Responses of Swimming Skipjack (*Katsuwonus pelamis*) and Yellowfin (*Thunnus albacares*) Tunas to Acute Hypoxia, and a Model of Their Cardiorespiratory Function

Peter G. Bushnell[1,*]
Richard W. Brill[2,†]
[1]John A. Burns School of Medicine, Department of Physiology, University of Hawaii, 1960 East-West Road, Honolulu, Hawaii 96822; [2]Southwest Fisheries Science Center Honolulu Laboratory, National Marine Fisheries Service, National Oceanic and Atmospheric Administration, 2570 Dole Street, Honolulu, Hawaii 96822-2396.

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Abstract

Heart rate and swimming-speed responses to acute hypoxia were measured in skipjack (Katsuwonus pelamis) *and yellowfin tunas* (Thunnus albacares). *Swimming speeds began to increase in both species when O_2 tension (P_{O_2}) reached approximately 124 mmHg. Bradycardia became significant in both species when P_{O_2} reached approximately 130 mmHg. Heart rate fell with P_{O_2} in yellowfin tuna, but, in skipjack tuna, it increased at the lowest O_2 levels reached (89–70 mmHg). Bradycardia occurred in both species despite concomitant increases in swimming speed. A continuous infusion dye dilution system was used to monitor changes in ventilation volume ($\dot{V}_g$) during hypoxia in yellowfin tuna. As P_{O_2} fell, $\dot{V}_g$ increased. At the lowest O_2 levels (109–90 mmHg), $\dot{V}_g$ was 45% higher than during normoxia. Ventilation volume increased despite no concomitant increases in swimming speed. Data from these experiments were used to develop a model capable of predicting O_2 demand and delivery, maximum sustainable (i.e., aerobic) swimming speeds, and minimum survivable O_2 levels for yellowfin and skipjack tunas. Results from the model indicate that the cardiorespiratory system of tunas is capable of maximum rates of O_2 delivery, even at low swimming speeds, that are approximately three times those of other active teleosts. We believe that, because the pelagic environment provides no place to hide and rest following exhaustive activity, the ability of the cardiorespiratory system of tunas to deliver O_2 to the tissues at high rates evolved for the rapid repayment of O_2 debts rather than to permit exceptionally high sustained swimming speeds.*

* Present address: Department of Zoology, University of British Columbia, Vancouver, British Columbia V6T 2A9, Canada.
† To whom correspondence should be sent.

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Introduction

Because of their daily vertical migrations, tunas often encounter reduced ambient O_2, and ambient O_2 levels appear to strongly influence their movements, depth distributions, and vulnerability to specific fishing-gear types (Ingham, Cook, and Hausknecth 1977; Barkley, Neill, and Gooding 1978; Sharp 1978; Sund, Blackburn, and Williams 1981; Hanamoto 1987). Dizon (1977) was apparently the first to directly observe the responses of tunas to lowered ambient O_2. He found that skipjack tuna (*Katsuwonus pelamis*) exposed to hypoxia (P_{O_2} 70–85 mmHg) increased swimming speed, whereas yellowfin tuna (*Thunnus albacares*) did not. He hypothesized that only the former were stressed at this level of hypoxia. Understanding this apparent interspecific difference was the impetus for experiments by Bushnell, Brill, and Bourke (1990) on the cardiorespiratory responses to acute hypoxia of spinally blocked (i.e., paralyzed) skipjack and yellowfin tunas. They found that both species responded to even mild hypoxia (115 mmHg) by increasing mouth gape and ventilation volume ($\dot{V}_g$) and decreasing heart rate (HR).

Using spinally blocked animals affords the opportunity to measure a number of physiological variables simultaneously; however, it eliminates a potentially important response to hypoxia of obligate ram-ventilating fishes like tunas, namely, an increase in swimming speed. By measuring HR, $\dot{V}_g$, and swimming speed of skipjack and yellowfin tunas during normoxia and hypoxia, this study sought to determine what influences the inability to swim has on the physiology of these tunas in normoxia and on their responses to hypoxia.

Because swimming tunas cannot tow multiple cannulas for extended periods (Jones, Brill, and Mense 1986; Jones et al. 1990), we undertook two separate experiments. The first evaluated changes in HR and swimming speed, and the second, changes in $\dot{V}_g$ and swimming speed in response to acute hypoxia. In the latter experiment, the O_2 tensions of exhalant water samples (P_{eO_2}) were analyzed to determine utilization (U, i.e., percent of O_2 in the inhalant water extracted by the gills) and O_2 consumption ($\dot{V}_{O_2}$). This is only the second time these parameters have been measured simultaneously in swimming tunas in normoxia (the first was by Jones et al. 1990) and the first time they have ever been measured in hypoxia. These data are relevant to evaluating the performance of the cardiorespiratory systems of tunas specifically and of obligate ram-ventilating fishes in general.

We also used our data to reanalyze Dizon's (1977) model that evaluated the effects of increasing swimming speed or increasing mouth gape on O_2

delivery during normoxia and hypoxia in skipjack tuna. His model was based on the assumptions that O_2 demand increases exponentially with speed, whereas, without an increase in gape, O_2 delivery increases linearly. Dizon (1977) concluded that increases in swimming speed, without concomitant increases in gape, would result in O_2 demand exceeding O_2 delivery during hypoxia. We undertook a reanalysis because many of the parameters Dizon (1977) used had not been measured or were based on data that have since been shown to be incorrect. In addition, we extended his model to include yellowfin tuna.

Material and Methods

Yellowfin and skipjack tunas were purchased from local commercial fishermen and maintained in outdoor tanks at the National Marine Fisheries Service's Kewalo Research Facility in Honolulu, Hawaii. Water temperature in the tanks was 25° ± 2°C. Animal procurement, handling, and maintenance procedures are further described in Nakamura (1972); Queenth and Brill (1983); and Chang, Brill, and Yoshida (1983). Food was provided daily but was withheld for 24 h prior to an experiment to allow sufficient time for gut clearance (Magnuson 1969).

Experiments were conducted in an indoor 5,300-L doughnut-shaped tank (outside diam, 6.1 m; inside diam, 4.4 m); the inner portion of the tank was dry. Water height was set by an external standpipe and maintained at 37 cm. Seawater (25°C) was introduced to the tank through two sets of concentric polyvinyl chloride (PVC) pipes on the bottom of the swimming channel. Evenly spaced holes in the pipes delivered water to all portions of the tank simultaneously and kept the water well mixed.

The P_{O_2} of the water supplying the test tank was switched among three sources: normoxic water (≈150 mmHg), hypoxic water (20–30 mmHg), and hyperoxic water (275–300 mmHg). The hyperoxic water was used to bring the tank water back to full O_2 saturation after a hypoxic trial. The P_{O_2} of the water in the test tank (P_{iO_2}) was continuously monitored with a Radiometer polarographic electrode, mounted in a flow-through cuvette and connected to a Radiometer PHM 71 blood-gas analyzer. Electrode temperature was controlled by pumping water from the test tank through a surrounding water jacket. The P_{iO_2} was recorded every minute, either by hand or by connecting the blood-gas analyzer's analog output to a Hewlett-Packard (HP) 3455A digital voltmeter. In the latter case, the analog signal was digitally converted to P_{O_2} by the voltmeter and recorded by an HP 5150A thermal printer. A Yellow Springs Instrument portable O_2 meter was

occasionally used to sample P_{O_2} around the tank to ensure that the tank water remained well mixed throughout an experiment.

Swimming speeds were measured by recording the time to complete one full lap around the tank (generally about 45 s). Swimming patterns were differentiated as laps swum close to the inside wall, in the middle of the channel, or near the outside wall; the corresponding circumferences were used to calculate swimming speed in centimeters per second ($cm \cdot s^{-1}$) and body lengths per second ($BL \cdot s^{-1}$).

Heart Rate Experiment

Heart rates were monitored with electrocardiogram (ECG) electrodes (silver-plated copper wires) inserted subcutaneously over the heart. Electrodes were held in place near their points of insertion and at the posterior margin of the first dorsal fin with sutures (yellowfin tuna) or tiny (no. 85 Eagle Claw) stainless steel hooks (skipjack tuna). Seven skipjack tuna (fork length, 41.8–48.5 cm; body weight, 1.062–2.240 kg) and six yellowfin tuna (fork length, 33.8–57.2 cm; body weight, 0.630–3.390 kg) were used.

For yellowfin tuna, ECG electrodes were implanted under general anesthesia. The anesthetic used was $NaHCO_3$-buffered tricaine methanesulfonate (MS-222). The animals were revived in the test tank by pumping fresh seawater over their gills. Complete anesthesia and revival procedures are described in Jones et al. (1986, 1990). Total elapsed time, from initial anesthesia to full recovery of swimming ability, was approximately 25 min. Fish were given an additional hour to recover before the first hypoxic trial.

For unknown reasons, skipjack tuna often do not survive for long when forced to swim following recovery from anesthesia. Skipjack tuna were therefore handled differently. To minimize stress, fish were moved to the test tank the night before they were to be used in an experiment. On the following day, they were dip netted and gently restrained, ventral side up, on a padded table while seawater was pumped over their gills. Electrocardiogram wires were inserted subcutaneously, and the fish were immediately returned to the test tank. The entire procedure generally took less than 3 min. The fish were allowed 1 h to recover before the first hypoxic trial.

The ECG signal was recorded either by connecting the electrodes to a hand-held FM radio transmitter or to a Grass P-51 amplifier. When we used the transmitter, the signal was received with a Narco Biosystems Biotelemetry receiver, amplified, filtered, and recorded on cassette tape. When we used the amplifier, the amplified ECG signal was recorded directly with a portable

cassette recorder. Between tests, the electrodes were allowed to trail behind the fish. During recording, an investigator retrieved the electrodes, connected them to the FM transmitter or amplifier, and then followed the fish by walking around the inside of the tank. Care was taken to leave the ECG wires slack but clear of the fins, so as not to interfere with the fish's swimming movements. At the conclusion of the experiment, heart rates (beats per minute, bt · min^{-1}) were measured by playing the cassette tape back through an HP 5308A frequency counter that was connected to an IBM AT computer. The frequency counter measured time intervals between approximately every other heart beat, and the computer converted these data to heart rates. Heart rates were averaged over 1-min intervals and then matched to the corresponding measurements of swimming speed and P_{IO_2} for statistical analysis.

Data were collected over 50-min experimental runs consisting of a 10-min control period, a 30-min hypoxic period, and a 10-min recovery period. About 15 min were required to reach the minimum P_{IO_2} of 70 mmHg. At the conclusion of the hypoxic period, the tank was returned to full O_2 saturation as quickly as possible. The ECG leads were disconnected from the amplifier or transmitter, and the fish was allowed a 1-h recovery period before the next test. Animals were exposed to no more than two hypoxic trials. No individual was reused in subsequent experiments.

Ventilation Volume Experiment

We measured $\dot{V}_g$ with a continuous infusion dye dilution technique, using one dye infusion and two (left and right side) exhaled water sampling catheters, as described by Bushnell et al. (1990) and Jones et al. (1990). Catheters were implanted under general anesthesia, and the animals were revived in the test tank by pumping fresh seawater over the gills as described in Jones et al. (1986, 1990). Fish were given approximately 2 h to recover before the first hypoxic trial. Six skipjack (fork length, 47.4–49.0 cm; body weight, 1.824–2.019 kg) and eight yellowfin tuna (fork length, 49.4–57.4 cm; body weight, 2.050–4.032 kg) were used.

Separate water samples were taken to determine P_{EO_2}, P_{IO_2}, and dye concentrations. A second Radiometer system, with a water-jacketed electrode maintained at the test-tank temperature, was used to measure P_{IO_2} and P_{EO_2}. Sampling exhalant water for dye dilution and P_{EO_2} generally required about 60 s. Water samples were taken and lap times recorded simultaneously. Dye dilution samples were stored and analyzed at the end of the day, whereas P_{IO_2} and P_{EO_2} samples were measured immediately. The P_{O_2} electrodes were recalibrated with air-saturated water before each set of measurements.

The P_{IO_2} was reduced after three sets of control measurements were completed. Samples for $\dot{V}_g$, P_{IO_2}, and P_{EO_2} were taken, and lap times were recorded, as often as possible during the entire hypoxic episode. The minimum P_{IO_2} reached was approximately 90 mmHg. One hour was allowed for recovery between tests. Animals were exposed at most to two hypoxic trials. No individual was reused in subsequent experiments.

Utilization (U, in percent) and $\dot{V}_{O_2}$ (mg $O_2 \cdot min^{-1}$) were calculated from measured values:

$$U = [(P_{IO_2} - P_{EO_2})/P_{IO_2}] \times 100,$$

and

$$\dot{V}_{O_2} = \dot{V}_g \times [(P_{IO_2} - P_{EO_2}) \times \alpha],$$

where α is the solubility of O_2 in seawater (mg $\cdot L^{-1} \cdot mmHg^{-1}$) and $\dot{V}_g$ is in L $\cdot min^{-1}$.

Data Reduction and Statistical Procedures

An unpaired Student's *t*-test was used to compare mean control (i.e., normoxia) P_{IO_2}, P_{EO_2}, HR, swimming speed, $\dot{V}_g$, U, and $\dot{V}_{O_2}$ between the two species and to compare data from swimming fish with those from spinally blocked tunas reported in Bushnell et al. (1990).

Because the large volume of water in the test tank precluded step changes in P_{IO_2}, data recorded during hypoxia were divided into P_{IO_2} ranges (>150, 149–130, 129–110, 109–90, and 89–70 mmHg). To reduce variability between fish, percent changes from control means were determined. Mean (± standard error of the mean, SEM) percent changes in P_{IO_2}, HR, swimming speed, $\dot{V}_g$, U, and $\dot{V}_{O_2}$ were calculated for each range.

To determine at what level of hypoxia swimming speed and HR became significantly different from control measurements, a "breakpoint P_{IO_2}" was calculated for each experimental run. This was done by calculating 95% confidence intervals for mean swimming speed and HR in normoxia and comparing those to swimming speed and HR during hypoxia. The breakpoint P_{IO_2} swimming speed was taken to be the P_{IO_2} at which swimming speed exceeded the upper limit of the 95% confidence interval of mean control swimming speed. Similarly, the breakpoint P_{IO_2} for HR was the P_{IO_2} at which HR was significantly lower than the lower limit of the 95% confidence interval of mean control HR. In general, in the heart rate experiments, once swimming speed began to increase and HR began to decrease, they contin-

ued to do so. In a few cases, inconsistent changes in HR, swimming speed, or both were observed during hypoxia. These animals were eliminated from the breakpoint analysis. Grand mean breakpoints for HR and swimming speed were calculated by combining data from all skipjack tuna or all yellowfin tuna.

Results

Heart Rate Experiment

Table 1 summarizes the data collected from skipjack and yellowfin tunas in normoxia. The only significant difference was in absolute mean swimming speed, which was higher in skipjack tuna. As for the effect of swimming speed on HR, only four yellowfin tuna showed enough variability in speed during normoxia to provide usable data. As shown in figure 1, higher swimming speeds are associated with higher HR. (Note that some swimming

TABLE 1

Summary of normoxic (control) values (± SEM) measured in swimming yellowfin and skipjack tunas during the heart rate experiment

Variable	Yellowfin Tuna	Skipjack Tuna
Mass (kg)	1.9 ± .5	1.3 ± .1
	(6)	(7)
Fork length (cm)	46.1 ± 4.4	44.7 ± .5
	(6)	(7)
Control Pio_2 (mmHg)	151.7 ± 1.1	153.6 ± 1.3
	(6)	(7)
Heart rate (beats · min^{-1})	67.9 ± 3.4	76.8 ± 7.0
	(6)	(7)
Swimming speed:		
cm · s^{-1}	52.9 ± 4.1	73.0 ± 6.1*
	(6)	(6)
Body lengths · s^{-1}	1.2 ± .2	1.6 ± .1
	(6)	(6)

Note. Numbers in parentheses are the number of fish from which data were obtained.
* Significant difference ($P < 0.05$) between yellowfin and skipjack tunas.

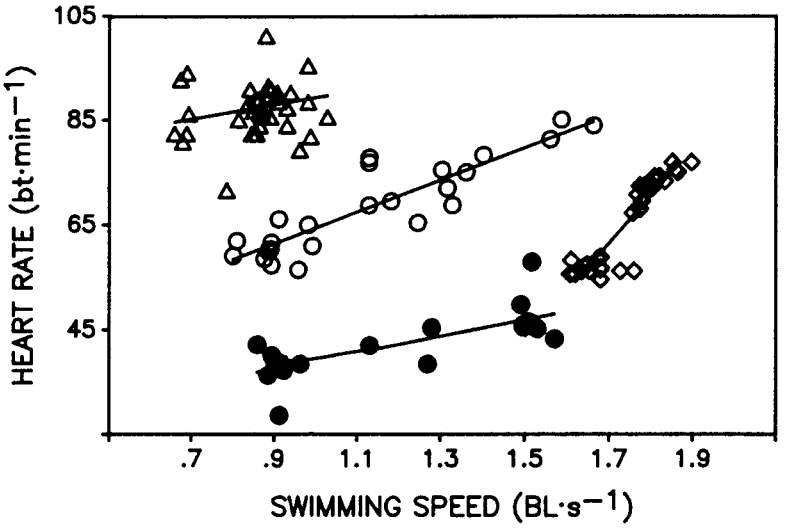

Fig. 1. Effects of voluntary increases in swimming speed on heart rate in four yellowfin tuna during normoxia. Solid lines are least-squares linear regressions.

speeds shown are below the predicted minimum hydrostatic equilibrium swimming speed [Magnuson 1978] for yellowfin tuna. This is most likely due to the fish's adopting a head-up tilting behavior as has been observed in Atlantic mackerel [*Scomber scombrus*, He and Wardle 1986], which, like tuna, are also negatively buoyant.)

The HR of an individual swimming yellowfin tuna is shown in figure 2*A*. The corresponding Pio_2, swimming speeds, and mean HR (averaged over 1-min intervals) are shown in figure 2*B*. The direct effect of hypoxia on swimming speed and heart rate can be clearly seen. All other yellowfin and skipjack tuna similarly tested also had a pronounced bradycardia when exposed to hypoxia. The bradycardia occurred despite concomitant increases in swimming speed, and, initially, heart rates slowed at rates similar to those seen in spinally blocked fish (Bushnell et al. 1990) (fig. 3*A*, *B*). However, when Pio_2 fell below 100 mmHg, the HR leveled off in yellowfin tuna and increased in skipjack tuna.

Breakpoint analyses showed that relatively modest declines in Pio_2 are enough to elicit significant reductions in HR and significant increases in swimming speed in both tuna species. The grand mean (± SEM) breakpoint Pio_2's for heart rate and swimming speed for skipjack tuna were 130.3 (± 7.5) and 123.6 (± 3.1) mmHg, respectively. The grand mean (± SEM) breakpoint Pio_2's for heart rate and swimming speed of yellowfin tuna were 129.9

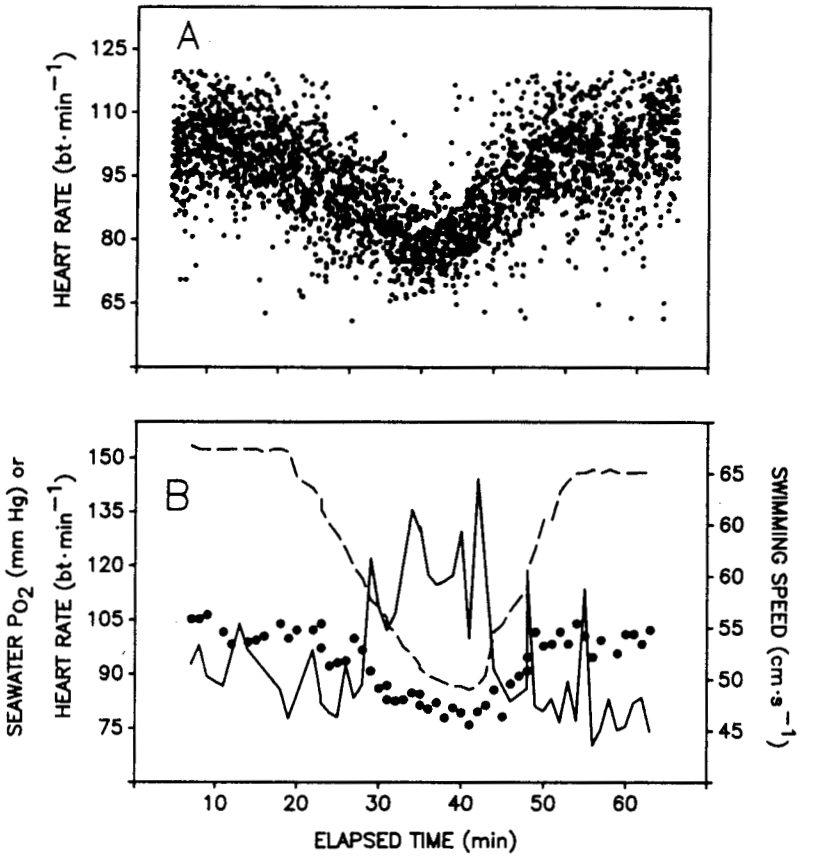

Fig. 2. (A) *The HR of a swimming yellowfin tuna, calculated from the time intervals between approximately every other heart beat; and* (B) *the corresponding* Pio_2 *(broken line), swimming speeds (solid line), and mean (averaged over 1-min intervals) heart rates (filled circles). The direct effect of hypoxia on swimming speed and heart rate can be clearly seen.*

(± 2.0) and 123.8 (± 6.5) mmHg, respectively. These were not significantly different in the two species or from the HR breakpoint Po_2 seen in spinally blocked fish (Bushnell et al. 1990).

Ventilation Volume Experiment

Of the six skipjack tuna used, only four regained the ability to swim at all following anesthesia, and none completed an entire hypoxia trial before losing equilibrium. Therefore, no data were recorded for skipjack tuna in

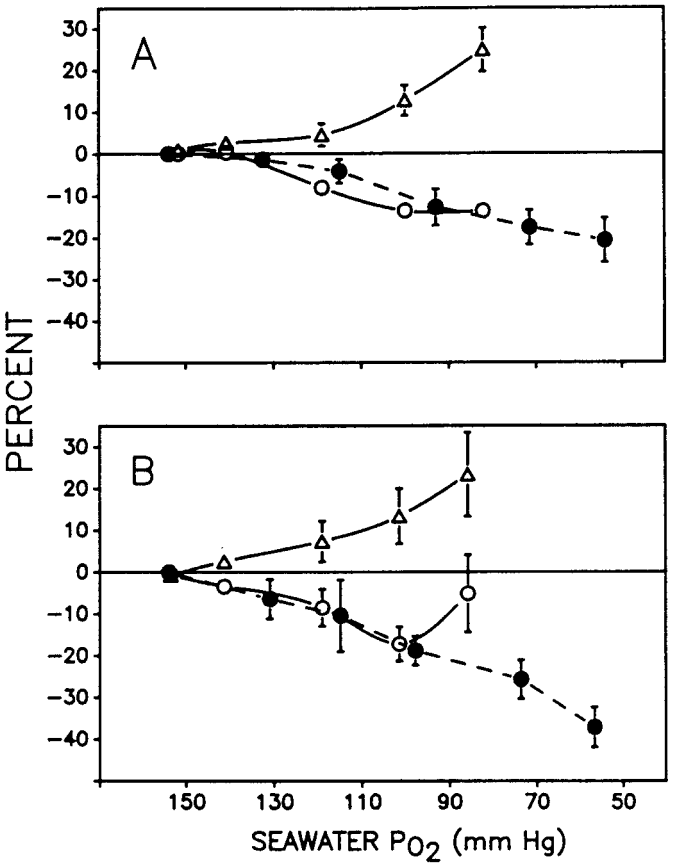

Fig. 3. Effect of hypoxia on relative changes (percent changes from mean values measured during normoxia) in swimming speed (open triangles) and heart rate (open circles) of swimming (A) *yellowfin tuna and* (B) *skipjack tuna. Changes in heart rate in response to hypoxia of spinally blocked yellowfin and skipjack tunas are shown by filled circles (data on spinally blocked tunas are from Bushnell et al. [1990]).*

hypoxia. In contrast, seven of the eight yellowfin tuna tested completed at least the first hypoxic trial.

With the exception of $\dot{V}_{O_2}$, all variables measured in yellowfin tuna during normoxia were significantly different from those measured in skipjack tuna (table 2). The P_{eO_2}, weight specific $\dot{V}_g$, and swimming speed were higher in latter; U was lower. Swimming speeds of skipjack tuna were higher during the ventilation volume experiment (2.2 ± 0.2 BL $\cdot$ s^{-1}) than during the heart rate experiment (1.6 ± 0.1 BL $\cdot$ s^{-1}). They were not significantly different in yellowfin tuna, however.

TABLE 2

Summary of mean normoxic control values (± SEM) measured in swimming yellowfin and skipjack tunas during the ventilation volume experiment

Variable	Yellowfin Tuna	Skipjack Tuna
Mass (kg)	2.8 ± .2 (7)	1.8 ± .1* (3)
Fork length (cm)	53.7 ± 1.0 (7)	47.2 ± 1.4* (3)
P_{IO_2} (mmHg)	154.7 ± .9 (7)	152.0 ± 1.2 (3)
P_{EO_2} (mmHg)	67.3 ± 2.6 (7)	85.1 ± 8.1* (2)
Utilization (%)	55.3 ± 2.0 (7)	44.3 ± 5.7* (2)
Ventilation volume:		
$L \cdot min^{-1}$	8.1 ± 1.1 (7)	10.2 ± 1.4 (3)
$L \cdot min^{-1} \cdot kg^{-1}$	2.9 ± .4 (7)	5.3 ± 1.0* (3)
Swimming speed:		
$cm \cdot s^{-1}$	68.0 ± 5.7 (7)	104.2 ± 10.0* (3)
Body lengths $\cdot s^{-1}$	1.3 ± .1 (7)	2.2 ± .2* (3)
Oxygen consumption ($mg\ O_2 \cdot h^{-1} \cdot kg^{-1}$)	624 ± 65 (7)	1,082 ± 398 (2)

Note. Numbers in parentheses are the number of fish from which data were obtained. Three sets of measurements of P_{IO_2}, P_{EO_2}, and swimming speed were obtained from each fish.
* Significant difference ($P \leq 0.05$) between yellowfin and skipjack tunas.

Figure 4 shows the changes in swimming speed, $\dot{V}_{O_2}$, U, and $\dot{V}_g$ in swimming yellowfin tuna. For comparison, data on the changes in $\dot{V}_g$ of spinally blocked fish (Bushnell et al. 1990) in response to hypoxia are also plotted. Note that the changes in $\dot{V}_g$, in response to hypoxia, are equivalent and that U decreased as $\dot{V}_g$ increased. Surprisingly, in contrast to the HR experiment,

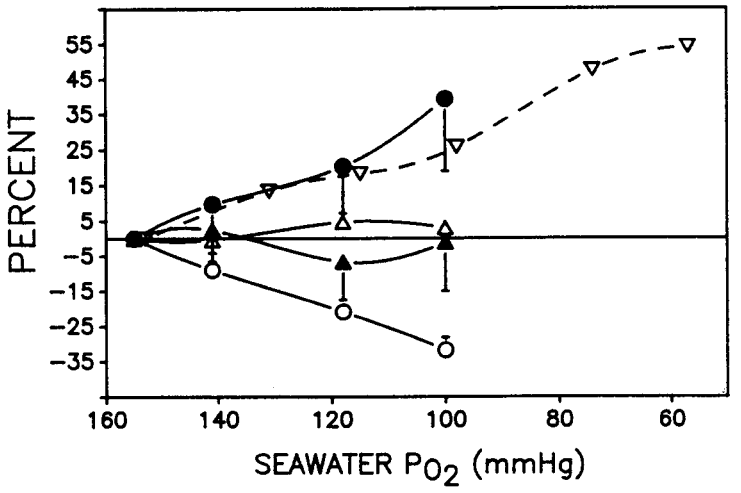

Fig. 4. Effect of hypoxia on relative changes (percent changes from mean values measured during normoxia) in utilization (open circles), ventilation volume (filled circles), swimming speed (open triangles), and oxygen consumption rate (filled triangles) of swimming yellowfin tuna (± SEM). Changes in ventilation volume (inverted triangles) of spinally blocked fish (from Bushnell et al. 1990) are also plotted.

swimming speed did not change. The $\dot{V}O_2$ also remained unchanged throughout the course of hypoxia.

Discussion

Normoxia

The swimming speeds of skipjack and yellowfin tunas in the heart rate experiment, and those of yellowfin tuna in the ventilation volume experiment, were similar to the swimming speeds of the uninstrumented animals used by Dizon (1977). Oxygen consumption rates of yellowfin tuna were comparable to those of the spinally fish used by Bushnell et al. (1990) but were almost double those of uninstrumented tunas studied by Boggs (1984).

Some stress, most likely associated with the general anesthesia required to implant the catheters, appeared to affect skipjack tuna in the ventilation volume experiments. They swam faster than the uninstrumented fish studied by Dizon (1977) and Boggs (1984), although their metabolic rate was not significantly different from those measured by Boggs (1984). Therefore, some anaerobic metabolism may have been involved, and this may explain why they did not survive even mild hypoxia.

There were some significant differences in cardiorespiratory variables measured in swimming yellowfin tuna and those measured in spinally blocked animals by Bushnell et al. (1990). The U in swimming fish (55%) was significantly higher than in spinally blocked fish (39%) but was essentially identical to those of swimming kawakawa (*Euthynnus affinis*) (55%) and swimming yellowfin tuna (56%) measured by Jones et al. (1990). The weight-specific $\dot{V}_g$ of swimming yellowfin tuna was significantly lower than the weight-specific $\dot{V}_g$ of spinally blocked fish (Bushnell et al. 1990). Therefore, spinally blocked yellowfin tuna, although able to set their own ventilation volume, apparently allow themselves to be overventilated. The U and weight-specific $\dot{V}_g$ of swimming skipjack tuna were not significantly higher than those of spinally blocked fish. Unfortunately, the small amount of data on the $\dot{V}_g$ of skipjack tuna, and their high variability, precludes detailed comparisons with spinally blocked fish.

The HR of swimming skipjack and yellowfin tunas were both 34% *lower* than the HR observed in spinally blocked fish (Bushnell et al. 1990) The reasons for this are unknown, but, in yellowfin tuna, this may be due to the apparent overventilation of spinally blocked animals rather than an effect of exercise. The effect of exercise on altering HR in teleost fish is not clear. A review by Jones and Randall (1978) presents evidence for decreases, increases, and no change in HR with exercise. In the four yellowfin tuna that, in normoxia, swam over a range of speeds great enough to show the effects of swimming speed on HR, HR and swimming speed were positively correlated. Moreover, the HR's we observed in swimming tunas were nowhere near the maximum heart rates (250–260 bt · min^{-1}) previously reported for swimming tunas (Kanwisher, Lawson, and Sundnes 1974) or for tunas paralyzed with a neuromuscular blocking agent and artificially ventilated (Brill 1987).

Hypoxia

Most previous studies measuring changes in swimming speeds of fish exposed to hypoxia used species capable of pumping water over their gills, and behavioral responses to hypoxia were found to depend on the experimental paradigm. For example, restrained dogfish sharks (*Scyliorhinus canicula*) became more active when exposed to hypoxia (Randall 1970), whereas unrestrained animals either became less active or remained inactive (Metcalf and Butler 1984). However, most fish reduce their activity during hypoxia (Randall 1970), presumably to reduce O_2 demand. Because tunas are obligate ram ventilators (Roberts 1978), this option has severe limitations. A decrease in speed would reduce $\dot{V}_g$ unless there were a concomitant

increase in mouth gape (i.e., mouth cross-sectional area). Although an increase in swimming speed is energetically expensive, it may be necessary if an increase in gape is not effective in maintaining O_2 delivery (this concept is more fully explored in the following sections where we expand Dizon's [1977] mathematical model of O_2 delivery in tunas). Examples of both kinds of responses were seen in this study. In the HR experiment, both species increased swimming speed in response to decreases in Pio_2. However, in the $\dot{V}_g$ experiment, yellowfin tuna increased their speed by an average of only 4%. Since $\dot{V}_g$ increased by 40%, these fish must have increased gape (and possibly also opercular flare). Moreover, the increases in $\dot{V}_g$ in swimming and spinally blocked yellowfin tuna (Bushnell et al. 1990) are essentially identical, indicating the suitability of the former technique for studying the responses and tolerances of tunas to hypoxia.

When compared with the uninstrumented fish used by Dizon (1977), the tunas in our HR study displayed a higher sensitivity to hypoxia. In other words, the Pio_2 at which they evinced an increase in swimming speed was higher. The breakpoint Pio_2 for swimming speed in *both* tuna species used in our study (ca. 124 mmHg) is higher than the 100 mmHg that Dizon (1977) reported as necessary to elicit swimming-speed changes in skipjack tuna.

As stated previously, Dizon (1977) found that yellowfin tuna did not increase swimming speed even when they were exposed to severe hypoxia. This is perplexing since his and our studies were conducted in the same tank with the same water system, using similarly sized fish, which, in normoxia, swam at identical swimming speeds. The major differences between protocols were that our fish towed ECG wires and were exposed to hypoxia much more rapidly. In Dizon (1977), the time required to go from normoxia to hypoxia ($Pio_2 \approx 70$ mmHg) and back to normoxia was approximately three times longer than in our study. The rate at which hypoxia deepens has been shown to be important in determining physiological responses in other fishes (Butler and Taylor 1971). Whether this is an important factor in the responses of tunas to hypoxia remains to be determined.

In swimming skipjack and yellowfin tunas exposed to hypoxia, HR appears to be influenced by two opposing drives: hypoxia and exercise. Initially, as Pio_2 falls, the hypoxia response dominates, because swimming speed has not yet increased very much. When Pio_2 has fallen enough to generate about a 10% increase in swimming speed, HR stops falling in yellowfin tuna and increases in skipjack tuna. At this point, the exercise drive to increase HR appears to begin to dominate. Regardless of this, the equivalent fall in HR in spinally blocked and swimming tunas clearly demonstrates the utility of the former technique for elucidating the responses to and tolerances of

tunas to hypoxia. The inability of spinally blocked tunas to swim does not appear to affect the HR response until severe levels of hypoxia are reached.

Model of O_2 Delivery in Tunas

Dizon (1977) created a mathematical model predicting the relationship among swimming speed, gape, O_2 demand, and O_2 delivery in skipjack tuna during normoxia and hypoxic. His results are replotted in figure 5*A*, *B*.

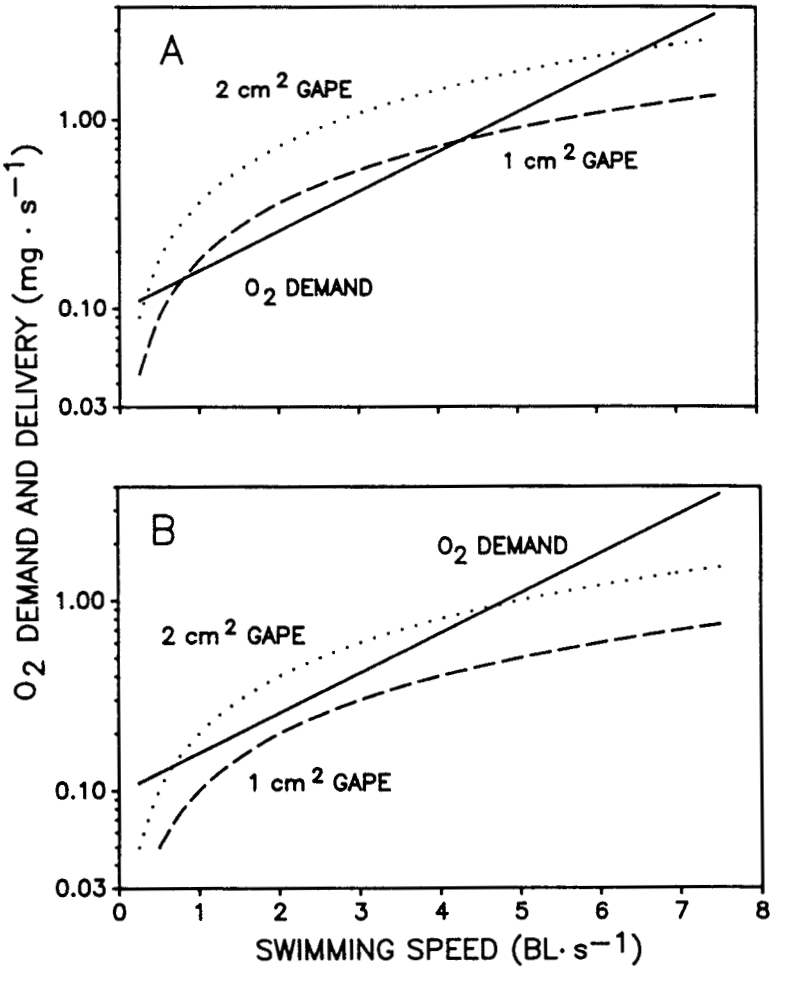

Fig. 5. Dizon's (1977) model of oxygen demand and effects of gape (i.e., mouth cross-sectional area) on oxygen delivery in skipjack tuna in (A) *normoxia and* (B) *hypoxia. In this model, hypoxia is defined as a seawater P_{O_2} = 85 mmHg.*

Since the model predicts that O_2 delivery rises linearly with speed and O_2 demand rises exponentially, a point is reached where O_2 demand exceeds delivery. Beyond this (the maximum aerobic swimming speed), further metabolic needs must be met anaerobically. During normoxia, the maximum aerobic swimming speed predicted by Dizon's (1977) model for a 40-cm fork length (1.4 kg) skipjack tuna is 4.5 BL · s^{-1} with a 1-cm^2 gape (dotted line, fig. 5*A*) and 6.5 BL · s^{-1} with a 2-cm^2 gape (dashed line, fig. 5*A*). Hypoxia is predicted to shift both O_2 delivery lines down (fig. 5*B*), such that oxygen delivery with a 1-cm^2 gape is below O_2 demand at all swimming speeds. His model therefore predicts that, during hypoxia, O_2 demand must be met by increasing gape rather than by increasing swimming speed alone.

Dizon's (1977) model contains two important parameters that we now know to be incorrect: (1) that O_2 consumption increases exponentially with swimming speed and (2) that U remains at 75% (i.e., U is independent of $\dot{V}_g$). The former was based on data given in Gooding, Neill, and Dizon (1981), and the latter on data provided in Stevens (1972). We therefore decided to reanalyze Dizon's (1977) model incorporating the following: data on swimming speed and metabolic rate provided by Boggs (1984) and Olson and Boggs (1986) and data on the effects of swimming speed on $\dot{V}_g$ and the effects of $\dot{V}_g$ and Pio_2 on U developed in the present study. We also decided to extend the model to include yellowfin tuna. All of the assumptions and specific equations used in our model are given in the Appendix.

Incorporating the relationships for U, $\dot{V}_g$, $\dot{V}o_2$, Pio_2, and swimming speed described in the Appendix results in a model with several noteworthy features (figs. 6, 7). As in Dizon's (1977) model (fig. 5), the slowest speeds that skipjack and yellowfin tunas can swim are predicted to be unconstrained by O_2 delivery. Oxygen delivery clearly exceeds demand even at speeds well below predicted minimum hydrostatic equilibrium swimming speeds (Magnuson 1973). The predicted maximum aerobic swimming speeds in normoxia are 5.6 and 3.5 BL · s^{-1} for yellowfin and skipjack tunas, respectively. Doubling the gape in normoxia (fig. 6) slightly increases the maximum aerobic swimming speed of skipjack tuna but reduces that of yellowfin tuna. This is a result of the reduction in U that is predicted at the higher $\dot{V}_g$. Maximum predicted $\dot{V}o_2$ at these speeds equaled 2,469 and 2,700 mg O_2 · kg^{-1} · h^{-1} (0.96 and 1.05 mg O_2 · s^{-1}) in yellowfin and skipjack tunas, respectively; these values are close to the maximum $\dot{V}o_2$ measured in freshly caught skipjack tuna (2,500 mg O_2 · kg^{-1} · h^{-1}; Gooding et al. 1981) and are roughly three times the maximum $\dot{V}o_2$ of other teleosts (1,000 ± 200 mg O_2 · kg^{-1} · h^{-1}; Brett 1972).

The maximum aerobic swimming speeds predicted by our model are quite high for most teleosts but appear low in light of the reputation of

tunas for prolonged, high-speed swimming. Beamish (1978), for instance, cites an example of a 40-cm skipjack tuna swimming at 20 BL · s^{-1} for 3 h. Anecdotal evidence from fishermen and others implies that schools of skipjack and yellowfin tunas are capable of swimming speeds in excess of 7–8 BL · s^{-1} for days. However, after reviewing documented tuna swimming speeds (Yuen 1970; Dizon 1977; Dizon, Brill, and Yuen 1978; Magnuson 1978; Gooding et al. 1981; Carey and Olson 1982; Boggs 1984; Holland, Brill, and Chang 1990), we found that, when swimming speeds surpass the maximal sustainable speeds predicted by our model, they always occurred for less than 200 min and therefore probably involved some degree of anaerobic metabolism (Beamish 1978).

In our model doubling gape does not appreciably extend maximum sustainable swimming speeds of skipjack tuna and reduces those of yellowfin tuna because of the concomitant decrease in U. Doubling the gape does, however, increase the O_2 delivery available for lactate metabolism (in fig. 6, the shaded areas between O_2 demand and delivery lines) by 36% in yellowfin tuna and 92% in skipjack tuna. At swimming speeds that are optimal for O_2 debt repayment, the maximum predicted O_2 delivery exceeds metabolic demand by 293%–525% in yellowfin tuna and 285%–393% in skipjack tuna, depending on gape.

In our model, increasing swimming speed during hypoxia (4.0 mg O_2 · L^{-1}, Pio_2 85 mmHg), if unaccompanied by an increase in gape, is predicted to be an ineffective response to hypoxia (fig. 7*A*, *B*). Doubling gape from 1 to 2 cm^2 results in enough oxygen delivery to allow both species to meet their metabolic needs aerobically at swimming speeds of 1.2–1.4 BL · s^{-1}. These velocities are right at the minimum hydrostatic equilibrium swimming speed of yellowfin tuna and substantially below that of skipjack tuna. In other words our model predicts that a Pio_2 of 85 mmHg is close to the minimum survivable O_2 tension for yellowfin tuna and is below the minimum survivable for skipjack tuna.

Even with a doubling of gape, increases in swimming speed beyond 1.2–1.4 BL · s^{-1} during hypoxia are predicted to not increase O_2 delivery. This agrees with experimental observations. When Gooding et al. (1981) exposed skipjack tuna to prolonged (4 h) levels of hypoxia (3.0–4.0 mg O_2 · L^{-1}, Pio_2 66–88 mmHg), all fish that swam between 1.5 and 2.9 BL · s^{-1} died within 20–155 min. The two fish that survived swam at a speed ($\approx$1 BL · s^{-1}) predicted by our model to be sustainable by aerobic metabolism. Presumably these fish were able to swim at speeds below their minimum predicted hydrostatic equilibrium speed by adopting a head-up tilting behavior similar to that observed in Atlantic mackerel (He and Wardle 1986). Although the ability of tunas to swim below their minimum predicted hydro-

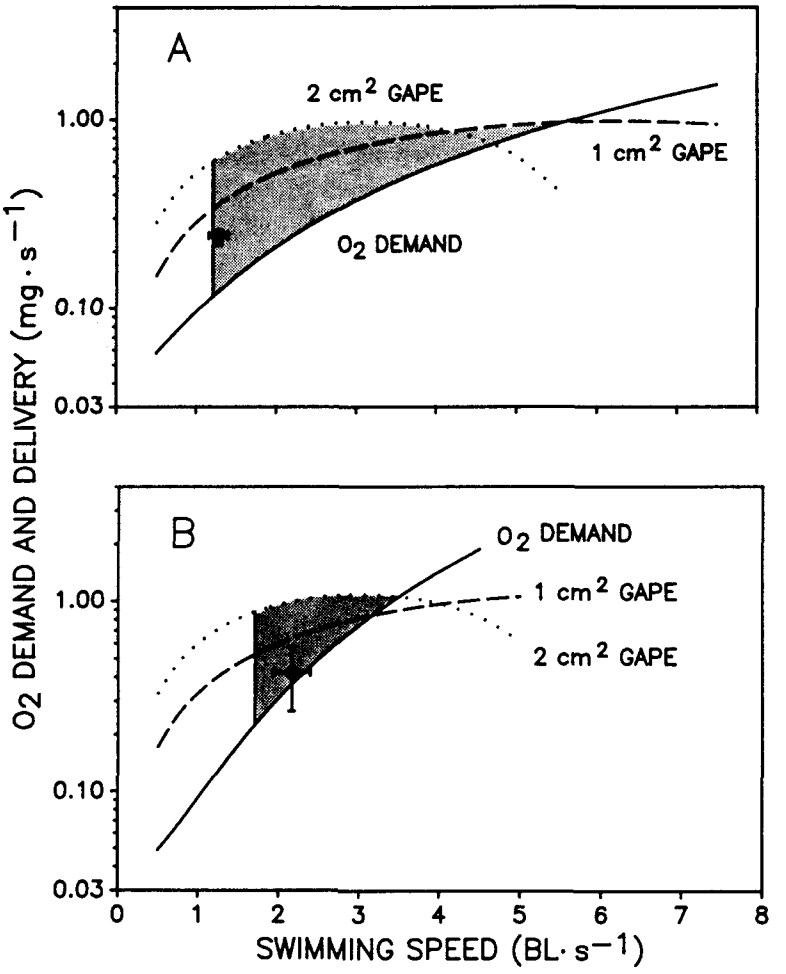

Fig. 6. Revised model of oxygen demand and effects of gape on oxygen delivery in (A) *yellowfin tuna and* (B) *skipjack tuna in normoxia. Note that, because of decreases in utilization, a larger gape (i.e., a higher ventilation volume) does not increase maximum sustainable (i.e., aerobic) swimming speeds. At speeds at which oxygen delivery exceeds demand (shaded areas), fish are capable of repaying oxygen debts. Single data points are the mean (± SEM) oxygen consumption rates and swimming speeds measured in this study during normoxia. Solid vertical lines show minimum hydrostatic equilibrium swimming speeds (from Magnuson 1973).*

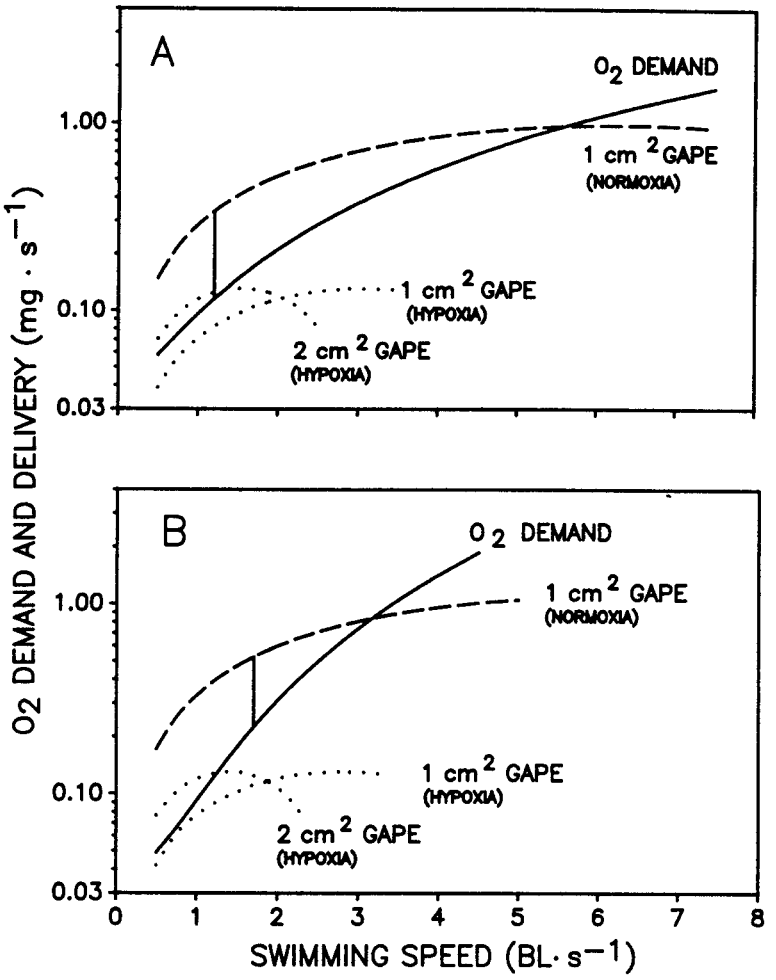

Fig. 7. Revised model of oxygen demand and effects of gape on oxygen delivery in (A) *yellowfin tuna and* (B) *skipjack tuna in normoxia (dashed lines) and hypoxia (dotted lines). Hypoxia in this model is defined as seawater* P_{O_2} *= 85 mmHg. Solid vertical lines show minimum hydrostatic equilibrium swimming speeds. In yellowfin tuna, a 2-cm² gape delivers enough oxygen to meet demand, but only at the minimum hydrostatic equilibrium speed, and there is little or no excess oxygen delivery available for repayment of oxygen debts. For skipjack tuna in hypoxia, even with a 2-cm² gape, oxygen delivery is well below oxygen demand at all speeds above minimum hydrostatic equilibrium speed.*

static equilibrium speeds makes prediction of lethal O_2 levels somewhat difficult, it is obvious that fish that must adopt these behaviors would not have the O_2 delivery capacity necessary to metabolize the high levels of white-muscle lactate known to be generated in feeding fish (Hochachka, Hulbert, and Guppy 1978). In other words, O_2 levels this low would not be survivable for prolonged periods.

The increases in swimming speeds in response to hypoxia observed in skipjack and yellowfin tunas in the HR portion of our study and in skipjack tuna in Dizon (1977) therefore are most likely a flight response. In other words increased swimming speed appears to be a behavior whose objective is to move the fish out of the hypoxic water, rather than an attempt to deliver enough O_2 to meet metabolic demand.

The picture of tuna swimming performance that results from our study and modeling efforts contradicts the currently held view that tunas are capable of very high, sustained speeds (Stevens and Dizon 1982). Tunas appear to be capable of high average swimming speeds—not as a consequence of an unusually large relative aerobic scope—but because a large glycolytic capacity is paired with an ability to repay O_2 debts at swimming speeds that are still quite high when compared with most teleosts. Furthermore, the capacity to provide large amounts of O_2 to the gills appears to be only one of several physiological adaptations for quick recovery from anaerobic exercise (Barrett and Connor 1964; Moon et al. 1986; Weber, Brill, and Hochachka 1986; Brill 1987). For example, studies by Perry et al. (1985) show that skipjack tuna can compensate for a 0.4-unit decrease in arterial blood pH (resulting from exhaustive exercise) within 30 min and that they can clear the metabolic proton load within 50 min. In other teleosts, these processes take 24 h or longer (Jones and Randall 1978).

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Appendix

To make the results of our model directly comparable to those of Dizon (1977), we also base our model 40-cm fork length (1.4-kg) fish and 24°C seawater containing 7.2 mg $O_2 \cdot L^{-1}$ at full saturation (Pio_2 = 154 mmHg). Hypoxia is defined as Pio_2 = 85 mmHg or 4.0 mg $O_2 \cdot L^{-1}$.

Oxygen Demand

Extensive studies by Boggs (1984) and Olson and Boggs (1986) show that, in tunas, $\dot{V}o_2$ (i.e., O_2 demand) is a power function, not an exponential function of speed, as presumed by Dizon (1977). Therefore, in our model, predicted energy demands are reduced at low swimming speeds (<2 BL $\cdot s^{-1}$) but increase dramatically at moderately higher speeds (>3.8 BL $\cdot s^{-1}$) when compared to Dizon's (1977) model. Equations 1 and 2 (from Boggs [1984]) also show that energy demands (PWR, W $\cdot g^{-1}$) of yellowfin (eq. [1]) and skipjack (eq. [2]) tunas decrease exponentially with fork length (FL, cm), are independent of weight (WT, g), and (as stated) increase exponentially with swimming speed (SPD, cm $\cdot s^{-1}$):

$$PWR = (4.45 \times 10^{-4} \times WT^{0.0}) + (1.59 \times 10^{-4} \times SPD^{1.64} \times FL^{-1.28}), \quad (1)$$

and

$$PWR = (4.26 \times 10^{-4} \times WT^{0.0}) + (0.91 \times 10^{-4} \times SPD^{2.36} \times FL^{-1.86}). \quad (2)$$

For use in our model, PWR was converted to O_2 demand (mg $O_2 \cdot s^{-1}$) by multiplying the former by body weight (1,400 g) and by 0.0654 mg $O_2 \cdot s^{-1} \cdot W^{-1}$ (Dejours 1975).

Oxygen Delivery

Predicted $\dot{V}o_2$ is a product of $\dot{V}_g$, O_2 content of the water ($[O_2]$, mg $\cdot L^{-1}$), and U (expressed as decimal fraction):

$$\dot{V}o_2 = \dot{V}_g \times [O_2] \times U. \quad (3)$$

In our model, $\dot{V}_g$ is a function of swimming speed and gape (i.e., mouth cross-sectional area), and U, in turn, is dependent on $\dot{V}_g$ and Pio_2.

Ventilation Volume

Because we could not control activity, we were unable to measure $\dot{V}_g$ over a wide range of swimming speeds. Therefore, we used mean $\dot{V}_g$ and swimming speeds (table 1) and assumed, like Dizon (1977), that $\dot{V}_g$ is directly proportional to speed. The resulting equations (4) and (5) (yellowfin and skipjack tuna, respectively) relating $\dot{V}_g$ and swimming speed are:

$$\dot{V}_g = 0.052 \times \text{SPD}', \tag{4}$$

and

$$\dot{V}_g = 0.057 \times \text{SPD}', \tag{5}$$

where SPD′ is swimming speed expressed in BL · s^{-1}. Because there are no measurements of mouth cross-sectional areas of swimming tunas, we assume that equations (4) and (5) are measured in tunas with a 1-cm^2 mouth cross-sectional area. We also assume, as did Dizon (1977), that $\dot{V}_g$ is directly proportional to mouth cross-sectional area. Also, based on data presented in Bushnell et al. (1990), we assume that both species are capable of doubling mouth cross-sectional areas from those required to provide adequate ventilation volumes at minimum O_2 demands.

Incorporating mouth cross-sectional area (GAPE) into equations (4) and (5) yields for yellowfin tuna,

$$\dot{V}_g = 0.052 \times \text{SPD}' \times \text{GAPE}, \tag{6}$$

and for skipjack tuna,

$$\dot{V}_g = 0.057 \times \text{SPD}' \times \text{GAPE}. \tag{7}$$

Utilization

Our measurements show that Dizon's (1977) predicted $\dot{V}_g$ for skipjack tuna is approximately 70% too low and that a 75% U is too high. The 75% U, measured by Stevens (1972) in skipjack tuna and used by Dizon (1977), was made in fish artificially ventilated at one-half the normal $\dot{V}_g$ of swimming animals. Data presented here also show that U is not fixed but is dependent on Pio_2 and $\dot{V}_g$ (fig. 4). Similar results have been reported for a variety of other teleosts and elasmobranchs (Saunders 1962; Hanson and Johansen 1970; Bushnell 1982). In our model, a fixed U is therefore replaced by a U that decreases with increasing $\dot{V}_g$ and decreasing Pio_2; the exact relationship was determined by a multiple linear regression of U on $\dot{V}_g$ and Pio_2 using data from yellowfin tuna. Because skipjack tuna did not survive during our attempts to measure $\dot{V}_g$ during hypoxia, the same regression equation is used for both species. Using the same regression equation appears reasonable since the U predicted for skipjack tuna by the multiple linear regression equation for yellowfin tuna is within 1% of the mean U measured in normoxia in spinally blocked and swimming skipjack tuna.

Using the data collected on yellowfin tuna in normoxia and hypoxia in a multiple linear regression of U on $\dot{V}_g$ (L · s^{-1}) and Pio_2 yields

$$\begin{aligned} &\text{U} = (Pio_2 \times 0.0029) - (\dot{V}_g \times 1.27) + 0.162, \\ &r^2 = 0.50. \end{aligned} \tag{8}$$

Tuna have a tremendous anaerobic capacity and can generate white-muscle lactate levels of 70–90 μmol · g^{-1} (Hochachka et al. 1978). To metabolize lactate, tuna

must deliver O_2 to the tissues at rates that exceed their usual metabolic demand. We believe that tuna are therefore capable of higher U than those shown by our data, when extra quantities of O_2 are needed to metabolize lactate. Higher U could be achieved by increasing effective gas exchange area of the gills (via lamellar recruitment) and by changes in intralamellar blood flow. Although these have never been demonstrated in tuna, both processes have been shown to be effective in increasing U in other teleosts (Nilsson 1986).

Maximum U and $\dot{V}O_2$ were incorporated into the model by increasing the intercept of equation (8) to achieve a U of 0.75 (i.e., 75%) at the $\dot{V}_g$ predicted to occur at minimum hydrostatic equilibrium swimming speeds of 1.4-kg skipjack and yellowfin tunas (0.097 and 0.062 L · s^{-1} at 1.7 and 1.2 BL · s^{-1}, respectively). Substituting normoxic PiO_2 (154 mmHg) and incorporating predicted $\dot{V}_g$ for both species (from eqq. [6] and [7]) yield for yellowfin tuna,

$$U = (PiO_2 \times 0.0029) - (\dot{V}_g \times 1.27) + 0.383, \quad (9)$$

and, for skipjack tuna,

$$U = (PiO_2 \times 0.0029) - (\dot{V}_g \times 1.27) + 0.427. \quad (10)$$

The assumptions made to forecast U in hypoxia were relatively simple. We assume that tunas in this environment maximize their O_2 delivery and are incapable of repaying O_2 debts. Therefore, the U predicted by the multiple linear regression of U on $\dot{V}_g$ and on PiO_2 (eq. [8]) is presumed to represent the highest U of which the fish are capable.

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