EFFECTS OF EXHAUSTING EXERCISE ON ACID-BASE REGULATION IN SKIPJACK TUNA (KATSUWONUS PELAMIS) BLOOD[1]

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The effects of exhausting exercise on acid-base balance of skipjack tuna blood were investigated. Following exercise, tuna displayed a mixed respiratory/metabolic acidosis with blood pH being reduced by ~0.4 units. The respiratory component (51% of the initial acidosis) was compensated following 20 min of recovery, while the blood metabolic acid load (H^+m; ~8 mM) was cleared after only 50 min. At that time, there was a great discrepancy between blood lactate load and H^+m load because blood lactate levels were still increasing. The significance of these results is discussed with reference to the tuna's habitat, behavior, and physiology.

INTRODUCTION

Skipjack tuna (*Katsuwonus pelamis*) have a remarkable capacity to maintain high cruising speeds for long periods of time. The estimated maximum sustainable speed for this species is 6–10 body lengths/s (Yuen 1970; Dizon, Brill, and Yuen 1978). While the sustainable velocities greatly exceed those of most other fish examined, the maximum swimming speeds attainable by skipjack tuna (15–20 body lengths/s; Brill and Dizon [1979]) do not differ greatly from those of other active teleosts (e.g., *Salmo gairdneri*, 15 body lengths/s [Webb 1971; Mosse 1979; Johnston 1982]). Similarly, the amount of time it takes to reach exhaustion during burst activity also does not differ greatly from those of other active fish. However, there are reasons to believe that tuna may be better adapted for recovery from burst-swimming activity than are other teleosts: tuna exhibit relatively rapid lactate clearance (1–2 h; [Barrett and Connor 1964]) compared to the 8–12 h or even longer period seen in other teleosts (e.g., 24 h for flounder [Wood, McMahon, and McDonald 1977] and >12 h for rainbow trout [Turner, Wood, and Clark 1983*a*]). Rapid acid-base recovery following burst swimming clearly would be advantageous to skipjack tuna, considering that they inhabit the open ocean (an environment that provides little shelter) and that high swimming speeds are a skipjack tuna's most potent defense against predators.

In the present study we have investigated blood acid-base changes in skipjack tuna blood following exhausting exercise. In these experiments we were interested in determining the respiratory and metabolic components of the acid-base disturbance following burst swimming and the method and time course of recovery. Particularly, our interest was in determining if protons and lactate formed in equimolar amounts by muscular anaerobic metabolism (Hochachka and Mommsen 1983) are cleared at the same rate or whether a discrepancy in proton/lactate loads develops, as has been observed in other fish species (Piiper et al. 1972; Turner et al. 1983*a*; Turner, Wood, and Hobe 1983*b*).

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MATERIAL AND METHODS

Skipjack tuna (*Katsuwonus pelamis*) were captured on hook and line by local fishermen and transported to Kewalo Research Facility (National Marine Fisheries Service, Southwest Fisheries Centre, Honolulu Laboratory), where all subsequent experiments were performed. Fish of either sex, weighing 1.2–2.3 kg (mean ± SE weight = 1.7 kg ± 0.2; n = 10) were maintained outdoors in large circular holding tanks and supplied with rapidly flowing seawater (temperature = 25 C). Animals were used within the first 3 days of capture and were not fed while in captivity.

EXPERIMENTAL PROTOCOL

Blood buffering capacity.—In order to sample blood, a fish was netted and quickly injected intramuscularly with 2.4–4.8 mg/kg body weight of the neuromuscular blocking agent gallamine triethiodide (Flaxedil; 20 mg/ml). On cessation of swimming, the fish was transferred to an operating table and submerged in water, and a tube that allowed continuous irrigation of the gills with aerated seawater (10–12 liters/min) was inserted into the mouth. The fish was surrounded by a tent of opaque plastic to eliminate visual disturbances. One milliliter of sodium heparin (10,000 U.S.P. units) was injected into the ventral aorta, and, following a 5-min mixing period, as much blood as possible (usually 30–50 ml) was withdrawn via "blind" ventral aortic or cardiac puncture.

To determine nonbicarbonate blood buffering value (β) and to construct CO_2 combining curves at various hematocrit (HCT) levels, blood from four fish was centrifuged and plasma removed. Erythrocytes were resuspended in plasma to give a range of HCTs of 0%–60%. Blood samples were placed into 50-ml tonometer shaker flasks (5 ml/flask) that were suspended in a constant-temperature (25 C) bath. Blood was equilibrated with humidified gas mixtures of CO_2 diluted with air (using flowmeters) to produce a range of P_{CO_2} ~0 to ~15 torr. Following a 45-min equilibration, 1-ml samples of blood or plasma were analyzed for pH, total CO_2 content (C_{CO_2}), and HCT. The pH measurements were made with a Radiometer PHM-71 digital acid-base analyzer and associated "micro" pH electrode at 25 C. C_{CO_2} was determined according to the method of Cameron (1971). P_{CO_2} and bicarbonate concentration [HCO_3^-] were calculated using a reorganization of the Henderson-Hasselbalch equation. The pK′ values of carbonic acid were obtained from Severinghaus, Stupfel, and Bradley (1956), and the solubility coefficients of CO_2 (αCO_2) were obtained from Albers (1970). Separate buffer curves for each HCT (n = 18) were plotted and β values ($dHCO_3^-/dpH$) determined. Finally, a curve relating buffering capacity to HCT was constructed using linear regression.

Effects of exhausting exercise.—Individual fish were exercised to exhaustion by chasing them around their holding tank, usually for periods of 5–10 min. Earlier studies (Hochachka, Hulbert, and Guppy 1978) have established that under such conditions skipjack tuna reach burst-swimming speeds of 20 body lengths/s and that white-muscle lactate concentration, [La^-], rises to extremely high levels. Following this exercise period, the fish was injected with Flaxedil (2.4–4.8 mg/kg body weight), transferred to an operating table, and maintained as described above. As quickly as possible (usually within 1 min of placement of the fish on the operating table) a 1-ml blood sample was taken from the ventral aorta and analyzed for pH, C_{CO_2}, [La^-], and HCT. The ventral aorta then was cannulated using an indwelling catheter, a procedure that took ~20 min. Additional blood samples were drawn immediately following cannulation and then at 10-min intervals for a further 30 min, at which time experiments were terminated and the fish sacrificed. Once again, blood was analyzed for pH, C_{CO_2}, [La^-], and HCT. Lactate levels were determined enzymatically according to the method of Hochachka et al. (1978). The concentration of metabolic H^+ ions added to the blood over any time period, [ΔH_m^+], was calculated according to the following equation (McDonald, Boutilier, and Toews 1980):

$$[\Delta H_m^+] = [HCO_3^-]_1 - [HCO_3^-]_2 - \beta(pH_1 - pH_2), \quad (1)$$

where β equals the nonbicarbonate buffering capacity of whole blood as determined in vitro. Blood metabolic H^+ load (H_m^+) was determined at any given time by summing the $[\Delta H_m^+]$'s, signs considered, from the preexercise sample onward. Blood lactate load was determined in a similar fashion.

Blood acid-base values for nonexercised (control) fish were obtained in a similar manner as were those for exercised fish, but care was taken to avoid stressing the animal prior to Flaxedil injection.

In the figures, variability of the data is indicated by ±1 SE. Sample means have been statistically analyzed using Student's *t*-test, and 5% was taken as the fiducial limit of significance.

RESULTS

CO_2 combining curves for separated plasma and whole blood at various HCTs are shown in figure 1A. The upward displacement of the curves with decreasing HCT indicates that the major fraction of total CO_2 in skipjack blood is carried in the plasma and not in erythrocytes. Similarly, the positive relationship between HCT and slope of the CO_2 combining curves reflects the greater buffering capacity as hemoglobin levels increase. Figure 1B illustrates the relationship between HCT

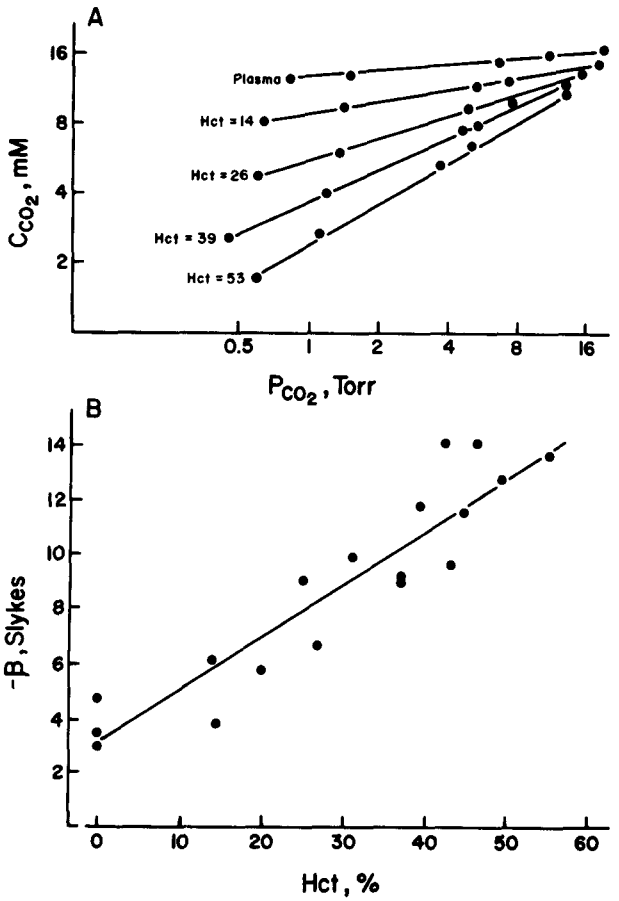

FIG. 1.—(*A*) CO_2 combining curves (log P_{CO_2} vs. log C_{CO_2}) for skipjack tuna blood of various hematocrit (HCT) values (n = 4, same fish for each curve) and (*B*) the relationship between nonbicarbonate buffering value (β) and HCT (18 determinations from blood of four fish). The regression equation is $-\beta = 0.196$ HCT + 3.11, $r = 0.93$.

TABLE 1

NONBICARBONATE BUFFERING CAPACITY (β) OF SKIPJACK TUNA BLOOD COMPARED WITH THAT OF VARIOUS OTHER FISH SPECIES

Species	$-\beta$ (slyke)	HCT (%)	Source
Katsuwonus pelamis	11.2	41	This study
Salmo gairdneri	10.3	26	Wood and Jackson (1980)
S. gairdneri	9.7	25	Wood, McDonald, and McMahon (1982)
S. gairdneri	8.5	. . .	Eddy (1976)
Salvelinus fontinalis	7.5	35	Packer and Sunkin (1979)
Catostomus commersoni	8.5	28	Wilkes et al. (1981)
Ictalurus punctatus	14.3	25	Cameron and Kormanik (1982)
Platichthys stellatus	5.2	16	Wood et al. (1977)
Hippoglossoides elassodon	6.7	14	Turner et al. (1983*b*)
Parophrys vetulus	6.1	25	McDonald et al. (1982)
Protopterus aethiopicus (water-breathing)	12.6	. . .	DeLaney et al. (1977)

and β and is given by the regression equation $-\beta = 0.196\,(\text{HCT}) + 3.11$. From in vivo determinations, HCT was 41% ± 2.2% ($n = 7$), giving a mean β of -11.2 slykes ($dHCO_3^-/dpH$; table 1). Plasma β is equal to the y-intercept of the regression line and is -3.11 slykes.

The effects of exhausting exercise on the acid-base status of tuna blood are shown in figure 2. After ~5 to ~10 min of forced burst-swimming activity, blood pH was greatly depressed from a preexercise value of 7.97 to a postexercise value of 7.54 (fig. 2A). The pH recovery following exercise was extremely rapid, taking only 30 min. When experiments were terminated, after 50 min of recovery, blood pH appeared to be greater than the preexercise value. This may indicate a slight acidosis in nonexercised (control) fish, probably owing to the stress of netting and Flaxedil injection. Alternatively, the higher pH following recovery from exercise may have been a result of overcompensation.

P_{CO_2} was greatly elevated following exercise (fig. 2D), but after 20 min of recovery P_{CO_2} had returned to the preexercise level. P_{CO_2} remained constant for the next 30 min, although at a lower level than the preexercise value, possibly indicating a condition of mild respiratory acidosis in nonexercised animals.

The changes in C_{CO_2} after exercise are shown in figure 2B. C_{CO_2} was lowered immediately postexercise and continued to decline for the initial 20 min of recovery. C_{CO_2} then increased during the next 30 min and was still rising on termination of the experiment.

Blood lactate concentration was significantly elevated following burst swimming and remained elevated throughout the postexercise period (fig. 2C). The preexercise blood $[La^-]$ is seemingly high (9.2 mM ± 2.4) when compared with blood $[La^-]$ of free-swimming skipjack tuna (4.7 mM; Hochachka et al. [1978]).

A pH-$[HCO_3^-]$ diagram displaying the temporal changes in blood acid-base status following exhausting exercise in skipjack tuna is illustrated in figure 3. The dashed line, AB_1, represents the in vitro buffer line (slope = -11.2 slykes). It is clear that the postexercise acidosis is both respiratory and metabolic in origin. Using an approach outlined in detail by Wood et al. (1977), we evaluated the relative contributions of alterations of P_{CO_2} and lactic acid to the total pH change observed. Immediately postexercise (fig. 3, point *B*) the metabolic component of the acidosis equaled 49% and the respiratory component of the total acidosis equaled 51%. After 20 min the respiratory acidosis was completely compensated, whereas blood H_m^+ load (see eq. [1]) remained elevated until 50 min postexercise, at which time there was a slight base excess (fig. 3). The changes in blood $[H_m^+]$ and $[La^-]$ during recovery from exercise are shown in figure 4. By definition, $\Delta[H_m^+]$ and $\Delta[La^-]$ in fish

at rest were equal to 0. Immediately postexercise, La^- load was significantly greater than H_m^+ load. La^- load continued to increase throughout the recovery period, while H_m^+ load declined gradually and was actually below the preexercise value at 50 min postexercise. The discrepancy between $\Delta[La^-]$ and $\Delta[H_m^+]$ could be caused by preferential removal of protons from the blood space and/or by a slower release of H^+ ions (with respect to La^-) from white muscle.

DISCUSSION

In the present study, a somewhat unorthodox method for blood sampling was employed. Clearly, this procedure did not allow us to measure true resting blood acid-base values or enable an analysis of blood acid-base changes in freely swimming animals following the forced exercise. Unfortunately, because of the fragile nature of skipjack tuna, all our attempts to anesthetize, catheterize, and recover tuna resulted in failure. Until very recently such a procedure was considered impossible by most tuna researchers. However, since completion of this study, successful recoveries of skipjack tuna from dorsal aortic catheterization have been reported (D. R. Jones, personal communication); however, 12 h was the longest that any fish survived following surgery and generally fish began to deteriorate after 3–4 h (D. R. Jones, personal communication), so it is also unlikely that true resting acid-base values can be obtained in this manner. Thus, it is apparent that until new surgical proce-

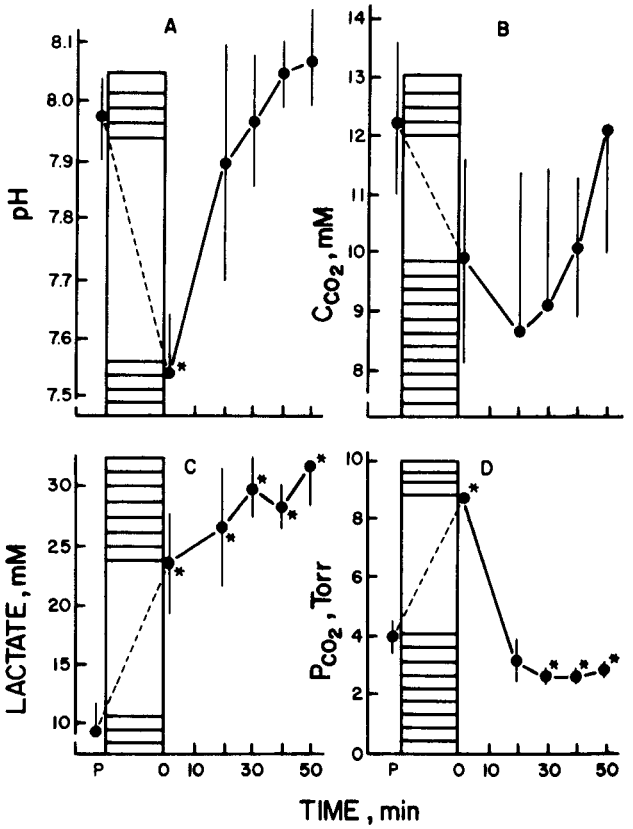

FIG. 2.—In vivo blood acid-base status of skipjack tuna at 25 C before and following exhausting exercise (burst swimming). Two separate groups of fish were used to determine preexercise (*P*) and postexercise values ($n = 3$ in both cases). Exercise period is represented by the striped bar; an asterisk indicates a significant difference from preexercise value.

dures are developed it will not be possible to use routine methodology to study blood acid-base status of tuna before and following exercise. It should be pointed out, however, that the methodology employed in the present study allowed arterial oxygen tension to be maintained at 70–80 torr, which is similar to values reported for freely swimming tuna (D. R. Jones, personal communication). Therefore, it would appear that forced ventilation does not limit gill O_2 transfer. Moreover, nonexercised tuna did not exhibit a marked blood acidosis (pH = 7.97), a condition normally associated with stressed fish.

The β of skipjack tuna blood (−11.2 slykes) is among the highest of all fish species examined (table 1). That the β is not greater is surprising, considering the high HCT (~40%) and hemoglobin concentration (14–20 g/100 ml; Klawe, Barrett, and Klawe [1963]) in this species. It is apparent, however, that hemoglobin is the major blood buffer (fig. 1). It also is clear from the negative relationship between CO_2 combining capacity and HCT in vitro that the major fraction of total CO_2 in the blood resides in the plasma, not in the erythrocytes. Similar results have been reported for trout (Eddy 1974) and flounder (Wood et al. 1977).

The changes in blood acid-base status immediately following strenuous exercise in skipjack tuna were similar to those reported for other active fish (e.g., *Scyliorhinus stellaris* [Piiper, Meyer, and Drees 1972]; *Salmo gairdneri* [Turner et al. 1983*a*; Wood, Turner, and Graham1983]). The large initial decrease in pH (~0.4 units) was a result of equal respiratory and

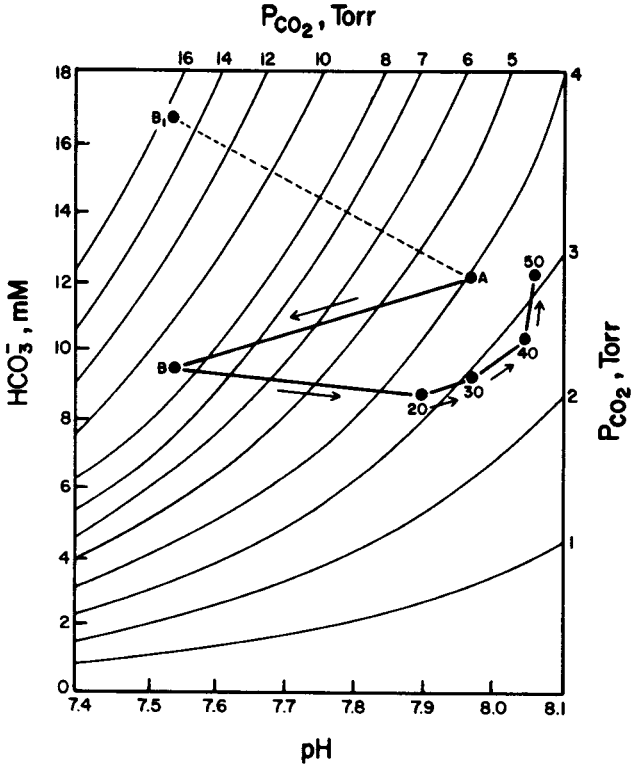

FIG. 3.—pH-[HCO_3^-] diagram showing temporal changes in blood acid-base status following exhausting exercise in skipjack tuna at 25 C (n = 3). A = the preexercise value from a separate group of fish (n = 3). B = the value immediately following exercise (usually within 1 min). Times (in min) of later blood samples are indicated at each successive data point. The dashed line AB_1 represents the in vitro buffer curve. See text for further details.

metabolic contributions. The respiratory component of the acidosis was compensated quickly, and following 20 min of recovery only the metabolic component remained. The increase in P_{CO_2} of venous blood during exercise probably is caused by greater aerobic metabolism as well as buffering of metabolic protons by HCO_3^-.

The ability of paralyzed skipjack tuna to recover from exhausting exercise is remarkable; after only 30 min blood pH was restored to preexercise levels, while H_m^+ load was cleared after only 50 min. Clearly, this is the most rapid metabolic acid clearance following burst-swimming activity ever reported for a fish. Normally, metabolic acid clearance in freely swimming fish requires ≥8 h (see review by Jones and Randall [1978]). The metabolic acid load immediately postexercise (~8 mM) was similar to values reported for rainbow trout (Turner et al. 1983*a*; Wood et al. 1983). The relatively low metabolic acid load is surprising considering that the tuna white-muscle contribution to burst swimming is supported almost entirely by the most intense glycolysis thus far known in nature (Hochachka et al. 1978). Typical values for white muscle [La^-] following burst swimming are ~100 μmol/g in skipjack tuna (Hochachka et al. 1978) versus ~40 μmol/g in rainbow trout (Turner et al. 1983*a*). The difference in white-muscle lactate levels between tuna and trout probably accounts for the much higher blood lactate concentration in skipjack tuna following exercise. As has been shown for dogfish and rainbow trout (Piiper et al. 1972; Turner et al. 1983*a*), blood lactate levels in skipjack tuna exceed blood metabolic acid levels during the recovery phase, which is exactly opposite to the situation observed in sluggish species (e.g., starry flounder [Wood et al. 1977]; flathead sole [Turner et al. 1983*b*]). However, the magnitude of the discrepancy between

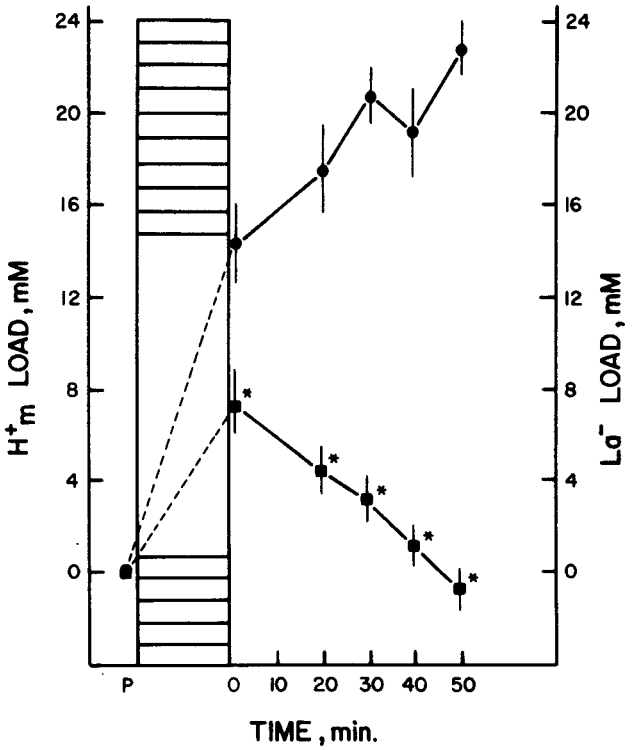

FIG. 4.—Changes in blood metabolic acid load (H_m^+ load; ■ —— ■) and lactate load (La^- load; ● —— ●) following exhausting exercise in skipjack tuna (n = 3). P = preexercise; an asterisk indicates a significant difference between H_m^+ load and La^- load at the same sample time.

[La^-] and [H^+_m] in skipjack tuna is far greater than other studies have shown (Piiper et al. 1972; Turner et al. 1983*a*; Wood et al. 1983). Since lactate ions and H^+ ions are produced in stoichiometrically equal amounts (Hochachka and Mommsen 1983), this discrepancy must be due to differential release of La^- and H^+ ions from white muscle and/or their differential removal from blood. Although it is difficult to differentiate between these two possibilities, our results do indicate that skipjack tuna are able to remove H^+ ions from the blood at a much faster rate than are other fish (50 min for tuna compared to 8–12 h for rainbow trout [Turner et al. 1983*a*]). Whether the metabolic acid is being excreted across the gills into the water (see Heisler 1980) or being translocated to another tissue (e.g., red muscle) is unclear. However, the large blood volume, cardiac output, and gill surface area in skipjack tuna (Muir 1969; Laars, Ulevitch, and Morrison 1978) are all factors that could enhance acid excretion. Nevertheless, we cannot ignore the possibility that the rapid acid-base regulation following exercise was in some way due to muscle paralysis, although we consider this unlikely. Indeed, muscular movements are thought to enhance local blood flow (Randall and Daxboeck 1982) and therefore would, if anything, probably enhance acid clearance. Hochachka et al. (personal communication) have proposed that the major site of lactate oxidation is red muscle. Thus, movement of H^+ ions from white muscle to red muscle could be important for the ultimate remetabolism of lactate. In trout, the discrepancy between [La^-] and [H^+_m] is believed to be due to differential release from white muscle (Turner et al. 1983*a*; Turner and Wood 1983). Studies using a perfused trunk preparation (Turner and Wood 1983) have shown that H^+ ions are retained in white muscle as a result of a continuing blood acidosis that inhibits proton efflux; under resting conditions, La^- and H^+ ions are released at similar rates. This would suggest that in skipjack tuna metabolic acid is not retained in white muscle, since blood pH is restored to resting levels in 30 min. It seems more likely that H^+ ions formed during anaerobic metabolism are eliminated rapidly from white muscle and removed from the blood by excretion and/or translocation. The fact that skipjack white muscle is well vascularized compared to that of other teleosts (Hulbert et al. 1979) supports this theory. A slower release of lactate ions from white muscle and/or their slower removal from the blood would explain the discrepancy between blood levels of these two ions following exercise. Clearly, measurements of acid excretion as well as intracellular pH of white and red muscle following burst activity and lactic acid infusion would help clarify this problem.

LITERATURE CITED

ALBERS, C. 1970. Acid-base balance. Pages 173–208 *in* W. S. HOAR and D. J. RANDALL, eds. Fish physiology. Vol. **4**. Academic Press, New York.

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

BRILL, R. W., and A. E. DIZON. 1979. Effect of temperature on isotonic twitch of isolated muscle and predicted maximum speeds of skipjack tuna. Environ. Biol. Fishes **4**:199–205.

CAMERON, J. N. 1971. Rapid method for determination of total carbon dioxide in small blood samples. J. Appl. Physiol. **31**:532–534.

CAMERON, J. N., and G. A. KORMANIK. 1982. The acid-base responses of gills and kidneys to infused acid and base loads in the channel catfish, *Ictalurus punctatus*. J. Exp. Biol. **99**:143–160.

DELANEY, R. G., G. LAHIRI, R. HAMILTON, and A. P. FISHMAN. 1977. Acid-base balance and plasma composition in the aestivating lungfish (*Protopterus*). Am. J. Physiol. **232**:10–17.

DIZON, A. E., R. W. BRILL, and H. S. H. YUEN. 1978. Correlations between environment, physiology and activity and its effects on thermoregulation in skipjack tuna, *Katsuwonus pelamis*. Pages 233–259 *in* G. D. SHARP and A. E. DIZON, eds. The physiological ecology of tunas. Academic Press, New York.

EDDY, F. B. 1974. *In vitro* blood carbon dioxide of the rainbow trout, *Salmo gairdneri*. Comp. Biochem. Physiol. **47A**:129–140.

———. 1976. Acid-base balance in rainbow trout (*Salmo gairdneri*) subjected to acid stresses. J. Exp. Biol. **64**:159–171.

HEISLER, N. 1980. Regulation of the acid-base status in fishes. Pages 123–162 *in* M. A. ALI, ed. Environmental physiology of fishes. Ser. A, vol. **35**. Nato Advanced Study Institute Series. Plenum, New York.

HOCHACHKA, P. W., W. C. HULBERT, and M. GUPPY.

1978. The tuna power plant and furnace. Pages 153–174 *in* G. D. SHARP and A. E. DIZON, eds. The physiological ecology of tunas. Academic Press, New York.

HOCHACHKA, P. W., and T. P. MOMMSEN. 1983. Protons and anaerobiosis. Science **219**:1391–1397.

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.

JOHNSTON, I. A. 1982. Physiology of muscle in hatchery raised fish. Comp. Biochem. Physiol. **13B**:105–124.

JONES, D. R., and D. J. RANDALL. 1978. The respiratory and circulatory systems during exercise. Pages 425–501 *in* W. S. HOAR and D. J. RANDALL, eds. Fish physiology. Vol. 7. Academic Press, New York.

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

LAARS, R. M., R. J. ULEVITCH, and D. C. MORRISON. 1978. Estimates of blood volume in the albacore tuna. Pages 135–139 *in* G. D. SHARP and A. E. DIZON, eds. The physiological ecology of tunas. Academic Press, New York.

MCDONALD, D. G., R. G. BOUTILIER, and D. P. TOEWS. 1980. The effects of enforced activity on ventilation, circulation and blood acid-base balance in the semi-terrestrial anuran, *Bufo marinus*. J. Exp. Biol. **84**:273–287.

MCDONALD, D. G., R. L. WALKER, P. R. H. WILKES, and C. M. WOOD. 1982. H^+ excretion in the marine teleost, *Parophrys vetulus*. J. Exp. Biol. **98**:403–414.

MOSSE, P. R. L. 1979. Capillary distribution and metabolic histochemistry of the lateral propulsive musculature of pelagic teleost fish. Cell Tissue Res. **203**:141–160.

MUIR, B. S. 1969. Gill dimensions as a function of fish size. J. Fisheries Res. Board Can. **26**(1):165–170.

PACKER, R. K., and A. L. SUNKIN. 1979. Blood acid-base balance in brook trout (*Salvelinus fontinalis*). J. Exp. Biol. **79**:115–126.

PIIPER, J., M. MEYER, and F. DREES. 1972. Hydrogen ion balance in the elasmobranch, *Scyliorhinus stellaris*, following exhausting activity. Respir. Physiol. **16**:290–303.

RANDALL, D. J., and C. DAXBOECK. 1982. Cardiovascular changes in the rainbow trout (*Salmo gairdneri*) during exercise. Can. J. Zool. **69**:1135–1142.

SEVERINGHAUS, J. W., M. STUPFEL, and A. F. BRADLEY. 1956. Variations of serum carbonic acid pK′ with pH and temperature. J. Appl. Physiol. **9**: 197–200.

TURNER, J. D., and C. M. WOOD. 1983. Factors affecting lactate and proton efflux from pre-exercised, isolated, perfused rainbow trout trunks. J. Exp. Biol. **105**:395–401.

TURNER, J. D., C. M. WOOD, and D. CLARK. 1983*a*. Lactate and proton dynamics in the rainbow trout (*Salmo gairdneri*). J. Exp. Biol. **104**:247–268.

TURNER, J. D., C. M. WOOD, and H. HOBE. 1983*b*. Physiological consequences of severe exercise in the inactive benthic flathead sole (*Hippoglossoides elassodon*): a comparison with the active pelagic rainbow trout (*Salmo gairdneri*). J. Exp. Biol. **104**:269–288.

WEBB, P. W. 1971. The swimming energetics of trout. I. Thrust and power output at cruising speeds. J. Exp. Biol. **55**:489–520.

WILKES, P. R. H., R. L. WALKER, D. G. MCDONALD, and C. M. WOOD. 1981. Respiratory, ventilatory, acid-base and ionoregulatory physiology of the white sucker, *Catostomus commersoni*: the influence of hyperoxia. J. Exp. Biol. **91**:239–254.

WOOD, C. M., and E. B. JACKSON. 1980. Blood acid-base regulation during environmental hyperoxia in the rainbow trout. Respir. Physiol. **42**:351–372.

WOOD, C. M., D. G. MCDONALD, and B. R. MCMAHON. 1982. The influence of experimental anaemia on blood acid-base regulation in vivo and in vitro in the starry flounder (*Platichthys stellatus*) and the rainbow trout (*Salmo gairdneri*). J. Exp. Biol. **96**:221–237.

WOOD, C. M., B. R. MCMAHON, and D. G. MCDONALD. 1977. An analysis of changes in blood pH following exhausting activity in the starry flounder, *Platichthys stellatus*. J. Exp. Biol. **69**:173–185.

WOOD, C. M., J. D. TURNER, and M. S. GRAHAM. 1983. Why do fish die after severe exercise? J. Fish Biol. **22**:189–201.

YUEN, H. S. H. 1970. Behavior of skipjack tuna, *Katsuwonus pelamis*, as determined by tracking with ultrasonic devices. J. Fisheries Res. Board Can. **27**:2071–2079.