black bass. Fisheries 28(7):10–17.

Wilde, G. R., C. E. Shavlik, and K. L. Pope. 2002. Initial mortality of black bass in B.A.S.S. fishing tournaments. North American Journal of Fisheries Management 22:950–954.

Young, J. L., S. G. Hinch, S. J. Cooke, G. T. Crossin, D. A. Patterson, A. P. Farrell, G. Van Der Kraak, A. G. Lotto, A. Lister, M. C. Healey, and K. K. English. 2006. Physiological and energetic correlates of en route mortality for abnormally early migrating adult sockeye salmon (*Oncorhynchus nerka*) in the Thompson River, British Columbia. Canadian Journal of Fisheries and Aquatic Sciences 63:1067–1077.