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ARTICLE

Overwinter Mortality of Gizzard Shad: Evaluation of Starvation and Cold Temperature Stress

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Abstract

Overwinter mortality of gizzard shad *Dorosoma cepedianum* has been attributed to starvation, cold stress, and predation; however, the interactions among these factors are not well understood and can change across winter. We evaluated possible causes for overwinter mortality of age-0 gizzard shad through a combination of experiments and field sampling during the winters of 2005–2006 and 2006–2007. In the first experiment, gizzard shad were placed into cages in Oneida Lake, New York, during three time periods prior to ice formation. The fish exhibited low mortality at temperatures above 8°C but high mortality (>75%) in all cages as temperature dropped below 8°C. We observed no consistent patterns of length-dependent mortality or changes in total percent dry weight (DW). However, the viscerosomatic index (VSI) decreased during all time periods, indicating disproportionate use of visceral tissues. In the second experiment, gizzard shad were exposed to temperature treatments of 1, 2, and 4°C in experimental cold rooms. Little mortality occurred as temperature dropped from 8°C to 4°C, but mortality increased after temperature reached 4°C and was highest in the two coldest treatments. Within a temperature treatment, small fish died faster, and mortality exhibited a weak negative correlation with VSI and percent DW. Average size of fish in field collections increased through the winter, indicating higher mortality of smaller individuals and proportional changes in somatic DW and visceral DW. Results from both experiments and the field collections suggest that cold stress and an inability to acclimate—rather than starvation—are driving gizzard shad mortality at low temperatures.

Overwinter mortality in fish can be a significant ecological force that affects population dynamics (Hurst and Conover 1998; O'Gorman et al. 2004; Ward et al. 2006) and limits geographic ranges (Shuter and Post 1990). Starvation and predation are commonly identified as major drivers of overwinter mortality rates (Adams et al. 1985; Sogard 1997; Fitzgerald et al. 2006), often resulting in size-selective mortality, as smaller fish tend to have fewer energy reserves (Oliver et al. 1979), metabolize at higher specific rates (Brett and Groves 1979), and are more susceptible to gape-limited predators (Sogard 1997). Substantial evidence supports starvation as a primary driver of mortality, as demonstrated by observed changes in length (Toneys and Coble 1979; Thompson et al. 1991; Miranda and Hubbard 1994; Bernard and Fox 1997; Pangle et al. 2004), energy content (Pierce et al. 1980; Flath and Diana 1985; Hurst et al. 2000), body composition (i.e., lipid content, protein, and ash-free dry weight [DW]; Thompson et al. 1991; Pratt and Fox 2002; Pangle et al. 2004), and various tissues (i.e., liver; Pastoureaud 1991; Van Dijk et al. 2005; Ibarz et al. 2007). However, additional factors, such as an inability to acclimate or function physiologically at cold temperatures, are also important mechanisms that drive overwinter mortality, despite being poorly understood (Hurst 2007; Donaldson et al. 2008). In most cases, overwinter mortality is probably affected by several interacting mechanisms that vary over time and space throughout the winter period (White et al. 1987; Bodensteiner

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and Lewis 1992; Hurst 2007; Donaldson et al. 2008). Identification of the relative strength of these mechanisms and the importance of their interactions remains difficult.

Recently, growing attention has focused on understanding the interactions among cold temperatures, starvation, and physiological stress in driving overwinter mortality (Hurst 2007; Donaldson et al. 2008). Starving fish may become increasingly sensitive to cold temperatures and may face greater risk of mortality attributed to reduced physiological function (Hurst 2007; Donaldson et al. 2008); however, physiological stress can also occur in fish with ample energy reserves. Physiological responses of cold-stressed fish include decreased cell membrane fluidity (Snyder and Hennessey 2003), osmoregulatory failure (Stanley and Colby 1971; Johnson and Evans 1996), and immunosuppression (Lepak and Kraft 2008). Physiological stress is often greatest at low temperatures (Johnson and Evans 1996; McCollum et al. 2003) or during rapid temperature changes, which can preclude adequate acclimation (Beitinger et al. 2000; Donaldson et al. 2008; but see Lankford and Targett 2001). At the northern extent of a species' range, winter temperatures can drop below levels that are easily tolerated by all individuals, thus resulting in mortality of fish that are unable to acclimate and function at extreme winter temperatures (Johnson and Evans 1996; Lankford and Targett 2001; Hurst and Conover 2002). These causes of mortality probably interact, and an understanding of the interactions is necessary to facilitate accurate predictions of population and species responses to projected climate change.

The gizzard shad *Dorosoma cepedianum* is a southern species with a northern range that historically extended into southern Ohio. Over the past century, the range of the gizzard shad has expanded by several hundred kilometers, and most of this expansion has occurred during periods of warm winter temperatures (White et al. 1987). High overwinter mortality of age-0 gizzard shad probably sets the northern extent of the species' range (Bodola 1955; White et al. 1987), and reports of massive winter die-offs are common (Bodola 1955; Walburg 1964; Wuellner et al. 2008). Gizzard shad are known to become disoriented at cold temperatures and are believed to suffer high mortality at temperatures below 4°C (Heidinger 1983; Adams et al. 1985; White et al. 1987). Overwinter mortality is often size selective such that larger gizzard shad have higher survival than smaller individuals (Adams et al. 1985; White et al. 1987; Ward et al. 2006; Michaletz 2010), and this has been attributed to size-related differences in vulnerability to cold stress (White et al. 1987) or starvation (Adams et al. 1985; White et al. 1987). Evidence supporting the role of starvation is based on comparisons of living and dying fish (Adams et al. 1985) and observed overwinter decreases in whole-fish DW, energy content, and percent lipid content (Pierce et al. 1980). Higher mortality of smaller fish is expected because gizzard shad have a nonlinear relationship between growth and storage of lipids: allocation of energy to lipid storage is greater among fish larger than 90 mm (Michaletz 2010). However, in Lake Erie, White et al. (1987) observed no change in percent lipid content for gizzard shad sampled throughout the winter; those authors found evidence that other factors were driving mortality, including an inability to acclimate enzyme and biological membrane function and an inability to mobilize stored lipids, which in turn can lead to liver glycogen and liver tissue depletion, liver failure, and death. A better understanding of the interaction between starvation and cold-stress-driven overwinter mortality is clearly needed to predict changes in gizzard shad abundance and the likely future range expansion associated with climate warming.

Here, we combine the results of a series of experiments and field collections conducted during the winters of 2005–2006 and 2006–2007 to explore the relative importance of cold stress and starvation on the overwinter mortality rates of age-0 gizzard shad. Specifically, we explored the effect of changing temperatures by using an in situ cage experiment to test for differences in gizzard shad mortality during three temperature regimes (warm and stable; declining; cold and fluctuating) prior to ice formation. Additionally, we evaluated the effect of temperature on gizzard shad survival and changes in DW composition in a controlled experimental setting at stable temperatures (1, 2, and 4°C) that are commonly observed after ice formation. To corroborate experimental results, we compare observations from the experiments with observations from field collections made during the winters of 2005–2006 and 2006–2007. Experiments were designed under the a priori hypothesis that gizzard shad mortality is driven by an inability to acclimate during changing temperatures and is driven by starvation when temperatures are cold and stable.

METHODS

Study site.—Oneida Lake, New York, is a large, shallow, mesotrophic lake (area = 207 km^2; maximum depth = 16.6 m; mean depth = 6.5 m) located in the fertile plain of Lake Ontario. Gizzard shad are not native to Oneida Lake and were first observed there during the mid-1950s (Forney 1980). Gizzard shad did not become common in any of the Cornell Biological Field Station's long-term monitoring programs until the early 1980s (Hall and Rudstam 1999; Fitzgerald et al. 2006; Rudstam and Jackson 2008). Overwinter mortality of age-0 gizzard shad in Oneida Lake has historically been high (close to 100%), and the severity and duration of Oneida Lake winter conditions are suspected to be responsible for this. During 1975–2008, ice duration averaged 93 d and ranged from 55 to 121 d, and only one winter was without complete ice cover (Rudstam and Jackson 2009). In recent years, ice duration has decreased by approximately 3 weeks and inconsistent ice cover during early winter has become more common. Formation of ice cover requires both sufficiently cold water temperatures and calm wind conditions, which often result in water temperatures reaching a "superchilled" state prior to ice formation. Winter water temperatures in the main lake are always less than 2°C and consistently drop

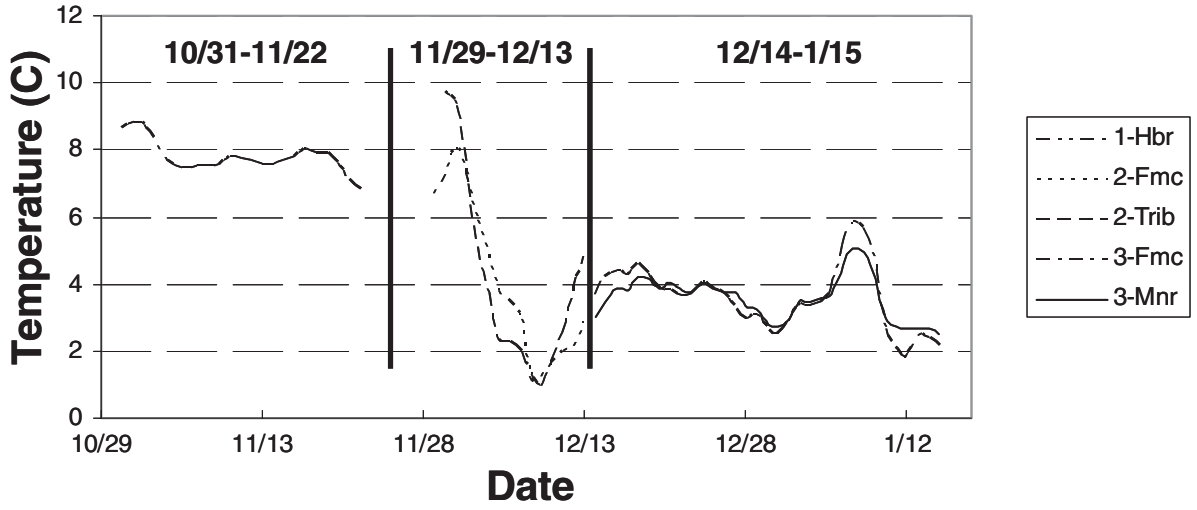

FIGURE 1. Temperature (°C) profiles from different sites over the duration of the cage experiment with gizzard shad in Oneida Lake, New York, 2006–2007 (1-Hbr = protected harbor site, time period 1; 2-Fmc = Fremac Marina, time period 2; 2-Trib = tributary [Chittenango Creek], time period 2; 3-Fmc = Fremac Marina, time period 3; 3-Mnr = Mariner's Landing [protected marina], time period 3).

below 1°C prior to ice formation and during periods when ice cover is inconsistent.

Cage experiment.—The cage experiment was designed to evaluate mortality of gizzard shad prior to ice formation, when temperatures decrease from 8°C to near 0°C and continue to fluctuate until ice cover forms. Mortality was evaluated in situ over three time periods, which were chosen to assess differences in energy use and mortality under different pre-ice temperature regimes (Figure 1). Experimental sites differed among time periods to reflect observed changes in gizzard shad habitat use (Fetzer 2009; Table 1) and included a shallow bay (Harbor), two marinas (Fremac Marina and Mariner's Landing), and a tributary (Chittenango Creek). Both marinas were approximately 1.3 ha and had an average depth of 2.5 m. During each time period, two cylindrical cages (2-m diameter × 1-m height) constructed from welded ground rods and plastic landscaping fence (6.35-mm mesh) were stocked with 200 age-0 gizzard shad. Only one cage was stocked during time period 1 (800 age-0 fish). The initial high stocking density of gizzard shad was used to evaluate potential problems with acclimation or crowding during confinement, but no such problems were observed (survival > 95%). Gizzard shad were collected at

TABLE 1. Summary of results from the in situ cage experiment with gizzard shad in Oneida Lake, New York. Significantly different changes from the initial reference sample (Dunnett's test: $\alpha = 0.05$) are indicated in bold italics (TL = total length;% DW = percent dry weight; RelSOM = relative somatic dry weight; RelVSC = relative visceral dry weight; dry VSI = viscerosomatic index, calculated as the ratio of visceral dry weight to somatic dry weight).

Time period (dates)	Location	Sample	Mortality (%)	*n*	TL (mm)	% DW	RelSOM	RelVSC	Dry VSI
1 (31 Oct–22 Nov 2006)	Harbor	Start		30	95.2 (1.64)	18.2 (0.30)	1.01 (0.03)	1.02 (0.03)	0.104 (0.002)
		Cage 1	4.4	30	***89.7 (2.05)***	***17.4 (0.22)***	***0.93 (0.02)***	***0.74 (0.01)***	***0.082 (0.001)***
2 (29 Nov–13 Dec 2006)	Fremac Marina	Start		29	99.7 (1.92)	17.3 (0.26)	0.97 (0.02)	0.84 (0.02)	0.093 (0.001)
		Cage 1	75.0	30	100.5 (1.65)	***16.5 (0.15)***	***0.91 (0.01)***	***0.73 (0.01)***	***0.087 (0.001)***
		Cage 2	75.0	30	98.5 (1.60)	17.8 (0.24)	0.95 (0.01)	0.80 (0.01)	0.090 (0.001)
2 (29 Nov–13 Dec 2006)	Chittenango Creek	Start		30	95.8 (2.13)	17.9 (0.27)	0.90 (0.02)	1.14 (0.05)	0.129 (0.005)
		Cage 1	99.5	1	101.9 (2.20)	***16.0 (0.27)***	0.85 (0.02)	***0.77 (0.03)***	***0.097 (0.002)***
		Cage 2	93.0	14					
3 (14 Dec 2006–15 Jan 2007)	Fremac Marina	Start		30	100.1 (2.35)	17.1 (0.26)	0.94 (0.02)	0.86 (0.02)	0.097 (0.001)
		Cage 1	91.0	18	***85.3 (1.63)***	16.8 (0.34)	***0.80 (0.03)***	***0.69 (0.02)***	***0.086 (0.002)***
		Cage 2	74.0	30	***92.4 (1.70)***	17.2 (0.21)	***0.84 (0.02)***	***0.72 (0.02)***	***0.088 (0.001)***
3 (14 Dec 2006–15 Jan 2007)	Mariner's Landing	Start		30	89.3 (1.96)	17.9 (0.17)	0.95 (0.02)	0.84 (0.02)	0.090 (0.001)
		Cage 1	68.5	30	91.0 (2.14)	***17.3 (0.21)***	***0.84 (0.02)***	***0.57 (0.01)***	***0.071 (0.001)***
		Cage 2	70.0	30	86.5 (2.41)	***16.4 (0.20)***	***0.77 (0.02)***	***0.52 (0.01)***	***0.070 (0.001)***

experimental sites by using a Smith-Root electrofishing boat (354-V pulsed DC; pulse rate = 60 Hz; 6 A of current into water) and were immediately stocked into the cages. Once stocked, the cages were lowered to the lake or tributary bottom and were left undisturbed until the experiment was terminated (14–32 d later). All cages except the cages at the Chittenango Creek site were visually inspected with an underwater camera 24 h after stocking to ensure that mortality related to shocking and handling was minimal. Due to deeper mooring in turbid water, it was not possible to inspect the tributary cages. Sampling throughout the experiment was also impossible, as disturbing the cages created stress in the gizzard shad, potentially biasing the results. At the beginning and end of each experiment, a sample of live gizzard shad was euthanized with an overdose of tricaine methanesulfonate (MS-222) and immediately frozen in water. Dead gizzard shad were not saved because they were often highly deteriorated and because the time since death could not be determined. Water temperature at each experimental location was continuously recorded with a HOBO temperature recorder (Onset Computer Corp., Bourne, Massachusetts).

For analysis, gizzard shad were thawed, measured for total length (TL), and weighed; fish were then dried at 65°C until a constant weight was achieved (typical drying period was 120 h). Visceral and somatic tissues were weighed and dried separately to calculate percent water, relative visceral and somatic DWs, and the viscerosomatic index (VSI; the ratio of visceral DW to somatic DW; Kaufman et al. 2007). Gizzard shad primarily store lipids in their somatic tissue (Shul'man 1974; White et al. 1987; Diana 2005); therefore, changes in visceral and somatic weights can be used to infer gizzard shad use of lipids (somatic) or alternative energy stores (visceral). Visceral tissue was defined as all internal organs from the esophagus to the anus. Relative visceral and somatic DWs were calculated as the ratio of observed tissue DW to the tissue DW predicted from standard TL–DW equations (similar to the index of relative weight). Standard visceral and somatic TL–DW equations were generated by log transformation of the DWs from fish collected at the beginning of time period 1 (31 October 2006; visceral: $\log_{10}\text{DW} = 2.99 \cdot \text{TL} - 6.78$, $F_{1,28} = 79.97$, $P < 0.001$, $r^2 = 0.74$, $n = 30$; somatic: $\log_{10}\text{DW} = 2.50 \cdot \text{TL} - 4.88$, $F_{1,28} = 75.78$, $P < 0.001$, $r^2 = 0.73$, $n = 30$); these equations were used in all analyses. To avoid bias in measurement of visceral wet weight or DW, fish were inspected for stomach contents. Five fish from all experiments and field collections were observed to have stomach contents, and these fish were removed from the analysis.

Analysis of variance was used to test for significant differences between the initial and final fish measurements for each experimental period. If a significant difference was detected, Dunnett's test was used to determine which cages showed significant differences between initial and final samples. A *t*-test was used in two cases: (1) for time period 1 because there was only one cage and (2) for period 2 at the Chittenango Creek site because the cages were pooled due to low survival.

Tank experiment.—A laboratory experiment was designed to evaluate gizzard shad mortality under lake conditions after ice formation, when temperatures are near zero and relatively constant. The experiment involved the application of different winter water temperatures (1, 2, and 4°C) to determine gizzard shad mortality rates and temperature thresholds. Nine flow-through tanks (800 L each) were evenly distributed across three cold rooms that were randomly assigned a temperature treatment; 100 age-0 gizzard shad were stocked into each tank. A common source of 8°C dechlorinated municipal water was used to fill a reservoir tank in each room, where water attained the treatment temperature before being fed to individual experimental tanks. Gizzard shad (72–133 mm TL) were collected in Oneida Lake when the water temperature reached 8°C (19 November 2006). Fish were collected by use of a beach seine (22.8 × 1.5 m; 6.35-mm, heavy-duty, 22-kg delta mesh) and were immediately transferred to the experimental facility. Prior to initiation of the tank experiment, gizzard shad were maintained at 8°C for 3 d to allow acclimation. Handling mortality was minimal and less than 5% in all but one tank during the 3 d. After acclimation, formaldehyde was added to each tank until a concentration of 25 mg/L was reached; this was done to minimize disease-related mortality. Formaldehyde was applied once every 48 h for a total of three treatments. Upon completion of the disease treatment, temperatures in the cold rooms were lowered at a rate of 0.33°C per day to allow adequate acclimation time and to avoid mortality due to rapid temperature change. Tank densities, the rate of temperature change, and the decision to conduct a disease treatment were based on preliminary data from a pilot experiment that was conducted during winter 2005–2006. Individual cold rooms were lowered to 4, 2, or 1°C (one room at each temperature) and were maintained within ±0.25°C of the treatment temperature once it was reached. A constant photoperiod of 10 h light : 14 h dark was maintained in each cold room throughout the experiment. Feed was not administered because a previous study indicated that gizzard shad do not feed at temperatures below 10°C (White et al. 1987).

Tank status was checked daily, and temperature, dissolved oxygen, and conductivity were recorded to ensure similar conditions across all tanks in each treatment. Dead fish were removed daily and were frozen in water, and the number of live fish remaining was recorded. Treatments were continued until all fish were dead (~46 d in the 1°C and 2°C rooms; ~74 d in the 4°C room). In addition to dead fish, five live fish were removed from each tank when the temperature reached 4°C; the fish were euthanized with an overdose of MS-222 and were frozen in water. An additional five live fish were removed on days 10, 20, and 45 if the numbers remaining were sufficient. Due to a limited number of survivors in the two coldest rooms, the number of live fish removed was less than five on multiple occasions. Analyses of live and dead fish followed the same protocol used in the cage experiment: individual fish TL, percent DW, relative visceral and somatic DWs, and VSI were determined. Standard visceral and somatic TL–DW equations were established by log transformation of the DWs from fish sacrificed at the beginning of the experiment (19 November 2006; visceral:

$\log_{10}$DW = 3.30·TL − 7.44, $F_{1,48} = 335.1$, $P < 0.001$, $r^2 = 0.87$, $n = 50$; somatic: $\log_{10}$DW = 2.85·TL − 5.60, $F_{1,48} = 265.1$, $P < 0.001$, $r^2 = 0.85$, $n = 50$).

Survival analysis (Cox proportional hazards model) was used to compare survival rates of live and dead fish across cold rooms (Lankford and Targett 2001; Hurst and Conover 2002) by using room, tank location within room, and TL as input parameters. The Cox proportional hazards model was chosen because the baseline hazard is unspecified and does not require knowledge of its underlying distribution and because covariates enter the model linearly. Tank location was included in the survival model to account for location-specific conditions within the cold rooms. Final treatment temperatures were reached 26 d after the initial stocking of tanks; therefore, all gizzard shad that died prior to this date were excluded from the survival analysis. Mortality thresholds were evaluated with logistic regression comparing fish that were removed alive with those that died during the experiment. Input parameters were percent DW, relative somatic DW, relative visceral DW, and VSI. All fish that were collected throughout the experiment were included in the logistic regression. The most parsimonious model for survival analysis and logistic regression were selected by use of Akaike's information criterion.

Field collections.—Age-0 gizzard shad were collected throughout the fall and winter of 2005–2006 and 2006–2007 from locations where gizzard shad were observed to congregate at high densities (i.e., protected harbors and marinas; Fetzer 2009). During 2005–2006, a beach seine (described above in Tank Experiment section) was used during periods of open water. During 2006–2007, consistent ice cover required employment of a variety of gear types to continue sample collections after establishment of ice cover. Prior to ice formation, samples were collected at the initiation of the cage and tank experiments by using an electrofishing boat (cages; described above in Cage Experiment section) or a beach seine (tanks; described above). After ice formation in 2006–2007, gizzard shad sampling was limited to small-mesh gill nets (21 × 6 m; containing seven 3-m-wide panels of the following bar mesh sizes: 6.2, 8.0, 10.0, 12.5, 15.0, 18.7, and 25.0 mm) that were fished under the ice for 4 h at the marinas (Fremac Marina and Mariner's Landing). Small-mesh gill nets were shortened to 1-m depth by using monofilament to tie the bottom 5 m of net to the leadline. Gill-net surveys were conducted every 2–3 weeks, and a target sample size of 30 fish was collected if possible. Surveys were discontinued in late February 2007 after marinas became anoxic (dissolved oxygen concentration < 1.0 mg/L) and after two additional gill-net surveys failed to catch any gizzard shad. Two additional electrofishing surveys were conducted in March and April 2007 after ice-out, but no gizzard shad were observed. All captured fish were euthanized with an overdose of MS-222 and immediately frozen in water. These fish were later processed in a manner identical to that used for the cage and tank experiments. Standard TL–DW relationships for visceral and somatic tissues were the same as those used in the cage experiment since the initial sampling date and samples were identical.

Analysis of variance was used to test for significant changes in TL, percent DW, relative somatic DW, relative visceral DW, and VSI across time for all sites in 2005–2006 and at three sites in 2006–2007. If a significant difference was detected, post hoc Tukey's honestly significant difference test was used to conduct pairwise comparisons. A *t*-test was used to analyze the 2005–2006 data for all indices except TL.

RESULTS

Cage Experiment

Gizzard shad mortality was low during time period 1, when temperatures were greater than 8°C, and high during time periods 2 and 3, when temperatures were lower than 8°C (Table 1). Within time periods 2 and 3, mortality was highest at the Chittenango Creek site (99.5% and 93.0%) and lowest at the marina sites (68.5–91.0%). Across all time periods, there was little evidence of higher mortality among smaller gizzard shad (Table 1); three cages showed a significant shift towards smaller TL distributions, indicating higher mortality of larger gizzard shad. Changes in percent DW were inconsistent (Table 1). Relative somatic DW decreased significantly during time periods 1 and 3 and showed inconsistent changes during time period 2 (Table 1). Relative visceral DW and VSI (Table 1) decreased across all time periods and sites except cage 2 from the Fremac Marina site during time period 2.

Tank Experiment

Gizzard shad mortality was low during the acclimation period, and fish showed no visible signs of stress. Throughout the experiment, fish swam continuously and they increasingly associated with the tank bottom as temperatures declined. Mortality increased as temperatures dropped below 4°C (Figure 2). Survival analysis and Akaike's information criterion indicated that all predictors (cold room, tank location, and TL) were necessary to predict the date of gizzard shad mortality. Room (i.e., temperature treatment) explained the greatest variability, followed by tank location and TL, respectively (Table 2). Survival was highest for fish in the 4°C room and lowest for those in the 1°C room; fish in the 2°C room exhibited survival similar to that of fish in the 1°C room, but the two values were significantly different (Table 2). Survival was greatest at tank location 2, intermediate at tank location 1, and lowest at tank location 3 (Table 2). Daily measurements of tanks indicated no significant difference in tank temperature across locations within a given cold room. However, cold room chillers blew air downward and out across the room, which may have led to faster and more dramatic temperature fluctuations in tank locations 1 and 3 relative to tank location 2. Unfortunately, temperature loggers were only placed in one tank per room, so we were unable to compare rates of temperature fluctuation across locations. Fish TL was the least important predictor of gizzard shad mortality in

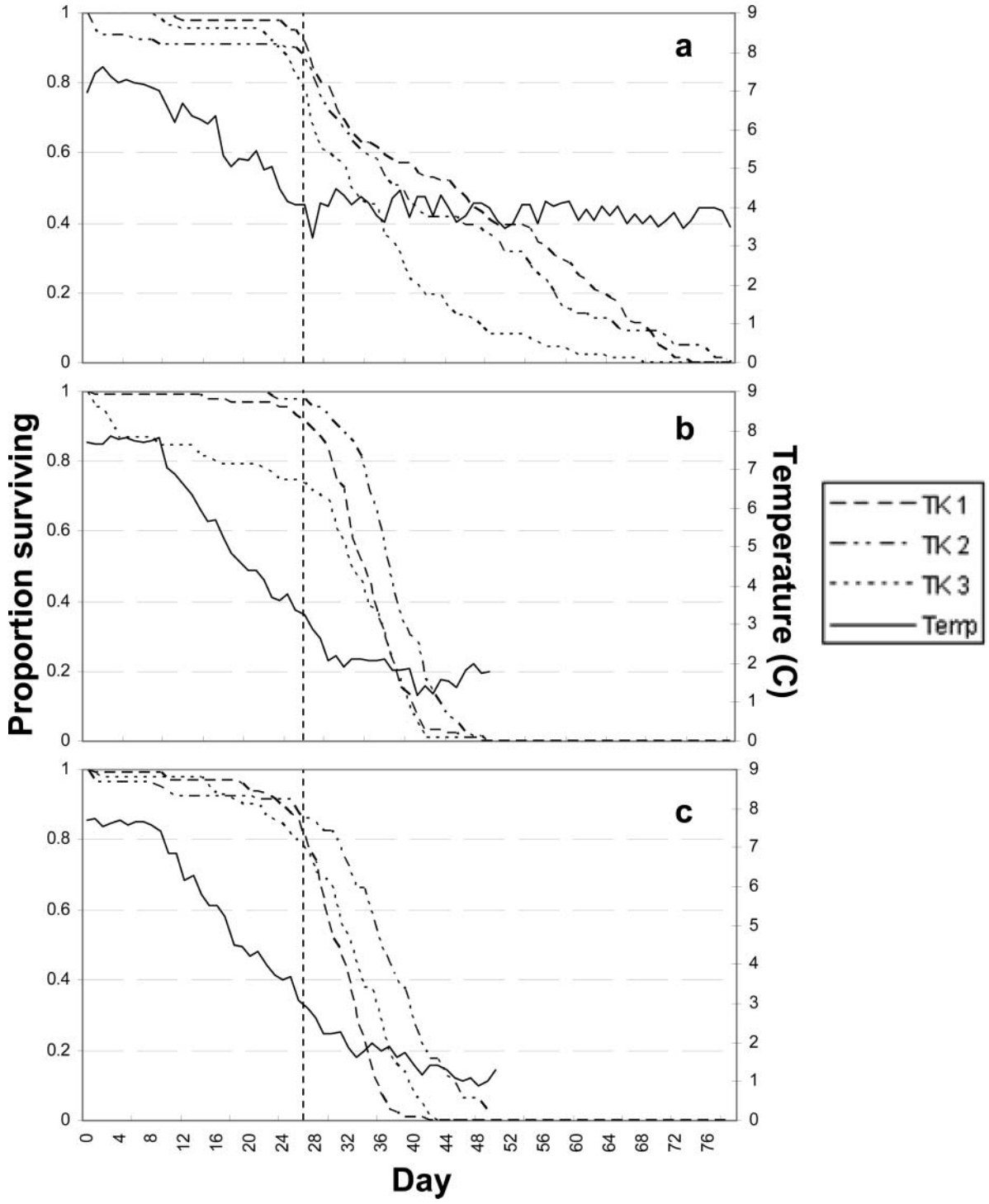

FIGURE 2. Proportion of gizzard shad surviving (tanks [TK] 1–3) and temperature (Temp, °C) over time across tanks from cold rooms at **(a)** 4°C, **(b)** 2°C, and **(c)** 1°C. The vertical dashed line on day 26 indicates when final treatment temperatures were reached across all cold rooms and corresponds to day 0 in the survival analysis.

the tank experiment (Table 2). Logistic regression indicated that VSI and percent DW were significant predictors of mortality (Table 3). The VSI explained the greatest amount of variation, followed by percent DW. Relative visceral and somatic DWs did not contribute significantly to the model.

Field Collections

Mean TLs of gizzard shad collected during the winters of 2005–2006 and 2006–2007 showed no significant change over the November–January period; however, in late January and early February (both years), gizzard shad mean TLs were

TABLE 2. Summary of coefficients and selection criteria for models of gizzard shad survival during a tank experiment that applied winter water temperatures of 1, 2, and 4°C (ΔAIC = difference in Akaike's information criterion between the given model and the best-performing model; TL = fish total length). Significant model coefficients are indicated in bold italics. Negative values increase survival time and positive values decrease survival time relative to that of fish in the 4°C room and fish at tank location 1.

		Model coefficient				
Model	ΔAIC	2°C room	1°C room	Tank location 2	Tank location 3	TL
Room + tank location + TL	0.00	***0.95***	***1.22***	***−0.48***	0.16	***−0.01***
Room + tank location	7.39	***0.10***	***1.21***	***−0.49***	0.14	
Room + TL	42.54	***0.87***	***1.13***			***−0.01***
Room	51.05	***0.88***	***1.11***			
Tank location	139.55			***−0.32***	***0.24***	
TL	161.18					***−0.01***

significantly larger in comparison with the mean TLs of earlier collections (Table 4). Percent DW, relative somatic DW, and relative visceral DW tended to be higher early in winter and to decrease over time (Table 4). There was little consistent change in VSI over the winter (Table 4).

DISCUSSION

Temperature

It is likely that temperature drives overwinter mortality of gizzard shad through rapid changes (i.e., cold shock; Cox and Coutant 1975; Donaldson et al. 2008) and through cumulative cold stress. Across experiments and field collections, gizzard shad consistently showed negative responses to changing temperatures and to temperatures below 4°C. Gizzard shad mortality rates during the cage experiment were high during periods of rapidly declining and near-freezing temperatures but were low when temperature was above 8°C. Our inability to sample fish throughout the cage experiment limits our ability to differentiate whether this mortality was driven by rapid declines in temperature or by exposure to lethal temperatures, but we suspect that both factors are important. Cox and Coutant (1975) observed cold shock in gizzard shad that were exposed to temperature changes of 3–5°C per day. In our cage experiment, the temperature change was more moderate at 1°C per day and the temperature declined to below 1°C, which probably led to the high rates of mortality. The importance of both factors is supported by comparing the mortality rates between time periods 2 and 3 in the Fremac Marina cages. Although total mortality was similar between the two periods (74–91%), mortality rates corrected for the durations of time periods 2 and 3 were nearly twice as high for time period 2 than for time period 3 despite the similarity in minimum temperatures (time period 2: 1°C; time period 3: 1.5°C) and average temperatures (time period 2: 4.0°C; time period 3: 3.6°C). These results suggest that the temperature decline during time period 2 increased the mortality rate, although this is purely speculative.

Dead gizzard shad were commonly observed as having washed up on shore in late fall before temperature declined to below 4°C, and we suspect that cold shock was the likely mechanism, since these mortality events tended to correspond

TABLE 3. Summary of coefficients and stepwise selection of a logistic model for gizzard shad mortality during the tank experiment (ΔAIC = difference in Akaike's information criterion between the given model and the best-performing model; dry VSI = viscerosomatic index based on dry weight;% DW = percent dry weight; RelSOM = relative somatic dry weight; RelVSC = relative visceral dry weight). Significant model coefficients are indicated in bold italics.

		Model coefficient				
Model	ΔAIC	Dry VSI	% DW	RelSOM	RelVSC	Intercept
Dry VSI + % DW	0.00	***−92.82***	***−48.81***			***18.81***
Dry VSI + % DW + RelVSC	1.98	***−92.84***	***−48.70***		−0.53	***19.36***
Dry VSI + % DW + RelSOM	1.99	***−92.85***	***−48.66***	−0.52		***19.35***
Dry VSI + % DW + RelSOM + RelVSC	3.96	***−92.72***	***−48.88***	4.54	−4.28	***18.55***
Dry VSI	38.67	***−75.89***				***9.16***
Dry VSI + RelVSC	40.59	***−75.94***			−1.06	***8.11***
Dry VSI + RelSOM	40.64	***−75.89***		0.78		8.37
% DW	154.00		***−21.33***			***4.98***
RelSOM	164.43			1.46		0.04
RelVSC	164.53				0.64	0.87

TABLE 4. Summary results from field collections of gizzard shad in Oneida Lake (TL = total length;% DW = percent dry weight; RelSOM = relative somatic dry weight; RelVSC = relative visceral dry weight; dry VSI = viscerosomatic index based on dry weight). For a given sampling location, means that share the same letter are not significantly different (pairwise comparisons of sampling dates; Tukey's honestly significant difference test: $\alpha = 0.05$; *t*-tests were used for 2005–2006 data).

Date	*n*	TL (mm)	% DW	RelSOM	RelVSC	Dry VSI
2005–2006 (all sites)						
22 Nov 2005	50	133.0 (2.14) z	0.250 (0.002) z	1.010 (0.012) z	1.010 (0.026) z	0.093 (0.002) z
11 Jan 2006	31	132.0 (2.17) z	0.227 (0.002) y	0.889 (0.012) y	0.758 (0.026) y	0.079 (0.002) z
16 Feb 2006	5	147.0 (5.23) y				
Mariner's Landing						
14 Dec 2006	30	89.3 (2.59) z	0.179 (0.003) z	0.950 (0.021) z	0.825 (0.018) z	0.089 (0.002) z
9 Jan 2007	30	94.9 (2.59) zy	0.169 (0.003) y	0.849 (0.021) y	0.714 (0.018) y	0.090 (0.002) z
24 Jan 2007	25	103.3 (2.84) y	0.167 (0.003) y	0.901 (0.023) zy	0.707 (0.017) y	0.087 (0.002) z
7 Feb 2007	30	104.0 (2.59) y	0.161 (0.003) y	0.861 (0.021) y	0.709 (0.016) y	0.092 (0.002) z
Fremac Marina						
29 Nov 2006	29	99.7 (1.92) zy	0.172 (0.002) z	0.970 (0.019) z	0.826 (0.017) z	0.092 (0.001) z
14 Dec 2006	30	100.1 (1.89) zy	0.170 (0.002) z	0.940 (0.019) zy	0.836 (0.016) z	0.096 (0.001) z
4 Jan 2007	31	94.6 (1.86) z	0.165 (0.002) zy	0.870 (0.019) yx	0.714 (0.016) y	0.086 (0.001) y
24 Jan 2007	9	101.3 (3.45) zy	0.151 (0.002) y	0.810 (0.035) x	0.660 (0.030) y	0.089 (0.002) zy
7 Feb 2007	22	103.9 (2.20) y	0.163 (0.002) zy	0.870 (0.022) yx	0.743 (0.019) y	0.094 (0.001) z
Chittenango Creek						
30 Nov 2006	30	95.8 (1.91) z	0.179 (0.002) z	0.898 (0.018) z	1.120 (0.028) z	0.128 (0.003) z
13 Dec 2006	30	89.2 (1.91) y	0.166 (0.002) y	0.856 (0.018) z	0.740 (0.028) y	0.089 (0.003) y
3 Jan 2007	30	87.6 (1.91) y	0.157 (0.002) x	0.784 (0.018) y	0.640 (0.028) x	0.084 (0.003) y

to unseasonably cold nights. Not surprisingly, gizzard shad appear to be less susceptible to acclimation stress during gradual temperature changes, as mortality was minimal when gizzard shad were acclimated at a rate of 0.33°C per day during the tank experiment. However, further research is required to fully evaluate the effect of temperature change on gizzard shad mortality. Interestingly, our results suggest that gradual temperature changes can reduce mortality during the acclimation process but do not prevent high mortality at low temperatures.

In the tank experiment, gizzard shad experienced high mortality rates at a temperature of 4°C or lower across all treatments, even when exposed to temperature declines of 0.33°C per day. These observations provide experimental evidence of previous claims that gizzard shad mortality increases rapidly below 4°C (Heidinger 1983; White et al. 1987) as the fish become increasingly susceptible to cold-stress-related mortality. Survival models developed from the tank experiment predict that (1) gizzard shad held at 4°C can survive roughly twice as long as gizzard shad held at 1°C or 2°C but (2) there is little difference in mortality between fish held at 1°C and those held at 2°C. It is unknown what drives the increase in mortality between 4°C and 1°C or 2°C. In our experiments, gizzard shad appeared to manifest both acute and chronic mortality. Osmoregulatory failure and enzymatic failure are likely causes for the acute mortality observed (as was suggested by White et al. 1987) but do not explain why mortality was drawn out in some cases. White et al. (1987) also suggested that gizzard shad are unable to mobilize all energy stores (i.e., lipids) at low temperatures and therefore may be limited to emergency energy stores (i.e., liver and muscle tissue), leading to chronic mortality at low temperatures. These observations suggest the existence of some interaction between temperature and starvation, but teasing apart the relationship remains difficult (see below).

Starvation

Starvation is known to be a major source of mortality for overwintering gizzard shad in southern reservoirs, as demonstrated by observed changes in whole-fish DW, energy content, and percent lipid content (Pierce et al. 1980) and by the reported differences between healthy and dying fish (Adams et al. 1985). Less is known about the importance of starvation at northern latitudes, where temperatures are consistently below 4°C for the duration of the winter. Across experiments and field collections, we found minimal evidence to support the idea that starvation is the primary driver of gizzard shad mortality.

In the cage experiment, we did not observe consistent patterns of size-selective mortality; thus, smaller fish were not displaying disproportionately higher mortality than larger fish, contrary to what would be expected if starvation was the primary cause of mortality. In fact, several cages exhibited negative size-selective mortality, indicating that the larger fish were experiencing higher mortality than small fish. Several researchers have identified conditions that promote negative size-selective mortality, particularly in fish that feed during winter months (Lankford and

Targett 2001; Connolly and Petersen 2003). In these cases, larger fish have a higher resource demand than smaller fish and may be unable to meet this demand during periods of resource limitation, which are common during winter months. We suspect that this was not the case in our experiments because only very limited feeding by gizzard shad was observed during winter and because food resources (detritus) are not limited. The cause of the observed decrease in gizzard shad TL over time in some of the cages is not known. These patterns may have resulted from experimental error, such as inadequate sample sizes or higher mortality of larger fish due to electrofishing-related stress. However, electrofishing stress seems unlikely, as cages were checked 24 h after stocking and large numbers of dead gizzard shad were not observed. Regardless of the cause, larger individuals in our cage experiment did not possess a survival advantage, making it unlikely that starvation was a factor driving mortality during the cage experiment. Additionally, during the cage experiment, gizzard shad had only recently begun to use energy stores accumulated throughout the growing season; thus, the energy stores should have been adequate to support survival over the short duration (2–3 weeks) of each experimental period.

The tank experiment also produced little evidence that mortality was primarily driven by starvation. If mortality was due to depletion of energy reserves, then gizzard shad in the warmest (4°C) room should have experienced higher mortality rates than fish in the colder rooms since fish metabolism increases at higher temperatures; however, this was not the case in our experiment. Although the gradual rate of mortality in the warmest room could be consistent with mortality from starvation or complications associated with starvation, the rapid die-off at 1°C and 2°C suggests that cold stress—rather than starvation—was driving the mortality in these treatments. The significance of TL in the model demonstrated size-selective mortality in overwintering gizzard shad, wherein better survival was predicted for larger individuals (Table 2); however, this relationship was weaker than those observed for other factors (i.e., temperature). This finding supports field observations that larger fish experience reduced mortality rates (e.g., Heidinger 1983; White et al. 1987) but suggests that extreme temperatures will result in high mortality regardless of size.

Although neither experiment identified starvation as the primary cause of overwinter mortality in gizzard shad, an inability to access available energy reserves may contribute to gizzard shad mortality during acclimation and prolonged exposure to cold temperatures (White et al. 1987). White et al. (1987) observed little change in percent lipid content of gizzard shad over the winter, but they identified declines in liver tissue and function. White et al. (1987) suggested that gizzard shad are unable to mobilize lipid stores at temperatures below 8°C and will instead utilize emergency energy sources, such as liver glycogen and liver tissue. Use of liver tissue and energy stores as an emergency energy source during food deprivation has been observed in several other species (Pastoureaud 1991; Power et al. 2000; Rios et al. 2007) and indicates that decreased liver function could lead to physiological failure and eventual death. Across experiments, we consistently observed disproportionate changes in visceral DW relative to somatic DW, as evidenced by changes in VSI. For the tank experiment, the VSI explained the greatest amount of variation in the logistic regression analysis comparing differences between dead and living gizzard shad. Additionally, percent DW, a metric of available lipid reserves, explained the least amount of variation in the model, suggesting that mortality is not correlated with decreasing lipid reserves but instead is associated with depletion of emergency energy sources, such as liver and muscle tissue. Gizzard shad store lipids within their muscle tissues; therefore, if mortality is related to the depletion of lipid reserves, then somatic tissue would be expected to show the greatest decrease during starvation (Shul'man 1974). The fact that somatic tissue did not exhibit such a decrease indicates that lipid reserves in the muscles are inaccessible to gizzard shad at low temperatures, thus forcing the fish to use smaller emergency energy stores (e.g., liver tissue), which ultimately leads to liver failure and death (White et al. 1987). Therefore, despite their high levels of stored lipids, gizzard shad may be functioning physiologically as fish with exhausted energy reserves. Additional information on the energy content and percent lipid of visceral and somatic tissues will be necessary to fully evaluate the use of lipids in muscle and liver tissue.

Temporal patterns in field collections provided support for the results from cage and tank experiments. Prior to ice formation, when temperatures were approaching 4°C, we observed no changes in the size distributions across sites during either year, despite the observations of large numbers of dead gizzard shad. After ice formation and temperature stabilization, size distributions began to shift toward larger fish; this finding indicates that the smaller fish in the population were suffering higher mortality rates, but we cannot determine whether the smaller fish were dying due to starvation or due to some other size-dependent mechanism. Surprisingly, consistent trends in energy use were not observed between experiments and field collections. Experimental fish showed disproportionate use of visceral tissues relative to somatic tissues, as evidenced by the decrease in VSI over time across experiments; in contrast, field-collected fish showed little change in VSI. These discrepancies could be due to (1) confinement stress associated with the experiments, (2) feeding by field-collected fish, or (3) biased field sampling of survivors over time. Confinement stress does not explain why confined fish would disproportionately rely on visceral energy stores and thus seems an unlikely mechanism for the differences. Feeding by gizzard shad in the field should not have influenced the calculation of VSI because the fish with stomach contents were removed from the analysis; feeding could have reduced the reliance on visceral energy stores, leading to the observed consistency in VSI over time in the field. However, the most likely explanation is that field collections consisted only of survivors. These fish may have been able to utilize visceral and somatic resources or may have begun the winter with higher relative somatic and visceral DWs, giving the appearance that

field-collected fish showed no decline in VSI since the fish in poorer condition had already died.

Synthesis

Within most of the species' native range, gizzard shad are rarely exposed to temperatures below 4°C for prolonged periods of time. Our experiments and field collections collectively support previous literature claims that gizzard shad are unable to tolerate the cold temperatures that are common during winter months in the northern portion of the species' range. Interestingly, temperature and starvation may interact to cause mortality if the gizzard shad are unable to access lipid energy reserves, forcing them to utilize emergency energy sources at cold temperatures. However, our results cannot definitively establish this relationship, and further analyses are needed.

The importance of temperature for influencing the ability of gizzard shad to survive winter indicates that system-specific availability of winter refugia is an important component of recruitment dynamics. In several species and systems, overwinter survival is linked to contributions from relatively small refuge habitats (Magnuson et al. 1985; Bodensteiner et al. 1992). Refuge habitats may buffer fish from stresses associated with temperature, predation, and starvation, and the availability of refuge habitats may influence fish overwinter survival and subsequent recruitment. We have demonstrated a major effect of temperature on the survival rates of gizzard shad; therefore, habitats with higher temperatures during winter should have a strong effect on survival. In Oneida Lake, gizzard shad apparently select marinas and backwater sloughs. These areas generally have higher temperatures and large amounts of detritus but are susceptible to midwinter anoxia if ice remains stable (Fetzer 2009). Further research should assess temperature and dissolved oxygen dynamics of these habitats within and across winters to evaluate their suitability as refuges for overwintering gizzard shad and to aid in the forecasting of gizzard shad responses to predicted climate change.

Currently, gizzard shad appear to have a positive impact on the Oneida Lake fishery. Age-0 gizzard shad provide a valuable prey resource for sport fish in Oneida Lake (Lantry et al. 2008); exhibit limited competition with native prey fish (Roseman et al. 1996); and, when abundant, act as a predation buffer for other prey fish (Fitzgerald et al. 2006). Additionally, high overwinter mortality of age-0 gizzard shad limits the populations of adult gizzard shad, which have been shown to exert negative impacts on water quality and fish communities in Ohio reservoirs (Vanni et al. 2005). A hydrodynamic model for Oneida Lake predicted higher summer water temperatures and decreasing frequency and duration of ice cover over the next century (DeStasio et al., in press). These changes will probably lead to age-0 gizzard shad entering winter at larger sizes and to a greater availability of potential temperature refuges if inconsistent ice cover reduces the development of anoxia in marinas and nearshore embayments. Based on the collective results of our study and previous work, we predict that these changes will lead to increased gizzard shad recruitment to age 1 and a greater role of gizzard shad in the Oneida Lake food web.

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