Metabolic and cardiac scope of high energy demand teleosts, the tunas

RICHARD W. BRILL
Southwest Fisheries Science Center Honolulu Laboratory, National Marine Fisheries Service, National Oceanic and Atmospheric Administration, 2570 Dole Street, Honolulu, HI 96822-2396, U.S.A.

AND

PETER G. BUSHNELL
Department of Zoology, University of British Columbia, 6270 University Boulevard, Vancouver, B.C., Canada V6T 2A9

Received June 25, 1990

BRILL, R. W., and BUSHNELL, P. G. 1991. Metabolic and cardiac scope of high energy demand teleosts, the tunas. Can. J. Zool. **69**: 2002–2009.

The maximum aerobic metabolic rates of tunas are far above those of other fishes. In this review we attempt to define features of the cardiovascular system that may account for this extreme performance. Tuna hearts are much larger than those of most other fishes and are half the mass of those of similarly sized mammals; however, the ratio of compact to spongy myocardium is not (except for one tuna species) higher than in other active teleosts. Myocardial aerobic enzyme activity levels are not significantly elevated. The cardiac outputs of spinally blocked skipjack tuna (*Katsuwonus pelamis*) and yellowfin tuna (*Thunnus albacares*) are roughly 4 times those of other active teleosts. Ventral aortic blood pressures are the highest among all fishes (more than twice those of rainbow trout (*Oncorhynchus mykiss*, formerly *Salmo gairdneri*)), and appear to be due to a high resistance to blood flow in the gills, since dorsal aortic blood pressures are not exceptional. Heart rates, cardiac output, and ventilation volume are high, but gill ventilation and perfusion appear to be well matched (i.e., gill ventilation : perfusion conductance ratios of ≈ 1). Blood hemoglobin concentrations of tunas are elevated, essentially identical with those of humans and twice those seen in other fishes. Because their blood oxygen carrying capacity is high and because skipjack and yellowfin tunas appear to be able to increase the minimum observed arterial to venous oxygen content differences approximately 3 times, we predict that they need to increase cardiac output only about 2 times during maximum rates of oxygen uptake. This ratio is not very different from those of other teleosts.

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Les taux de métabolisme aérobique maximaux des thons sont beaucoup plus élevés que ceux d'autres poissons. Dans cette synthèse, nous tentons de définir les caractéristiques du système vasculaire qui sont probablement responsables des cette performance remarquable. Les thons ont le coeur beaucoup plus gros que celui de la plupart des autres poissons, mais n'ont que la moitié de la masse de mammifères de même taille; cependant, le rapport entre la fraction compacte et la fraction spongieuse de leur myocarde n'est pas (sauf chez une espèce) plus élevée que chez d'autres téléostéens actifs. L'activité des enzymes aérobiques dans le myocarde n'est pas particulièrement élevée chez les thons. Le débit cardiaque de Thonines à ventre rayé (*Katsuwonus pelamis*) et d'Albacores à nageoires jaunes (*Thunnus albacares*) soumis à un blocage spinal est environ 4 fois plus élevé que celui d'autres téléostéens actifs. Chez les thons, la pression sanguine dans l'aorte ventrale est beaucoup plus élevée que chez tous les autres poissons (environ 3 fois plus élevée que celle de la Truite arc-en-ciel, *Oncorhynchus mykiss*, autrefois *Salmo gairdneri*) et semble attribuable à une forte résistance à la circulation dans les branchies, puisque la pression sanguine dans l'aorte dorsale n'est pas particulièrement élevée. Le rythme cardiaque, le débit cardiaque et le volume respiratoire sont élevés, mais la respiration branchiale et la perfusion semblent bien équilibrées (i.e, rapport respiration branchiale : conductance de perfusion ≈ 1). Les concentrations d'hémoglobine dans le sang des thons sont élevées, à peu près identiques à celles qui prévalent chez l'homme, et environ 2 fois plus élevées que celles des autres poissons. La capacité de transport d'oxygène du sang est élevée chez les thons et, comme la thonine et l'albacore semblent capables de tripler les différences minimales observées entre le contenu en oxygène des artères et celui des veines, nous croyons que ces poissons n'ont à augmenter leur débit cardiaque que par un facteur de 2 au cours des périodes où le taux de consommation d'oxygène est maximal. Ce rapport ne diffère pas beaucoup de celui enregistré chez d'autres téléostéens.

[Traduit par la rédaction]

Introduction

Tunas are pelagic (i.e., open ocean) fishes. In some ways, their environment is like outer space—vast and, on the human visual scale, almost empty. The one characteristic of the pelagic environment that we believe has most shaped the unique anatomy, physiology, and biochemistry of tunas is that it is a place where animals can run but cannot hide. We disagree with Carey (1973) and Graham (1975), who argue that the adaptations of tunas evolved to permit them to reach exceptionally high maximum burst swimming speeds, and with Stevens and Dizon (1982), who argue that the adaptations of tunas evolved to permit them to maintain exceptionally high (10 body lengths (BL)/s) cruising speeds. Because the pelagic environment provides no place for tunas to hide and rest while repaying an exercise- or hypoxia-incurred oxygen debt, we believe that tunas evolved the anatomical, physiological, and biochemical characteristics (Table 1) necessary to achieve exceptionally high maximum aerobic metabolic rates, to metabolize white muscle lactate as quickly as possible while maintaining relatively high (approximately 3–5 BL/s) swimming speeds (Bushnell and Brill 1991). Other advantages conferred by the ability to sustain high rates of aerobic metabolism are discussed in Brill (1987).

Although the exercise physiology of some groups of teleosts, such as salmonids, has been well investigated (Kiceniuk and Jones 1977; Jones and Randall 1978; Wood and Perry 1985), almost no data are available on the exercise physiology of tunas. Species that have been well studied are usually easily obtained, easily maintained in the laboratory, and easily induced to swim

Printed in Canada / Imprimé au Canada

TABLE 1. Adaptations of tunas for achieving high maximum aerobic metabolic rates

Adaptation	Reference
Large gill surface area	Muir and Hughes 1969
Thin secondary lamellae in the gills	Hughes 1984
High blood oxygen carrying capacity	Jones *et al.* 1986; Brill and Bushnell 1991
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

in a tunnel or flume. Tunas, on the other hand, are expensive to acquire, difficult to maintain (Queenth and Brill 1983), and currently available at only one laboratory in the world, the National Marine Fisheries Service's Kewalo Research Facility in Honolulu, Hawaii (Chang *et al.* 1983). A few studies involving tunas swimming in a tunnel (Brill and Dizon 1979; Graham and Laurs 1982; Graham *et al.* 1989) or a doughnut-shaped tank (Jones *et al.* 1986, 1990; Bushnell 1988) have been published; however, none has specifically investigated cardiovascular, respiratory, or metabolic scope. There has been some work on the ability of tunas to generate exceptional white muscle lactate levels (Guppy *et al.* 1979) and to rapidly recover from strenuous exercise (Barrett and Connor 1964; Perry *et al.* 1985; Weber *et al.* 1986), but all that is known about tuna cardiorespiratory physiology is based on data from anesthetized, paralyzed (with neuromuscular blocking agents), or spinally blocked animals, or from animals swimming at or near their minimum hydrostatic equilibrium speeds (Stevens 1972; Magnuson and Weininger 1978; Lai *et al.* 1987; Bushnell 1988; White *et al.* 1988; Bushnell and Brill 1991).

Some aspects of tuna biology, however, have been well studied (Sharp and Dizon 1978). The standard metabolic rates (i.e., metabolic rate at zero overt muscular activity) of tunas are approximately 3–5 times those of other active teleosts and about 10 times those of sluggish bottom-dwelling species (Brill 1979, 1987). Similar ratios also are found when the few data available on the maximum recorded or predicted aerobic metabolic rates of tunas (Gooding *et al.* 1981; Bushnell and Brill 1991) are compared with those of other teleosts. (As explained in Brill (1987), high maximum aerobic metabolic rates are inexorably linked to high standard metabolic rates in teleosts.) It is therefore plausible that the cardiovascular system of tunas exhibits similarly exceptional levels of function, a possibility that will be examined in this review. Finally, although some data are available on the physiology and biochemistry of marlins (*Makaira* spp. and *Tetrapturus* spp.) (Davie and Daxboeck 1984; Daxboeck and Davie 1986; Suarez *et al.* 1986), the other group of large predatory pelagic (and presumably high energy demand) fishes, no *in vivo* physiological data are available, so this group will not be included.

Cardiac anatomy and biochemistry

The tuna heart, like those of other teleosts, consists of four chambers, the sinus venosus, atrium, ventricle, and bulbus arteriosus, all contained within a fluid-filled pericardial cavity. However, the tuna heart is remarkably large compared with those of most other fish species. As a proportion of body mass, the tuna heart is approximately half the size of the hearts of birds and mammals. The relation between ventricle mass and body mass for a series of terrestrial and aquatic vertebrates is shown in Fig. 1. For all regression lines, the mass exponent is less than 1, indicating that relative ventricle size is allometric, rather than isometric, with body size in all vertebrates. Thus, comparisons of relative heart size should be made over similar body sizes or by means of analysis of covariance procedures (Packard and Boardman 1988).

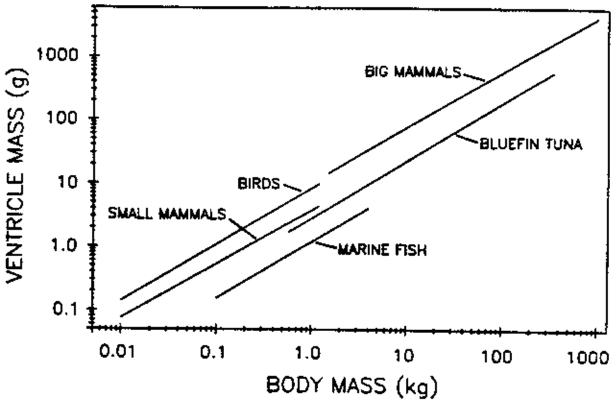

FIG. 1. Relationship betwen heart mass (g) and body mass (kg) for different groups of animals. The data for birds and mammals come from Poupa and Ostadal (1969) and for tuna and marine fishes from Poupa *et al.* (1981). The regression equations are as follows: birds, $y = 0.89 \cdot x + 93$; large mammals, $y = 0.87 \cdot x + 1.02$; small mammals, $y = 0.85 \cdot x + 0.58$; tuna, $y = 0.90 \cdot x + 0.47$; fish, $y = 0.89 \cdot x + 0.08$; where y is log (heart mass in g) and x is log (body mass in kg).

Table 2 presents more detailed data on the relative ventricle sizes of teleosts. Body sizes for all species listed are not available, but those of cod (*Gadus morhua*), sea bass (*Dicentrarcus labrax*), and sea raven (*Hemitripterus americanus*) are probably in the 0.1- to 10.0-kg range. Although these data are confounded somewhat by the fact that ventricle size may show strong seasonal (i.e, adaptation temperature) alterations (Farrell *et al.* 1988), tunas still clearly have a relative ventricle mass approximately 3 times greater than that of other active species such as striped bass (*Morone saxatilis*) and rainbow trout (*Oncorhynchus mykiss*, formerly *Salmo gairdneri*), and an order of magnitude greater than that of sluggish bottom-dwelling species such as flounder (*Pleuronectes* spp.). It appears also that all parts of the teleost heart increase in size roughly in proportion to the increase in ventricle size, because species as diverse as Pacific blue marlin (*Makaira nigricans*), flounder, and Atlantic mackerel (*Scomber scombrus*) all have ventricle masses that account for approximately 60–80% of total heart mass (Poupa *et al.* 1974; Santer *et al.* 1983; Tota 1983; Davie and Daxboeck 1984).

In teleosts the ventricle is composed either entirely of spongy (also referred to as trabecular) myocardium, or of an inner spongy myocardium surrounded by an outer compact myocardium. Of the 93 teleost species examined by Santer and Greer Walker (1980), only 20 species (21.5%) had hearts composed of both spongy and compact myocardium. The presence of compact myocardium seems to be due not to phylogenetic position but rather to mode of life. In general, active species have compact myocardium, whereas sluggish species do not.

Tuna ventricles clearly contain compact myocardium, but the

TABLE 2. Relative ventricle sizes of tunas and other teleosts

	Body size (kg)	Ventricle size (% body mass)[a]	Reference
Bluefin tuna (*Thunnus thynnus*)	0.57–350 (112)	0.312–0.164	Poupa *et al.* 1981
Yellowfin tuna (*Thunnus albacares*)	0.280–2.666 (88)	0.290±0.005	R. W. Brill and R. Bourke, unpublished data
Atlantic mackeral (*Scomber scombrus*)	≈0.1–1.2 (35)	0.167±0.005	Santer *et al.* 1983
Stribed bass (*Morone saxatilis*)	na (7)	0.124±0.004	Sidell and Driedzic 1985
Atlantic cod (*Gadus morhua*)	na (10)	0.079±0.0003	Sidell and Driedzic 1985; Hansen and Sidell 1983
Sea bass (*Dicentrarcus labrax*)	na (7)	0.077±0.003	Sidell and Driedzic 1985; Zammit and Newsholme 1979
Rainbow trout (*Oncorhynchus mykiss*)	≈0.002–1.0 (≈60)	0.08–0.13	Farrell *et al.* 1988
Sea raven (*Hemitripterus americanus*)	0.600–1.500 (7)	0.069±0.002	Sidell and Driedzic 1985; Driedzic and Stewart 1982
Flounder (*Pleuronectes platessa*)	≈0.1–1.2	0.035±0.002	Santer *et al.* 1983

NOTE: Numbers in parentheses show the number of observations. na, data not available.

[a] Mean ± SE, except for *T. thynnus* and *O. mykiss*, where the range is given.

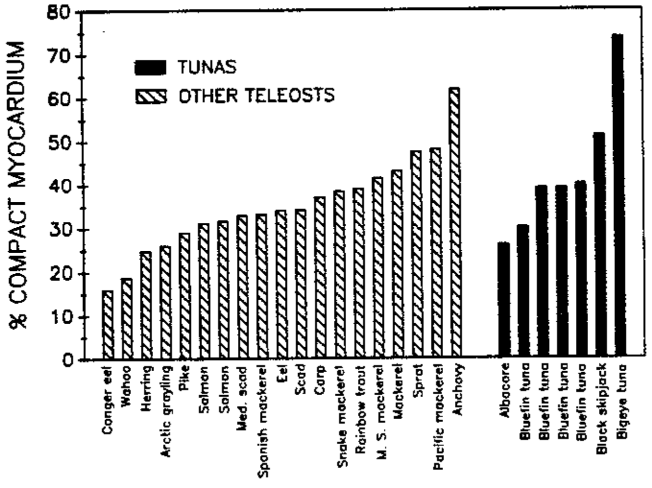

FIG. 2. The fraction of the ventricle composed of compact myocardium, based on cross-sectional areas, in various teleosts. Data are from Table 2 (p. 34) in Santer (1985). Common names given match those listed in that table. Additional data for tuna are from Tota (1978), Tota *et al.* (1983), and Sanchez-Quintana and Hurle (1987). Note that, except for bigeye tuna, the fraction of the ventricle made up of compact myocardium in tunas falls approximately mid-range of that seen in other teleosts.

percentage of compact myocardium generally does not appear to be higher in the ventricles of tunas than in the those of other teleosts. As shown in Fig. 2, when compact myocardium is measured as a proportion of the area of a transverse section of the ventricle, only in the heart of bigeye tuna (*Thunnus obesus*) is the proportion exceptionally large (74%) (Santer and Greer Walker 1980). In the ventricles of other tuna species, such as bluefin tuna (*T. thynnus*) and black skipjack tunas (*Euthynnus lineatus*), the fraction of compact myocardium (30–51%) is approximately in the middle of the range of values observed in other teleosts (Fig. 2) (Tota 1978; Santer 1985; Sanchez-Quintana and Hurle 1987). Indeed, in albacore (*T. alalunga*) the fraction of the ventricle composed of compact myocardium (26%) is at the lower end of the range observed in other teleosts. These data agree with those of Breisch *et al.* (1983), who measured the dry mass of compact myocardium in albacore and found it to be 18% of the ventricular dry mass. In rainbow trout, the dry mass of the compact myocardium composes 30–45% of the ventricular dry mass (Farrell *et al.* 1988), depending upon the age of the fish; this also agrees with the proportion of compact myocardium determined by measuring relative cross-sectional areas (38.9%, Santer and Greer-Walker 1980). Some evidence indicates that the thickness of the compct layer of the heart increases with age and size (Bass *et al.* 1973; Poupa *et al.* 1974; Farrell *et al.* 1988), and since tunas tend to be large when caught, this too argues against the supposition that tuna ventricles have a greater proportion of compact myocardium than those of other teleosts.

As described in Cimini *et al.* (1977), Santer (1985), and Sanchez-Quintana and Hurle (1987), a number of structural modifications are apparent in tuna ventricles. In some teleosts, such as rainbow trout, the compact myocardium consists of two muscle layers, an apically directed outer layer and a circular inner layer. However, Tota (1978) states that bluefin tuna have four different muscle fiber layers in their compact myocardium, although he does not describe them. For this species, Santer (1985) describes three muscle layers, with longitudinally, circularly, and transversely running fiber bundles. Sanchez-Quintana and Hurle (1987) describe the fiber orientation of tuna compact myocardium in some detail. Superficial fibers lie parallel to the ventricular borders in the lateral face and circularly in the caudal face. The inner fibers are arranged circularly around the anterior–posterior axis, and transversely in

TABLE 3. Enzyme activity profiles of hearts from skipjack tuna (*Katsuwonus pelamis*), sea raven (*Hemitripterus americanus*), ocean pout (*Macrozoarces americanus*), and rainbow trout (*Oncorhynchus mykiss*)

Enzyme	Skipjack tuna[a]	Sea raven[b]	Ocean pout[b]	Rainbow trout[c]
Citrate synthase	25.8	34.1	36.1	30.1
Malate dehydrogenase	884.2	410.9	638.1	na
Hexokinase	4.7	7.1	6.9	17.1
Phosphofructokinase	20.6	3.7	3.3	na
Pyruvate kinase	126.6	104.8	102.8	na
Lactate dehydrogenase (10 mM pyruvate)	449.0	437.5	361.4	na

NOTE: Activity levels are given in micromoles of substrate converted to product per minute per gram of tissue at 25°C. Activity levels for hearts of sea raven and ocean pout were measured at 10°C and converted to activity levels at 25°C assuming a Q_{10} of 2. Data for rainbow trout are mean activity levels from compact and spongy myocardia.

[a] From Hochachka *et al.* 1978.

[b] From Driedzic and Stewart 1982.

[c] From Farrell *et al.* 1990.

the lateral face. They are, however, transverse in the caudal face. Therefore, unlike those of other teleost ventricles, the longitudinal, transverse, and dorsal–ventral dimensions of the ventricle of tunas are all probably reduced during systole. Furthermore, since many fibers insert on the bulbo-ventricular fibrous ring, the bulbo-ventricular orifice may be opened actively (Sanchez-Quintana and Hurle 1987).

In other fishes, only the compact layer of the myocardium receives an arterial coronary vascular supply. The spongy myocardium is supplied by venous blood from the lacunary circulation in the ventricular cavity (Tota 1983, 1989). Tunas, sharks, and swordfish (*Xiphias gladius*) have well-developed arterio-luminal vessels, which connect the arterial coronary vessels to the ventricular lumen, as well as vessels localized in the subendocardium (Tota 1978, 1989; Tota *et al.* 1983). This suggests a high aerobic dependency of both layers of the myocardium. The fact that the spongy layer in adult bluefin tuna has mitochondria with a higher density of cristae and higher aerobic respiratory enzyme activities implies that it is perhaps more aerobic than the compact layer (Basile *et al.* 1976; Tota 1983). In fact, the spongy layer may expedite the metabolism of excess lactate produced by the somatic musculature after strenuous exercise (Gesser and Poupa 1973; Basile *et al.* 1976). (In rainbow trout, extracellular lactate can fuel almost the entire maximum work output of the heart (A. P. Farrell, personal communication).) Surprisingly, Modigh and Tota (1975) report that in younger bluefin tuna, mitochondria isolated from the outer layer have higher aerobic metabolic activity than those isolated from the inner layer.

The metabolic zonation of spongy and compact myocardia aside, tuna hearts are not very different biochemically from those of other fishes, even quite sluggish ones (Table 3). Malate dehydrogenase and phosphofructokinase activities are slightly elevated in skipjack tuna hearts compared with those of sea raven and ocean pout (*Macrozoarces americanus*). Even so, these differences are insignificant when compared with the differences seen in the white muscle enzyme activity levels. For example, the lactate dehydrogenase, pyruvate kinase, and phosphofructokinase activity levels in white muscle are orders of magnitude higher in skipjack tuna than in ocean pout (5492, 1[illegible] and 25.0 mM substrate converted per minute per gram at 2[illegible] (Hochachka *et al.* 1978) versus 177, 67.5, and 2.51 mM substrate converted per minute per gram at 10°C, respectively (Driedzic and Stewart 1982).) It therefore appears that the higher energy demands of the tuna heart (discussed below) are met by increased ventricle size rather than elevated enzyme activity levels (or necessarily a larger proportion of compact myocardium). This suggests that there are upper limits to enzyme activity levels in tissues required to produce constant work (i.e., to function aerobically).

Mention should also be made of the high levels of myoglobin in tuna hearts. Bluefin tuna (ca. 1–300 kg body mass) have ventricular myoglobin levels (ca. 6–20 $\mathrm{mg \cdot g^{-1}}$; Giovane *et al.* 1980) which, at their upper range, are one or two orders of magnitude higher than those of sluggish species such as sea raven (1.10 $\mathrm{mg \cdot g^{-1}}$) and ocean pout (0.08 $\mathrm{mg \cdot g^{-1}}$), and 3 times higher than those of active species such as striped bass (6.2 $\mathrm{mg \cdot g^{-1}}$; Driedzic 1983). Whether these high myoglobin levels function in the tuna heart, as they do in tuna red muscle, to increase rates of oxygen delivery to the mitochondria (Stevens 1982) or to maintain contractility in the face of low extracellular oxygen levels (Driedzic 1983, 1988) remains to be determined.

Cardiac energy output

The minimum cardiac energy output (calculated by multiplying mean cardiac output by mean ventral aortic pressure) of three species of tunas and two species of other teleosts is given in Table 4. Note that the cardiac energy output of skipjack tuna and yellowfin tuna (*Thunnus albacares*) is over 20 times higher than that of a sluggish species such as sea raven, and approximately 10 times higher than that of an active species such as rainbow trout. The higher cardiac energy outputs of these two tunas are obviously due to their higher blood pressures and higher cardiac outputs. The cardiac energy output of albacore is significantly lower, perhaps because measurements were made at 15–18°C rather than 25°C as with the other tuna species.

The minimum cardiac energy demands (calculated by dividing cardiac energy output by muscle efficiency) of three species of tunas and two species of other teleosts are also given in Table 4. Cardiac energy demand has been calculated as a fraction of the measured standard metabolic rate, assuming an oxycalorific equivalent of 3.37 $\mathrm{cal \cdot mg\ O_2^{-1}}$ (Dejours 1975) and a cardiac muscle efficiency of 20% for all species. (Note that cardiac muscle efficiency has been determined for rainbow trout (Farrell and Milligan 1986; Graham and Farrell 1990) and sea raven (Farrell *et al.* 1985) hearts and found to range from 13 to 26%. It is possible, although we believe it to be unlikely, that tuna hearts could have much higher efficiency and therefore much

TABLE 4. Cardiac output (CO), mean ventral aortic pressure (VAP), mean dorsal aortic pressure (DAP), cardiac energy output (CEO, cardiac output multiplied by mean ventral aortic pressure), and cardiac energy demand (CED, cardiac energy output divided by 20% muscle efficiency) expressed as a fraction of standard metabolic rate (SMR)

	CO ($mL \cdot min^{-1} \cdot kg^{-1}$)	VAP (mmHg)	DAP (mmHg)	CEO ($mW \cdot kg^{-1}$)	CED (% of SMR)	References
Sea raven (*Hemitripterus americanus*)	15.5	39–42	na	1.0	na	Farrell *et al.* 1982; Sidell and Driedzic 1985
Rainbow trout (*Oncorhynchus mykiss*)	17–37	39–42	31–34	1.5–2.9	2.3–4.4	Wood and Shelton 1980; Kiceniuk and Jones 1977; Bushnell *et al.* 1984
Skipjack tuna (*Katsuwonus pelamis*)	132.3	87.3	40.2	25.5	2.3–7.9	Bushnell 1988; Brill 1987
Yellowfin tuna (*Thunnus albacares*)	115.4	87.9	32.6	22.9	3.5–10.2	Bushnell 1988; Brill 1987
Albacore (*T. alalunga*)	29.4	82.3	48–80	6.6	5.7	White *et al.* 1988; Graham and Laurs 1982; Lai *et al.* 1987

NOTE: The value for cardiac muscle efficiency (20%) was based on data presented in Farrell (1985), Farrell *et al.* (1985), and Farrell and Milligan (1986). Cardiac energy demand was converted to oxygen demand on the basis of an oxycalorific equivalent of 3.37 $cal \cdot mg\ O_2^{-1}$. na, data not available.

lower cardiac energy demand. This could be a useful area of investigation.) When expressed as a fraction of the metabolic rate of the spinally blocked tunas, where cardiac output and blood pressure were measured (Bushnell 1988), the cardiac energy demands of skipjack and yellowfin tunas (2.3 and 3.5%, respectively) are no higher than those of other fishes (Farrell and Steffensen 1987). However, when expressed as a fraction of the lower metabolic rate of fishes paralyzed with a neuromuscular blocking agent, the relative cardiac energy demand of tunas is roughly double that of rainbow trout.

The elevated ventral aortic pressures seen in skipjack and yellowfin tunas are not caused by high blood viscosity due to elevated hematocrit. The hematocrits of yellowfin tuna and rainbow trout are roughly the same (28 and 23%, respectively) and that of skipjack tuna is only somewhat higher (34%) (Kiceniuk Jones 1977; Bushnell 1988). Also, elevated ventral aortic blood pressure is not caused by the blood having to traverse two relatively small sets of blood vessels, one in the gills and the other in the vascular countercurrent heat exchangers (Stevens *et al.* 1974; Graham and Diener 1978), as has been hypothesized by Tota (1978). Mean ventral aortic blood pressures as high as 75 mmHg have been observed in the bluefish (*Pomatomus saltatrix*) (Ogilvy *et al.* 1988), a species that lacks vascular countercurrent heat exchangers.

The dorsal aortic pressures of skipjack and yellowfin tunas are only 1.3–1.6 times those of rainbow trout, whereas the ventral aortic pressures are more than double (Table 4). Therefore, the relative resistance to blood flow offered by the gills of skipjack and yellowfin tunas is much higher than that offered by the gills of rainbow trout. This is surprising, given that it has been hypothesized that the short, oblique blood-flow channels, unique to the secondary lamellae of tuna gills, ensure a very low (ca. 4 mmHg) pressure drop across the gills (Muir and Brown 1971). In paralyzed or spinally blocked tunas, where dorsal and ventral aortic blood pressures have been measured, blood flow in the gills is apparently through the much longer (and therefore higher resistance) marginal blood flow channels of the secondary lamellae. Lai *et al.* (1978) have recorded ventral and dorsal aortic blood pressures in paralyzed albacore similar to those of skipjack and yellowfin tunas. However, Breisch *et al.* (1983) and White *et al.* (1988) have observed a small blood pressure drop (ca. 5 mmHg) across the gills in this species. Whether blood flow in the gills of tunas can be modified during exercise, so that gill resistance and the blood pressure drop across the gills are reduced, remains to be determined.

Metabolic and cardiac scope

Gooding *et al.* (1981) have measured aerobic metabolic rates ($\dot{V}_{O_2}$) as high as 32 $mL\ O_2 \cdot min^{-1} \cdot kg^{-1}$, in 1.8- to 2.2-kg skipjack tuna, and according to recent mathematical models of the cardiorespiratory system of skipjack and yellowfin tunas, both species should be able to reach slightly higher $\dot{V}_{O_2}$ values (Bushnell and Brill 1991). The maximum aerobic metabolic rate of skipjack tuna is therefore approximately 8–9 times higher than the standard metabolic rate. This is less than the increase of 12–15 times found in other teleosts (Wood and Perry 1985), but given that the standard metabolic rates of tunas are anywhere from 4 to 10 times those of other teleosts, the actual increases in metabolic rate of which skipjack tuna are capable are extraordinary indeed. The obvious question is, how are the cardiorespiratory systems of tunas able to accommodate these remarkable maximum $\dot{V}_{O_2}$ values?

As stated previously, there are no data on the cardiac scope (i.e., maximum cardiac outputs) of tunas, although some information on maximum heart rates is available. Heart rates in tunas swimming at low speeds (90–130 beats/min; Bushnell 1988; Bushnell and Brill 1991) are similar to those of anesthetized or spinally blocked animals (Breisch *et al.* 1983; Lai *et al.* 1987; Bushnell *et al.* 1991). Heart rates as high as 250–260 beats/min have been recorded both in swimming tunas (Kanwisher *et al.* 1974) and in paralyzed (with a neuromuscular blocking agent), artificially ventilated fish (Brill 1987). For comparison, rainbow trout (at 15°C) have a resting heart rate of 38 beats/min, which increases to only 51 beats/min at maximum sustainable swimming speed (Kiceniuk and Jones 1977).

It is also of some interest that spinally blocked skipjack and yellowfin tunas show continuous blood flow in their ventral aortas (Fig. 3) (and therefore in their gills), as do other teleosts (Jones *et al.* 1974; Farrell 1982), because of the elasticity of the bulbus arteriosus. Note, however, that although the bulbus arteriosus of yellowfin tuna is extremely elastic, it is not much more so than that of carp (*Cyprinus carpio*) or rainbow trout

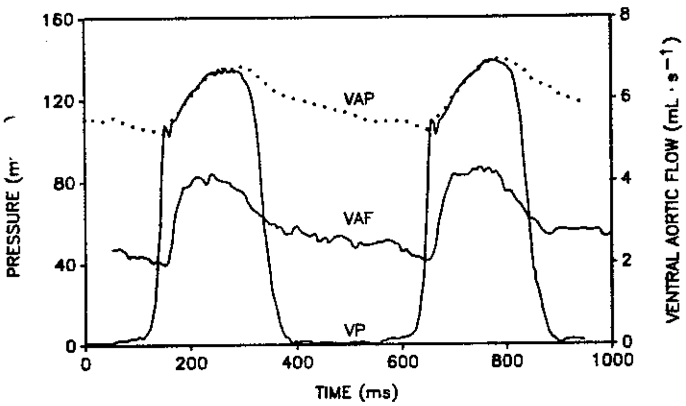

FIG. 3. Ventral aortic pressure (VAP), ventral aortic flow (VAP), and ventricular pressure (VP) recorded in spinally blocked yellowfin tuna. VAF was recorded just outside the bulbus arteriosus and was continuous throughout diastole (from Jones *et al.*[1]).

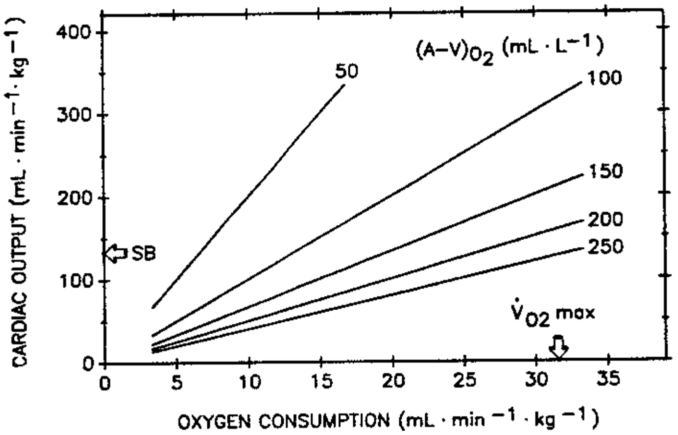

FIG. 4. Recorded and predicted values of cardiac output of skipjack tuna. The isopleths show predicted cardiac output based on various arterial to venous oxygen content differences, $(A - V)_{O_2}$. The arrow labeled "SB" indicates cardiac output of spinally blocked (i.e., nonswimming) fish (Bushnell 1988), and the arrow labeled "$\dot{V}_{O_2}$ max" indicates maximum measured oxygen consumption rate (Gooding *et al.* 1981).

when comparisons are made over the pressure ranges occurring in the ventral aorta of each species (Licht and Harris 1973; Priede 1976; Farrell 1979; Jones *et al.*[1]).

Although there are no data on the maximum ventilation volumes of tunas, the ventilation volumes measured in spinally blocked or slow-swimming (<1.9 BL/s) skipjack and yellowfin tunas range from 1.5 to 6.7 $L \cdot min^{-1} \cdot kg^{-1}$ (Bushnell 1988; Jones *et al.* 1990). These ventilation volumes far exceed those of resting rainbow trout (0.17 $L \cdot min^{-1} \cdot kg^{-1}$; Davis and Cameron 1971). However, because of the higher cardiac output and blood oxygen capacity (discussed below) of skipjack and yellowfin tunas, gill ventilation and perfusion appear well matched because ventilation:perfusion conductance ratios in spinally blocked animals are 1.09 and 0.73, respectively (Bushnell 1988). These are close to the theoretically ideal ratio of 1 (Piiper and Schied 1984).

More importantly, blood hemoglobin concentrations in skipjack and yellowfin tunas (15.0 $g \cdot dL^{-1}$) are approximately e those seen in other fishes (Bushnell 1988; Yamamoto and Itazawa 1989; Brill and Bushnell 1991) and identical with those of humans (Lloyd 1976). It appears reasonable to assume that the hematocrit of tunas could increase to nearly 50% during periods of exercise or stress, because of red cell ejection from the spleen, red cell swelling, loss of plasma volume, or, more likely, a combination of all three (Yamamoto and Itazawa 1989). Based on measured hemoglobin concentrations of tuna blood (Brill and Bushness 1991), we believe it is plausible that tunas have a *maximum* blood oxygen carrying capacity of approximately 250 $mL \cdot L^{-1}$ under these conditions. If tunas could produce a venous oxygen content of zero, meeting a metabolic oxygen demand of 30 $mL\ O_2 \cdot min^{-1} \cdot kg^{-1}$ would hypothetically require a cardiac output of 127 $mL \cdot min^{-1} \cdot kg^{-1}$, or almost no increase in the cardiac output above that seen in spinally blocked (i.e., nonswimming) fish.

Since skipjack and yellowfin tunas are unlikely to be able to produce a zero venous oxygen content, predicted cardiac output at maximum $\dot{V}_{O_2}$ then becomes a function of predicted minimum venous oxygen content (i.e., predicted maximum arterial to venous oxygen content difference, or $(A - V)_{O_2}$; Fig. 4). Although no data are available on venous oxygen content of tunas at maximum $\dot{V}_{O_2}$, a value of 80 $mL\ O_2 \cdot L^{-1}$ has been observed in spinally blocked fish during severe hypoxia (Bushnell 1988). It seems reasonable to assume that tunas can achieve an $(A - V)_{O_2}$ of at least 150–200 $mL\ O_2 \cdot L^{-1}$, or about 3–4 times the values seen in spinally blocked fish. Therefore, the maximum cardiac output required to meet maximum oxygen demand in tunas is predicted to be approximately 150–200 $mL \cdot min^{-1} \cdot kg^{-1}$, approximately 1.5–2.0 times the cardiac output seen in spinally blocked (i.e., "resting") animals (Fig. 4). These values are close to the roughly 3-fold increase (above resting) in cardiac output seen in rainbow trout during maximum exercise (Kiceniuk and Jones 1977).

In summary, the high blood oxygen carrying capacity of tunas appears to enable these fishes to achieve remarkably high maximum $\dot{V}_{O_2}$ values without the need for undue increases in cardiac output (i.e., unreasonably large cardiac scope). Measurements of cardiorespiratory function and blood gas values of tunas at various exercise levels are obviously needed to test this hypothesis.

Acknowledgements

The authors thank David R. Jones, Anthony P. Farrell, and Steffan Nilsson for reviewing the manuscript, providing helpful comments and suggestions, bringing several key papers to our attention, and granting permission to use unpublished data. The authors also gratefully acknowledge the editorial help of Leslie C. Williams.

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