Metabolic fright responses of different-sized largemouth bass (*Micropterus salmoides*) to two avian predators show variations in nonlethal energetic costs

Steven J. Cooke, Jeff Steinmetz, Jacob F. Degner, Emily C. Grant, and David P. Philipp

Abstract: Recently, researchers have identified that nonlethal costs of predation may arise not only from lost energy intake but also potentially from increased energetic expenditure. During periods of heightened stress following unsuccessful predation attempts, organisms may remain in an altered physiological state with elevated metabolism for some time. Few studies have quantified these nonlethal energetic costs of predation. We monitored the cardiac response (cardiac output (Q), heart rate (f_H), and stroke volume (SV_H)) of largemouth bass, *Micropterus salmoides*, ranging in size from 200 to 425 mm, to simulated avian predation attempts by great blue heron, *Ardea herodias*, and osprey, *Pandion haliaetus*. Fear bradycardia during a 30-s predation attempt varied depending upon the size of the fish and the type of predator. The magnitude of the bradycardia decreased with increasing size of the fish; however, the disturbances were more extreme in response to osprey than to blue heron models. Maximal cardiac disturbance following simulated predation attempts by osprey were consistent among size classes of bass. However, the magnitude of the disturbance following heron predation attempts reduced as the size of the fish increased. Size-specific trends were even more extreme for cardiac-recovery durations. Largemouth bass of all sizes exposed to osprey predation attempts required ~40 min for Q and f_H and ~30 min for SV_H to return to predisturbance levels. Although small bass exposed to heron predation attempts required recovery times similar to fish exposed to osprey predation attempts, as the size of largemouth bass exposed to the heron model increased above ~300 mm, the recovery time decreased significantly. We conclude that the size-specific response of largemouth bass to different predators is reflective of their ability to assess the risk posed by different predators. In addition, the nonlethal costs of predation can be substantial and should be considered in future bioenergetics models.

Résumé : Récemment, des chercheurs ont découvert que les coûts non létaux de la prédation peuvent être reliés non seulement à la baisse de l'apport d'énergie, mais aussi à l'augmentation de la dépense énergétique. Durant les périodes de stress important qui suivent des essais infructueux de prédation, les organismes demeurent dans un état physiologique perturbé avec un métabolisme élevé pendant un certain temps. Peu de chercheurs se sont attardés à quantifier ces coûts énergétiques non létaux de la prédation. Nous avons mesuré la réaction cardiaque (rendement (Q), rythme (f_H) et volume systolique (SV_H)) chez des achigans à grande bouche (*Micropterus salmoides*) de 200 à 425 mm à des attaques simulées de prédation par le grand héron bleu, *Ardea herodias*, et par le balbuzard pêcheur, *Pandion haliaetus*. La bradycardie causée par la peur reliée à une tentative de prédation de 30 s variait en fonction de la taille du poisson et du type de prédateur. L'amplitude de la bradycardie diminuait en fonction inverse de la taille du poisson et les perturbations étaient plus importantes en présence de modèles de balbuzards que de hérons. La perturbation cardiaque maximale qui sévissait à la suite de l'attaque simulée d'un balbuzard était la même chez tous les poissons de la même classe de taille. L'amplitude de la perturbation à la suite d'une tentative de prédation par un héron, cependant,

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S.J. Cooke.[1,2] Program in Natural Resources Ecology and Conservation Biology, Department of Natural Resources and Environmental Sciences, University of Illinois, 607 East Peabody Drive, Champaign, IL 61820, U.S.A., and Center for Aquatic Ecology, Illinois Natural History Survey, 607 East Peabody Drive, Champaign, IL 61820, U.S.A.
J. Steinmetz. Program in Ecology and Evolutionary Biology, Department of Animal Biology, University of Illinois, 607 East Peabody Drive, Champaign, IL 61820, U.S.A., and Center for Aquatic Ecology, Illinois Natural History Survey, 607 East Peabody Drive, Champaign, IL 61820, U.S.A.
J.F. Degner and E.C. Grant. Center for Aquatic Ecology, Illinois Natural History Survey, 607 East Peabody Drive, Champaign, IL 61820, U.S.A.
D.P. Philipp. Program in Ecology and Evolutionary Biology, Department of Natural Resources and Environmental Sciences, University of Illinois, 607 East Peabody Drive, Champaign, IL 61820, U.S.A., and Center for Aquatic Ecology, Illinois Natural History Survey, 607 East Peabody Drive, Champaign, IL 61820, U.S.A.

[1]Corresponding author (e-mail: scooke@interchange.ubc.ca).
[2]Present address: Centre for Applied Conservation Research, Department of Forest Sciences, The University of British Columbia, 2424 Main Mall, Vancouver, BC V6T 1Z4, Canada.

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diminuait en fonction inverse de la taille du poisson. Les tendances spécifiques à la taille étaient encore plus extrêmes pour ce qui est de la durée de la récupération cardiaque. Chez les achigans à grande bouche de toutes les tailles soumis aux tentatives de prédation des balbuzards, il fallait ~40 min pour que Q et f_H et ~30 min pour que SV_H retrouvent leurs valeurs initiales d'avant l'attaque. Bien que les petits achigans exposés aux attaques du héron aient requis le même temps pour récupérer que les poissons exposés au balbuzard, à mesure que la taille des poissons exposés au héron augmentait au-delà de ~300 mm, le temps nécessaire à la récupération diminuait significativement. Nous concluons que, chez les achigans à grande bouche, la réaction spécifique à la taille à différents prédateurs reflète leur capacité d'évaluer les risques que posent les diverses espèces de prédateurs. De plus, les coûts non létaux de la prédation peuvent être élevés et devront désormais être pris en compte dans les modèles de bioénergétique.

[Traduit par la Rédaction]

Introduction

Predation has long been a focus of ecological research (Volterra 1926; Brooks and Dodson 1965; Paine 1966). Traditionally, ecologists have focused on the obvious lethal effects of predation (Volterra 1926; Brooks and Dodson 1965). In recent years, however, there has been a growing interest in nonlethal effects of predation attempts on prey (e.g., Lima and Dill 1990; Fraser and Gilliam 1992; Peckarsky and McIntosh 1998). Nonlethal costs occur when predators alter the physiology or behavior of the prey in such a way as to reduce growth rates, increase energetic demand, and ultimately decrease fitness (Godin and Sproul 1988; Lima and Dill 1990; Werner and Anholt 1996). Such nonlethal costs have been found in a variety of taxa, including frogs (Werner and Anholt 1996), mayflies (Peckarsky and McIntosh 1998), water striders (Krupa and Sih 1998), and fish (Mittelbach and Chesson 1987; Fraser and Gilliam 1992).

Nonlethal predator effects on fish include altered or restricted habitat use (e.g., Power 1984; Reinhardt and Healey 1997; Allouche and Gaudin 2001); changes in foraging (e.g., Miliniski and Heller 1978; Martel and Dill 1993), territorial (Martel and Dill 1993; Martel 1996), and respiratory (Kramer et al. 1983; Smith and Kramer 1986) behavior; decreased growth rate (Power 1984; Diehl and Eklöv 1995); and lower reproductive output (Fraser and Gilliam 1992). As the above studies demonstrate, part of the predator-induced decrease in growth rate and reproductive output is the result of lost foraging opportunities and decreased energy intake. Some of the nonlethal costs, however, may occur not as lost energy intake but as increased energy output. Few studies have explored potential metabolic costs associated with predator attacks, owing in part to the difficulty of monitoring metabolic rates in free-ranging animals at the temporal scale required.

Perhaps the most successful approach to estimating metabolic rate in fish (and indeed in other vertebrates) is through monitoring cardio-respiratory parameters; it is the only method that provides minute-to-minute information (Lucas et al. 1993). The primary role of the cardiovascular system is oxygen delivery to aerobic tissues, but it also transports hormones and metabolites arising from stress (Satchell 1991). Many of these cardio-respiratory parameters, including ventilation rates and heart rate, are correlated with metabolic rate and serve as sensitive stress indicators (Lucas et al. 1993). Energetic assessments are particularly effective for quantifying individual-level consequences of stress (Maltby 1999). Several studies employing this approach have been conducted on predation risk in different taxa, including mammals (Jacobsen and Stuart 1978; Moen et al. 1978) and fish (Metcalfe et al. 1987; Johnsson et al. 2001). Collectively, these papers indicate that predation threat can be metabolically costly and may contribute to the decreased growth rate reported in earlier studies.

A prey's ability to compensate for these lost foraging opportunities and increased energetic costs will depend upon both the frequency of predator attacks and how quickly the prey returns to normal physiological and behavioral states. If predator attacks are infrequent, or if prey return to normal states quickly after the predator attacks, there may be little lost foraging opportunities or energetic losses. Conversely, if predation risk is persistent, or if the prey take a long time to return to a normal state, a large net loss of energy is possible. The magnitude of the predation risk may also vary for a given species depending upon its size. For example, small larval tadpoles show greater responses to predation risk than large tadpoles (Eklöv 2000). Ideally, prey could assess their risk, based on factors such as their size or the type of predator, and adjust their behavioral and physiological responses appropriately. A number of studies have shown that prey do differentiate among predators (Krupa and Sih 1998; Peckarsky and McIntosh 1998; Eklöv 2000), but few studies have shown different physiological responses to multiple predators.

The goals of this study were to (*i*) determine whether the threat of predation induced a cardiac response in prey, (*ii*) determine the recovery time of the prey to a pre-attack state, (*iii*) examine whether the cardiac response and recovery time differed between two types of predators, and (*iv*) determine if fish exhibit size-specific responses to different predator types. By monitoring the cardiac output (Q), heart rate (f_H), and stroke volume (SV_H) of fish before, during, and after encounters with model predators, we determined how simulated predator attacks altered the metabolic rate of the fish. Specifically, we used models of a great blue heron, *Ardea herodias*, and an osprey, *Pandion haliaetus*, to test whether largemouth bass, *Micropterus salmoides*, respond to simulated predator attacks by altering the cardiac parameters indicative of metabolic rate.

Materials and methods

Study site and animals

Largemouth bass used in this study were angled (landed within 20 s) from Lake Opinicon, located in eastern Ontario,

between 17 and 26 June 2001. Lake Opinicon is a shallow oligotrophic lake (maximum depth 11 m, surface area 890 ha) with extensive vegetated littoral zones. The lake has reasonable densities of both osprey and heron that are frequently observed "fishing" in the regions where the test fish were collected. During the 2001 breeding period, seven osprey nests were located on Lake Opinicon (Queen's University Biological Station (QUBS) staff, personal communication) and the ospreys were observed to fish in littoral areas. Heron were also observed frequently in littoral areas. A heron rookery is located on the QUBS property within 3 km of Lake Opinicon (QUBS staff, personal communication). Upon capture, fish were placed in darkened aerated coolers (60 L) and transported immediately to the laboratory. Fish ranging in size from 200 to 425 mm were targeted for these experiments. Fish were held in large aquaria (200 L) for 24 h prior to surgery, to ensure that they had acclimated to laboratory conditions and were in a post-absorptive state. During this period, fish were continuously provided with fresh lake water and exposed to the ambient photoperiod. Water temperatures during the experiments ranged between 23.4 and 25.1°C.

Cardiovascular monitoring

Surgical procedures and the *Q* apparatus are described in detail elsewhere (Schreer et al. 2001). Briefly, each fish was anaesthetized with 60 ppm clove oil (emulsified with ethanol, 9:1 ethanol : clove oil) until the fish lost equilibrium and was nonresponsive. Water containing a maintenance concentration of anaesthetic (30 ppm clove oil) was pumped over the gills during surgery. A flexible silicone cuff-type Doppler flow probe (subminiature 20 MHz piezoelectric transducer; Iowa Doppler Products, Iowa City, Iowa, U.S.A.) was placed around the aorta. The lead wire from the probe was then sutured to the side of the fish in six locations, to prevent shifting of the cuff. Using a flowmeter (545C-4 Directional Pulsed Doppler Flowmeter; Bioengineering, The University of Iowa, Iowa City, Iowa, U.S.A.) and a digital strip-chart recorder (LabVIEW, Version 4.0.1, National Instruments Corporation, Austin, Texas, U.S.A.), we monitored cardiac parameters using the techniques outlined in Schreer et al. (2001).

Following surgery, individual fish were immediately placed in a 70-L tank (50 × 50 cm) and monitored until they regained equilibrium. Fish were allowed to recover from surgery and to acclimate to the tank for at least 18 h. The experimental tanks were continuously supplied with fresh lake water at 24.1 ± 0.3°C. No cover was provided for the tanks. The fish had unobstructed views of the freshly painted ceiling, which was off-white. All experiments were conducted between the hours of 1000 and 1600, to minimize the effects of differing light levels and diel activity patterns. Cardiac parameters were recorded continuously for at least 1 h prior to the simulated predation attempts (resting), during the predation attempts, and for at least 4 h post disturbance (recovery). Access to the laboratory was restricted during resting and recovery, to prevent external disturbance.

Avian predator models

Model avian predators have been used successfully in a number of other studies to elicit behavioral responses in fish (herons: Smith and Kramer 1986; Godin and Sproul 1988; kingfishers: Millinski and Heller 1978; Gotceitas and Godin 1991; Litvak 1993). The great blue heron model was a life-sized taxidermy mount of an adult male bird. The mounted heron was standing in a foraging position, with wings folded and the neck half extended. The beak was facing down and away from the body. The heron model was brought from outside the tank room to the tank edge. It was held in this position with occasional subtle turning from side to side for 30 s, at which time the model was tilted, bringing the beak down rapidly so that it broke the surface of the water. The heron was immediately lifted back to an upright position and slowly removed from the room.

The osprey model was carved from blue insulation foam board and was painted. The model was less than life-sized, measuring 74 cm from wing tip to wing tip. The wings were fully outstretched and pointing slightly upwards, symmetrically. The beak was facing forward and downward. The talons were open and outstretched and the tail was slightly spread. The model was attached to a 2-m pole under the left wing. The model was carried into the room and the pole was used to "fly" the model slowly in a circle 1–2 m above the tanks for 30 s. The final pass was made just above the tank and the talons were made to break the surface of the water. The model was then removed from the room. The operator remained beyond the line of sight of the fish.

Data analysis

After a 5-h recovery period, fish were euthanized with an overdose of anaesthetic (180 ppm clove oil) and a postmortem calibration was conducted to convert Doppler shift (in volts) to actual blood flow ($mL \cdot min^{-1}$) (Schreer et al. 2001). This method has proven reliable for determining resting cardiovascular rates in other studies of largemouth bass (e.g., Cooke et al. 2003*b*) and the resting cardiovascular values we observed in this study compare favorably with those data. To determine baseline cardiac values, magnitudes of response, and recovery times, traces for Q, f_H, and SV_H were first plotted for each fish as 60-s means and evaluated visually. A fish was considered to be recovered when values returned to resting and became stable (within 5% of resting values) (Schreer et al. 2001). The maximal disturbance was determined as the maximal change in a cardiac parameter (either positive or negative from predisturbance levels) during recovery. For some analyses, response variables were divided among three size classes of test fish (small, 201–275 mm; medium-sized, 276–350 mm; large, 351–425mm).

Data were assessed visually for normality, using quantile plots, and for homogeneity of variance, using residual plots (SYSTAT, version 8.0; SAS Institute Inc., Cary, North Carolina, U.S.A.). The premise of all analyses was to test two null hypotheses: (1) that there were no differences in response variables among the two predators for each size class and (2) that there were no differences within each of the species across the size classes. We used two-way analysis of variance to look for the effects of species and size on the cardiac responses (JMP, version 4.0; SAS Institute Inc.). Planned contrasts were used to examine where specific differences of interest occurred, particularly in the presence of interactions. Where appropriate, we also used least-squares

linear regression to examine the relationship between size of fish and the response variable of interest. We then used analysis of variance to test whether the slope of the line differed from zero. Tests were considered significant at $\alpha = 0.05$.

Results

Mean water temperatures (24.1 ± 0.3°C) were similar among experiments conducted on both treatment groups and among size classes (Table 1). The total length of fish differed significantly among size classes, as expected (Table 1), increasing from small (201–275 mm) to medium-sized (276–350) to large (351–425) (Table 1). Within each size-specific group, the mean total length of fish was similar for the two predator treatment groups (Table 1). Basal cardiac values were similar for the two predator treatments and did not differ among the three size classes of largemouth bass for all cardiac parameters that we monitored (Table 1). The mean basal cardiac values for all 45 fish used in this series of experiments were 37.8 ± 0.8 $mL \cdot kg^{-1} \cdot min^{-1}$($Q$); 48.2 ± 0.7 beats/min (f_H); 0.772 ± 0.011 $mL \cdot kg^{-1}$ (SV_H,).

The immediate cardiac response of fish exposed to either of the avian predators was a substantial bradycardia (reduced f_H). Bradycardia began within 2 s of the predator being introduced above the tank. During this period, fish were believed to be stationary based upon auditory observations (i.e., fish were not heard splashing or banging against the tank sides). The magnitude of the bradycardia varied by type of predator and the size class of the fish (Fig. 1A, Table 2). For both predator types, the bradycardia was more extreme in small fish than in large fish (Fig. 1B). There was also a consistent pattern of less-extreme bradycadia in fish exposed to the heron (27.5 ± 0.9 beats/min) than those exposed to the osprey (23.8 ± 0.9 beats/min) (Fig. 1). The fear bradycardia experienced by the fish terminated when the model predators simulated attacks. At this time, a small number of the fish attempted to avoid the predator as shown by them bumping against the tank wall. However, the relatively small size of the tank minimized any opportunity for locomotory escape behaviours. Upon the subsequent removal of the predator, all f_Hs became tachycardic (higher than basal levels), resulting in maximal cardiac disturbance within 3 min.

Maximal cardiac disturbance in largemouth bass differed by predator on a size-specific basis (Table 2). Cardiac output and f_H following a simulated osprey attack increased ~30 and ~45%, respectively, over basal levels for all size classes of largemouth bass (Figs. 2A, 2B). Maximal Q disturbance following a simulated heron attack, however, was ~30% for small and medium-sized fish but only ~15% for larger fish (Fig. 2A). Heart rate exhibited a similar pattern following a simulated heron attack (~50% for small fish, ~40% for medium-sized fish, and only ~25% for larger fish; Fig. 2B). There was no relationship between either maximal Q or f_H and fish size for largemouth bass exposed to simulated osprey attacks. However, fish exposed to simulated heron attacks exhibited a strong negative relationship between fish size and both maximal Q and f_H (Figs. 2D, 2E). Stroke volume differed from f_H and Q in that it decreased in response to disturbance. Although SV_H following a simulated osprey attack decreased to ~75% of basal levels for all size classes, maximal disturbance following heron attack decreased to ~80% of basal levels for small and medium-sized fish and only ~85% for larger fish (Table 2, Fig. 2C). The maximal change in SV_H of largemouth bass exposed to simulated osprey attacks was independent of fish size (Fig. 2F); however, largemouth bass exposed to simulated heron attacks exhibited a strong positive relationship between fish size and maximal change in SV_H (Fig. 2F).

Following the maximal change in cardiac parameters in response to the simulated predator attacks, Q and f_H gradually decreased, whereas SV_H rose as values returned to basal levels. The time required for cardiac parameters to return to basal levels varied for type of predator and size of fish (Table 2). Cardiac output and f_H required ~40 min to recover across all three size classes following a simulated osprey attack, whereas following a simulated heron attack, recovery time was most rapid for the largest fish (~20 min), slowest for the smallest fish (~40 min), and intermediate for medium-sized fish (~30 min) (Figs. 3A, 3B). Recovery duration differed significantly between heron and osprey attacks for fish of medium and large size classes for both Q and f_H (Figs. 3A, 3B). There was no relationship between either Q or f_H recovery time and size of fish following osprey attack, but recovery time for both Q and f_H following heron attack decreased significantly with increasing size of fish (Figs. 3D, 3E). Stroke volume generally recovered more rapidly than Q and f_H, although patterns of recovery were similar among all three parameters (Fig. 3C). Stroke volume required ~30 min to recover across all three size classes following simulated osprey attack, whereas following heron attack, recovery time was most rapid for the largest fish (~15 min), slowest for the smallest fish (~35 min), and intermediate for medium-sized fish (~25 min) (Table 2, Fig. 3C). Recovery duration differed between heron and osprey attacks for medium-sized fish and large size classes. Stroke volume recovery time following osprey attack did not differ across all sizes of fish, whereas following heron attack, recovery time decreased significantly with increasing size of fish (Fig. 3F).

Discussion

Although other studies have examined the nonlethal effects of predators on prey, ours is the first to use Q as an indicator of the metabolic costs of predation attempts on fish. Part of the decreased growth rate and lowered reproductive output found in other studies of nonlethal effects of predators is undoubtedly attributable to lost foraging opportunities and decreased energy intake. During the period of cardiac recovery following disturbance, it is likely that bass do not consume food (Cooke et al. 2002). Indeed, other studies have shown decreased foraging occurs in the presence of predators (Milinski and Heller 1978; Metcalfe et al. 1987; Martel and Dill 1993). Using Q as a correlate of energy expenditure, however, it is clear that some of this loss of growth is not only due to lost energy intake but to increased energy expenditure associated with increased fear and alertness.

Largemouth bass had very different size-specific responses to the simulated attacks of two different predators, ospreys and herons. This difference is probably due to size-specific differences in the vulnerability of largemouth bass to these two predators. Osprey can consume prey of between 130 and

Table 1. Meristics of largemouth bass (*Micropterus salmoides*) exposed to simulated avian predator attacks.

Predator	Size class	*N*	Water temperature (°C)	Total length (mm)	Basal cardiac output ($mL \cdot kg^{-1} \cdot min^{-1}$)	Basal heart rate (beats/min)	Basal stroke volume ($mL \cdot kg^{-1}$)
Heron	Small	5	24.2 ± 0.2	237 ± 6 aA	40.2 ± 2.1	48.3 ± 2.5	0.753 ± 0.027
	Medium-sized	11	24.1 ± 0.1	310 ± 4 bB	38.3 ± 1.3	51.0 ± 1.8	0.763 ± 0.022
	Large	9	24.2 ± 0.2	383 ± 4 cC	35.7 ± 1.5	45.8 ± 2.5	0.793 ± 0.022
Osprey	Small	7	24.0 ± 0.2	231 ± 5 aA	35.9 ± 2.3	48.3 ± 1.9	0.801 ± 0.034
	Medium-sized	7	23.9 ± 0.1	305 ± 5 bB	37.7 ± 2.9	47.3 ± 1.6	0.738 ± 0.021
	Large	6	24.3 ± 0.2	372 ± 5 cC	34.8 ± 2.6	47.9 ± 1.5	0.807 ± 0.027

Note: Values are means ± 1 SEM for specific predators and size classes. Because water temperature and all basal cardiac parameters did not differ by predator or size class, no designations are provided. Total length, however, differed significantly among size classes ($F_{[2,39]} = 408.0$, $P < 0.001$). For total length, dissimilar lowercase letters indicate significantly different ($P < 0.05$) values within a predator type across the size classes; dissimilar uppercase letters indicate significant differences ($P < 0.05$) among predator types for specific size classes.

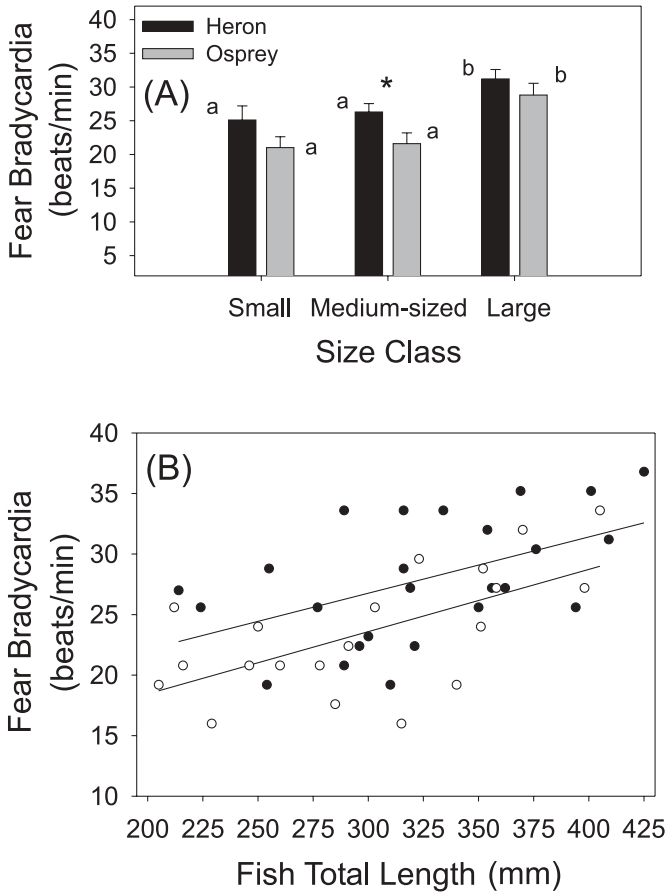
Table 2. Analysis of variance model parameters for largemouth bass magnitude of fear bradycardia, maximal cardiac disturbance, and recovery time, following exposure to simulated predator attacks; for all cardiac variables monitored the main effect was species of predator and the secondary effect was size of the fish.

Parameter	Source	SS	df	*F*	*P*
Magnitude of bradycardia	SPP	141.24	1	7.6158	0.009
	Size	388.27	2	10.4677	<0.001
	SPP × size	11.17	2	0.3011	0.742
	Error	723.29	39		
Cardiac output maximal disturbance	SPP	453.30	1	12.2705	0.001
	Size	676.15	2	9.1514	0.001
	SPP × size	278.60	2	3.7708	0.032
	Error	1440.76	39		
Heart rate maximal disturbance	SPP	415.41	1	6.4191	0.015
	Size	1080.66	2	8.3495	0.001
	SPP × size	991.89	2	7.6636	0.002
	Error	2523.86	39		
Stroke volume maximal disturbance	SPP	160.57	1	4.2102	0.046
	Size	94.31	2	1.2365	0.302
	SPP × size	264.63	2	3.4695	0.041
	Error	1487.36	39		
Cardiac output recovery time	SPP	722.58	1	33.8671	<0.001
	Size	669.36	2	15.6864	<0.001
	SPP × size	777.23	2	18.2143	<0.001
	Error	832.09	39		
Heart rate recovery time	SPP	589.45	1	27.9454	<0.001
	Size	608.79	2	14.4313	<0.001
	SPP × size	859.22	2	20.3673	<0.001
	Error	822.62	39		
Stroke volume recovery time	SPP	350.99	1	16.2309	0.003
	Size	707.39	2	16.3558	<0.001
	SPP × size	565.31	2	13.0709	<0.001
	Error	843.38	39		

600 mm (Swenson 1978; Chubbs and Trimper 1998; Glahn et al. 1999). Although great blue herons typically consume fish less than 200 mm long, larger herons can eat fish of up to 300 mm (Forbes 1987; Butler 1992; Stickley et al. 1995; Glahn et al. 1999). Because all sizes of largemouth bass tested were vulnerable to osprey, they showed a similar f_H response during the simulated predator attacks. Largemouth bass, however, become increasingly less vulnerable to great blue herons as they begin to exceed 200 mm in total length, which explains why the cardiac response decreased with increasing fish size.

We observed varying degrees of bradycardia when model predators were first introduced but before they actually "attacked." All sizes of fish tested with the osprey model showed a similar magnitude of bradycardia, whereas larger fish had less pronounced f_H reductions than smaller fish when responding to simulated heron attacks. This difference in response may indicate that the bass are attempting to remain more cryptic or have more fear of ospreys than of herons. The bradycardia was indicative of an overall circulatory depression, because there was no way SV_H could compensate for such large reductions in f_H. Some evidence suggests that respiratory activity inhibits normal vagal tone (Satchell 1991). A cessation of breathing, such as is observed during

Fig. 1. (A) Bradycardia magnitude following simulated predator attacks in three size classes of largemouth bass (*Micropterus salmoides*). All values are means ± 1 SEM. Dissimilar letters indicate significantly different ($P < 0.05$) values within a predator type across the size classes. Asterisks indicate significant differences ($P < 0.05$) in bradycardia magnitude among predator types for specific size classes. (B) Relationship between magnitude of bradycardia and total length of largemouth bass exposed to simulated heron (●) attacks (bradycardia = 12.81 + 0.05x, $r^2 = 0.26$, $F_{[1,23]} = 8.0641$, $P < 0.009$) and simulated osprey (○) attacks (bradycardia = 8.16 + 0.051x, $r^2 = 0.39$, $F_{[1,18]} = 11.7353$, $P = 0.003$).

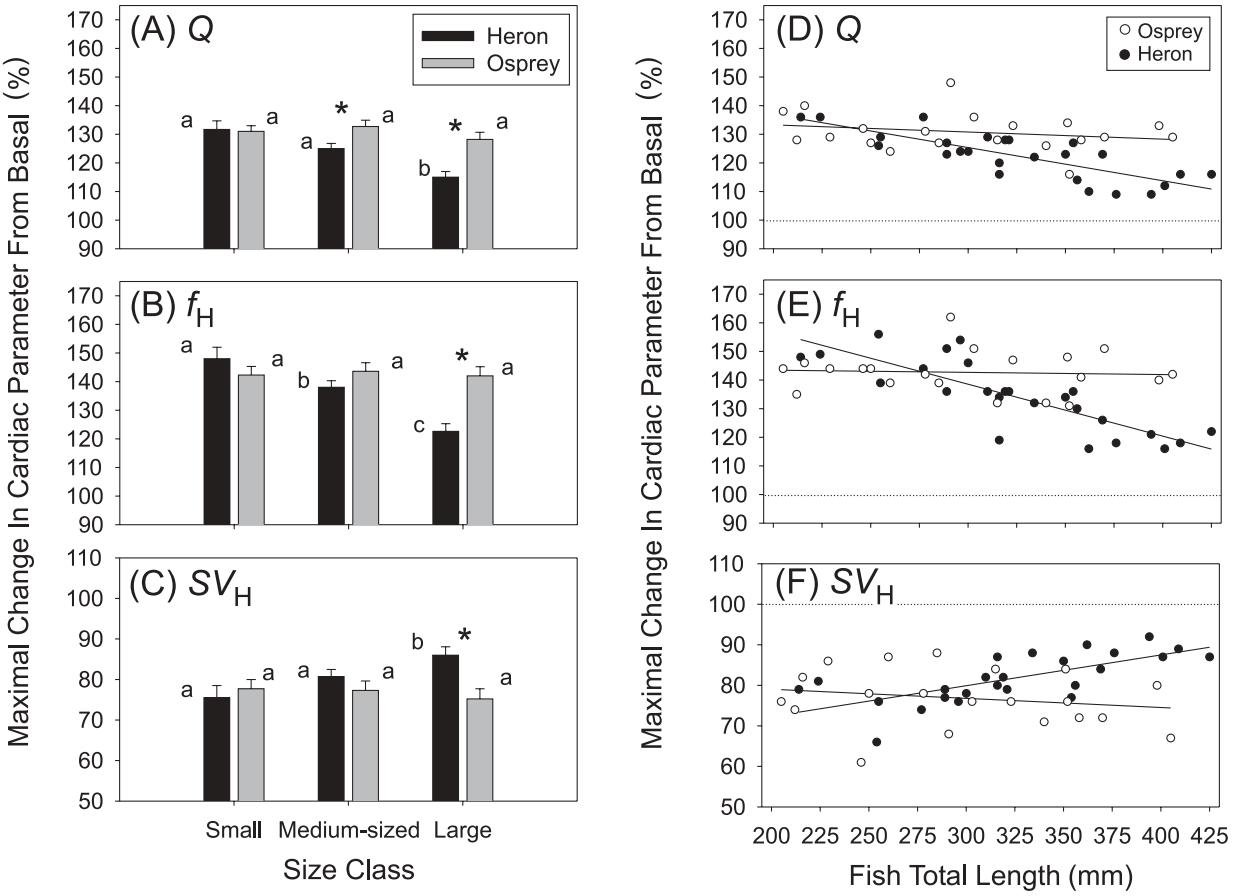


the initial "freeze" of fish exposed to predators, results in a rise in vagal tone to the heart (Satchell 1991), resulting in bradycardia. Although we did not directly observe ventilation rates, they are strongly coupled with f_H, suggesting that ventilation rates slowed during the freeze response. Interestingly, Metcalfe et al. (1987) observed that when exposed to a predator threat, fish were hyperactive, creating erratic patterns of opercular contraction. Temporary reductions in cardiorespiratory activity are adaptive if they obscure the presence of the fish to the predator. It is also possible that the bradycardia was anticipatory, as has been observed in lingcod, *Ophiodon elongatus*, prior to spontaneous activity (Farrell 1982). Physiologically, bradycardia is an important mechanism for delaying the phasing up of cardiac pumping rates following disturbance without creating major peaks in flow that could damage delicate vessels or tissue.

The levels of cardiac disturbance that we present here were more extreme than those reported in other accounts of cardiac disturbance due to predation threat (Höjesjö et al. 1999; Johnsson et al. 2001). Höjesjö et al. (1999) reported that juvenile rainbow trout, *Oncorhynchus mykiss*, exhibited very minor increases in f_H following simulated predation by a heron. Johnsson et al. (2001) monitored f_H responses of juvenile Atlantic salmon, *Salmo salar*, and found that, in age 1+ wild salmon, a return to basal values had not occurred after a 15-min monitoring period. Conversely, in domesticated salmon, a return to basal levels was seen within several minutes of the attack. The fish typically experienced a bradycardia during the attack and, in some cases, a tachycardia post attack but, in general, f_H only increased by a maximum of about 15%. More often, the change in f_H was closer to 5%. In our study, f_H increased by as much as ~50% above resting values. However, consistent with our results, Johnsson et al. (2001) reported that age 1 (~18 cm) Atlantic salmon had more pronounced f_H responses compared with age 2 fish (~30 cm). They concluded that the general reduction in antipredator response in age 2+ salmon may be due to their larger size. Consistent with this supposition, these sizes of fish (2+) also correspond to the upper limit of herons gape size. The relatively minimal cardiac response observed by Johnsson et al. (2001) may be due to the limitations imposed by only monitoring f_H in salmonids. When exposed to exercise or stress, f_H is usually rather static in salmonids (e.g., Anderson et al. 1998), while the changes in Q are usually due to increases in SV_H (see Farrell 1991; Thorarensen et al. 1996). Conversely, when largemouth bass are exposed to exercise or stress, SV_H is static or even decreases, whereas f_H typically increases (Schreer et al. 2001; S.J. Cooke, unpublished data). Even with a decrease in SV_H, the massive increase in f_H is sufficient to increase Q. In our study, we measured all three cardiac parameters of which the resting cardiac variables compared favorably with other data collected in our laboratory (Cooke et al. 2003*b*). If Höjesjö et al. (1999) and Johnsson et al. (2001) had monitored other cardiac paramenters, they might have seen larger effects. Indeed, in Johnsson et al. (2001), there is evidence for size-specific responses in f_H to avian predation attempts; however, they were not statistically significant.

The magnitude of cardiac disturbance associated with avian predation attempts falls in the intermediate range of responses to other stressors experienced by largemouth bass (Table 3). Most of the stressors that produced the greatest disturbances and the longest recovery periods are stressors and (or) activites that are faced by fish on rare occasions. In the field, largemouth bass may experience such levels of exhaustive exercise during riverine migrations, negotiation of fishways or obstructions, or when angled. Conversely, localized intermediate swimming activity, social interactions, and predator encounters are activities that are experienced more regularly by fish. Although avian predation attempts only require intermediate recovery periods relative to exhaustive exercise, predation attempts would likely be encountered more frequently. Although social interactions and low-speed swimming also elicit cardiac responses (Cooke et al. 2002), the fish are able to recover very rapidly compared with predator attacks (Table 3). Johnsson et al. (2001) and Höjesjö et al. (1999) suggest that the high postfright f_H may be adaptive, because the high f_H will supply the body with oxygen to pre-

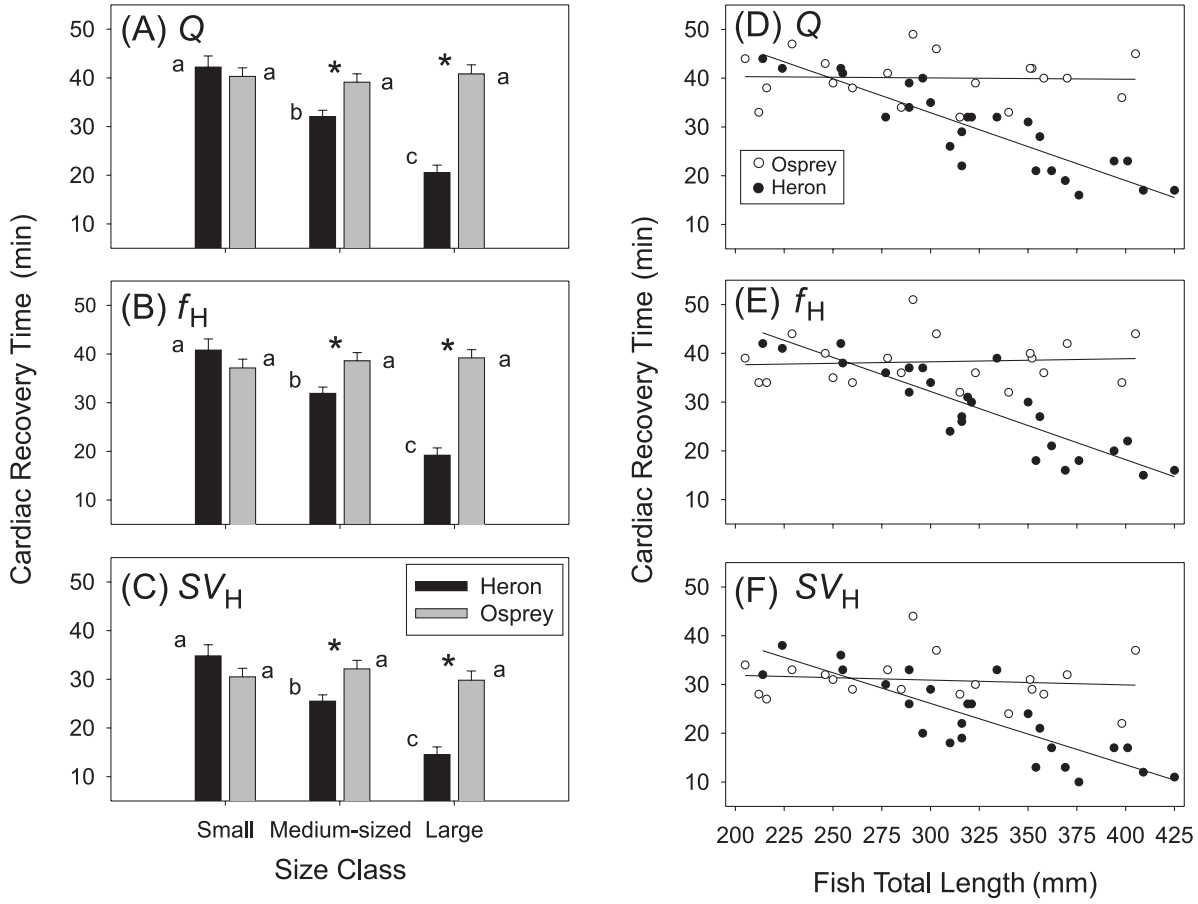
Fig. 2. Maximal change in cardiac parameters (*Q*, cardiac output (A); f_H, heart rate (B); and SV_H, stroke volume (C)) following simulated predator attacks in three size classes of largemouth bass. All values are means ± 1 SEM. Dissimilar letters indicate significantly different ($P < 0.05$) values within a predator type across the size classes. Asterisks indicate significant differences ($P < 0.05$) in maximal cardiac disturbance among predator types for specific size classes. Relationship between magnitude of cardiac disturbance in three cardiac parameters (*Q* (D); f_H (E); and SV_H (F)) and total length of largemouth bass exposed to simulated heron (●) attacks (*Q* disturbance = 160.13 – 0.12*x*, $r^2 = 0.64$, $F_{[1,23]} = 40.4807$, $P < 0.001$; f_H disturbance = 193.02 – 0.18*x*, $r^2 = 0.68$, $F_{[1,23]} = 48.2199$, $P < 0.001$; SV_H disturbance = 57.14 – 0.08*x*, $r^2 = 0.50$, $F_{[1,23]} = 22.7163$, $P < 0.001$) and simulated osprey (○) attacks (*Q* disturbance = 138.26 – 0.025*x*, $r^2 = 0.06$, $F_{[1,18]} = 1.0403$, $P = 0.321$; f_H disturbance = 147.57 – 0.018*x*, $r^2 = 0.02$, $F_{[1,18]} = 0.1524$, $P = 0.705$; SV_H disturbance = 83.53 – 0.023*x*, $r^2 = 0.04$, $F_{[1,18]} = 0.7135$, $P = 0.409$).



pare for flight should the predator appear again (Ydenberg and Dill 1986). We agree that the initial adaptive value of the fight–flight response should aid in survival. When cardiac activity (and therefore active metabolic rate) remains heightened for an extended period of time, however, the fish has less metabolic scope to escape from predators or to deal with other stressors (Priede 1977; Priede 1985). It is during this prolonged period of heightened f_H and recovery from oxygen debt that the fish may be particularly vulnerable to predators. Priede (1977) suggests that the more time fish metabolic rates are exceeding the normal range, the greater is the chance for negative impact on fitness and probability for mortality. Frequent avian predation attempts, even if resulting in no physical injury, could result in direct fitness impacts and increased mortality risk from elevated metabolic activity and a decrease in available scope to respond to other stressors. Furthermore, the increases in metabolic rate have obvious energetic costs that will result in less energy for somatic growth or reproduction.

Our study, and other controlled predator-attempt experiments (e.g., Höjesjö et al. 1999; Johnsson et al. 2001), have been conducted in relatively confined conditions. As a result, behavioural responses, including bursts of locomotion, may have been reduced over what could be expected in free-swimming fish. Sparse field data suggest that a 20-s burst of maximal locomotory activity required to escape a predator equals (in energy expenditure) 15 min of active metabolism (at maximum sustainable swimming speed) or 3 h of standard metabolism (Brett and Groves 1979). Other researchers (Adams et al. 1982; Soofiani 1983) have suggested that costs associated with foraging and predator escape activities in active metabolism may reach ~40% of energy intake. Rice (1990) used a combination of empirical and theoretical data to examine the bioenergetic consequences of different stressors on largemouth bass. The models suggested that a 10% chronic increase in metabolic rate reduced net growth by 22%. Intermediate intermittent stressors that resulted in zero food consumption and a 50% increase in respiration rate for 1 day every 3 weeks resulted in a 23% reduction in net growth over the season. Indeed, acute mild stress is

Fig. 3. Cardiac recovery time for three cardiac parameters (Q, cardiac output (A); f_H, heart rate (B); and SV_H, stroke volume (C)) following simulated predator attacks in three size classes of largemouth bass. All values are means ± 1 SEM. Dissimilar letters indicate significantly different ($P < 0.05$) values within a predator type across the size classes. Asterisks indicate significant differences ($P < 0.05$) in recovery time among predator types for specific size classes. Relationship between cardiac recovery time in three cardiac parameters (Q (D); f_H (E); and SV_H (F)) and total length of largemouth bass exposed to simulated heron (●) attacks (Q recovery = 74.65 – 0.14x, r^2 = 0.79, $F_{[1,23]}$ = 87.8587, $P < 0.001$; f_H recovery = 74.11 – 0.14x, r^2 = 0.77, $F_{[1,23]}$ = 80.4594, $P < 0.001$; SV_H recovery = 63.86 – 0.13x, r^2 = 0.71, $F_{[1,23]}$ = 55.5245, $P < 0.001$) and simulated osprey (○) attacks (Q recovery = 40.88 – 0.002x, r^2 = 0.01, $F_{[1,18]}$ = 0.0227, P = 0.882; f_H recovery = 38.22 – 0.001x, r^2 = 0.01, $F_{[1,18]}$ = 0.0007, P = 0.979; SV_H recovery = 33.85 – 0.019x, r^2 = 0.02, $F_{[1,18]}$ = 0.2932, P = 0.595).



known to impair feeding and can increase metabolic rates of fish by 50–121% over basal levels (Smart 1981; Barton and Schreck 1987). Collectively, these findings indicate that relatively few attack episodes in the life of the fish may result in significant proportions of metabolic expenditure. Additionally, while Q (metabolic rate) is heightened, blood is diverted from the gut region leading to reduced or halted disgestion (Randall and Daxboeck 1982). Although there are no studies that have explicitly examined gut blood flow following predation attempts, several researchers have documented substantial decreases in gut blood flow in response to spontaneous struggling (Farrell et al. 2001). It may take up to 20 min to return to routine gut flow levels following these reductions in blood flow in response to spontaneous struggle (Farrell et al. 2001). Prior to recovery from the predation attempt, therefore, not only is prey consumption unlikely, but food previously consumed is not likely to be digested. During stress, energy may also be mobilized, reducing energy stores (Barton and Schreck 1987).

In general, prey do not compensate for the energetic losses associated with predator attacks (Fraser and Gilliam 1992; Diehl and Eklöv 1995; Reinhardt and Healey 1997; Allouche and Gaudin 2001), although several studies suggest that compensation may be possible (Russell and Wootton 1992; Nicieza and Metcalfe 1997). A prey's ability to compensate for lost foraging opportunities and increased energetic costs will depend upon both the frequency of predator attacks and how quickly the prey returns to a normal physiological and behavioral state following such an attack. Little is known about natural predation rates of ospreys, but one study shows a predation rate of 2.1 trout/day at hatcheries in the northeastern U.S. (Glahn et al. 1999). Predation rates of herons vary: 13.6 prey/ha each day for Midwestern streams (Steinmetz et al. 2003), 0.14 catfish/h at some hatcheries (Glahn et al. 2000), 0.8 catfish/h at other catfish farms (Stickley et al. 1995), and 2.2 trout/h at aquaculture facilities in the northeastern U.S. (Glahn et al. 1999). Conservatively, if largemouth bass in these systems are exposed to one osprey attack and one heron attack every day, this translates into ~90 min of the day that the bass spends not foraging

Table 3. Summary of largemouth bass cardiac responses to a variety of different stressors for comparison with magnitude of disturbance and recovery time following avian predation attempts.

Water temperature (°C)	Stressor or activity	Relative increase in heart rate (% resting)	Cardiac recovery time (min)	Source
24	Predation attempt by osprey (large bass)	145	39	This study
	Predation attempt by osprey (small bass)	144	38	This study
	Predation attempt by heron (large bass)	120	20	This study
	Predation attempt by heron (small bass)	150	42	This study
	Frontal display to conspecific	133	3	Cooke et al. 2002
	Potential prey opportunity	144	2	Cooke et al. 2002
	Displacement (10 s of swimming)	128	2	Cooke et al. 2002
3	Exhaustive exercise	430	140	Cooke et al. 2003*a*
13	Exhaustive exercise and air exposure	178	134	Cooke et al. 2003*b*
17	Exhaustive exercise and air exposure	188	135	Cooke et al. 2003*b*
21	Exhaustive exercise and air exposure	202	134	Cooke et al. 2003*b*
25	Exhaustive exercise and air exposure	218	129	Cooke et al. 2003*b*
21	Graded hypoxia followed by normoxia	84	>180	Furimsky et al. 2003
24	Angling tournament	195	–180	C.D. Suski et al.[1]
25	Forced swimming (0.5 BL/s)	–120	na	S.J. Cooke, unpublished data
	Forced swimming (1.0 BL/s)	–140	na	S.J. Cooke, unpublished data
	Forced swimming (1.5 BL/s)	–170	na	S.J. Cooke, unpublished data
	Forced swimming (2.0 BL/s)	–190	na	S.J. Cooke, unpublished data
	Forced swimming (2.5 BL/s)	–210	na	S.J. Cooke, unpublished data

Note: The summary is limited to other studies by our laboratory, to ensure similarity of the analyses and methodologies. For comparative purposes, we focused only on f_H, because some of the data we report are from f_H transmitters. BL/s is body length per second.

[1]C.D. Suski, S.S. Killen, S.J. Cooke, J.D. Kieffer, D.P. Philipp, and B.L. Tufts. Magnitude of physiological disturbance associated with different components of largemouth bass and walleye live-release angling tournaments. Unpublished manuscript.

and experiencing higher energetic expenditures. Interestingly, peak activity and feeding times of largemouth bass (i.e., crepuscular; Demersl et al. 1996) coincide with periods when ospreys and great blue herons are also most active (Butler 1992), potentially magnifying nonlethal costs to largemouth bass.

In conclusion, we show that largemouth bass are able to differentiate between attacks by two different predators and adjust their physiological response accordingly. The overall cardiac response to predators is intermediate among the suite of natural and anthropogenically induced stressors that largemouth bass may encounter. Depending on the predator attack rate in the area, predators may impose significant nonlethal costs for largemouth bass. Indeed, the nonlethal costs of predation require further study and incorporation into the energetics models of many taxa. Future studies should examine this response over a larger size range of prey and integrate the lost foraging opportunities (decreased energy intake) with the physiological response (increased energy expenditure), to better understand the mechanisms affecting nonlethal responses of prey to predators. In addition, future studies should also monitor Q, f_H, and SV_H and develop relationships between these cardiovascular variables and oxygen consumption, to estimate the actual costs of different nonlethal predator scenarios.

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