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BRIEF COMMUNICATION

Hypoxia tolerance variance between swimming and resting striped bass *Morone saxatilis*

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Individual striped bass *Morone saxatilis* were each exposed in random order to aquatic hypoxia (10% air saturation) either while swimming at 50% of their estimated critical swimming speed (U_{crit}) or while at rest until they lost equilibrium. Individuals were always less tolerant of hypoxia when swimming ($P < 0{\cdot}01$); the average fish was over five times more tolerant to the same hypoxia exposure when not swimming. There was no relationship between an individual's rank order of hypoxia tolerance (HT) under the two flow regimes, suggesting that different factors determine an individual's HT when at rest than when swimming.

Key words: environmental oxygen concentration; flow; individual; loss of equilibrium.

Environmental oxygen concentration [O_2] is generally considered a limiting resource for fish (Fry, 1971). As [O_2] decreases, the difference between maximum metabolic rate (MMR) and resting routine metabolic rate (RMR) or metabolic scope decreases (Claireaux *et al.*, 2000) limiting an animal's capacity to engage in metabolically expensive activities such as swimming and digestion. If an animal exceeds its metabolic scope, energy demand must be met anaerobically. Anaerobic metabolism captures only *c.* 8% of the energy from food that can be extracted aerobically (Gnaiger, 1993) and is generally not sustainable. Thus, oxygen scarcity (hypoxia) can not only cause direct mortality in fishes due to suffocation (Rice *et al.*, 2013), but can also have indirect effects such as inefficient energy use, behavioural abnormalities and reduced swimming capacity and growth (Domenici *et al.*, 2012). Environmental [O_2] can also be an important contributor to the spatial distribution of fishes in the environment (Pihl *et al.*, 1991; D'Amours, 1993), at times forcing fishes to occupy marginal habitats (Coutant, 1985; Eby & Crowder, 2002; Thompson *et al.*, 2010; Rice *et al.*, 2013).

In regions where dissolved oxygen is limiting, the fitness of an organism may depend on its hypoxia tolerance (HT), which is known to vary among (Pihl, *et al.*, 1991; Wannamaker & Rice, 2000) and within (Claireaux & Lagardere, 1999; Mandic *et al.*, 2009;

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Claireaux *et al.*, 2013) species. This HT variance and its implications for fitness are also contextually dependent upon the physiology, behaviour and environment of the fishes (Richards, 2011). For example, La Pointe *et al.* (2014) show that striped bass *Morone saxatilis* (Walbaum 1792), infected with a common Chesapeake Bay bacterium, have a higher critical oxygen tension and greater loss of aerobic scope than uninfected conspecifics. The co-familiar European sea bass *Dicentrarchus labrax* (L. 1758) showed disorientation and reduced ability to respond to stimuli under moderate hypoxia [50% air saturation (AS)] , which would affect predator and prey interactions (Lefrancois & Domenici, 2006). Furthermore, *D. labrax* were also shown to increase their risk-taking behaviour under hypoxic conditions in a manner that was dependent on individual RMR (Killen *et al.,* 2011). Thus, differential survival (mortality selection) and the ability to carry on routine biological functions leading to fecundity (*e.g.* growth) in waters that experience hypoxia may depend on relative HT.

HT has generally been tested under minimal flow conditions, either with large groups of fish together or with an individual fish in a respirometer or fish box (Robb & Abrahams, 2003; Farwell *et al.*, 2007; McKenzie *et al.*, 2008; Claireaux *et al.*, 2013). Such experiments usually result in a wide range of HTs for individuals of similar size and from the same population (Miller *et al.*, 2002; Davies *et al.*, 2011; Killen *et al.*, 2011; Claireaux *et al.*, 2013). Loss of equilibrium (LOE) is one commonly used endpoint in these tests, which, if done carefully, are not lethal, allowing multiple tests on the same individual. While this type of trial may accurately assess HT in relatively inactive, demersal or small species, pelagic schooling species such as *M. saxatilis* are more likely to encounter hypoxia while moving through hypoxic layers or trying to escape advancing hypoxic zones (Rice *et al.*, 2013).

Juvenile *M. saxatilis* are commonly found in hypolimnetic regions of Chesapeake Bay, an important nursery ground for the entire Atlantic stock (Waldman *et al.*, 1997), where hypoxic zones occur annually from April to October (Hagy *et al.*, 2004; Kemp *et al.*, 2005). Thermal stratification, combined with salinity gradients, creates a hypolimnion that is isolated from atmospheric oxygen by a turbid epilimnion for much of this time. Density differences between the two layers create a stable pycnocline that limits mixing and oxygenation. By combining this with minimal hypolimnetic photosynthetic oxygen production and organic matter fallout from the epilimnion, net oxygen depletion can occur in the hypolimnion. Hypoxia created thus in estuarine environments has dramatically increased in recent years as a result of cultural eutrophication (Diaz & Breitburg, 2009). These hypoxic zones are not static and can be driven suddenly into normoxic waters by winds (seiches) and tidal currents (Breitburg, 1990), overwhelming a first line of hypoxia defence, behavioural avoidance, leading to the death of fish (Rice *et al.,* 2013).

Due to the commercial and recreational importance of *M. saxatilis*, it is imperative to have a realistic understanding of this species' ability to cope with and respond to hypoxia, as hypoxic regions are predicted to expand with climate change (Keeling *et al.*, 2010). While studies of how hypoxia influences swimming performance are legion and have been performed on many fish species (Chapman & McKenzie, 2009; Domenici *et al.*, 2012), studies of how HT is influenced by swimming are almost unknown (McKenzie *et al.*, 2007). Here, hypoxia challenge tests were performed on the same individuals under two flow regimes in Brett-type swim tunnels (Nelson, 1989) to test the hypothesis that individual *M. saxatilis* have identical HT regardless of swimming activity.

A total of 13 juvenile *M. saxatilis*, 123–183 mm total length (L_T), were collected by the Maryland Department of Natural Resources trawl survey from the main channel of the Chesapeake Bay and transported to Towson University in Chesapeake Bay water at 4° C. Fish were brought to the experimental temperature of 20·1 ± 1·0° C (mean ± S.D.) by increasing the water temperature 2° C per day. Salinity was kept at 0·0092 ± 0·0012 and at 12L:12D photoperiod throughout the study. Fish were acclimated to the laboratory for 4 months in a common 355 l tank with moderate flow (fish generally swam) before being anaesthetized with MS-222 (120 mg l^{-1}, buffered with Na^+; HCO_3^-) weighed, measured and individually marked by injecting a passive integrated transponder (PIT; Biomark; www.biomark.com) into the abdominal cavity. An antibiotic cocktail of tetracycline HCl, ampicillin and cephalexin monohydrate (Thomas Laboratories; www.thomaslabs.com) was applied to the wound site after tagging and individuals were allowed a minimum of 2 weeks recovery from tagging before being subjected to hypoxia.

Hypoxia trials were conducted in Brett-type swim tunnels (Nelson, 1989) under both (1) minimal flow and (2) 50% of critical swimming speed (U_{crit}) flow conditions. Randomly selected individuals were allowed to fast for 24 h prior to being transferred to the swim tunnel without air exposure, and were not fed during a subsequent 24 h acclimation to the swim tunnel. Individuals were exposed to a minimal water speed (<3 cm s^{-1}) during the 24 h acclimation period. Approximately half of the individuals were tested first under minimal flow ($n = 7$), whereas the other six fish were tested first under flowing conditions to control for trial sequence. A given fish's second trial was also determined randomly, resulting in different time intervals between the two tests for each individual, but 2 weeks was adopted as a minimum inter-test interval.

Minimal flow conditions were a current speed of <3 cm s^{-1} to allow adequate mixing but did not require the fish to swim to maintain station. During flow trials, fish were swum at 50% of their estimated U_{crit}, a speed thought reasonable for fish to escape an encroaching hypoxic water mass. The U_{crit} of different sized fish was estimated from Beamish (1970) using data from largemouth bass *Micropterus salmoides* (Lacépède 1802) of similar size (19·7 ± 3·7 cm), also at 20° C. The equation used to predict 50% of U_{crit} in *M. saxatilis* was $v = (10^{1\cdot4465})\,(10^{0\cdot0137L})(0\cdot5)$, where v is 50% of the estimated critical swimming speed in cm s^{-1} that the fish were swum at, and L is the most recent L_T of the fish in cm. Flow was controlled with a rheostat. The swim tunnel was calibrated before the experiment using a Marsh-McBirney 2000 (www.hach.com) flow meter by measuring at 18 points in the swimming section of the tunnel region at each of the 12 variable transformer settings spanning the velocity range of the tunnel (a total of 216 measurements).

A flow trial was initiated by increasing the velocity of water flow by 5 cm s^{-1} every minute until 50% of their estimated U_{crit} was achieved. Fish then remained at that speed for 5 min, at which point oxygen tension was reduced over *c.* 30 min to 10% AS. A light was placed at the back of the swimming chamber to deter fish from resting. If a fish tried to rest on the downstream retaining screen, it was gently touched with a blunt plastic probe. LOE often occurred while the animal was swimming, but if an individual impinged on the downstream retaining screen did not resume swimming after three touches, this was also considered incapacitation and the trial was stopped.

Oxygen concentration was reduced to 10% AS over *c.* 30 min (29·9 ± 1·3 min), a speed indicative of a rapid hypoxic zone incursion (Breitburg, 1992), by bubbling nitrogen gas into the descending arm of the swim tunnels. Oxygen concentration decreased to 10% AS as an exponential function with an average instantaneous slope of $3{\cdot}12 \pm 0{\cdot}38\%$ AS min^{-1}. If an animal did not lose equilibrium after 4 h at 10% AS, the oxygen concentration was lowered further by 2% AS every hour until they did. Two galvanic oxygen-sensing probes were used to determine the level of AS in the swim tunnel (one anterior and one posterior to the swimming section). The probes were calibrated before each trial. One probe was connected through a digital converter box to a solenoid valve attached to an air stone, which maintained dissolved oxygen saturation at the desired level (Oxy-Reg System, Loligo Systems; www.loligosystems.com). HT was recorded as cumulative oxygen deficit (D_{CO}). If oxygen concentration is plotted as a function of time, D_{CO} is the difference between the area under the curves of a hypothetical animal remaining at 100% AS throughout the experiment and the experimental animal's actual oxygen exposure until the time that it lost equilibrium (LOE). D_{CO} is recorded in the units of per cent times minutes and included the initial period of reduction to 10% AS. For example, a theoretical animal that lost equilibrium at exactly 4 h at 10% AS after a 30 min reduction to 10% AS during which the experimental animal had an oxygen deficit of 1350% times minutes (difference between 100% saturation and the area under the exponential reduction curve) would have a D_{CO} of: D_{CO} $((30T \times 100\%X) - 350\%T) + (240T \times 100\%X) - (240T \times 10\%X) = 23\,250\%T$, where T is time in min and X is AS.

Immediately as an animal lost equilibrium, it was removed, measured, weighed and transferred to a recovery tank at 100% AS. Trial order for both tests was randomly determined, but a minimum separation time between trials for an individual of at least 2 weeks was adopted. The mean time between trials for each individual was 8·4 weeks.

All statistical analyses were conducted with an α level of 0·05 in SPSS (www.01.ibm.com/software/analytics/spss) or Statistica 5.0 (www.statsoft.com). The D_{CO} values were not normally distributed, so a Wilcoxon signed rank test was conducted to determine if HT of individuals was significantly different in individual fish swimming at 50% of U_{crit} than while resting. Spearman's rank correlation analysis was used to explore relationships between HT and body size (Dytham, 2011). A Kruskal–Wallis test was used to determine if the order in which individuals were exposed to the two conditions had an effect on HT. This experiment complied with all Towson University animal care regulations and guidelines (IACUC #102510 JN-11).

HT of juvenile *M. saxatilis* was significantly higher under minimal flow conditions (Wilcoxon; $Z = 3{\cdot}18$, $n = 13$, $P < 0{\cdot}001$) (Fig. 1). Each individual was more tolerant of hypoxia at low flow than while swimming at 50% of estimated U_{crit}. The mean HT (D_{CO}) for minimal flow trials was $29\,718{\cdot}8 \pm 2744{\cdot}9\%T$, whereas the average HT for 50% of U_{crit} trials was only $5535{\cdot}8 \pm 1479{\cdot}9\%T$. Interestingly, there was absolutely no correlation between each individual's HT in minimal flow water and while swimming (Spearman $\rho = 0{\cdot}0$, $n = 13$, $P > 0{\cdot}05$). In other words, being very tolerant to hypoxia at rest did not translate into exceptional tolerance while swimming. HT was more variable among the individual fish when swimming (c.v. = 96·4%) than when the animals were tested in minimal flow water (c.v. = 33·3%). There was no correlation between an individual's mass at the time of the trial and HT for either minimal flow (Spearman's rank, $\rho = -0{\cdot}291$, $n = 13$, $P > 0{\cdot}05$) or 50% of U_{crit} (Spearman's rank, $\rho = 0{\cdot}401$, $n = 13$, $P > 0{\cdot}05$). The mean HT of individuals did not depend on exposure order for either test (Kruskal–Wallis; $P > 0{\cdot}05$ for both).

Juvenile *M. saxatilis* from this study were relatively tolerant of hypoxia compared with other marine species (Nilsson & Ostlund-Nilsson, 2004), having the ability to

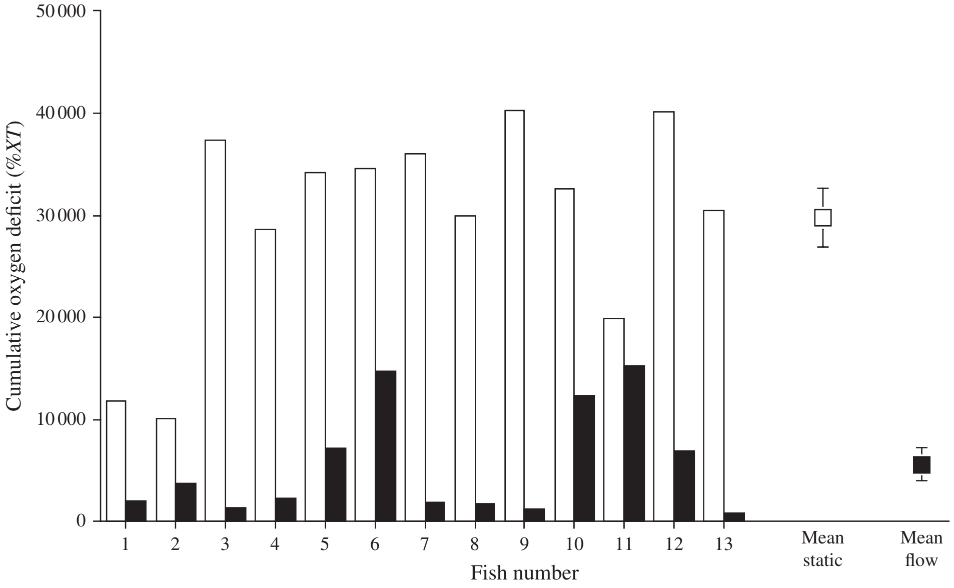

FIG. 1. Hypoxia tolerance (HT), measured as time (*T*) times severity of hypoxia exposure (% air saturation; *X*) until loss of equilibrium occurred for 13 juvenile *Morone saxatilis*. Animals were lowered to 10%*X* over *c.* 30 min and then remained at 10%*X* for 4 h, followed by subsequent hourly decrements of 2%*X*. Hypoxia challenges came under either static flow (<3 cm s^{-1}; ☐) or a flow of 50% of their estimated critical swimming speed (U_{crit}; ■). Tick marks on the abscissa represent the 13 individual fish. Mean ± S.E. HT for each flow condition is also plotted on the far right of the graph.

withstand an oxygen level of 10% AS for more than 4 h when resting, with two individuals able to reach 2% AS before losing equilibrium. This result is consistent with Chittenden (1971) who reduced oxygen levels in a static tank to an average of 0·95 mg l^{-1} (*c.* 10% AS) before LOE was observed in young-of-the-year *M. saxatilis* (70–110 mm L_T) and compares favourably with the co-familiar *D. labrax* that loses equilibrium *c.* 6 h after a 1 h reduction to 20% AS followed by a further 2% reduction in AS per hour (Claireaux *et al.*, 2013). The intraspecific variability in HT was also similar between *M. saxatilis* (this study) and *D. labrax* (Claireaux *et al.*, 2013).

Each juvenile *M. saxatilis* was less tolerant of hypoxia when swimming (Fig. 1). The average resting fish required a D_{CO} of 29 717·7%*T* before it lost equilibrium, roughly five times the D_{CO} required to cause LOE while swimming at 50% of U_{crit} (5607%*T*). These same animals had significantly repeatable times to LOE in four group trials conducted in static water ($P < 0·05$ Kendall Concordance; unpubl. data). There are no direct comparison studies, but this result compares favourably with that of Vagner *et al.* (2008) who reported an approximate 50% drop in time to fatigue in grey mullet *Mugil cephalus* L. 1758 swimming at 45 cm s^{-1} when [O_2] is lowered to 15% AS. McKenzie *et al.* (2007) reported an interesting and relevant disparity between swimming and resting Adriatic sturgeon *Acipenser naccarii* Bonaparte 1836 exposed to hypoxia. They measured oxygen consumption during progressive hypoxia and reported that swimming fish oxyregulated, whereas sedentary fish oxyconformed. While the study subjects of McKenzie *et al.* (2007) are quite phylogenetically distant from *M. saxatilis*, their results could help explain the dramatic differences in HT between swimming and resting *M. saxatilis*. If *M. saxatilis* are only oxyregulating while swimming, this coupled with the increased oxygen demand while swimming would account for the much more rapid advancement to equilibrium loss. The results are also predictable from the 'limiting oxygen concentration' modelling of Claireaux & Lagardere (1999) for juvenile *D. labrax*. This reduced HT while swimming is not necessarily an intuitive result. LOE from at least one other environmental stressor (heat) is unaffected by swimming activity in *M. saxatilis*. The critical thermal maximum (CT_{max}) of 11 similar-sized *M. saxatilis* was not affected by swimming at 50% of U_{crit}, despite the fact that dissolved oxygen was also decreasing with the rising temperature of the CT_{max} test (J. A. Nelson, unpubl. data).

An important finding of this study was that there was no relationship between an individual's HT in a minimal flow environment and while swimming (insignificant rank order correlation; Spearman $P > 0{\cdot}05$), which suggests different mechanisms whereby equilibrium is lost under the two conditions. This relates well to the different metabolic scaling coefficients seen between resting and exercising animals (Darveau *et al.*, 2002; Glazier, 2009). Because different metabolic processes such as protein turnover and Na^+/K^+ ATPase activity are the dominant consumers of energy at rest, and processes such as myosin and Ca^{++} ATPases dominate during exercise, there is no *a priori* reason to expect an animal to have equal relative HT at each of the activity levels. As reduction in oxygen consumption can be observed in hypoxic fishes both at rest (Speers-Roesch *et al.*, 2010) and during exercise (Fu *et al.*, 2011), there may be further intraspecific variation in how they selectively arrest metabolic processes and make cardiovascular adjustments, producing further shuffling of their relative HTs (*i.e.* a hypoxia response–swimming interaction term) at different levels of hypoxia and exercise. Supporting this idea, Urbina & Glover (2013) showed a change in the aerobic metabolic scaling coefficient in a large number and size range of an oxyconforming galaxid under hypoxia as the animals differentially transitioned into anaerobic metabolism to meet their energy needs.

HT in fishes is a complex function of an animal's ability to extract oxygen from the environment and supply it to key tissues, control aerobic metabolic rate and to recruit anaerobic metabolism. Thus, in various studies, HT has been found to be related to resting metabolic rate (Claireaux & Lagardere, 1999; McKenzie *et al.*, 2008), ability to depress aerobic metabolism (Corkum & Gamperl, 2009) and recruit anaerobic metabolism (Almeida-Val *et al.*, 2000). Marras *et al.* (2010) showed that a cohort of *D. labrax* undergoing repetitive constant acceleration tests had much larger variation in their anaerobic capacity than in their aerobic capacity. Whether that result applies to the larger variability of *M. saxatilis* HT while swimming is material for future study.

Size did not affect HT under either flow condition. These results conform to Chittenden (1971) who found no effect of fish size on oxygen levels at LOE or death in *M. saxatilis*. Although various studies have reported divergent effects of body size on HT, Nilsson & Ostlund-Nilsson (2008) reviewed the subject and concluded that there is no scaling of HT until fish transition to anaerobic metabolism, when larger fish would have an advantage due to their lower mass-specific metabolic rate and ability to store fermentable substrates.

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