FAU Institutional Repository

http://purl.fcla.edu/fau/fauir

This paper was submitted by the faculty of FAU's Harbor Branch Oceanographic Institute.

Notice: ©1991 Rosenstiel School of Marine and Atmospheric Science, University of Miami. This manuscript is available at http://www.rsmas.miami.edu/bms and may be cited as: Peterson, M. S. & Gilmore, R. G., Jr. (1991). Eco-physiology of juvenile snook *Centropomus undecimalis* (Bloch): life-history implications. *Bulletin of Marine Science, 48*(1), 46-57.

BULLETIN OF MARINE SCIENCE, 48(1): 46–57, 1991

ECO-PHYSIOLOGY OF JUVENILE SNOOK *CENTROPOMUS UNDECIMALIS* (BLOCH): LIFE-HISTORY IMPLICATIONS

Mark S. Peterson and R. Grant Gilmore, Jr.

ABSTRACT

Hypoxia is an environmental factor that affects a myriad of behavioral, physiological and habitat-use aspects of the life-history of fish. Snook, *Centropomus undecimalis* (Bloch), exhibit marked ontogenetic differences in habitat-use that we propose are based on physiological constraints and environmental conditions that change as the fish grow. To test this hypothesis, we compared known life-history data with laboratory tests of hematocrit, osmotic and chloride ion regulation, weight-dependent oxygen consumption and ventilation rates, and mortality. These data were also compared to field tests of size-dependent mortality under normoxic and hypoxic conditions. Under hypoxic conditions snook showed significant increases in hematocrit and decreases in plasma chloride ion concentration. Snook also exhibited significantly reduced metabolic rates, concomitant increases in ventilation rates, and increased mortality in hypoxic conditions. These ontogenetic physiological differences correlate with juvenile snook migration from impounded mangrove swamp habitat at sizes between 100–150 mm SL (about 45.0 g WW) along Florida's east-central coast.

Reduced oxygen availability may adversely affect fish reproduction, growth, mortality, foraging ability and habitat use (Davis, 1975). Hypoxia is a major cause of fish mortality in mangrove swamp habitats, particularly in coastal south-central Florida where a large percentage of these swamps have been impounded for mosquito control (Carlson et al., 1985; Gilmore, 1987). Many fishes have adapted physiologically and/or behaviorally to survive low oxygen conditions. Mosquitofish, *Gambusia holbrooki* (=*G. affinis*; Wooten et al., 1988) (Subrahmanyam, 1980) and *G. affinis* (location of original stock unknown; Cech et al., 1985), other poeciliids (Weber and Kramer, 1983; Kramer, 1987) and striped mullet, *Mugil cephalus* (Moore, 1976), can behaviorally adjust their oxygen uptake by using aquatic surface respiration and striped mullet can take atmospheric oxygen (Hoese, 1985). The tarpon, *Megalops atlanticus*, is an obligate air breather (at least during its juvenile stage) and thus is well adapted to living in hypoxic habitats (Shlaifer and Breder, 1940; Wade, 1962).

Snook, *Centropomus undecimalis* is an important euryecious piscine predator which inhabits mangrove swamps during a portion of its early life-history (Harrington and Harrington, 1961; Fore and Schmidt, 1973; Gilmore et al., 1983; McMichael et al., 1989). In the Indian River lagoon, 15–20 mm standard length (SL) juvenile snook recruit into freshwater and brackish habitats from offshore spawning areas between late April and January (Gilmore et al., 1983; Tucker and Campbell, 1988). They inhabit these habitats for 4–6 months where they grow at a rate of about 1.0 mm·day^{-1} then leave the mangroves in late winter or early spring at about 100–150 mm SL (Gilmore et al., 1983).

The impact of hypoxia on juvenile snook during residency in mangrove swamp habitats is not well understood. We hypothesized that juvenile snook are affected by hypoxia during this residency period and that as they grow, hypoxic effects constrain the use of this habitat at some upper range of body weight, thus promoting movement out of mangrove swamps. Thus, our objectives were to: 1) document hypoxia-induced changes in hematocrit, plasma osmolality, plasma chloride ion concentration and ontogenetic changes in oxygen consumption and

ventilation rates; 2) assess survival in low-oxygen tensions; and 3) review these physiological changes in juvenile snook in light of its known life-history pattern.

Materials and Methods

General Field and Laboratory Protocol.—Experimental and prey fishes were collected from impounded and unimpounded mangrove swamps in the Indian River lagoon, Florida. Experimental animals were held in 76-liter aquaria at salinities of 30 ± 1‰, 30 ± 1°C and under a 12L:12D photoperiod centered at 1230, for at least 7 days (see details within each sub-section). Snook were fed natural prey species ad libitum daily. Experimental salinities were produced using filtered (5-μm) Atlantic Ocean seawater diluted with aged reverse osmosis water and were checked daily. For all experiments except the survival part, fish were not fed for 24 h prior to testing.

Normoxic/Hypoxic Blood Constituents Experimental Protocol.—Snook (N = 40) were held in the desired salinity and temperature conditions for 7.6 ± 0.5 days prior to experimentation. Fish length was measured to the nearest mm SL. All blood samples were obtained by blotting each individual dry and severing its caudal peduncle. The incision was blotted and blood from the caudal artery was drawn into a heparinized micro-capillary tube and centrifuged for 4 min at 13,460 × g in an International Micro-capillary Centrifuge (Model MB) for hematocrit determination. All fish were processed within 30 min and individual blood collection was completed within 1 min to reduce handling effects on blood constituents (Robertson et al., 1987). All individuals were sacrificed between 0800–0900. Plasma osmolality (mOsm kg^{-1}) was determined on a 10 μl sample with a Wescor Vapor Pressure Osmometer (Model 5500). Plasma chloride ion concentration (meq/liter) was determined from a 10 μl sample on a Buchler Digital Chloridometer (Model 4-2500). Hypoxic conditions were produced by bubbling nitrogen gas directly into the aquarium through an air-stone for 3.0 h, thus gradually reducing the oxygen tension to hypoxic conditions (38.5 ± 2.6 mm Hg; about 26% saturation; 1.6 ppm). Snook were kept at the new P_{O_2} tension for 2.5 h. Experimental P_{O_2} were measured by injecting a single water sample into a calibrated Radiometer PHM-73/D616/E5046 oxygen analyzer system.

Oxygen Consumption Rates.—Oxygen consumption was measured with a flow-through respirometer consisting of an acrylic tube (33.7 cm × 7.94 cm ID diameter or 18.1 cm × 7.94 cm ID diameter) with PVC caps sealed by a rubber O-ring. Inhalent water was supplied from a 456-liter headbox via vinyl tubing through a small acrylic chamber (6.98 cm × 7.94 cm ID diameter; vol. 300 ml) leading into the respirometers. Exhalent water also entered a small acrylic chamber which enclosed a small stirring bar driven by a submersible stirrer. Water samples were taken from these chambers with a syringe and needle mounted through a rubber stopper. The large respirometers were flushed on average every 27.9 min while the small respirometers flushed on average every 14.4 min (59.1 ± 7.7 ml·min^{-1}). Due to flow rates and respirometer size, steady state was achieved rapidly (Propp et al., 1982). The entire system was enclosed in a heated water bath to maintain water temperatures. Four respirometers (three experimental and one control) were enclosed within a PVC and wood frame with black plastic partitions and an outer cover to reduce external influences on fish activity. The top of the frame was a one-way mirror for observing the fish and counting opercular movements. A small fluorescent light mounted above the tubes maintained the 12L:12D photoperiod.

Water from the headbox was de-oxygenated by an in-line oxygen stripper (Cameron, 1986) made of PVC (152.4 cm × 7.94 cm ID diameter) and filled with glass marbles to increase surface area for diffusion. Nitrogen gas was introduced through a gas diffuser from the bottom, thus producing a counter-current flow. Regulation of the counter flow rate brought about the desired oxygen tensions.

Oxygen consumption rates (VO_2: $mg \cdot O_2 \cdot g^{-1} \cdot h^{-1}$) were calculated by the equation: $VO_2 = [(P_{O_{2i}} - P_{O_{2e}})\,(a)\,(1.428)\,V] \cdot WW^{-1}$ (g) (Lampert, 1984); where ($P_{O_{2i}}$) = oxygen tension of inhalent water, ($P_{O_{2e}}$) = oxygen tension of exhalent water, a = solubility coefficient (from tables in Cameron, 1986), 1.428 converts $ml \cdot liter^{-1}$ to $mg \cdot liter^{-1}$ oxygen, V = flow rate ($ml \cdot h^{-1}$), and WW = wet weight (g). Results were considered routine metabolic rates (Brett and Groves, 1979). Each experiment took 43.5 h (Fig. 1). An individual was placed in each of the three respirometers at 1330 and allowed to adjust to the chamber for 18.5 h in normoxic water (177.1 ± 13.9 mm Hg). Between 0800–0900, initial and final P_{O_2} readings as well as flow rate were measured for each respirometer. A respirometer without fish was used to adjust for microbial respiration. The fish remained in the chamber for an additional 18.5 h and then the P_{O_2} was gradually reduced over the next 3.0 h to 41.2 ± 2.1 mm Hg. Once the desired oxygen tension was obtained, the fish were allowed 2.5 h to adjust to the lower oxygen tension. The initial and final hypoxic readings were taken between 0800–0900. Individuals (N = 14) were used only once.

To remove some variability in our oxygen consumption rates, experiments were set up as follows: 1) to remove potential effects of circadian patterns in osmoregulation (Peterson and Gilmore, 1988), all individuals were run between 0800–0900; 2) the effect of the "lights on/off" phenomenon on oxygen consumption rates (Saint-Paul, 1988) was mitigated by setting the photoperiod to come on at 0630

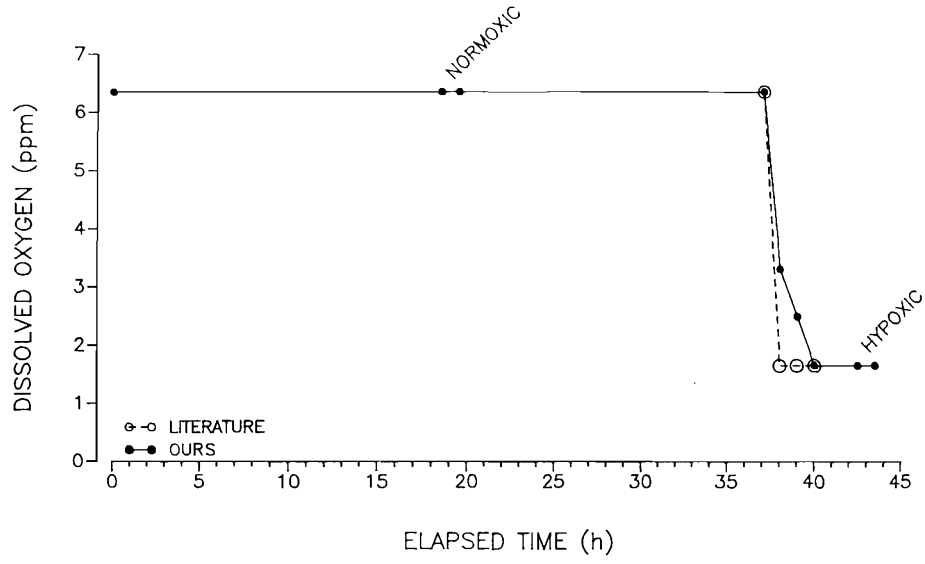

Figure 1. Schematic of the strategies used in the oxygen consumption experiments.

(staggered over a 15-min period) which allowed at least 1.25 h before the determinations were initiated; and 3) the P_{O2} was gradually reduced (Burton et al., 1980) over a 3.0-h time period, instead of the more typical 0.5–1.0-h period. This gradual reduction allowed the fish to adjust to the low dissolved oxygen conditions for 2.5 h before beginning the oxygen consumption experiments.

Ventilation Rates and Survival Experiments.—Survival experiments were conducted at 60 (about 2.5 ppm) and 40 (about 1.6 ppm) mm Hg. Snook (N = 5 for each weight group) were held in the experimental conditions for 12.6 ± 2.5 days. Individual fish were placed into either the 0.85-liter or 1.65-liter respirometer and given 18.5 h to adjust to the tubes under flowing, normoxic (162.3 ± 13.6 mm Hg) water. Between 0730–0800, the oxygen tension was rapidly reduced to a P_{O2} of 60 (59.6 ± 1.2 mm Hg) or 40 (39.6 ± 1.2 mm Hg) and death was monitored at intervals of 1, 2, 4, 6, 8, 10, 12 and 24 h. Death was established when a fish did not move its opercula for 1 min. The 1.65-liter tubes flushed on average every 13.6 min and the 0.85-liter tubes flushed on average every 7.02 min (at 121.1 ± 9.0 $ml \cdot min^{-1}$). Ventilation rates were recorded during this phase of the study. Normoxic rates were obtained immediately prior to decreasing the oxygen tension while hypoxic rates were taken 1 h after the tension was reduced.

Field Test.—An in situ test, simulating a hypoxic event, was conducted to document size-dependent mortality in snook. A pump used to both aerate and pump water into a closed impounded mangrove swamp was turned off, allowing hypoxic conditions to develop within the impoundment. Snook behavior/mortality was observed as ambient dissolved oxygen concentrations were measured. After the event, all snook were collected and measured.

Statistical Treatments.—Comparisons of fish size, plasma osmolality, plasma chloride ion concentration (all $\log_{10}$ transformed), and percent hematocrit (arcsine transformed) were analysed by P_{O2} treatment with analysis of variance (ANOVA; alpha = 0.05). Oxygen consumption and ventilation rates were analysed by a paired *t*-test (alpha = 0.05); oxygen consumption rates versus wet weight were examined by linear regression (Jensen, 1986). The mean difference between the control and treatment responses is denoted as $\bar{D}$ and applies only to the paired *t* statistic. Mean survival after 24 h (arcsine transformed) was analysed by a *t*-test. All values are expressed as $\bar{x}$ ± 1 SD.

Results

Normoxic/Hypoxic Blood Constituents.—Snook ranged in size from 54–107 mm SL (normoxia) and 57–103 mm SL (hypoxia). There were no significant size or plasma osmolality differences between the normoxic and hypoxic individuals (ANOVA; $P > 0.05$; Fig. 2). Significantly elevated hematocrits ($P < 0.05$) and

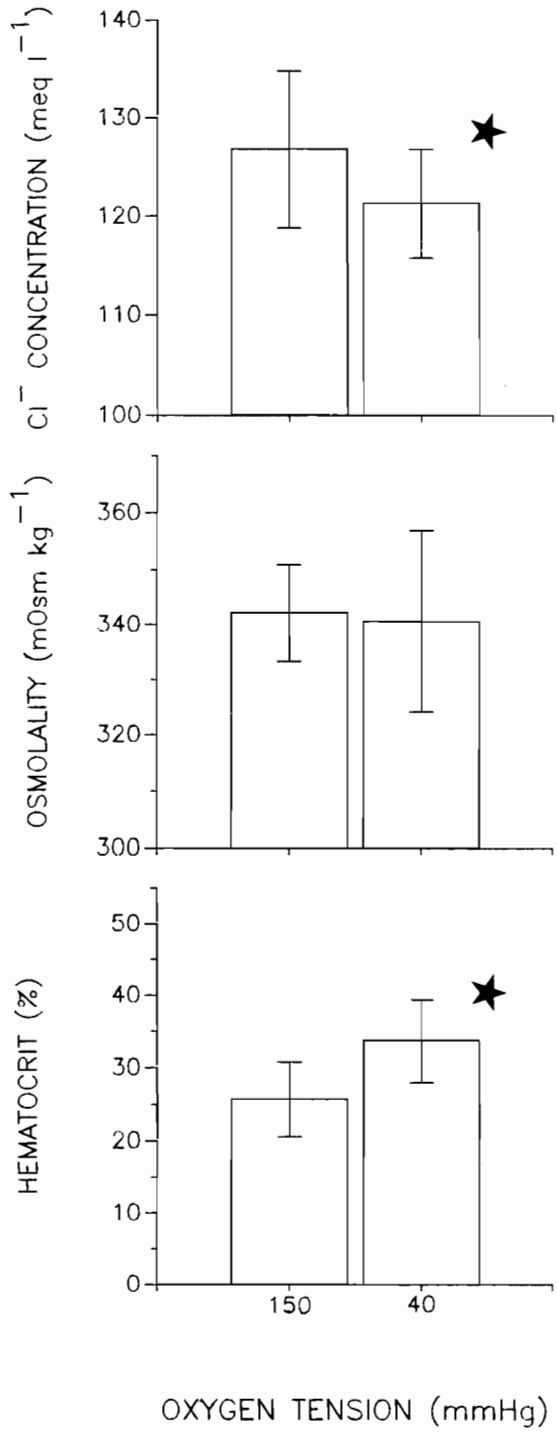

Figure 2. Comparison of hematocrit, osmolality and chloride ion concentration in normoxic (150 mm Hg) and hypoxic (40 mm Hg) conditions for snook (N = 40). Star = significant difference ($P < 0.05$) between oxygen tensions. Values are $\bar{x} \pm SD$.

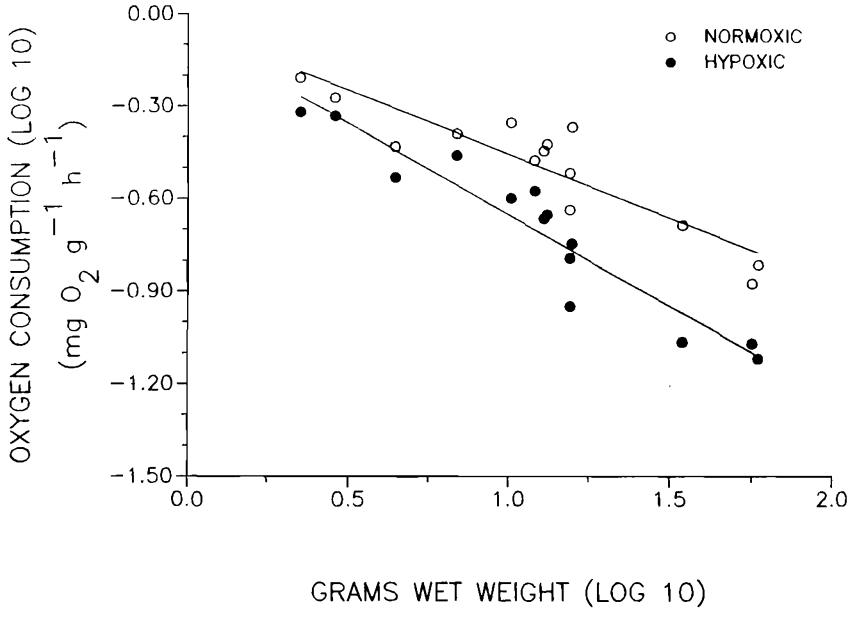

Figure 3. Comparison of snook oxygen consumption rates by wet weight in normoxic and hypoxic conditions (N = 14).

reduced plasma chloride values were documented for snook under hypoxic conditions (Fig. 2).

Oxygen Consumption Rates.—Snook ranged in weight from 2.25–58.72 g WW (55–173 mm SL; N = 14). A significant relationship between wet weight and oxygen consumption was documented in both normoxic and hypoxic conditions (ANOVA; $P < 0.001$; Fig. 3). Although we could determine normoxic rates for individuals larger than about 50–60 g WW (65.6 ± 19.5 g WW and 175.7 ± 17.1 mm SL; N = 6), we were not able to keep the same individuals alive under hypoxic conditions. We did not include these six individuals in the statistical analyses. For normoxia ($r = 0.82$), the relationship was: log $VO_2 = -0.4004127 - 0.4164017$ *log WW; whereas, for hypoxia ($r = 0.90$), the relationship was: log $VO_2 = -0.6130052 - 0.5924819$*log WW. Significant reductions in VO_2 (pooled across body weight) between normoxic and hypoxic conditions were documented (paired $t = 9.67$, df = 13, $\bar{D} = 0.3270$; $P < 0.001$).

Ventilation Rates and Survival.—Ventilation rates (beats/min) were significantly greater for small snook (< about 50–60 g WW; about 150–160 mm SL; Table 1) under hypoxia (40 mm Hg; paired $t = -4.45$, df = 4, $\bar{D} = -41.40$, $P < 0.05$)

Table 1. Mean ($\bar{x}$ ± SD) snook size used in survival and ventilation experiments. SL = standard length (mm); WW = wet weight (g)

		60 mm Hg	40 mm Hg
Large snook	WW:	83.8 ± 13.2	92.1 ± 22.3
	SL:	189.4 ± 15.6	190.2 ± 16.7
Small snook	WW:	42.9 ± 5.3	36.2 ± 10.9
	SL:	152.8 ± 10.0	139.4 ± 16.2

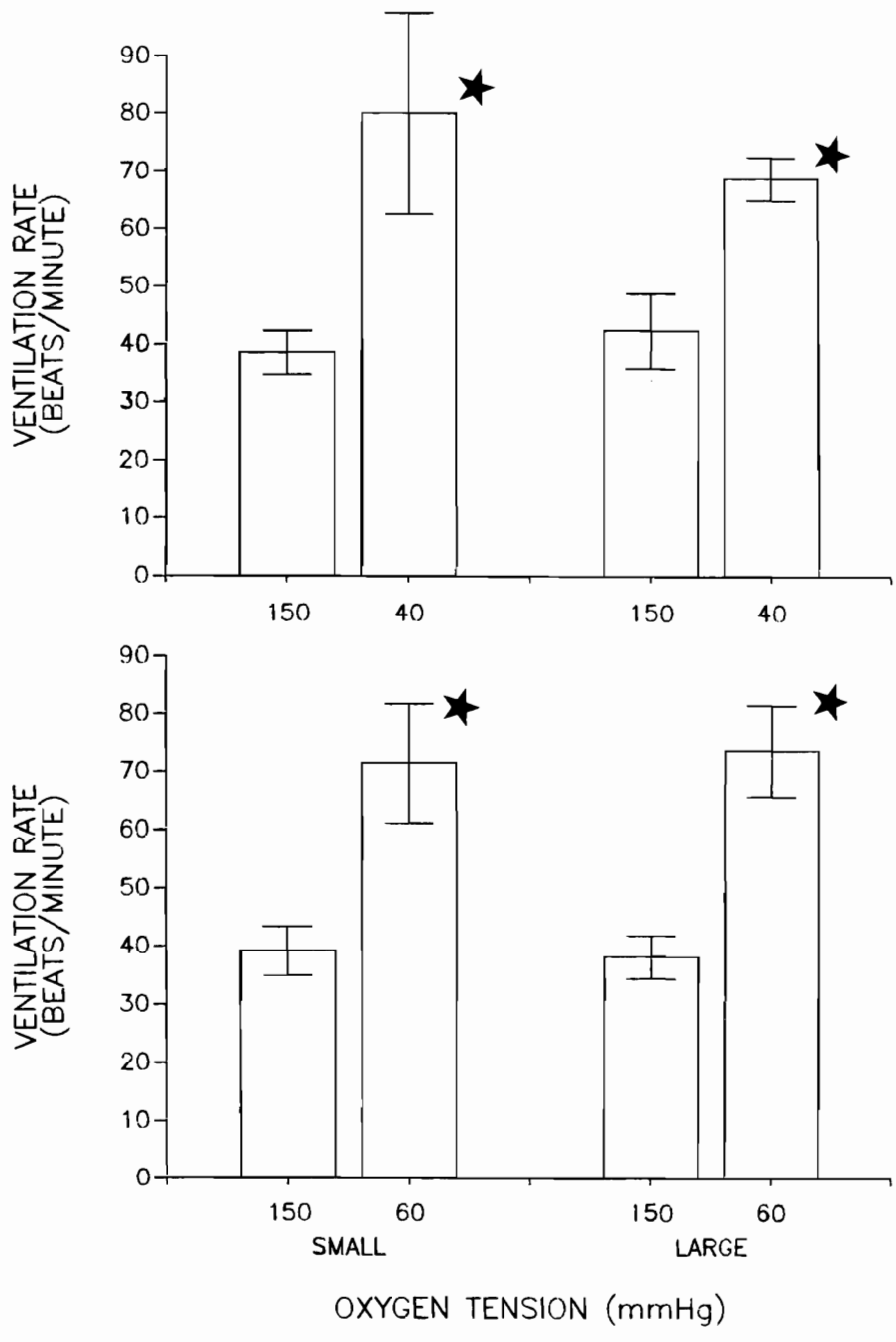

Figure 4. Comparison of snook ventilation rates ($\bar{x} \pm SD$; N = 5 per size group) between small and large snook in normoxic and hypoxic conditions. Star = significant difference ($P < 0.05$) within a size group.

and moderate hypoxia (60 mm Hg; paired $t = -7.76$, df = 4, $\bar{D} = -32.20$, $P < 0.01$) than normoxia (Fig. 4). Ventiliation rates of large snook (> about 50–60 g WW; Table 1) were also significantly greater in hypoxia (paired $t = -15.61$, df = 4, $\bar{D} = -26.40$, $P < 0.001$) and moderate hypoxia (paired $t = -10.28$, df = 4, $\bar{D} = -35.40$, $P < 0.01$) than normoxia (Fig. 4).

Both reduced oxygen tensions were lethal for juvenile snook and there was an ontogenetic difference in survival (Fig. 5). For small snook (about <50–60 g WW), there was 80% survival in 60 mm Hg after 24 h, whereas in 40 mm Hg survival decreased to 80% after only 3 h. Survival of small snook at 40 mm Hg was 60% between 9 and 24 h (Fig. 5). For large snook (about >50–60 g WW), only 60% survived after 7 h in 60 mm Hg, whereas in 40 mm Hg, all died within 3 h (Fig. 5). There was significantly greater mortality at 40 than 60 mm Hg for both small ($t = 3.54$, df = 88, $P < 0.01$) and large snook ($t = 7.90$, df = 88, $P < 0.001$; Fig. 6). There was also significantly greater mean mortality between the two size groups at 40 mm Hg ($t = 7.05$, df = 88, $P < 0.001$) and 60 mm Hg ($t = 3.09$, df = 88, $P < 0.01$; Fig. 6).

Field Test.—A large number of small and large juvenile snook rose to the surface to breath near the air-water interface. All individuals were obviously stressed by hypoxia but it was only lethal to individuals >145 mm SL (N = 50).

Discussion

Juvenile snook undergo ontogenetic changes in hypoxia tolerance. This pattern correlates with ontogenetic habitat use for the same east-central Florida snook population (Gilmore et al., 1983). We documented significant increases in hematocrit for snook under hypoxic conditions. This secondary stress-response is an initial response to hypoxia (Swift and Lloyd, 1974). Swift (1981) documented a significant increase in hematocrit for rainbow trout, *Oncorhynchus mykiss* (=*Salmo gairdneri*; Smith and Stearley, 1989) exposed to 2.3 mg·liter^{-1} dissolved oxygen or lower for periods longer than 3 h and suggested (Swift, 1982) that increased hematocrit might be adaptive to fishes by allowing them to bind more available oxygen when it is environmentally low. Elevated hematocrits may be caused by an increased production of erythrocytes, fluid loss to the tissues with a subsequent decrease in the plasma volume, and/or swelling of the erythrocytes (Swift and Lloyd, 1974; Swift, 1981; Fievet et al., 1987).

The significant decrease in plasma chloride ion concentration in snook held under hypoxic conditions suggests two mechanisms. First, a well-defined acidosis occurs in fish blood under hypoxic conditions that promotes a rapid increase in red blood cell (RBC) volume due to a large net uptake of Na^+ and Cl^- from the blood (Fievet et al., 1987). This Cl^- uptake by the RBC not only increases RBC volume but reduces plasma Na^+ and Cl^-, as was documented for Cl^- in our study. Secondly, as hypoxic conditions prevailed, diuresis occurred and chloride and possibly other ions were lost via the urine. Significant decreases in plasma chloride under low oxygen conditions (<2.3 mg·liter^{-1}; Swift, 1981) and decreases in plasma sodium, potassium and chloride ions under salinity stress (Johnston and Cheverie, 1985) have been documented for rainbow trout, *O. mykiss*. Chloride has also been shown to be less tightly regulated than other ions in teleosts (Lutz, 1972; Nordlie, 1987). Hunn (1969) documented significant loss of chloride, sodium, magnesium and potassium following hypoxia in rainbow trout and suggested that it may be due to reduced tubular reabsorptive capacity when oxygen is reduced.

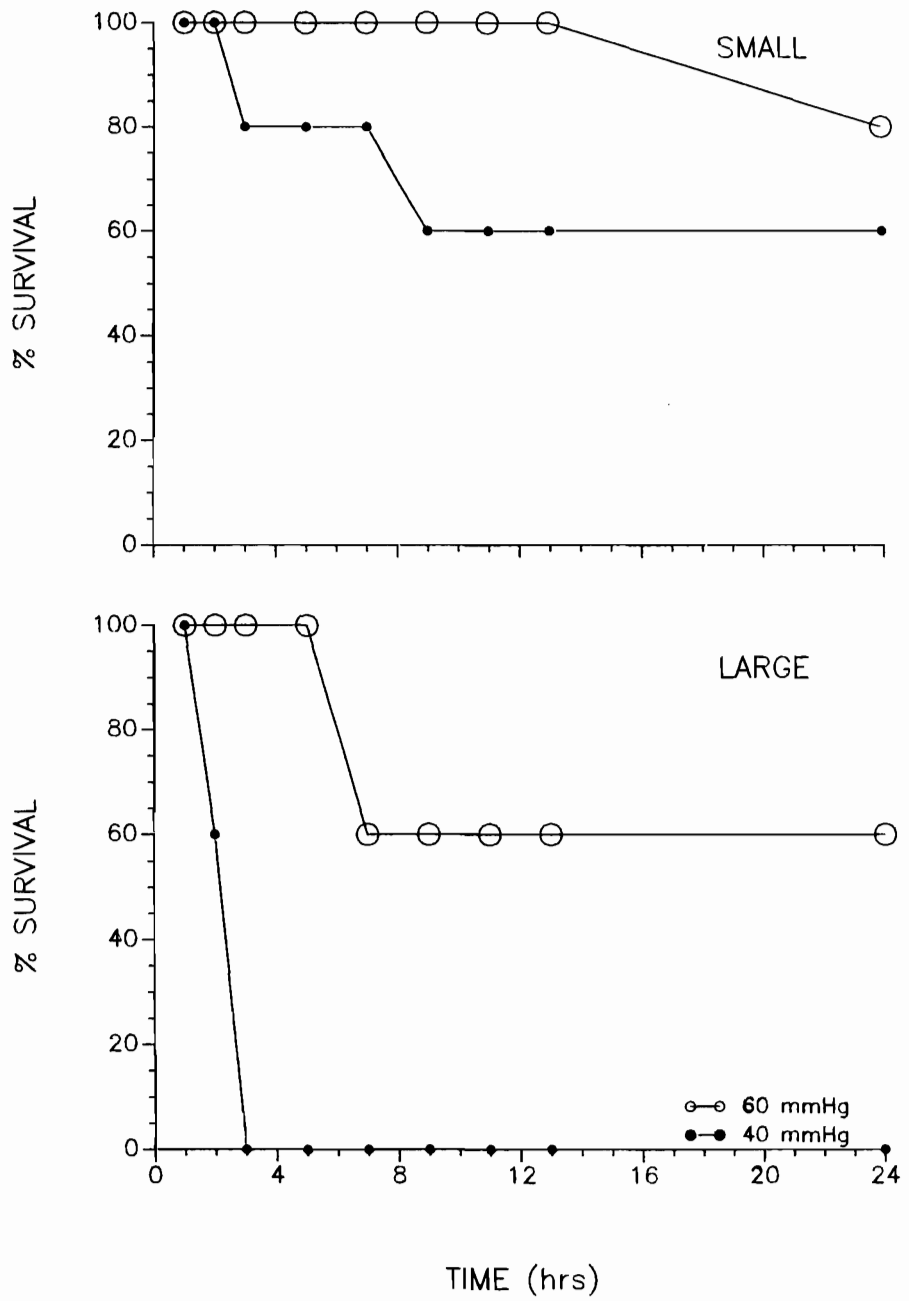

Figure 5. Comparison of survival data ($\bar{x} \pm SD$; N = 5 per size group) over a 24-h period for small snook and large snook.

Juvenile snook also exhibited significantly reduced metabolic rates, concomitant increases in ventilation rates and increased mortality in hypoxic conditions. The documented weight-dependent respiration rates correlate well with movement out of mangrove swamp habitats by large snook and thus support the ontogenetic

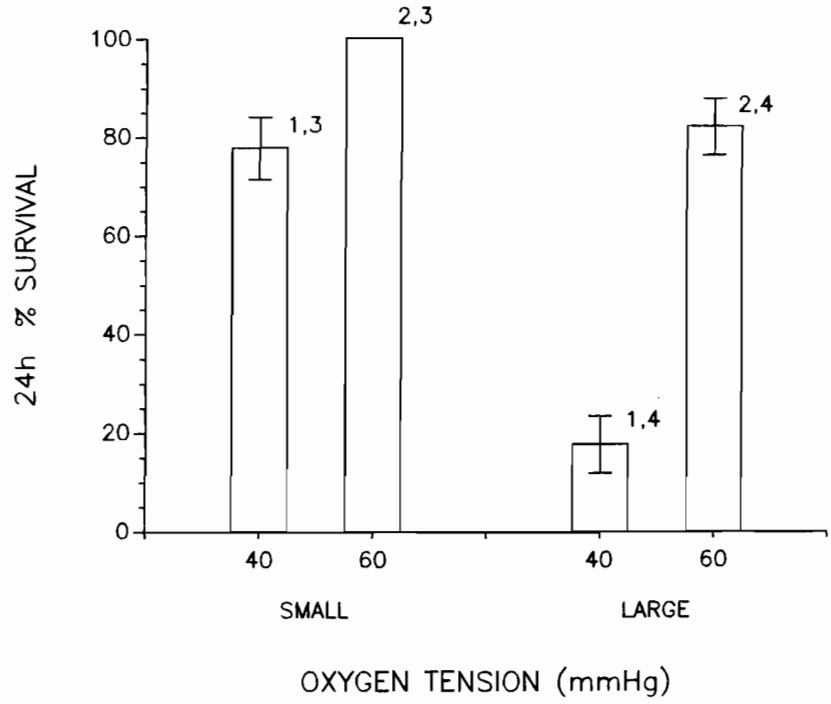

Figure 6. Mean ($\bar{x} \pm SD$; N = 5 per size group) survival over a 24-h period for small and large snook in the two oxygen tensions. The 1 and 2 = survival is different ($P < 0.05$) due to oxygen tensions (within a size group). The 3 and 4 = survival is different ($P < 0.05$) due to size (within a tension). To calculate a mean survival for the small size group in tension 60, we reduced survival of one individual to 99.9%. This allowed us to statistically treat differences within and between sizes.

habitat-use pattern documented by Gilmore et al. (1983). The mortality of large snook exposed to hypoxia during the oxygen consumption experiments adds support to this hypothesis. A number of other studies (Lomholt and Johansen, 1979; Cech et al., 1985; Eccles, 1985; Boese, 1988) have documented reduced metabolic rates with increasing hypoxia and weight. Similarly, survival of juvenile snook in low-oxygen tensions is weight-dependent.

Snook had significantly greater ventilation rates and more rhythmic opercular movement with lower activity in hypoxic water. Increases in gill ventilation rates probably result in an increase of energy expenditure for a number of fishes (Lomholt and Johansen, 1979; Boese, 1988). Indeed, Randall and Daxboeck (1984) have estimated that the metabolic cost of ventilation is about 10% of oxygen uptake in fishes, indicating that as oxygen tension decreases and ventilation increases, the energetic cost proportionally increases for snook. A decrease in general activity under hypoxic conditions, as documented in this study, has been implicated as an energy conservation characteristic in fishes (Lomholt and Johansen, 1979; Subrahmanyam, 1980). Lower metabolic rates in low oxygen tensions apparently cannot support large snook metabolic requirements and death occurs in tensions as high as 60 mm Hg.

Our in situ test of size-dependent snook mortality supported our laboratory data and further indicates that large snook have more difficulty surviving hypoxic conditions than small snook in hypoxic impounded mangroves. Prior to impoundment of the swamp, larger snook could leave the diurnally and/or seasonally

hypoxic mangrove swamp habitat for better conditions in the adjacent estuary, thus avoiding these stressful and/or lethal time periods.

Juvenile snook (<30 mm SL) that recruit into tidal freshwater and/or mangrove habitats between late-spring and winter leave these habitats for the adjacent seagrass beds after reaching about 100–150 mm SL. Gilmore et al. (1983) indicated that juvenile snook in swamp habitats averaged 67 mm SL (4.7 g WW) and ranged from 10–174 mm SL (0.02–76.1 g WW). We have documented decreased tolerance of hypoxia with increasing fish size which correlates well with the movement of juvenile snook greater than about 50–60 g WW (about 100–150 mm SL) from low-oxygen mangrove habitats.

The differential physiological adaptations to hypoxia of various size classes of juvenile snook correspond not only with ontogenetic movement between habitats but also with the change in abiotic parameters within the mangrove swamp habitat. Early juvenile snook typically enter mangrove swamp habitats in the Indian River Lagoon during periods of high water which floods the high marsh between late August and December and possibly in spring (March–May) when mean water levels are aperiodically high (Provost, 1973). Water levels at all other times of the year are not high enough to connect the low and high marsh. Thus, snook that entered the high marsh, ponds or depressions during the fall could be trapped there until the following spring or potentially the following fall flooding. Snook migration from the high marsh to the estuary may be a response to decreasing water levels (depth) along with greater fish size and correlated weight-dependent physiological changes documented in this study.

Habitat-use patterns of fishes can be explained in part by knowledge of their eco-physiology in light of realistic environmental constraints. This idea suggests that as the environmental conditions change so will the habitat-use patterns of the species. This was documented for snook in this study and is supported by studies on Amazonian floodplain fishes by Saint-Paul and Soares (1987).

Acknowledgments

We wish to thank Drs. K. Bird and R. Laughlin for the use of laboratory space and equipment respectively. D. Scheidt, R. Brockmeyer, Jr., J. Luczkovich, D. Carlson, P. O'Brian, D. Tremain and J. Schaffer aided with field work. N. Brown-Peterson, S. W. Ross and Drs. M. L. Moser, F. G. Nordlie, W. J. Diehl and R. Altig commented on the manuscript. Funding was provided by a post-doctoral fellowship to M.S.P. from the Harbor Branch Institution, Inc. and a grant from the Indian River Mosquito Control District to R.G.G. This is contribution #773 of the Harbor Branch Oceanographic Institution, Inc.

Literature Cited

Boese, B. L. 1988. Hypoxia-induced respiratory changes in English sole (*Parophrys vetulus* Girard). Comp. Biochem. Physiol. 89A: 257–260.

Brett, J. R. and T. D. Groves. 1979. Physiological energetics. Pages 279–352 *in* W. S. Hoar and D. J. Randall, eds. Fish physiology, Vol. VIII. Academic Press, Inc., Orlando.

Burton, D. T., L. B. Richardson and C. J. Moore. 1980. Effect of oxygen reduction rate and constant low dissolved oxygen concentrations on two estuarine fish. Trans. Amer. Fish. Soc. 109: 552–557.

Cameron, J. 1986. Principles of physiological measurement. Academic Press, Inc., New York. 278 pp.

Carlson, D. B., R. G. Gilmore and J. R. Rey. 1985. Salt marsh impoundment management on Florida's central east coast: reintegrating isolated high marshes to the estuary. Pages 47–63 *in* F. J. Webb, ed. Proceedings of the 12th annual conference on wetlands restoration and creation, Tampa, Florida.

Cech, J. J., M. J. Massingill, B. Vondracek and A. L. Linden. 1985. Respiratory metabolism of mosquitofish, *Gambusia affinis*: effects of temperature, dissolved oxygen, and sex difference. Env. Biol. Fishes 13: 297–307.

Davis, J. C. 1975. Minimal dissolved oxygen requirements of aquatic life with emphasis on Canadian species: a review. J. Fish. Res. Bd. Can. 32: 2295–2332.

Eccles, D. H. 1985. The effect of temperature and mass on routine oxygen consumption in the South African cyprinid fish *Barbus aeneus* Burchell. J. Fish Biol. 27: 155–165.

Fievet, B., R. Motais and S. Thomas. 1987. Role of adrenergic-dependent H^+ release from red cells in acidosis induced by hypoxia in trout. Amer. J. Physiol. 252: R269–R275.

Fore, P. L. and T. W. Schmidt. 1973. Biology of juvenile and adult snook, *Centropomus undecimalis,* in the Ten Thousand Islands, Florida, Chap. 16. *In* Ecosystems analyses of the Big Cypress Swamp and estuaries. U.S. EPA, Surveillance and Analysis Division, Athens, Georgia. Pp. 1–18.

Gilmore, R. G. 1987. Fish, macrocrustacean and avian population dynamics and cohabitation in tidally influenced impounded subtropical wetlands. Pages 373–394 *in* W. R. Whitman and W. H. Meredith, eds. Waterfowl and wetlands symposium. Delaware Dept. Nat. Res. Env. Control, Dover.

———, C. J. Donohoe and D. W. Cooke. 1983. Observations on the distribution and biology of east-central Florida populations of the common snook, *Centropomus undecimalis* (Bloch). Fla. Sci. 46: 313–336.

Harrington, R. W., Jr. and E. S. Harrington. 1961. Food selection among fishes invading a high subtropical salt marsh: from onset of flooding through the progress of a mosquito brood. Ecology 42: 646–666.

Hoese, H. D. 1985. Jumping mullet: the internal diving bell hypothesis. Env. Biol. Fishes 13: 309–314.

Hunn, J. B. 1969. Chemical composition of rainbow trout urine following acute hypoxic stress. Trans. Amer. Fish. Soc. 98: 20–22.

Jensen, A. L. 1986. Functional regression and correlation analysis. Can. J. Fish. Aquat. Sci. 43: 1742–1745.

Johnston, C. E. and J. C. Cheverie. 1985. Comparative analysis of ionoregulation in rainbow trout (*Salmo gairdneri*) of different sizes following rapid and slow salinity acclimation. Can. J. Fish. Aquat. Sci. 42: 1994–2003.

Kramer, D. L. 1987. Dissolved oxygen and fish behavior. Env. Biol. Fishes 18: 81–92.

Lampert, W. 1984. The measurement of respiration. Pages 416–468 *in* J. A. Downing and F. H. Rigler, eds. A manual on methods for the assessment of secondary productivity in fresh waters. Blackwell Sci. Publs., Oxford.

Lomholt, J. P. and K. Johansen. 1979. Hypoxia acclimation in carp—how it affects O_2 uptake, ventilation, and O_2 extraction from water. Physiol. Zool. 52: 38–49.

Lutz, P. L. 1972. Ionic patterns in the teleost. Comp. Biochem. Physiol. 42A: 719–773.

McMichael, R. H., Jr., K. M. Peters and G. R. Parsons. 1989. Early life history of snook, *Centropomus undecimalis,* in Tampa Bay, Florida. Northeast Gulf Sci. 10: 113–125.

Moore, R. H. 1976. Seasonal patterns in the respiratory metabolism of the mullets *Mugil cephalus* and *Mugil curema.* Contr. Mar. Sci. 20: 133–146.

Nordlie, F. G. 1987. Plasma osmotic, Na^+ and Cl^- regulation under euryhaline conditions in *Cyprinodon variegatus* Lacepede. Comp. Biochem. Physiol. 86A: 57–61.

Peterson, M. S. and R. G. Gilmore, Jr. 1988. Hematocrit, osmolality and ion concentration in fishes: consideration of circadian patterns in the experimental design. J. Exp. Mar. Biol. Ecol. 121: 73–78.

Propp, M. V., M. R. Garber and V. I. Ryabuscko. 1982. Unstable processes in the metabolic rate measurements in flow-through systems. Mar. Biol. 67: 47–49.

Provost, M. W. 1973. Mean high water mark and use of tidelands in Florida. Fla. Sci. 36: 50–66.

Randall, D. J. and C. Daxboeck. 1984. Oxygen and carbon dioxide transfer across fish gills. Pages 263–314 *in* W. S. Hoar and D. J. Randall, eds. Fish physiology, Vol. 10A. Academic Press, Inc., Orlando.

Robertson, L., P. Thomas, C. R. Arnold and J. M. Trant. 1987. Plasma cortisol and secondary stress responses of red drum to handling, transport, rearing density, and a disease outbreak. Prog. Fish-Cult. 49: 1–12.

Saint-Paul, U. 1988. Diurnal routine O_2 consumption at different O_2 concentrations by *Colossoma macropomum* and *Colossoma brachypomum* (Teleosti: Serrasalmidae). Comp. Biochem. Physiol. 89A: 675–682.

——— and G. M. Soares. 1987. Diurnal distribution and behavioral responses of fishes to extreme hypoxia in an Amazon floodplain lake. Env. Biol. Fishes 20: 91–104.

Shlaifer, A. and C. M. Breder. 1940. Social and respiratory behavior of small tarpon. Zoologica 25: 493–512.

Smith, G. R. and R. F. Stearley. 1989. The classification and scientific names of rainbow and cutthroat trouts. Fisheries 14: 4–10.

Subrahmanyam, C. B. 1980. Oxygen consumption of estuarine fish in relation to external oxygen tension. Comp. Biochem. Physiol. 67A: 129–133.

Swift, D. J. 1981. Changes in selected blood component concentrations of rainbow trout, *Salmo gairdneri,* exposed to hypoxia or sublethal concentrations of phenol or ammonia. J. Fish Biol. 19: 45–61.

———. 1982. Changes in selected blood component concentrations of rainbow trout, *Salmo gairdneri,* following the blocking of the cortisol stress response with betamethasone and subsequent exposure to phenol or hypoxia. J. Fish Biol. 21: 269–277.

——— and R. Lloyd. 1974. Changes in urine flow and haematocrit value of rainbow trout *Salmo gairdneri* (Richardson) exposed to hypoxia. J. Fish Biol. 6: 379–387.

Tucker, J. W., Jr. and S. W. Campbell. 1988. Spawning season of common snook along the east central Florida coast. Fla. Sci. 51: 1–6.

Wade, R. A. 1962. The biology of the tarpon, *Megalops atlanticus,* and the ox-eye, *Megalops cyprinoides,* with notes on larval development. Bull. Mar. Sci. Gulf Carib. 12: 545–622.

Weber, J. M. and D. L. Kramer. 1983. Effects of hypoxia and surface access on growth, mortality, and behavior of juvenile guppies, *Poecilia reticulata.* Can. J. Fish. Aquat. Sci. 40: 1583–1588.

Wooton, M. C., K. T. Scribner, and M. H. Smith. 1988. Genetic variability and systematics of *Gambusia* in the southeastern United States. Copeia 1988: 283–289.

DATE ACCEPTED: May 21, 1990.

ADDRESSES: *Division of Marine Science, Harbor Branch Oceanographic Institution, Inc., 5600 Old Dixie Highway, Ft. Pierce, Florida 34946;* PRESENT ADDRESS: *(M.S.P.) Department of Biological Sciences, P.O. Drawer GY, Mississippi State University, Mississippi State, Mississippi 39762.*