ON THE STANDARD METABOLIC RATES OF TROPICAL TUNAS, INCLUDING THE EFFECT OF BODY SIZE AND ACUTE TEMPERATURE CHANGE

RICHARD W. BRILL[1]

ABSTRACT

The standard metabolic rates (SMR's) of fishes and the effect of body weight on SMR's are important input parameters to energetics, growth, and population models. This study was undertaken to obtain these data for the tropical tuna species, yellowfin tuna, *Thunnus albacares*, and kawakawa, *Euthynnus affinis*. These data compliment similar SMR measurements from skipjack tuna, *Katsuwonus pelamis*, previously published. The effect of acute temperature change on the SMR of all three species was also determined.

The SMR was estimated by directly measuring the oxygen uptake rate of animals paralyzed with a neuromuscular blocking drug, rather than by the more commonly used method of extrapolation of swimming speed-metabolic rate curves back to zero swimming speed. To test the adequacy of this technique, the SMR's of aholehole, *Kuhlia sandvicensis*, and rainbow trout, *Salmo gairdneri*, were determined using similar methodology. The SMR's measured in this way were not significantly different from the published SMR's of these species determined by extrapolation of swimming speed-metabolic rate curves back to zero swimming speed.

All three tuna species have very high SMR's, over five times higher than other active teleost species such as salmon and trout. The effect of body size on the SMR is similar in all three tuna species, but the weight specific SMR of tuna decreases more rapidly with increasing body size than in other fishes. Based on SMR's measured at 20° and 25°C, the Q_{10}'s were 3.16, 2.31, and 2.44 for yellowfin tuna, kawakawa, and skipjack tuna, respectively. These are similar to Q_{10} values found for the SMR's of other teleosts.

Tunas can achieve exceptionally high maximum aerobic metabolic rates. This ability requires a complete set of anatomical, physiological, and biochemical adaptations. I hypothesize that one of these adaptations, large gill surface areas, causes tunas to have exceptionally high energy demands even at rest. Tunas' high SMR's are an inevitable consequence of their ability to achieve exceptionally high maximum aerobic metabolic rates.

The standard metabolic rate (SMR) (the metabolic rate of a postabsorptive animal completely at rest) and the effect of body size on SMR are important input parameters to growth, energetics, and population models (e.g., Sharp and Francis 1976; Kitchell et al. 1978). This study was therefore undertaken to obtain these data for yellowfin tuna, *Thunnus albacares*, and kawakawa, *Euthynnus affinis*. These measurements were designed to directly compliment the SMR measurements for skipjack tuna, *Katsuwonus pelamis*, that had been previously published (Brill 1979). The effect of acute temperature change on the SMR of skipjack tuna, yellowfin tuna, and kawakawa was also determined. The effect of acute temperature change, as opposed to the effect of temperature adaptation, is relevant to tuna because of the 5° to 15°C water temperature changes these species normally experience during the daily vertical movements which are a constant feature of their behavior in the open ocean (Dizon et al. 1978; Carey and Olson 1982; Yonemori 1982).

In other teleosts, SMR's have been determined by extrapolating metabolic rate-swimming speed curves back to zero swimming speed (e.g., Brett 1965). Although Graham and Laurs (1982) have successfully measured the metabolic rate of albacore, *T. alalunga*, (a temperate tuna species) swimming in a water tunnel, this methodology is presently not possible with tropical tunas (skipjack tuna, yellowfin tuna, and kawakawa). Attempts to get these species to swim in several prototype water tunnel designs have shown that they will do so for only very short periods (Brill and Dizon 1979 and unpublished observations). As a result, measuring the SMR's of tropical tunas directly in animals paralyzed with a neuromuscular blocking agent is currently the only method available to obtain these data.

To validate this technique, the SMR's of rainbow

[1]Southwest Fisheries Center Honolulu Laboratory, National Marine Fisheries Service, NOAA, 2570 Dole Street, Honolulu, HI 96822-2396.

Manuscript accepted November 1986.
FISHERY BULLETIN: VOL. 85, NO. 1, 1987.

trout, *Salmo gairdneri*, and aholehole, *Kuhlia sandvicensis*, were also measured using paralyzed animals. These two species were chosen because they are available in Hawaii and because there are published data on their SMR's based on extrapolation of swimming speed-metabolic rate curves back to zero swimming speed (Muir et al. 1965; Bushnell et al. 1984).

The SMR of a 1 kg skipjack tuna (412 mg O_2/h, Brill 1979), is almost five times greater than that of a 1 kg sockeye salmon, *Oncorhynchus nerka* (83 mg O_2/h, Brett and Glass 1973). The former measurements were made at 25°C and the latter at 20°C, because 25°C is the upper lethal temperature for salmon (Brett 1972). However, a 5°C temperature difference could not account for this SMR difference because the Q_{10}'s for the SMR's of fishes are generally about 2 (Robinson et al. 1983). The maximum sustainable aerobic metabolic rate (MMR, the metabolic rate at the maximum swimming speed sustainable for at least 1 h) of a 1 kg sockeye salmon at 20°C is 796 mg O_2/(kg·h), whereas 1.8-2.2 kg skipjack tuna at 24°C have been shown to be able to achieve active metabolic rates over 2,000 mg O_2/(kg·h) (Gooding et al. 1981). Although there are no metabolic rate measurements available for tunas at their maximum sustainable swimming speeds, two conclusions are still obvious: 1) skipjack tuna have very high SMR's even when compared with other active equal sized teleosts and 2) skipjack tuna are capable of very high aerobic metabolic rates.

I hypothesize that the high SMR's of tunas are primarily a result of their large gill surface areas (Hughes 1979). In other words, adaptations that permit high maximum sustainable rates of oxygen uptake (i.e., high MMR's) obligate tunas to have high SMR's. Analogous arguments with respect to the resting and maximal metabolic rates of terrestrial vertebrates have been presented by Bennett and Ruben (1979).

MATERIALS AND METHODS

SMR Measurements-Tuna

Live skipjack tuna, yellowfin tuna, and kawakawa were purchased from local fishermen and maintained at the Kewalo Research Facility (Southwest Fisheries Center Honolulu Laboratory, National Marine Fisheries Service, NOAA). Animal procurement, handling, and maintenance procedures at this facility are described by Nakamura (1972), Queenth and Brill,[2] and Chang et al.[3] Fishes were maintained in outdoor tanks for a few days to over 1 yr before use. Temperature of the seawater supplied to the holding tanks was 25°C (±2). Food was presented daily; however, individuals were not fed for at least 20 h prior to use in an experiment. This allowed sufficient time for gut clearance and for blood glucose level to return to prefeeding levels (Magnuson 1969).

Each experimental animal was removed from its holding tank by dip net and injected intramuscularly with 1-3 mg/kg of the neuromuscular blocking agent Flaxedil[4] (gallamine triethiodide). The animal was quickly returned to its holding tank, and when it could no longer swim, it was immediately rushed into the laboratory and placed in a Plexiglas flow-through box respirometer similar to that used by Stevens (1972). The respirometer was equipped with a movable partition which was placed immediately behind the fish to reduce the respirometer's volume and, thus, reduce the lag time between actual and measured changes in metabolic rate to only minutes (Niimi 1978). Water flow through the respirometer was maintained at 3-7 L/(kg·min) and was measured every 30-60 min by recording the time to fill a 1 L graduated cylinder. Water temperature was controlled by a chiller and freshwater heat exchanger and by a quartz heater mounted in the inflow seawater line. Temperature control was ±0.3°C.

Unlike the previous study on the SMR of skipjack tuna (Brill 1979), the spinal cord was not cut to stop all overt muscular activity. Rather, an 18-gauge hypodermic needle was placed intramuscularly and connected to the outside of the respirometer via a short length of polyethylene tubing. Through this tube, 0.1-0.3 mL doses of Flaxedil were administered when the fish began to show any slight tail movements. To monitor heart rate, electrocardiogram leads were mounted subcutaneously on the ventral body surface. Heart rate was determined by timing the interval between successive beats with a Hewlett-Packard (HP) 5308A frequency counter. Thermistors were used to measure fish muscle and water temperatures. With the aid of an 18-gauge hypodermic needle, a thermistor bead mounted in 0.9 mm diameter polyethylene tubing was inserted

[2]Queenth, M. K. K., and R. W. Brill. 1983. Operations and procedures manual for visiting scientists at the Kewalo Research Facility. Southwest Fisheries Center Honolulu Laboratory, National Marine Fisheries Service, NOAA, Honolulu, HI 96822-2396, Administrative Report H-83-7, 16 p.

[3]Chang, R. K. C., R. W. Brill, and H. O. Yoshida. 1983. The Kewalo Research Facility, 1958 to 1983—25 years of progress. Southwest Fisheries Center Honolulu Laboratory, National Marine Fisheries Service, NOAA, Honolulu, Hawaii 96822-2396, Administrative Report H-83-14, 28 p.

[4]Reference to trade names does not imply endorsement by the National Marine Fisheries Service, NOAA.

into the red muscle immediately adjacent to the spinal column. Thermistor probes were also mounted in the incoming seawater line and in the respirometer box itself. Red muscle and water temperatures were determined by measuring the resistance of the various thermistors with an HP 3456A digital multimeter.

Oxygen concentration (milligrams per liter) of the water upstream and downstream of the fish was determined with a dissolved oxygen meter (Yellowsprings Instrument, model 51A) equipped with a Clark-type polarographic electrode oxygen-temperature probe. The probe was normally in the outflow seawater line, but was moved to the inflow seawater line to determine inflow seawater oxygen levels every 30-60 min. The analog output of the oxygen meter was also measured with the HP digital multimeter. An HP 9825A computer was used to control an HP 5930A six-channel relay actuator which permitted the digital multimeter to determine sequentially the resistances of various thermistors and the analog output of the oxygen meter. Seawater oxygen level, red muscle and water temperatures, metabolic rate, and heart rate were calculated and printed by the computer at 5-min intervals.

After being sealed, the respirometer box was covered with black plastic to minimize disturbance to the fish. Temperature of the seawater supplied to the respirometer was maintained at 21°-22°C for the first 1-2 h because reduced water temperature has been shown to help tuna survive after handling (Barrett and Connor 1964). Seawater temperature was then changed to either 20° or 25°C, and the fish maintained at the test temperature until its metabolic rate remained relatively stable for at least 1 h. The SMR was estimated by averaging the last 5-12 metabolic rate measurements. The standard deviations of the metabolic rate measurements used to estimate SMR were <11% of the mean (i.e., SMR) in all cases, and in 70% of the cases, the standard deviations were <5% of the mean.

To determine the SMR at a second temperature, the water temperature was changed to either 20°, 25°, or 30°C, and metabolic rate measurements continued again until the fish's metabolic rate remained stable for 1 h.

SMR Measurements-Aholehole and Rainbow Trout

Aholehole were obtained from Sea Life Park (Waimanalo, HI) and rainbow trout from a commercial fish farmer (through the University of Hawaii, Hilo). The former were maintained in an outdoor tank with running seawater at 25°C (±2) and the latter, in an indoor tank with running freshwater at 15°C (±2). Both species were fed daily, but individuals were not fed for at least 20 h prior to use in an experiment.

The respirometer used for aholehole was essentially identical to that used by Davis and Cameron (1971) and Jones and Schwarzfeld (1974) to measure water flow and gas exchange across the gills of rainbow trout. The aholehole were anesthetized in 1:10,000-1:30,000 MS222 (Tricaine methanosulfonate). A thin, rubber membrane was sutured around the fish's mouth and sealed with a small amount of tissue glue (Histoacryl, B. Braun Melsungen AG, West Germany). The fish was then placed in a black Plexiglas box that was open at both ends. This box was then placed in a larger tank that was divided into two chambers by a partition with a hole through it. The membrane sealed around the fish's jaws was attached to the edge of the hole and sealed in place with a Plexiglas plate held with stainless steel wing nuts. This system allowed separation of the inspired and expired water, yet allowed the fish to make normal respiratory movements. Water level in the two chambers was maintained by standpipes (constant level drains). Ventilation volume was determined by measuring the water flow rate from the standpipe in the chamber containing the fish. By lowering this standpipe, the fish could be force-ventilated.

Water samples were drawn from the anterior chamber, and from the black Plexiglas box containing the fish, approximately every 15 to 20 min. Water oxygen level was determined with a water-jacketed oxygen electrode (Radiometer, Copenhagen) maintained at 25°C. Metabolic rate was calculated using the oxygen content difference between inspired and expired water and the ventilation volume.

Aholehole were given 2 h to recover from the anesthesia before metabolic rate measurements were begun. A series of metabolic rate measurements were made with the water level in the two chambers even and the fish actively pumping water over its gills, until the its metabolic rate remained relatively stable for at least 1 h. The water level in the chamber containing the fish was then lowered and measurements taken while the animal was being force-ventilated, continuing again until the metabolic rate stablized. Finally, the fish was given 0.1-0.3 mL Flaxedil (intramuscularly) and metabolic rate measurements continued while the animal was paralyzed and force-ventilated. In two cases, the fish was left in the respirometer overnight on forced ventilation to allow the effects of Flaxedil to wear off. Metabolic rate measurements were made again

before and after Flaxedil injection the next day. The SMR was calculated as the mean of the last four to six metabolic rate measurements. Water temperature was maintained at 25°C (±0.3) throughout the experiment.

The SMR of rainbow trout was directly determined in the same respirometry box as that used for tunas, using essentially identical methodology, except freshwater was used, inspired and expired water were sampled, and oxygen levels were measured with a water-jacketed Radiometer oxygen electrode.

RESULTS

Effects of Body Size on SMR

The SMR's of 21 kawakawa (0.540-2.153 kg) and 13 yellowfin tuna (0.585-3.890 kg) were determined at 25°C. Regression lines of SMR versus body weight were fitted by Gauss-Newton iteration (Biomedical Computer Programs, Program BMDP 3R), rather than a log-log transformation of the data (Fig. 1). The advantages of the former and disadvantages of the latter method are discussed by Zar (1968) and Glass (1969).

The best fitting allometric equations are

1) Kawakawa:
   $SMR = 392.5\ (\pm 32.3)\ W^{0.496\ (\pm 0.145)}$
   $n = 21$
2) Yellowfin tuna:
   $SMR = 286.8\ (\pm 26.9)\ W^{0.573\ (\pm 0.116)}$
   $n = 13$.

For comparison, the allometric equation relating

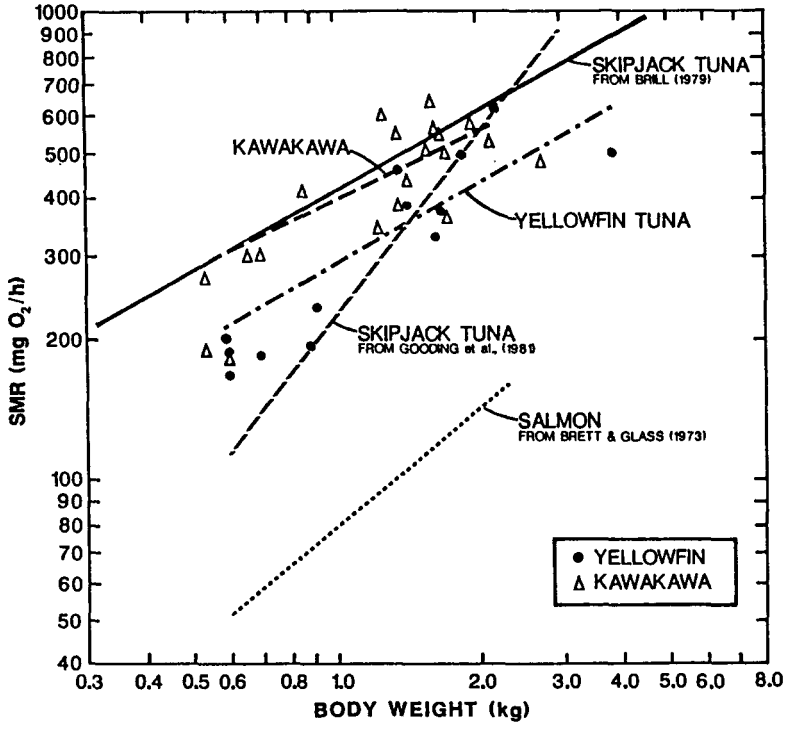

FIGURE 1.—A double logarithmic plot of the standard metabolic rates (SMR) of 13 yellowfin tuna and 21 kawakawa. The lines represent the allometric equations: $SMR = 286.8\ W^{0.573}$, $SMR = 392.5\ W^{0.496}$, and $SMR = 412.0\ W^{0.563}$ for yellowfin tuna, kawakawa, and skipjack tuna, respectively, where the SMR is mg O_2/h and W is body weight in kilograms. The line for skipjack tuna is from Brill (1979). For comparison, the regression lines based on swimming skipjack tuna (Gooding et al. 1981) and for salmon at 20°C (Brett and Glass 1973) are also shown. All tuna data is from fish at 23°-25°C.

SMR and body weight in skipjack tuna is (Brill 1979)

$$SMR = 412.0\ (\pm 27.1)\ W^{0.563\ (\pm 0.07)}$$
$$n = 33.$$

The SMR is in mg O_2/h and W is body weight in kilograms. The values in parentheses are the standard errors of the parameters.

Effects of Acute Temperature Change on SMR, Heart Rate, and Excess Red Muscle Temperature

A total of 8 kawakawa, 12 yellowfin tuna, and 5 skipjack tuna were subjected to 5°C temperature changes. Most temperature changes were made between 20°C and 25°C, which all fish survived. Ten fish were exposed to 25° and 30°C, but only four survived long enough at 30°C to provide usable data. Because of the expense and difficulty in obtaining live tunas, the latter treatment was not pursued.

The SMR's and mean heart rates at 20°, 25°, and 30°C are given in Table 1. The Q_{10}'s of SMR for water temperatures changes from 20° to 25°C were variable and ranged from 5.82 to 1.39. The mean Q_{10}'s (±95% confidence intervals) were 2.44 ± 0.97, 2.31 ± 0.51, and 3.16 ± 0.93 for skipjack tuna, yellowfin tuna, and kawakawa, respectively.

The range of mean excess red muscle temperatures are given in Table 2. These excess muscle temperatures are lower than those measured in free swimming yellowfin and skipjack tunas (Dizon and Brill 1979). This is as expected because in paralyzed tunas, most of the heat production (i.e., energy consumption) most likely occurs at the heart and gills where the heat would not be retained by the vascular countercurrent heat exchangers.

The SMR of Aholehole and Rainbow Trout

Aholehole, unlike rainbow trout, will not sit quietly in a darkened respirometer box nor stop breathing movements when force-ventilated. Because Flaxedil

TABLE 1.—Effect of temperature on the standard metabolic rate and heart rate of yellowfin tuna, kawakawa, and skipjack tuna.

Species Weight	SMR (mg O_2 h) 20°C	25°C	30°C	Q_{10} 20°-25°C	25°-30°C	Heart rate (min^{-1}) 20°C	25°C	30°C	Q_{10} 20°-25°C	25°-30°C
Yellowfin tuna										
2.215	470 ± 47	625 ± 53	—	1.77	—	89 ± 1	133 ± 2	—	2.21	—
1.438	258 ± 8	386 ± 21	605 ± 13	2.24	2.46	98 ± 1	146 ± 2	176 ± 1	2.22	1.45
1.635	161 ± 17	330 ± 17	—	4.20	—	81 ± 2	138 ± 2	—	2.89	—
3.890	311 ± 12	501 ± 32	—	2.60	—	57 ± 2	90 ± 2	—	2.49	—
0.704	112 ± 6	184 ± 6	—	2.70	—	99 ± 4	149 ± 1	—	2.26	—
0.877	150 ± 6	193 ± 6	—	1.66	—	77 ± 1	121 ± 3	—	2.45	—
0.599	103 ± 3	166 ± 6	—	2.60	—	73 ± 6	138 ± 8	—	3.55	—
0.595	153 ± 6	187 ± 6	—	1.49	—	74 ± 6	137 ± 5	—	3.41	—
0.585	154 ± 9	199 ± 3	266 ± 10	1.67	1.73	118 ± 1	159 ± 3	200 ± 2	1.82	1.58
1.290	333 ± 4	493 ± 17	—	2.19	—	106 ± 1	160 ± 2	—	2.28	—
Mean:				2.31	2.10				2.56	1.59
Standard deviation:				0.80	0.52				0.56	0.09
Kawakawa										
1.439	258 ± 12	431 ± 19	—	2.79	—	128 ± 1	205 ± 8	—	2.57	—
1.713	331 ± 12	497 ± 11	—	2.25	—	147 ± 2	213 ± 2	—	2.10	—
0.870	170 ± 14	410 ± 27	—	5.82	—	147 ± 4	217 ± 6	—	2.18	—
1.283	379 ± 9	598 ± 25	—	2.49	—	146 ± 14	206 ± 23	—	1.99	—
1.623	363 ± 27	640 ± 23	—	3.11	—	118 ± 4	175 ± 3	—	2.20	—
1.377	—	543 ± 35	761 ± 23	—	1.96	—	200 ± 14	272 ± 27	—	1.85
1.653	309 ± 7	560 ± 34	—	3.28	—	155 ± 1	221 ± 4	—	2.03	—
0.700	195 ± 10	300 ± 11	—	2.37	—	183 ± 4	253 ± 15	—	1.91	—
Mean:				3.16	1.96				2.14	1.85
Standard deviation:				1.23	—				0.22	—
Skipjack tuna										
1.069	140 ± 7	263 ± 22	—	3.53	—	78 ± 28	202 ± 4	—	6.71	—
0.582	214 ± 8	282 ± 6	386 ± 7	1.87	1.87	148 ± 2	237 ± 9	275 ± 20	2.56	1.35
0.425	173 ± 6	226 ± 11	—	1.71	—	122 ± 6	191 ± 5	—	2.45	—
0.448	179 ± 9	211 ± 8	—	1.39	—	134 ± 8	197 ± 3	—	2.16	—
0.629	113 ± 6	217 ± 7	—	3.71	—	145 ± 3	212 ± 3	—	2.14	—
Mean:				2.44	1.87				3.74	1.35
Standard error:				1.09	—				2.11	—

TABLE 2.—Range of mean (±SD) excess muscle temperatures in paralyzed tuna.

Tuna	20°C Mean	20°C SD	25°C Mean	25°C SD	30°C Mean	30°C SD
Kawakawa	0.7(±0.2)-1.9(±0.3)		0.5(±0.2)-1.5(±0.3)		[1]1.0(0.1)	
Yellowfin	0.3(±0.2)-1.4(±0.1)		0.0(±0.1)-0.7(±0.2)		0.2(±0.1)-0.4(±0.1)	
Skipjack	0.2(±0.1)-0.6(±0.1)		0.0(±0.2)-1.0(±0.2)		[1]0.6(±0.2)	

[1]Only one fish survived long enough to provide useful data.

stops all movements, all fish showed a decrease in metabolic rate after injection. The decrease ranged from 10 to 52% (mean 36%).

The directly measured SMR's from four aholehole and four rainbow trout paralyzed with Flaxedil are given in Table 3.

DISCUSSION

Adequacy of Directly Measured SMR

Muir et al. (1965) provided a regression equation for SMR versus weight for aholehole adapted to 23°C freshwater, based on extrapolation of swimming speed-metabolic rate curves back to zero swimming speed. The predicted freshwater SMR's based on their regression equation was increased by 75% to account for the higher osmoregulatory costs of seawater adapted animals (Nordlie and Leffler 1975). No correction was made for temperature. As shown in Table 3, in all cases but one, the directly measured SMR's are close to the SMR's based Muir et al.'s data when corrected for seawater adapted animals. With respect to rainbow trout, in all cases but one, directly measured SMR's are within one standard deviation of the SMR's obtained by extrapolation to zero swimming speed for rainbow trout at 15°C obtained by Bushnell et al. (1984). Therefore, directly measuring SMR's in Flaxedil-paralyzed aholehole and rainbow trout yields data that are similar to data obtained by the more widely used method of determining SMR by extrapolation of a swimming speed-metabolic rate curve back to zero swimming speed.

Tropical tuna species such as yellowfin, skipjack, and kawakawa will survive in a swimming tunnel for only short periods of time. Although other methods to control swimming speed (such as weighting and fin clipping, Dizon and Brill 1979; Boggs 1984) have been tried, they have met with only limited success. Therefore, direct measurement of SMR's using Flaxedil-paralyzed animals is for now the only way to obtain these data for tropical tuna species. As the data from aholehole and rainbow trout show, direct measure of SMR's using paralyzed animals yields results similar to that obtained by the more commonly used method of extrapolating swimming speed-metabolic rate curves back to zero swimming speed.

The heart rates (+1 SE, at 25°C) observed in this study were 230+20, 206+36, 132+17/min for skipjack tuna, kawakawa, and yellowfin tuna, respectively. These heart rates are higher than those observed for lightly anesthetized skipjack tuna (Stevens 1972), and are 60 and 39% higher than heart rates measured in skipjack and yellowfin tunas (respectively) that have been immobilized by spinal blockade with lidocaine and are force ventilated (unpubl. obs.). The heart rates measured in Flaxedil-paralyzed animals are, however, within the range exhibited by free-swimming skipjack tuna (80-240 beats/min, Kanwisher et al. 1974). The higher heart

TABLE 3.—Standard metabolic rate of aholehole and rainbow trout.

Oxygen consumption (mg O_2 kg^{-1} h^{-1}) ± SD					
Aholehole			Rainbow trout		
Weight (g)	Measured SMR	Predicted SMR[1]	Weight (g)	Measured SMR	Predicted SMR[2]
65.5	264(±12.2)	118	289	53.3(±3.8)	82.5±(27.4)
80.9	146(±16)	113	401	78.8(±7.0)	82.5±(27.4)
91.2	[3]135/127(±9.9/8.8)	111	403	60.5(±10.9)	82.5±(27.4)
108.5	[3]114/162(±11.1/12.0)	106	568	55.5(±9.9)	82.5±(27.4)

[1]Based on Muir et al. (1965) and corrected for saltwater adapted fish based on Nordlie and Leffler (1975).

[2]From Bushnell et al. (1984), for 250-350 g fish adapted to 15°C. No corrections for the weight dependence of SMR were provided.

[3]SMR determinations made approximately 20 h apart.

rates observed in paralyzed tunas may be due to the vagolytic action of Flaxedil (Grollman and Grollman 1970). However, as the data from aholehole and rainbow trout show, estimating SMR using animals paralyzed with Flaxedil and by extrapolation of swimming speed-metabolic rate curves back to zero swimming speed yield similar results.

Effect of Body Size and Acute Temperature Change on SMR

In Figure 1, it appears that the SMR's of yellowfin tuna are lower than those of skipjack tuna and kawakawa. However, based on the 95% confidence intervals, the heights of the regression lines (at mean body weights) are not significantly different from each other. Based on the 95% confidence intervals, the weight exponents of the regression equations for kawakawa, yellowfin tuna, and skipjack tuna also are not significantly different over the size ranges tested (Fig. 1). In other words, the effect of body weight on the SMR is not significantly different among the three tuna species. The exponent in the allometric equation describing the effect of body size on the SMR of other teleosts ranges from approximately 0.65 to >1 (Winberg 1956; Fry 1957; Beamish 1964; Beamish and Mookherjii 1964; Glass 1969; Brett 1972). The lower values of the exponents for tunas indicate that the weight specific SMR[5] (i.e., mg O_2/(g·h)) of tunas decreases more rapidly as body size increases than it does for other teleosts.

Gooding et al. (1981) also estimated the SMR of skipjack tuna. When converted to the same units used in this study (SMR in mg O_2/h and W in kg), the relationship they found for the effect of body weight on SMR was

$$\text{SMR} = 234\ W^{1.19}.$$

The exponent greater than one means that they predict the weight specific SMR to increase with increasing body size. As shown in Figure 1, Gooding et al.'s predicted SMR's are lower than mine for small fish, but exceed my estimates above approximately 2.5 kg body weight because of the large weight exponent.

To estimate SMR, Gooding et al. (1981) used a multiple linear regression equation of the logarithm of metabolic rate versus swimming speed and the logarithm of body weight, and then extrapolated back to zero swimming speed. Their data and extrapolations were based on several groups of different-sized fish swimming at voluntary speeds in a tank respirometer. This methodology is not equivalent to the more conventional one of estimating SMR based on swimming speed-metabolic rate curves that are constructed by forcing one fish, swimming in a tunnel respirometer, to undergo stepwise increases in swimming speed during which the fish remains for at least 1 h at each speed (Brett 1972). Furthermore, Gooding et al. (1981) expressed swimming speeds in body lengths per second. Boggs (1984) has shown that this will cause appreciable bias when fitting multiple linear regression equations because the effect of the body size on active metabolic rate is different at different swimming speeds.

The Effect of Acute Temperature Change on SMR and Heart Rate

As shown in Table 1, the Q_{10}'s (effect of temperature) for the SMR's of skipjack tuna, yellowfin tuna, and kawakawa are the same. They are also close to the Q_{10}'s for SMR's of other teleost species subjected to acute temperature change (Q_{10} = 2.16, Moffitt and Crawshaw 1983; Q_{10} = 2.10, Boehlert 1978), and for the effect of temperature on SMR where fish were acclimated to each test temperature (Q_{10} = 2.48, Ott et al. 1980; Q_{10} = 1.82-2.83, Duthie 1982).

This result was not expected since studies on the effect of temperature change on the metabolic rate of isolated red and white muscle samples (Gordon 1968, 1972a, 1972b), volitional swimming speed (Dizon et al. 1978), and preliminary work on active metabolic rate of skipjack tuna showed all three to be unaffected by temperature.

Comparing the metabolic rate (1,052 mg O_2/h, from Gooding et al. 1981) of a 2.0 kg skipjack tuna at its minimum swimming speed (1.4 body lengths/s) to its directly measured SMR (608 mg O_2/h, from Brill 1979), shows that the SMR constitutes 58% of the minimum swimming metabolic rate. Because skipjack tuna's SMR constitutes a large fraction of their metabolic rate at minimum swimming speeds and increases as temperature increases, whereas swimming metabolic rate and volitional swimming speed do not, increases in muscle efficiency (i.e., increases in thrust developed by the caudal propeller per unit of O_2 uptake), reductions in hydrodynamic drag (perhaps due to reduction in water viscosity), or unknown physiological adjustments must occur

[5]If the allometric equation to describe the effect of body size on whole body standard metabolic rate (SMR) is $\text{SMR} = aW^b$, then the corresponding equation to describe weight-specific SMR versus body weight is $\text{SMR}/W = aW^b/W$ or $\text{SMR}' = aW^{b-1}$; where SMR' = weight-specific SMR, W = body weight, and a and b are fitted parameters.

when ambient temperature increases to keep active metabolic rate temperature independent.

The effect of water temperature (20°-25°C) on heart rate was variable (Q_{10}'s ranged from 6.71 to 1.82). The mean values (±95% confidence intervals) of 3.74 (±1.9), 2.56 (±0.35), 2.14 (±0.17) for skipjack tuna, yellowfin tuna, and kawakawa, respectively, are not significantly different from each other and are close to the Q_{10} (2-3) found for the effect of temperature on the heart rate of lingcod, *Ophiodon elongatus*, (Stevens et al. 1972).

Why Are The SMR's of Tunas So High?

Also shown in Figure 1 is the SMR-body weight relationship for sockeye salmon at 20°C, taken from Brett and Glass (1973). Even with the differences in the slopes of the lines, it is still apparent that tunas have remarkably high SMR's. In the following paragraphs, I argue (as did Stevens and Neill 1978; Stevens and Dizon 1982) that tunas are "energy speculators", gambling high rates of energy expenditure against high rates of energy return. I also hypothesize that tunas' physiology and anatomy have evolved to increase maximum sustainable (i.e., aerobic) metabolic rates (MMR's) and that high SMR's are an inevitable consequence of this ability. In other words, high SMR's are a result of anatomical and physiological adaptations (primarily large gill surface areas) associated with high MMR's. Tunas have high MMR's and high SMR's, whereas sluggish bottom-dwelling flatfish (e.g., *Platichthys flesus*) have low MMR's and low SMR's (Duthie 1982). Active fish like salmon have MMR's and SMR's intermediate between these two extremes (Brett 1972).

Advantages of High Maximum Metabolic Rates

Tunas live in the open ocean, an environment which provides no shelter and where patches of forage are widely scattered (Sund et al. 1981). In this environment, high sustainable swimming speeds (i.e., high MMR's) enable tunas to travel quickly between food patches and to search large volumes of water in the least amount of time. Also, tunas have been shown to have very high rates of digestion (Magnuson 1969), which is advantageous for species that must be able to fully exploit a food patch whenever one is found. Since digestion is an energy consuming process, high rates of oxygen delivery and blood flow are required for high rates of digestion.

Because the pelagic environment provides tuna no place to hide and rest while repaying an oxygen debt, the ability to quickly metabolize lactate is also advantageous. High MMR's therefore allow tuna to rapidly repay an oxygen debt when one is accumulated. Tuna's only defense against predators such as blue marlin, *Makaira nigricans*, is presumably a burst of maximum (i.e., anaerobic) swimming. Prey capture by tunas also must involve some high speed swimming. Coulson (1979) has argued that the ability to achieve high rates of anaerobic glycolysis allows vertebrate ectotherms to successfully compete with vertebrate endotherms, which are capable of much higher rates of aerobic metabolism. However, most vertebrate ectotherms, whether terrestrial or aquatic, must spend long quiescent periods to metabolize lactate (Coulson et al. 1977). Yet tunas have the ability to metabolize some of the highest muscle lactate levels ever recorded in vertebrates in only a few hours (Barrett and Connor 1964; Hochachka et al. 1978). Other teleosts may take as long as 24 h to recover from severe exercise even though they accumulate lower white muscle lactate concentrations (Black et al. 1961; Wardle 1978). Tunas' vascular heat exchangers appear to also aid the rapid movement of lactate from the white muscle where it is produced to the red muscle where it is presumably metabolized (Stevens and Carey 1981).

Although using different terminology, McNab (1980) citing terrestrial vertebrates and Pauly (1981) citing fishes, both argue that given certain constraints, high MMR's are advantageous because rates of somatic and gonadal growth are dependent upon rates of delivery of oxygen and substrate to the tissues. Indeed, Pauly (1981) has shown that the growth rates of fishes are proportional to, and perhaps controlled by, gill surface area. Furthermore, he suggests that it is maximum rate of oxygen delivery to the tissues, rather than food supply, that limits growth rates and that species like tunas, which have the largest gill surface areas, have the highest growth rates. Koch and Wieser (1983) have shown that fish reduce activity levels during periods of gonadal growth. Tunas cannot make this trade off. For tunas, it is probably necessary to maintain a high rate of activity during gonadal synthesis which, in turn, requires respiratory and cardiovascular systems capable of delivering oxygen and metabolic substrates to the tissues at high rates.

Adaptations of Tunas For Achieving High Maximum Metabolic Rates

In a series of studies on the MMR's in land mammals (see Taylor and Weibel 1981, and the papers

that follow), pulmonary diffusing capacity, mitochondrial volume and capillary density in muscles were shown to be limiting factors in achieving high MMR's. From these studies Weibel et al. (1981) proposed that, at maximum rates of aerobic metabolism, there is no excess capacity at any level in the respiratory chain. In other words, to achieve high MMR's, a complete series of anatomical/physiological/biochemical adaptations must be present. And, as shown in Table 4, these adaptations are present in tunas.

TABLE 4.—Adaptations of tunas for high maximum metabolic rates.

Large gill surface areas	Muir and Hughes 1969
Thin secondary lamella in the gills	Muir and Brown 1971
High hematocrit, high hemoglobin levels (i.e., high blood O_2 carrying capacity)	Klawe et al. 1963; Jones et al. 1986
High maximum cardiac output	Poupa and Lindstrom 1983
Elevated muscle temperatures	Stevens and Neill 1978 Stevens 1982
High muscle myoglobin levels	George and Stevens 1978 Stevens and Carey 1981
High muscle mitochondrial density	George and Stevens 1978 Hulbert et al. 1979
High muscle capillary density	Hulbert et al. 1979
High muscle aerobic enzyme activity levels	Guppy et al. 1979

One of tunas' adaptations for high MMR's are gills with large respiratory surface areas. However, high rates of oxygen uptake are inexorably linked with high osmoregulatory costs, since gills that permit high rates of oxygen uptake must also permit high rates of water and ion movements. This is especially true in marine fishes like tunas where seawater and blood osmolality are approximately 1,000 and 400 mosm, respectively (Bourke 1983). Rao (1968), Farmer and Beamish (1969), Nordlie and Leffler (1975), and Furspan et al. (1984) estimated that the cost of osmoregulation can account for 27 to 50% of the SMR. The gills are a main osmoregulatory effector organ (Evans 1979), and Daxboeck et al. (1982) found that gill tissue respiration alone can account for 27% of the SMR in trout. The SMR, therefore, is obviously strongly influenced by osmoregulatory cost, which in turn is strongly influenced by gill surface area. Ultsch (1973, 1976) came to a similar conclusion after finding that the SMR's of aquatic (i.e., gill breathing) salamanders were controlled by respiratory (i.e., gill) surface area.

Muir and Hughes (1969) measured the total secondary lamellar gill surface (i.e., respiratory) area in skipjack tuna, yellowfin tuna, and bluefin tuna, *Thunnus thunnus*. They found total secondary lamellar areas for 1 kg tunas to be an order of magnitude or more larger than 1 kg bass or roach. Also, they found gill areas were proportional to body weight and the exponent to be 0.85 for the combined data from the three tuna species. This exponent is significantly different from the exponents I found for the effect of body weight on SMR's. It appears that in tunas, the SMR is not strictly determined by secondary lamellar surface area, although high osmoregulatory costs are most likely the main cause of tunas' high SMR's. Also, the difference between the effct of body size on SMR and gill respiratory area implies that larger tunas have greater scope of activity than smaller fishes, as has been shown to occur in other teleosts (Hughes 1984).

ACKNOWLEDGMENTS

I wish to thank Christofer H. Boggs, Peter G. Bushnell, Terry Foreman, Carol Hopper, Robert Olson, and E. Don Stevens for reviewing this paper and providing useful criticisms and suggestions; Robert E. Bourke for obtaining aholehole and rainbow trout; and David Jones for lending the respirometer for aholehole.

LITERATURE CITED

BARRETT, I., AND A. R. CONNOR.
1964. Muscle glycogen and blood lactate in yellowfin tuna, *Thunnus albacares*, and skipjack, *Katsuwonus pelamis*, following capture and tagging. Inter-Am. Trop. Tuna Comm. Bull. 9:219-268.

BEAMISH, F. W. H.
1964. Respiration of fishes with special emphasis on standard oxygen consumption. II. Influence of weight and temperature on respiration of several species. Can. J. Zool. 42: 177-188.

BEAMISH, F. W. H., AND P. S. MOOKHERJII.
1964. Respiration of fishes with special emphasis on standard oxygen consumption. I. Influence of weight and temperature on respiration of goldfish, *Carassius auratus* L. Can. J. Zool. 42:161-175.

BENNETT, A. F., AND J. A. RUBEN.
1979. Endothermy and activity in vertebrates. Science (Wash., D.C.) 206:649-654.

BLACK, E. C., A. C. ROBERTSON, AND R. R. PARKER.
1961. Some aspects of carbohydrate metabolism in fish. *In* A. W. Martin (editor), Comparative physiology of carbohydrate metabolism in heterothermic animals, p. 89-124. Univ. Washington Press, Seattle.

BOEHLERT, G. W.
1978. Changes in the oxygen consumption of prejuvenile rockfish, *Sebastes diploproa*, prior to migration from the surface to deep water. Physiol. Zool. 51:56-67.

BOGGS, C. H.
1984. Tuna bioenergetics and hydrodynamics. Ph.D. Thesis, Univ. Wisconsin-Madison, Madison, 198 p.

BOURKE, R. E.
1983. Causes of mortalities in skipjack tuna (*Katsuwonus pelamis*) following capture stress. M.S. Thesis, Univ.

Hawaii, Honolulu, 82 p.

BRETT, J. R.
1965. The relation of size to rate of oxygen consumption and sustained swimming speed of sockeye salmon (*Oncorhynchus nerka*). J. Fish. Res. Board Can. 22:1491-1501.
1972. The metabolic demand for oxygen in fish, particularly salmonids, and a comparison with other vertebrates. Respir. Physiol. 14:151-170.

BRETT, J. R., AND N. R. GLASS.
1973. Metabolic rates and critical swimming speeds of sockeye salmon (*Oncorhynchus nerka*) in relation to size and temperature. J. Fish. Res. Board Can. 30:379-387.

BRILL, R. W.
1979. The effect of body size on the standard metabolic rate of skipjack tuna, *Katsuwonus pelamis*. Fish. Bull., U.S. 77:494-498.

BRILL, R. W., AND A. E. DIZON.
1979. Red and white muscle fibre activity in swimming skipjack tuna, *Katsuwonus pelamis* (L.). J. Fish Biol. 15:679-685.

BUSHNELL, P. G., J. F. STEFFENSEN, AND K. JOHANSEN.
1984. Oxygen consumption and swimming performance in hypoxia-acclimated rainbow trout *Salmo gairdneri*. J. Exp. Biol. 113:225-235.

CAREY, F. G., AND R. J. OLSON.
1982. Sonic tracking experiments with tunas. Collective Volume of Scientific Papers, International Commission for the Conservation of Atlantic Tunas (SCRS-1981) 17:458-466.

COULSON, R. A.
1979. Anaerobic glycolysis: The Smith and Wesson of the heterotherms. Perspect. Biol. Med. 22:465-479.

COULSON, R. A., T. HERNANDEZ, AND J. D. HERBERT.
1977. Review. Metabolic rate, enzyme kinetics *in vivo*. Comp. Biochem. Physiol. 56A:251-262.

DAVIS, J. C., AND J. N. CAMERON.
1971. Water flow and gas exchange at the gills of rainbow trout, *Salmo gairdneri*. J. Exp. Biol. 54:1-18.

DAXBOECK, C., P. S. DAVIE, S. F. PERRY, AND D. J. RANDALL.
1982. Oxygen uptake in a spontaneously ventilating, blood-perfused trout preparation. J. Exp. Biol. 101:35-45.

DIZON, A. E., AND R. W. BRILL.
1979. Thermoregulation in tunas. Am. Zool. 19(1):249-265.

DIZON, A. E., R. W. BRILL, AND H. S. H. YUEN.
1978. III. Correlations between environment, physiology, and activity and the effects on thermoregulation in skipjack tuna. *In* G. D. Sharp and A. E. Dizon (editors), The physiological ecology of tunas, p. 233-259. Acad. Press, N.Y.

DUTHIE, G. G.
1982. The respiratory metabolism of temperature-adapted flatfish at rest and during swimming activity and the use of anaerobic metabolism at moderate swimming speeds. J. Exp. Biol. 97:359-373.

EVANS, D. H.
1979. Osmotic and ionic regulation by freshwater and marine fishes. *In* M. A. Ali (editor), Environmental physiology of fishes, p. 93-122. NATO Advanced Study Institute, Series A: Life Sciences 35.

FARMER, G. J., AND F. W. H. BEAMISH.
1969. Oxygen consumption of *Tilapia nilotica* in relation to swimming speed and salinity. J. Fish. Res. Board Can. 26: 2807-2821.

FRY, F. E. J.
1957. The aquatic respiration of fish. *In* M. E. Brown (editor), The physiology of fishes, Vol. 1, p. 1-63. Acad. Press, N.Y.

FURSPAN, P., H. D. PRANGE, AND L. GREENWALD.
1984. Energetics and osmoregulation in the catfish, *Ictalurus nebulosus* and *I. punctatus*. Comp. Biochem. Physiol. 77A: 773-778.

GEORGE, J. C., AND E. D. STEVENS.
1978. Fine structure and metabolism adaptation of red and white muscles in tuna. Environ. Biol. Fishes 3:185-191.

GLASS, N. R.
1969. Discussion of calculation of power function with special reference to respiratory metabolism in fish. J. Fish. Res. Board Can. 26:2643-2650.

GOODING, R. M., W. H. NEILL, AND A. E. DIZON.
1981. Respiration rates and low-oxygen tolerance limits in skipjack tuna, *Katsuwonus pelamis*. Fish. Bull., U.S. 79: 31-48.

GORDON, M. S.
1968. Oxygen consumption of red and white muscles from tuna fishes. Science (Wash., D.C.) 159:87-90.
1972a. Comparative studies on the metabolism of shallow-water and deep-sea marine fishes. I. White-muscle metabolism in shallow-water fishes. Mar. Biol. (Berl.) 15:222-237.
1972b. Comparative studies on the metabolism of shallow-water and deep-sea marine fishes. II. Red-muscle metabolism in shallow-water fishes. Mar. Biol. (Berl.) 15:246-250.

GRAHAM, J. B., AND R. M. LAURS.
1982. Metabolic rate of the albacore tuna *Thunnus alalunga*. Mar. Biol. (Berl.) 72:1-6.

GROLLMAN, A., AND E. F. GROLLMAN.
1970. Pharmacology and therapeutics. Lea & Febiger, Philadelphia, PA.

GUPPY, M., W. C. HULBERT, AND P. W. HOCHACHKA.
1979. Metabolic sources of heat and power in tuna muscles. II. Enzyme and metabolite profiles. J. Exp. Biol. 82:303-319.

HOCHACHKA, P. W., W. HULBERT, AND M. GUPPY.
1978. I. The tuna power plant and furnace. *In* G. D. Sharp and A. E. Dizon (editors), The physiological ecology of tunas, p. 153-174. Acad. Press, N.Y.

HUGHES, G. M.
1979. Morphometry of fish gas exchange organs in relation to their respiratory function. *In* M. A. Ali (editor), Environmental physiology of fishes p. 33-56. Plenum Press, N.Y.
1984. Scaling of respiratory areas in relation to oxygen consumption of vertebrates. Experientia (Basel) 40:519-524.

HULBERT, W. C., M. GUPPY, B. MURPHY, AND P. W. HOCHACHKA.
1979. Metabolic sources of heat and power in tuna muscles. I. Muscle fine structure. J. Exp. Biol. 82:289-301.

JONES, D. R., R. W. BRILL, AND D. C. MENSE.
1986. The influence of blood gas properties on gas tensions and pH of ventral and dorsal aortic blood in free-swimming tuna, *Euthynnus affinis*. J. Exp. Biol. 120:201-213.

JONES, D. R., AND T. SCHWARZFELD.
1974. The oxygen cost to the metabolism and efficiency of breathing in trout (*Salmo gairdneri*). Respir. Physiol. 21: 241-254.

KANWISHER, J., K. LAWSON, AND G. SUNDNES.
1974. Acoustic telemetry from fish. Fish. Bull., U.S. 72: 251-255.

KITCHELL, J. F., W. H. NEILL, A. E. DIZON, AND J. J. MAGNUSON.
1978. III. Bioenergetic spectra of skipjack and yellowfin tunas. *In* G. D. Sharp and A. E. Dizon (editors), The physiological ecology of tunas, p. 357-368. Acad. Press, N.Y.

KLAWE, W. L., I. BARRETT, AND B. M. H. KLAWE.
1963. Hemoglobin content of the blood of six species of scombroid fishes. Nature 198:96.

KOCH, F., AND W. WIESER.
1983. Partitioning of energy in fish: Can reduction of swimming activity compensate for the cost of production? J. Exp. Biol. 107:141-146.

MAGNUSON, J. J.
1969. Digestion of food consumption by skipjack tuna (*Katsuwonus pelamis*). Trans. Am. Fish. Soc. 98:379-392.

MCNAB, B. K.
1980. Food habits, energetics, and the population biology of mammals. Am. Nat. 116:106-124.

MOFFITT, B. P., AND L. I. CRAWSHAW.
1983. Effects of acute temperature changes on metabolism, heart rate, and ventilation frequency in carp *Cyprinus carpio* L. Physiol. Zool. 56:397-403.

MUIR, B. S., AND C. E. BROWN.
1971. Effects of blood pathway on the blood-pressure drop in fish gills, with special reference to tunas. J. Fish. Res. Board Can. 28:947-955.

MUIR, B. S., AND G. M. HUGHES.
1969. Gill dimensions for three species of tunny. J. Exp. Biol. 51:271-285.

MUIR, B. S., G. J. NELSON, AND K. W. BRIDGES.
1965. A method for measuring swimming speed in oxygen consumption studies on the aholehole *Kuhlia sandvicensis*. Trans. Am. Fish. Soc. 94:378-382.

NAKAMURA, E. L.
1972. Development and uses of facilities for studying tuna behavior. *In* H. E. Winn and B. L. Olla (editors), Behavior of marine animals. Current perspectives in research 2: Vertebrates, p. 245-277. Plenum Publ. Corp.

NIIMI, A. J.
1978. Lag adjustment between estimated and actual physiological responses conducted in flow-through systems. J. Fish. Res. Board Can. 35:1265-1269.

NORDLIE, F. G., AND C. W. LEFFLER.
1975. Ionic regulation and the energetics of osmoregulation in *Mugil cephalus* Lin. Comp. Biochem. Physiol. 51A:125-131.

OTT, M. E., N. HEISLER, AND G. R. ULTSCH.
1980. A re-evaluation of the relationship between temperature and the critical oxygen tension in fresh water fishes. Comp. Biochem. Physiol. 67A:337-340.

PAULY, D.
1981. The relationships between gill surface area and growth performance in fish: a generalization of von Bertalanffy's theory of growth. Meeresforschung 28:251-282.

POUPA, O., AND L. LINDSTROM.
1983. Comparative and scaling aspects of heart and body weights with reference to blood supply of cardiac fibers. Comp. Biochem. Physiol. 76A:413-421.

RAO, G. M. M.
1968. Oxygen-consumption of rainbow trout (*Salmo gairdneri*) in relation to activity and salinity. Can. J. Zool. 46: 781-786.

ROBINSON, W. R., R. H. PETERS, AND J. ZIMMERMANN.
1983. The effects of body size and temperature on metabolic rate of organisms. Can. J. Zool. 61:281-288.

SHARP, G. D., AND R. C. FRANCIS.
1976. An energetics model for the exploited yellowfin tuna, *Thunnus albacares*, population in the eastern Pacific Ocean. Fish. Bull., U.S. 74:36-51.

STEVENS, E. D.
1972. Some aspects of gas exchange in tuna. J. Exp. Biol. 56:809-823.
1982. The effect of temperature on facilitated oxygen diffusion and its relation to warm tuna muscle. Can. J. Zool. 60:1148-1152.

STEVENS, E. D., G. R. BENNION, D. J. RANDALL, AND G. SHELTON.
1972. Factors affecting arterial pressures and blood flow from the heart in intact, unrestrained lingcod, *Ophiodon elongatus*. Comp. Biochem. Physiol. 43A:681-695.

STEVENS, E. D., AND F. G. CAREY.
1981. One why of the warmth of warm-bodied fish. Am. J. Physiol. 240:R151-R155.

STEVENS, E. D., AND A. E. DIZON.
1982. Energetics of locomotion in warm-bodied fish. Annu. Rev. Physiol. 44:121-131.

STEVENS, E. D., AND W. H. NEILL.
1978. Body temperature relations of tunas, especially skipjack. *In* W. S. Hoar and D. J. Randall (editors), Fish physiology, Vol. VII, p. 316-360. Acad. Press, N.Y.

SUND, P. N., M. BLACKBURN, AND F. WILLIAMS.
1981. Tunas and their environment in the Pacific Ocean: A review. Oceanogr. Mar. Biol., Annu. Rev. 19:443-512.

TAYLOR, C. R., AND E. R. WEIBEL.
1981. Design of the mammalian respiratory system. I. Problem and strategy. Respir. Physiol. 44:1-10.

ULTSCH, G. R.
1973. A theoretical and experimental investigation of the relationships between metabolic rate, body size, and oxygen exchange capacity. Respir. Physiol. 18:143-160.
1976. Respiratory surface area as a factor controlling the standard rate of O_2 consumption of aquatic salamanders. Respir. Physiol. 26:357-369.

Wardle, C. S.
1978. Non-release of lactic acid from anaerobic swimming muscle of plaice *Pleuronectes platessa* L.: A stress reaction. J. Exp. Biol. 77:141-155.

WEIBEL, E. R., C. R. TRACY, P. GEHR, H. HOPPELER, O. MATHIEU, AND G. M. O. MALOIY.
1981. Design of the mammalian respiratory system. IX Functional and structural limits for oxygen flow. Respir. Physiol. 44:151-164.

WINBERG, G. G.
1956. Rate of metabolism and food requirement of fishes. [In Russ.] Nauch. Tr. Beloruss. Gos. Univ. Minsk., 253 p. (Translated, 1960, Fish. Res. Board Can. Trans. Ser. 194, 202 p.)

YONEMORI, T.
1982. Study of tuna behavior, particularly their swimming depths, by the use of sonic tags (Cho-onpa hoshiki-ho ni yoru maguro-rui no yuei suishin no kenkyu). *From* Enyo (Far Seas) Fish. Res. Lab. (Shimizu) Newsl. 44:1-5. (Translated by Tamio Otsu, 1982, 7 p., Translation No. 70; available Southwest Fish. Cent. Honolulu Lab., Natl. Mar. Fish. Serv., NOAA, Honolulu, HI 96822-2396).

ZAR, J. H.
1968. Calculation and miscalculation of the allometric equation as a model in biological data. Bioscience 18:1118-1120.