J. exp. Biol. ***179****, 115–129 (1993)*
Printed in Great Britain © The Company of Biologists Limited 1993

INTESTINAL BLOOD FLOW IN SWIMMING CHINOOK SALMON *ONCORHYNCHUS TSHAWYTSCHA* AND THE EFFECTS OF HAEMATOCRIT ON BLOOD FLOW DISTRIBUTION

H. THORARENSEN[1], P. E. GALLAUGHER[1], A. K. KIESSLING[2], and A. P. FARRELL[1]

[1]*Simon Fraser University, Department of Biological Sciences, Burnaby, British Columbia, Canada V5A 1S6* and [2]*Department of Fisheries and Oceans, Biological Sciences Branch, Biotechnology, Genetics and Nutrition Section, West Vancouver Laboratory, 416 Marine Drive, West Vancouver, British Columbia, Canada V7V 1N6*

Accepted 19 February 1993

Summary

Blood flow in the intestinal artery (q_{IA}), the rate of oxygen consumption ($\dot{V}_{O_2}$) and a number of haematological variables were measured in chinook salmon, *Oncorhynchus tshawytscha*, while they swam up to the critical swimming velocity (U_{crit}). The fish used in this study had previously been exposed to one of two different exercise-training regimes, swimming for 8 months at either $1.5 bl\,s^{-1}$ (HS) or $0.5 bl\,s^{-1}$ (LS) (where bl is body length). During this period, growth rate was the same in both groups.

At rest, q_{IA} was approximately 36% of cardiac output. q_{IA} was inversely related to $\dot{V}_{O_2}$, indicating that blood flow was gradually redistributed from the viscera as the oxygen demands of the locomotory muscles increased. Both $\dot{V}_{O_2}$ and q_{IA} were relatively constant at swimming velocities less than 50% U_{crit}, but at U_{crit}, q_{IA} had decreased by 60–70% as $\dot{V}_{O_2}$ reached a maximum. Blood flow redistribution away from the intestine contributed significantly to the oxygen supply for locomotory muscles, since it was estimated that the oxygen-transporting capacity of this redistributed blood flow was enough to support 12–18% of the maximum internal oxygen consumption (total $\dot{V}_{O_2}$ − gill $\dot{V}_{O_2}$).

Following exercise training, haematocrit (Hct) in the HS group (27.1%) was significantly higher than in the LS group (23.3%). However, neither the maximum $\dot{V}_{O_2}$ nor U_{crit} was significantly different in the two groups. q_{IA} was inversely related to Hct but, in spite of lower q_{IA} at rest, oxygen transport to the intestines was greater at all swimming speeds in the HS than in the LS training group. In addition, blood flow in the HS group was better maintained as the swimming speed was increased. As a result of the higher Hct in the HS-trained group, oxygen transport to the intestines was similar in both groups at their respective training velocities. Therefore, we suggest that, by increasing Hct and thereby maintaining oxygen delivery to the intestines, the HS group maintained normal intestinal function while swimming at the higher velocity, enabling overall growth rate to be the same as in the LS group.

Key words: chinook salmon, *Oncorhynchus tshawytscha*, intestinal blood flow, blood flow distribution, haematocrit, metabolic rate, exercise training.

Introduction

Salmon migrate to the ocean to exploit the more abundant food resources found there. When foraging they must maintain normal body functions and grow, whilst swimming more or less continuously. This is a challenge to the cardiovascular system, which must transport oxygen to working locomotory muscles and, at the same time, support the substantial metabolic maintenance requirements for feeding and growth. Following feeding, oxygen consumption of resting fish may double because of the metabolic costs of digestion, absorption and assimilation (Brett and Zala, 1975; Jobling, 1981; Brown and Cameron, 1991), such that the postprandial metabolic rate may represent up to 43% of the active metabolic rate (Brett and Zala, 1975). Even though it is unlikely that salmonids can maintain intestinal blood flow while swimming maximally, they can maintain relatively high swimming speeds (1.5–2bodylengths s^{-1} ($bl\,s^{-1}$) without compromising growth (see Davison, 1989; Christiansen and Jobling, 1990).

The cardiovascular system of most vertebrates does not have the capacity to support simultaneously maximum blood flow demands for locomotion, feeding and maintenance functions (Farrell, 1991). Consequently, blood flow is redistributed from organs such as the intestines when the demands of locomotory muscles for oxygen increase (Vatner *et al.* 1974; Vatner, 1978; Laughlin and Armstrong, 1982; Armstrong *et al.* 1987; Eriksen *et al.* 1990; McKirnan *et al.* 1991). The blood flow requirement depends on blood oxygen content, which is in part determined by Hct (Lindenfeld *et al.* 1985). Therefore, Hct may affect how much blood flow has to be redistributed from the viscera to locomotory muscles during exercise (Vatner *et al.* 1972, 1974).

In resting fish, intestinal blood flow constitutes a significant (30–40%) proportion of cardiac output and, postprandially, splanchnic blood flow increases by 60–70 % (Axelsson *et al.* 1989; Axelsson and Fritsche, 1991; H. Thorarensen and A. P. Farrell, in preparation). However, during exercise fish reduce blood flow to the intestines (Randall and Daxboeck, 1982; Axelsson *et al.* 1989; Axelsson and Fritsche, 1991), and this reduction in intestinal blood flow may ultimately limit nutrient absorption and transport from the absorptive surfaces (Mailman, 1982). In addition, seawater fish must drink to regain water lost across the gills to the hypertonic environment (Eddy, 1982; Kirsch *et al.* 1984), an uptake process that probably depends on adequate intestinal blood flow. Since reduced blood flow to the intestines can limit energy intake and the ability to osmoregulate, the swimming speeds that can be sustained over an extended period may be determined by the ability of the cardiovascular system to maintain intestinal blood flow.

The purpose of this research was to study to what extent intestinal blood flow can be maintained in chinook salmon as swimming speed increases. This is probably determined by the oxygen-transporting capacity of the cardiovascular system, which is set by cardiac output and blood oxygen content. During exercise, the maximum oxygen-transporting capacity determines how much blood flow can be delivered to other organs in excess of what is required to meet the oxygen demand of the locomotory musculature. Exercise training of fish is known to increase heart size (Hochachka, 1961; Greer-Walker and Emerson, 1978; Farrell *et al.* 1990), enhance cardiac performance (Farrell *et al.* 1991) and

increase haemoglobin content of blood (Hochachka, 1961; Farlinger and Beamish, 1978); all of these effects may contribute to increased oxygen-transporting capacity of the cardiovascular system. Thus, in addition to the general changes in intestinal blood flow associated with swimming, we were interested in defining whether exercise training caused any cardiovascular adjustments that affected intestinal blood flow. Therefore, two groups of fish were compared following 8 months of exercise training, at either high or low swimming speeds, in order to identify any changes that might improve the fishes' ability to maintain blood flow to the intestine while swimming.

In this study we measured blood flow in the intestinal artery (IA), one of four arteries that supply the gastrointestinal tract of chinook salmon (Thorarensen *et al.* 1991). Judging from the diameters of these vessels, the IA carries at least 80–90% of the total blood flow. The IA, through its various branches, supplies blood to the liver, the pyloric caecae, the spleen and the intestine. Since the IA carries most of the blood flow to the gut, it was assumed that, during exercise, blood flow to the IA reflected total blood flow to the digestive tract.

Material and methods

Experimental animals

An all-female (feminized) stock of Chinook salmon [*Oncorhynchus tshawytscha* (Walbaum)] was obtained from Big Qualicum Hatchery, Qualicum Beach, British Columbia, and kept in outdoor tanks under natural photoperiod for the duration of the experiment. The chinook were exercise trained for 8 months (December to July), swimming continuously at either 0.5bodylengths s^{-1} ($bl\,s^{-1}$) (low speed: LS) or 1.5$bl\,s^{-1}$ (high speed: HS) in annular swimming channels (outer diameter 3.35m; inner diameter 2.90m). The water velocity was measured in the centre of the flume and regular checks indicated that variability in cross-sectional flow was less than 10%. The velocity in the channels was adjusted every month as the fish increased in size. Ample supplies of sea water at ambient temperature (8–11°C) and salinity (30±1‰) were provided throughout the training period. The fish were fed satiation levels of dry pellets twice daily (Biodiet dry, Oregon Bioproducts). Further descriptions of the training tanks and husbandry will be provided by A. K. Kiessling and D. Higgs (in preparation).

Growth of fish in both groups was identical. The initial mean masses were 76.5g and 76.4g, respectively, for the LS and HS groups. The final masses were 368±30.2 g (±S.E.M., N=25) and 362±15.9g (N=21), and the lengths were 30.3±0.34cm and 30.1±0.36cm respectively. However, the HS fish consumed 25% more food.

Critical swimming speed and the respirometer

U_{crit} and $\dot{V}_{O_2}$ were measured at 10±1°C in a Brett-type swim-tunnel respirometer. The fish were allowed to recover for more than 5h in the respirometer before U_{crit} was determined by increasing the swimming speed, first in steps of 0.5$bl\,s^{-1}$ and then of 0.25$bl\,s^{-1}$. Each velocity step was maintained for either 30min or until the fish fatigued, i.e. when the fish could not swim off the rear grid. We did not induce fish to swim by applying electric shocks. The velocity settings on the swim tunnel were calibrated

regularly with a flow meter. U_{crit} was calculated as described by Brett (1964) and corrected for the blocking effect of the fish in the tunnel (Bell and Terhune, 1970).

Oxygen tension ($P_{W_{O_2}}$) in the swim tunnel was monitored continuously by an oxygen electrode (Radiometer E5046) thermostatically controlled at the experimental temperature and connected to a PM71 unit (Radiometer, Copenhagen). Water from the swim tunnel was drawn past the electrode by a roller pump and the $P_{W_{O_2}}$ was recorded every second by a computer. $\dot{V}_{O_2}$ was measured by closing off the tunnel for 6min while $P_{W_{O_2}}$ was recorded. A least-squares procedure was used to calculate the slope or $dP_{W_{O_2}}/dt$. Oxygen consumption was calculated as:

$$\dot{V}_{O_2} = V \times dP_{W_{O_2}} \times dt^{-1} \times \alpha \times m^{-1}, \quad (1)$$

where V is the volume of the tunnel (39.1l), α is the solubility constant for oxygen at the experimental temperature and salinity ($\mu mol\, O_2\, l^{-1}\, kPa^{-1}$), and m is body mass. The $P_{W_{O_2}}$ was never allowed to decrease by more than 1kPa while $\dot{V}_{O_2}$ was recorded.

Arterial cannulation and implantation of flow probes

The fish were anaesthetized in a 1:2000 solution of 2-phenoxyethanol (Sigma Chemical Co., St Louis, Missouri) in sea water and anaesthesia was maintained by irrigating the gills continuously with chilled 1:4000 2-phenoxyethanol in sea water. The fish were cannulated in the dorsal aorta (DA) with polyethylene tubing (PE-50). The cannula was inserted with a trocar between the second and third gill arches and sutured to the roof of the mouth. The cannula was externalized in front of the nasal openings, secured with stitches in front of the dorsal fin, and filled with saline (0.9% NaCl) containing heparin at $150 i.u.ml^{-1}$.

Two types of flow probes were used to measure blood flow in the IA: pulsed Doppler flow probes (PDP) (TMI, Iowa City, Iowa) and Transonic flow probes (TFP) (Transonic Inc., Ithaca, New York), depending on whether blood flow was being measured in resting or swimming fish. The PDP only measures the velocity of blood in the vessel, whereas the TFP measures absolute flow. However, the TFP has bulkier leads than the PDP and is therefore not as well suited for measuring blood flow in swimming fish. The flow probes were placed on the intestinal artery (IA) through a lateral incision, beginning 2cm ventral to the lateral line, directly above the pectoral fin and extending 4cm ventrally in parallel with the myotomes. The skin was cut with a scalpel and the myotomes were carefully separated with blunt dissection. A short section of the intestinal artery was freed from the surrounding fascia and the probe cuff placed on the vessel. The incision was closed with 3-0 silk thread, using interrupted stitches through both muscles and skin. The probe leads were secured on the body surface and anchored dorsally with the DA cannula. The total time for surgery was less than 30min. The fish were allowed to recover for at least 24 h before experiments were performed.

Haematological variables

Each time blood samples were taken from swimming fish, 1ml of blood was removed to measure arterial P_{O_2} ($P_{a_{O_2}}$), oxygen content ($C_{a_{O_2}}$), haematocrit (Hct), and

haemoglobin concentration [Hb]. $P_{a_{O_2}}$ was measured with a thermostatted electrode (E5046) connected to a PM71 unit. $C_{a_{O_2}}$ was measured in 30 μl blood samples by the method of Tucker (1967). Sigma diagnostic kits numbers 525A and 826-UV were used to measure blood haemoglobin (in 20 μl samples) and plasma lactate (in 100 μl samples) respectively. The blood that was used to measure $P_{a_{O_2}}$ and any leftover blood from the initial 1ml sample (total amount around 0.8ml) was reinjected into the fish and enough saline was added to make the total volume 1ml. Before the fish fatigued, 4–5 blood samples were taken, resulting in approximately 1.0ml of blood being replaced by saline.

Experimental protocol

Three sets of experiments were performed. In the first set, the critical swimming speed (U_{crit}) and oxygen consumption ($\dot{V}_{O_2}$) of unoperated fish were determined. In the second set, fish were cannulated in the dorsal aorta (DA) and a pulsed Doppler flow probe (PDP) was implanted on the intestinal artery (IA). The fish were then swum to U_{crit} while relative blood flow in the IA (q_{IA}), blood pressure in the DA (P_{DA}), heart rate (f_H) and $\dot{V}_{O_2}$ were measured after each increment in swimming speed. Arterial blood samples for $P_{a_{O_2}}$, $C_{a_{O_2}}$, Hct and [Hb] were taken at rest (5–10cm s^{-1} water velocity in the swim tunnel with the fish not swimming) and at a swimming speed of 1.5bl s^{-1}. When the fish appeared to be approaching their maximum swimming speed, as indicated by the more frequent bursts of swimming that were required to maintain position, blood samples were taken at every velocity step. Thus, blood was sampled 4–5 times from every fish before it fatigued and then after 1h of recovery. Samples for lactate were taken at rest, as the fish fatigued, and after 1h of recovery. For statistical analysis, haematological data recorded between 75 and 95% of U_{crit} (mean 85%) were pooled, as were samples taken above 95% of U_{crit}.

The third set of experiments was performed on a separate group of LS fish. Resting levels of blood flow in the intestinal artery were measured with TFP, to provide absolute values for blood flow in the IA and thus an estimate of q_{IA} for the fish in experiment 2. The fish with TPFs were placed in dark holding boxes and, after 24h of recovery, blood flow in the IA was recorded overnight by a computer every minute. Between 04:00h and 08:00h IA blood flow was the most stable, so these values were averaged to represent resting blood flow. Following this period, Hct and $C_{a_{O_2}}$ were measured and the 1ml of blood removed from the fish was replaced with saline to reduce Hct. Hct was reduced in a progressive fashion by repeating the same procedure one to three times at 24h intervals and the relationship between Hct and absolute values of q_{IA} was established for individual fish. A few attempts were made to increase Hct with a transfusion of packed red blood cells, but only two were successful.

Data acquisition and statistics

Signals from flow meters, pressure transducers and oxygen meters were amplified and monitored by a Grass chart recorder (model 7PCP B, Grass Instruments, Quincy, Massachusetts) and stored in a computer. The computer sampled q_{IA} and P_{DA} signals at a rate of 5Hz and recordings were averaged over 6min for each swimming velocity. Labtech Notebook software (Laboratory Technology Corporation, Massachusetts) was used to convert the signals to digital form, to process the signals and to calculate f_H.

Statistical analyses were performed using the General Linear Models procedure and Proc Corr in SAS (Version 6, SAS Institute). q_{IA}, P_{DA}, f_H, splanchnic vascular resistance and the haematological variables were compared at different swimming speeds, in the two training groups, by a two-way analysis of variance (ANOVA) with repeated measures on individual fish. Other models are described as they are presented in the Results section. Values are presented as mean ± S.E.M. throughout text and figures. The fiducial limits for accepting significance were $P<0.05$.

Results

Critical swimming speed and oxygen consumption

Neither the $\dot{V}_{O_2max}$ (HS: 319.9±12.8 μmolmin^{-1}kg^{-1}; LS: 290±15.3 μmolmin^{-1} kg^{-1}) nor the U_{crit} (HS: 87.5±2.2cm s^{-1}; LS: 85.5±1.9cm s^{-1}) of the two training groups differed significantly following 8 months of exercise training (N=10 fish from each group). There was significant correlation between the U_{crit} and $\dot{V}_{O_2max}$ of individual fish ($P<0.0024$).

A second-order polynomial modelled $\dot{V}_{O_2}$ (r^2=0.86, Fig. 1) as a function of swimming speed (U) in cm s^{-1}. No parameters of the model were significantly different in the two training groups and, therefore, the data were pooled. This model accounted for individual variability by allowing a different intercept (I_f) for each fish:

$$\dot{V}_{O_2} = I_f - 1.37U + 0.043U^2. \quad (2)$$

The equation implies that the energy budget of swimming fish consists of two components: one that increases with the square of the swimming speed and another that decreases as a function of swimming speed. This model gave a closer fit to the data than a linear regression of the logarithm of $\dot{V}_{O_2}$ on swimming speed (r^2=0.79).

The U_{crit} of the operated fish from the two groups was not significantly different, being 68.8±3.1bl s^{-1} for the HS group (N=11) and 65.0±2.8bl s^{-1} for the LS group (N=15). Similarly, the $\dot{V}_{O_2max}$ was not significantly different in the two groups (250±22 μmolmin^{-1}kg^{-1} in LS and 318±25 μmolmin^{-1}kg^{-1} in HS). The $\dot{V}_{O_2max}$ was not significantly different in operated and unoperated (experiment 1) fish, but the U_{crit} was 23% lower in operated fish compared with unoperated fish. When $\dot{V}_{O_2}$ of cannulated fish was modelled as a function of swimming speed, Hct could be incorporated into the model as a significant variable ($P<0.008$), indicating that fish with higher Hct have higher $\dot{V}_{O_2}$:

$$\dot{V}_{O_2} = I_f + 3.72\text{Hct} - 1.84U + 0.065U^2. \quad (3)$$

Relative changes in intestinal blood flow and haematological variables of swimming fish

In both training groups, q_{IA} was progressively reduced as swimming speed increased above 30–40cm s^{-1} (Fig. 2A) because of a concomitant increase in splanchnic vascular resistance (Fig. 2B). A comparison with ANOVA of the mean changes in q_{IA} as swimming speed was increased showed that, in the HS group, blood flow was significantly less reduced ($P<0.0003$) than in the LS group. Since the number of

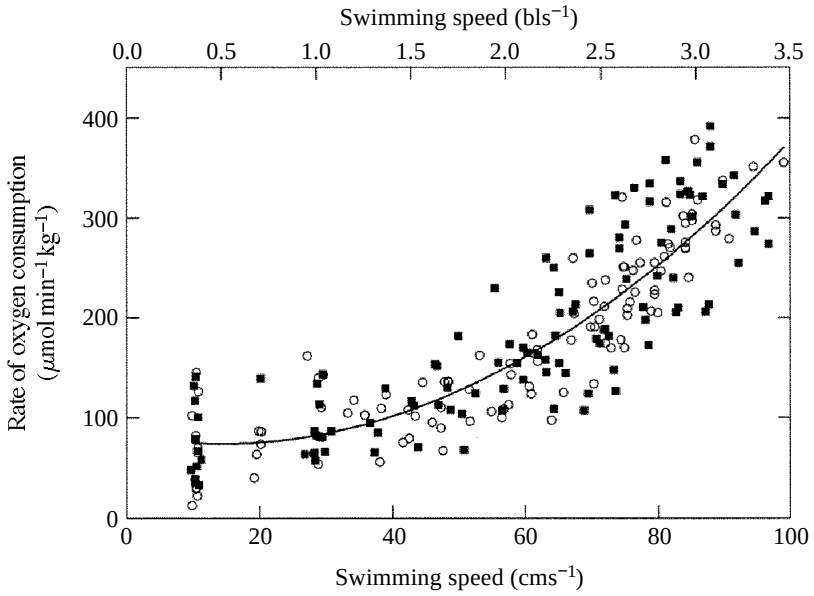

Fig. 1. The rate of oxygen consumption of unoperated chinook salmon. Open symbols: fish trained at 0.5bl s^{-1} (LS); filled symbols: fish trained at 1.5bl s^{-1} (HS). N=10 fish from each group.

observations for each point in Fig. 2 is reduced at the highest swimming speeds, observations made at swimming speeds greater than the mean U_{crit} were not included in the analysis. However, the values for the best swimmers have been included in the figure. P_{DA} did not change significantly with swimming speed (Fig. 2C), but f_H increased by 21% (Fig. 2D, P<0.0001). Neither P_{DA} nor f_H was significantly different between the two training groups.

There was a negative linear relationship (r^2=0.83) between $\dot{V}_{O_2}$ and q_{IA} (Fig. 3), suggesting that intestinal blood flow was gradually reduced as the oxygen demands of locomotory muscles increased. The slopes of this relationship for the two groups in Fig. 3 were not significantly different.

The mean haematocrit of the HS training group was significantly (P<0.0001) higher than that of the LS group (Table 1). This difference was also reflected in the significantly higher (P<0.0001) [Hb] and C_{aO_2} (Table 1). Mean cell haemoglobin concentration (MCHC) was slightly lower (P<0.05) in the LS group (Table 1). Neither C_{aO_2} nor [Hb] changed significantly with increased swimming speed. P_{aO_2} was, however, significantly (P<0.0001) reduced at swimming speeds higher than 85% of U_{crit}, but there was no significant difference between the two training groups (Table 1).

Absolute blood flow in the intestinal artery

Blood flow in the intestinal artery of resting fish was inversely (P<0.0002) related to Hct. In spite of significant variability among individuals, all fish showed this same relationship and there was no significant difference between the slopes for individual fish.

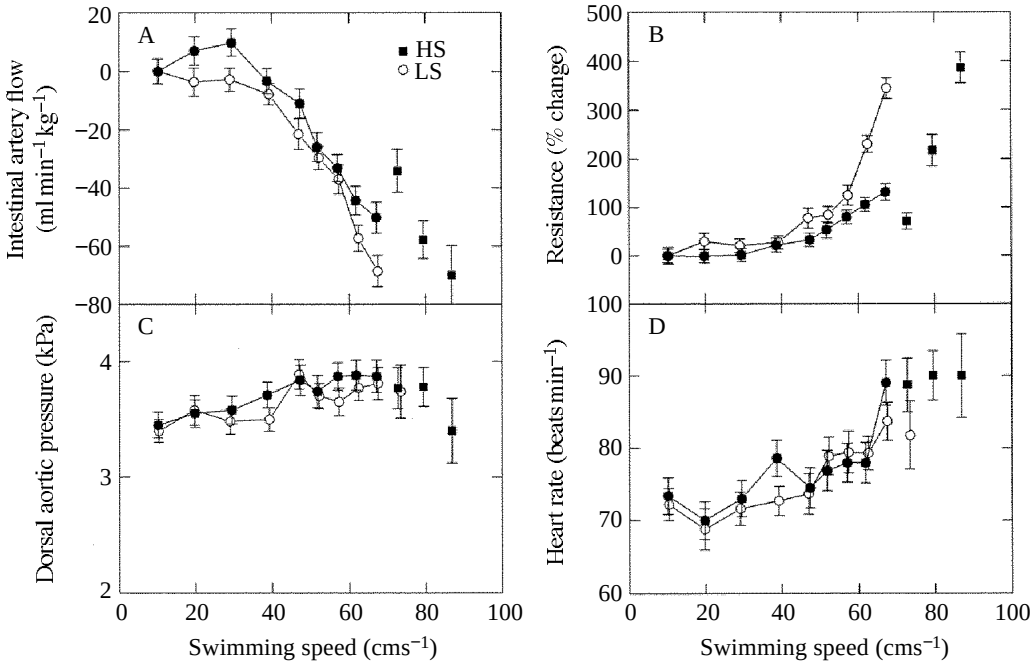

Fig. 2. Cardiovascular changes during exercise in chinook salmon, swimming with a DA cannula and a Doppler flow probe on the intestinal artery. Bars show ± S.E.M., N=10 for each group of fish shown connected with a line. Individual points represent mean values for 2–3 fish of the group that swam faster than the remainder. (A) Blood flow in the intestinal artery, expressed as the percentage change from the resting level. There is significant difference between groups (P<0.0003) and significant changes in both groups as swimming speed increases (P<0.0001). (B) Relative resistance of the splanchnic vascular bed. Resistance increases significantly with swimming speed in both groups (P<0.0001). (C) Pressure in the dorsal aorta. The results for the two groups are not significantly different and there are no significant changes with swimming speed. (D) Heart rate shows a significant increase with swimming speed (P<0.0001), but heart rate in the two groups was not significantly different.

A linear regression of q_{IA} *versus* Hct that assumed the same slope (S.E.M. ±0.12) for all fish (N=10) but different intercepts, explained 86% of the total variance:

$$q_{IA} = I_f - 0.57\text{Hct}. \quad (4)$$

The mean intercept was 27.5 (±1.52). Recordings of q_{IA} made at Hcts lower than 20 % were not included in the analysis since, at lower Hct, q_{IA} appeared to decrease, but this phenomenon was not explored further. The highest recorded Hct was 36%.

Discussion

At rest, intestinal blood flow was quite variable among individuals, but q_{IA} of all fish increased as Hct and blood oxygen-carrying capacity were reduced. This indicates that blood flow is adjusted to maintain oxygen delivery to the intestines. The resting q_{IA} values of the LS and HS training groups, as predicted by equation 4 (and mean intercept

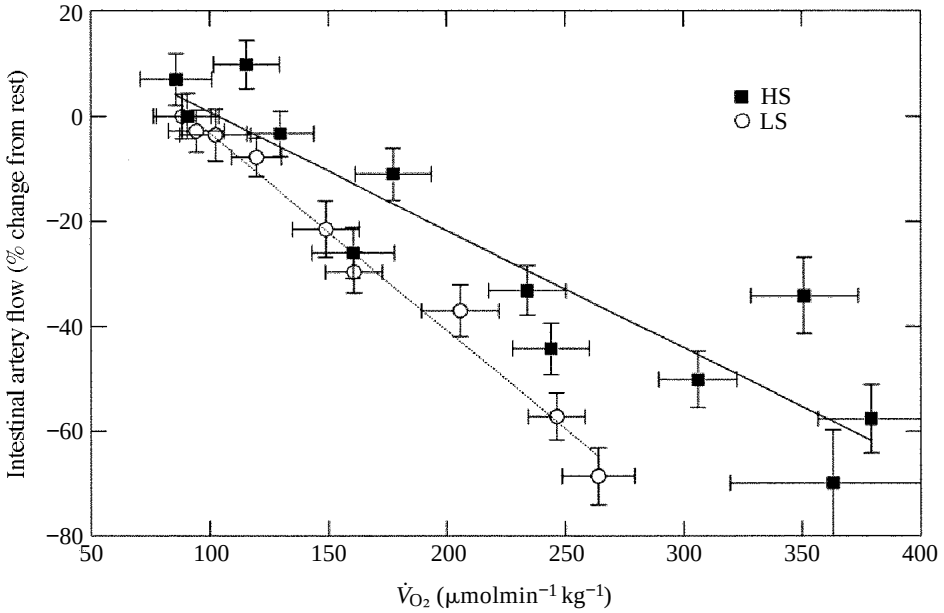

Fig. 3. Blood flow in the intestinal artery as a function of oxygen uptake. Horizontal and vertical bars show ± S.E.M., *N*=10 in each group.

for all fish), were 14.2 and 12.0mlmin^{-1}kg^{-1}, respectively, indicating that resting intestinal blood flow was not appreciably changed by the two exercise training regimes.

Assuming that q_{IA} is 80–90% of the total gastrointestinal blood flow, blood flow to the gut should amount to 13–18mlmin^{-1}kg^{-1} in chinook salmon. This compares well with the 13mlmin^{-1}kg^{-1} measured in the hepatic portal vein of anaesthetized rainbow trout (McLean and Ash, 1989), but it is substantially higher than the resting gut blood flow reported for the sea raven (5.6mlmin^{-1}kg^{-1}) and the cod (7.6mlmin^{-1}kg^{-1}) (Axelsson *et al.* 1989; Axelsson and Fritsche, 1991; Axelsson, 1991). However, the cardiac output of chinook is also considerably higher than that of sea raven (18.8mlmin^{-1}kg^{-1}) and cod (18–19mlmin^{-1}kg^{-1}), being 33mlmin^{-1}kg^{-1} at rest in fish with a Hct of 30 % (H. Thorarensen, unpublished observation). The predicted q_{IA} for a fish with this Hct would be 12mlmin^{-1}kg^{-1}, so the total gastrointestinal blood flow constitutes about 36% of cardiac output. The proportion of cardiac output going to the gastrointestinal tract is, therefore, very similar to the values reported for sea raven (30%) and for cod (40%) (Axelsson *et al.* 1989; Axelsson and Fritsche, 1991; Axelsson, 1991). Direct measurements of gastrointestinal blood flow appear to indicate consistently that a higher proportion of cardiac output goes to the gut than the 10–20% estimated using radiolabelled microspheres (Daxboeck, 1981; Barron *et al.* 1987; H. Thorarensen, unpublished observations). Barron *et al.* (1987) have suggested that the microsphere method may underestimate visceral blood flow.

Our results imply that the pumping capacity of the heart of chinook salmon is not enough to allow the fish to maintain maximum aerobic swimming velocity without redistributing blood flow from the viscera to the locomotory muscles. Reduced gut blood

Table 1. *Haematocrit (Hct), blood oxygen content (Ca_{O_2}), haemoglobin concentration ([Hb]), arterial P_{O_2} (Pa_{O_2}), mean cell haemoglobin concentration (MCHC) and plasma lactate concentration in fish at various swimming speeds*

		Swimming speed					
	Training speed	Rest	1 bl s^{-1}	85 % U_{crit}	U_{crit}	Recovery	Mean
Hct	LS	23.3 (0.9)	23.8 (0.8)	24.4 (0.6)	24.5 (0.9)	22.1 (1.0)	23.3†
	HS	27.1 (1.0)	27.2 (0.9)	28.4 (0.7)	28.3 (1.0)	26.7 (1.0)	27.6
Ca_{O_2}	LS	8.3 (0.5)	9.3 (0.4)	9.1 (0.3)	8.5 (0.5)	8.3 (0.5)	8.7†
(% vol)	HS	10.9 (0.6)	11.0 (0.5)	11.5 (0.4)	10.5 (0.6)	10.6 (0.5)	10.9
[Hb]	LS	7.16 (0.3)	6.98 (0.3)	7.22 (0.2)	7.21 (0.3)	6.57 (0.3)	7.0†
($g\,dl^{-1}$)	HS	8.61 (0.3)	8.24 (0.3)	8.61 (0.2)	8.37 (0.4)	8.07 (0.3)	8.4
Pa_{O_2}	LS	15.9 (0.5)	15.0 (0.5)	13.1 (0.4)	11.3 (0.5)	15.3 (0.5)	14.1‡
(kPa)	HS	15.7 (0.6)	14.8 (0.5)	12.2 (0.4)	10.4 (0.6)	15.2 (0.6)	13.6‡
MCHC	LS	304 (5)	290 (4)	294 (3)	295 (5)	297 (5)	296*
($g\,l^{-1}$)	HS	312 (6)	303 (5)	305 (5)	295 (6)	303 (5)	304
Lactate	LS	0.0 (0.5)			1.5 (0.4)	2.6 (0.3)	1.4‡
($\mu mol\,l^{-1}$)	HS	0.1 (0.5)			1.7 (0.4)	2.1 (0.4)	1.3‡

The fish were trained at either 0.5bl s^{-1} (LS, N=15) or 1.5bl s^{-1} (HS, N=11).
Numbers in parentheses are ± S.E.M.
*Significant difference between training groups (P<0.05).
†Significant difference between training groups (P<0.0001).
‡Significant difference among swimming speeds (P<0.0001).
Recovery, measurements were taken after 1h of recovery.

flow has also been reported for swimming rainbow trout (Randall and Daxboeck, 1982), sea raven (Axelsson *et al.* 1989) and cod (Axelsson and Fritsche, 1991). However, this is the first time that intestinal blood flow has been shown to be linearly related to $\dot{V}_{O_2}$ (Fig. 3). This indicates that, at swimming velocities over 50 % U_{crit}, q_{IA} is regulated in relation to the metabolic demands of the locomotory muscles. At lower swimming velocities, $\dot{V}_{O_2}$ did not increase appreciably (Fig. 1) and q_{IA} remained relatively constant, but at swimming speeds higher than 50% of U_{crit}, q_{IA} was reduced as $\dot{V}_{O_2}$ increased, because of a concurrent increase in splanchnic vascular resistance (Fig. 2B). The exact control mechanism for this is unknown. However, the resistance of the intestinal vasculature of fish increases in response to adrenergic, cholinergic and peptidergic stimulation (Holmgren and Nilsson, 1974; Axelsson *et al.* 1989; Olson and Meisheri, 1989; Axelsson and Fritsche *et al.* 1991; Holmgren *et al.* 1992; H. Thorarensen, unpublished observations).

The absolute intestinal blood flow in swimming fish was estimated (Fig. 4A) from the predicted resting blood flow and the relative changes in q_{IA} shown in Fig. 2A. In addition, oxygen transport to the intestines was estimated by multiplying q_{IA} by Ca_{O_2} (Fig. 4B). The HS-trained fish maintained adequate intestinal oxygenation while swimming at 1.5bl s^{-1} by increasing Hct. Increased Hct was observed in earlier studies of exercise training in fish (Hochachka, 1961; Farlinger and Beamish, 1978). The increased Hct elevated the oxygen-carrying capacity of the blood, and thus oxygen transport to the

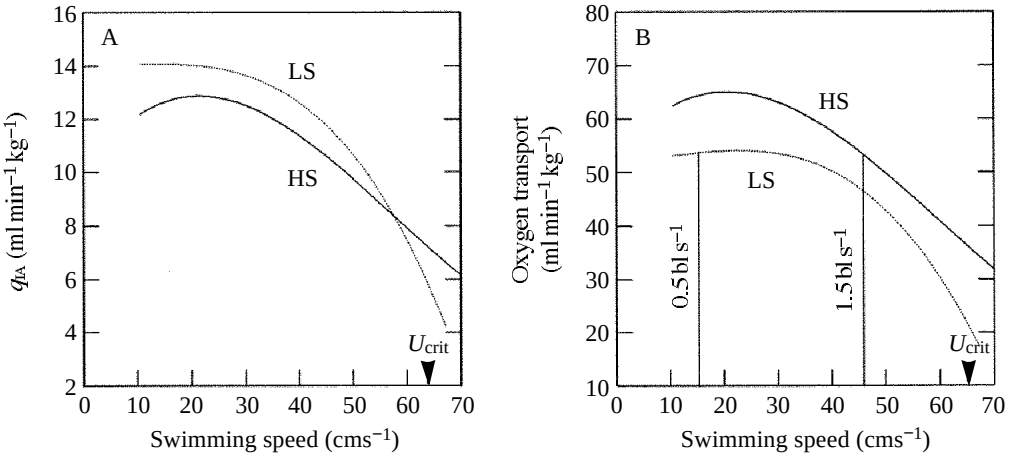

Fig. 4. Predicted oxygen transport and blood flow in the intestinal artery (q_{IA}) of swimming chinook salmon. (A) Predicted blood flow in the intestinal artery. (B) Predicted oxygen transport (blood flow × blood oxygen content). Dotted line: fish trained at 0.5bl s^{-1}; solid line: fish trained at 1.5bl s^{-1}. The vertical lines show the training velocity in the two groups.

intestines was higher in the HS-trained group at all swimming speeds (Fig. 4B), in spite of lower q_{IA} at rest (Fig. 4A). Hence, any loss in intestinal oxygen transport which would otherwise have occurred because of reduced intestinal blood flow was compensated for by the increased Hct.

An additional advantage of an elevated Hct is that less blood flow is required to meet the oxygen demands of the locomotory muscles and thus there is less of a need to redistribute blood flow away from the viscera as swimming speed is increased (Fig. 4A). This is consistent with our finding that q_{IA} was reduced less in the HS-trained group as swimming velocity was increased (Fig. 2A). The elevated Hct may also produce a greater scope for increasing intestinal blood flow postprandially without compromising oxygen delivery to locomotory muscles. We suggest, therefore, that a significant role of the elevated Hct for the HS group was to maintain oxygen transport to the intestines, and consequently normal intestinal function, while swimming continuously at a relatively high velocity.

Even though oxygen consumption was 50% higher at the HS training velocity than at the LS velocity (Fig. 1), the growth of fish in both training groups was similar. This suggests that intestinal function was indeed maintained in the HS-trained fish. Since food intake increased by only 25%, food utilization must also have increased. Earlier studies have indicated that food utilization does indeed increase when salmonids are exercise-trained (see Davison, 1989). However, 1.5bl s^{-1} must be close to the upper limit of the swimming velocity that the fish can sustain without compromising growth, because at higher swimming speeds intestinal blood flow is reduced rapidly (Fig. 2A), resulting eventually in negative energy and, possibly, fluid balances.

The higher Hct in the HS-trained fish did not improve their aerobic swimming performance compared with the LS-trained fish. This confirms the results of Gallaugher *et al.* (1992), which suggest that swimming performance of rainbow trout is not limited

Table 2. *Importance of redistribution of intestinal blood flow during exercise for fish trained at 0.5bl s^{-1} (LS) and 1.5bl s^{-1} (HS)*

	LS	HS
Internal $\dot{V}_{O_2}$* (μmolmin^{-1}kg^{-1})	175	223
O_2-transporting capacity of blood flow redistributed from the intestinal artery at U_{crit} (μmolmin^{-1}kg^{-1})†	37	31
Contribution to tissue $\dot{V}_{O_2}$ (μmolmin^{-1}kg^{-1})‡	32	27
Percentage of internal $\dot{V}_{O_2}$	18	12

*Internal $\dot{V}_{O_2}$ is oxygen consumption of tissues other than gills, assumed to be 70% of total $\dot{V}_{O_2}$ (Daxboeck *et al.* 1982).

†Total redistribution from the intestines could be as much as 10–20% higher.

‡Assuming that average tissue oxygen extraction is 86% (Kiceniuk and Jones, 1977).

by blood oxygen-carrying capacity within their normal Hct range. However, the results of the present study indicate that the significance of the Hct response to exercise is to allow other functions, such as digestion and perhaps osmoregulation, to be maintained while the fish are swimming.

In order to assess the relevance of the redistribution of blood flow away from the intestines for muscle oxygen delivery, at high swimming speeds, an estimate was made of the oxygen-transporting capacity of the blood flow redirected away from the intestines during swimming (Table 2). It was assumed that the internal oxygen consumption ($\dot{V}_{O_2}$ − gill oxygen consumption) was 70% of $\dot{V}_{O_2}$ (Daxboeck *et al.* 1982) and that, at U_{crit}, extraction of oxygen from blood was 86% (Kiceniuk and Jones, 1977). These calculations indicate that blood flow redistributed away from the IA can transport at least 12% of the total oxygen consumed at U_{crit} in the HS-trained group and 18% in the LS-trained group. Since the IA may only carry 80–90% of the total intestinal blood flow, the actual values are likely to be higher than our estimates. It is evident, therefore, that redistribution of blood flow away from the intestines contributes significantly to the oxygen supply of aerobic muscles at swimming velocities close to U_{crit}, and that the relative importance of this redistribution is greater in the LS-trained group with a lower Hct.

The redistribution of blood flow from the intestines will undoubtedly reduce the oxidative metabolism of these tissues. This suggestion is consistent with the polynomial model of $\dot{V}_{O_2}$ as a function of swimming speed, namely the component that decreases linearly with swimming velocity. Other studies have also indicated that less oxygen is allocated to the maintenance functions of fish as swimming speed increases (Furnell, 1987; Kaufmann and Wieser, 1992). It has been suggested that the oxygen demand of swimming fish may exceed the rate at which oxygen can be delivered, resulting in a temporary shift of energy allocation from maintenance to locomotory functions (Wieser, 1989). Our results indicate that this shift is at least accompanied by, if not a result of, redistribution of blood flow. Furthermore, the degree to which this happens may be set by the factors, such as Hct, that alter the oxygen-transporting capacity of the cardiovascular system.

It is assumed in the polynomial model that the cost of locomotion increases in proportion to U^2. In previous studies, the metabolic cost of swimming has been estimated to increase in proportion to $U^{1.6}$–U^2 (see Jones and Randall, 1978; Kaufmann, 1990). However, these models implicitly assume that the standard metabolic rate remains the same at all swimming speeds. This no longer appears to be a reasonable assumption. If energy allocation to maintenance functions is reduced as swimming velocity increases, it is obvious that the metabolic cost of swimming will be underestimated, i.e. the value of the exponent should be higher. Therefore, our assumption that the oxygen consumption of swimming muscles increases in proportion to U^2 is in keeping with the earlier estimates of the aerobic cost of swimming.

As the chinook salmon swam to U_{crit}, Hct did not increase significantly. This has also been observed in coho salmon (Brauner *et al.* 1993). However, an increase in Hct has repeatedly been observed in rainbow trout and in brown trout swimming to U_{crit} in fresh water (Thomas *et al.* 1987; Butler *et al.* 1992; Gallaugher *et al.* 1992). Whether this is a reflection of interspecies differences, different environment (sea water *versus* fresh water) or some other factor is unknown. It has been suggested that the significance of the increased Hct in rainbow trout is to maintain $C_{a_{O_2}}$ at U_{crit} when $P_{a_{O_2}}$ is reduced (Thomas *et al.* 1987; Butler *et al.* 1992; Gallaugher *et al.* 1992). However, in the chinook salmon the $C_{a_{O_2}}$ was not reduced at U_{crit}, in spite of a substantial reduction in $P_{a_{O_2}}$ and unchanged Hct.

In conclusion, our results show that intestinal blood flow is approximately 36% of cardiac output in resting chinook salmon. During exercise, blood flow is redistributed from the viscera to meet the increased oxygen demands of the skeletal muscles. Intestinal blood flow is relatively constant at swimming speeds lower than 40–50% of U_{crit}, when $\dot{V}_{O_2}$ increases only slightly. At U_{crit}, however, q_{IA} is reduced by 60–70%. A significant proportion of muscle oxygen consumption during exercise can be supported by this blood flow redistribution away from the viscera.

High-speed exercise training resulted in an increased Hct and oxygen-carrying capacity of the blood, thus facilitating oxygen transport to the viscera at all swimming speeds. Since less blood flow is required to meet the oxygen demands of locomotory muscles in fish with an elevated Hct, this indirectly helps to maintain intestinal blood flow while the fish are swimming because there is less need to redistribute blood flow. Therefore, increased Hct allows the fish to swim continuously at higher speeds without compromising growth and maintenance functions.

This study was supported by an NSERC operating grant to A.P.F. A.K.K. received support from the Swedish Science Council and H.T. and P.G. were supported by Graduate Fellowships from Simon Fraser University.

References

ARMSTRONG, R. B., DELP, M. D., GOLJAN, E. F. AND LAUGHLIN, M. H.(1987). Distribution of blood flow in muscles of miniature swine during exercise. *J. appl. Physiol.* **62**, 1285–1298.

AXELSSON, M. (1990). On the cardiovascular control in representatives of three vertebrate groups: Effects of exercise and feeding. PhD thesis, University of Gothenburg.

AXELSSON, M., DRIEDZIC, W. R., FARRELL, A. P. AND NILSSON, S. (1989). Regulation of cardiac output and gut blood flow in the sea raven, *Hemitripterus americanus. Fish Physiol. Biochem.* **6**, 315–326.

AXELSSON, M. AND FRITSCHE, R. (1991). Effects of exercise, hypoxia and feeding on the gastrointestinal blood flow in the Atlantic cod *Gadus morhua. J. exp. Biol.* **158**, 181–198.

BARRON, M. G., TARR, B. D. AND HAYTON, W. L.(1987). Temperature dependence of cardiac output and regional blood flow in rainbow trout, *Salmo gairdenri* Richardson. *J. Fish Biol.* **31**, 735–744.

BELL, W. M. AND TERHUNE, L. D. B.(1970). Water tunnel design for fisheries research. *Tech. Rep. Fish. Res. Bd Can.* **195**, 69pp.

BRAUNER, C. J., SHRIMPTON, J. M. AND RANDALL, D. J. (1993). The effects of elevated plasma ion concentrations on swimming performance in coho salmon (*Oncorhynchus kisutch*) parr. *Can. J. Fish. aquat. Sci.* (in press).

BRETT, J. R.(1964). The respiratory metabolism and swimming performance of young sockeye salmon. *J. Fish. Res. Bd Can.* **21**, 1183–1226.

BRETT, J. R. AND ZALA, C. A. (1975). Daily pattern of nitrogen excretion and oxygen consumption of sockeye salmon, *Oncorhynchus nerka*, under controlled conditions. *J. Fish. Res. Bd Can.* **32**, 2479–2486.

BROWN, C. R. AND CAMERON, J. N. (1991). The induction of specific dynamic action in channel catfish by infusion of essential amino acids. *Physiol. Zool.* **64**, 276–297.

BUTLER, P. J., DAY, N. AND NAMBA, K.(1992). Interactive effects of seasonal temperature and low pH on resting oxygen uptake and swimming performance of adult brown trout *Salmo trutta. J. exp. Biol.* **165**, 195–212.

CHRISTIANSEN, J. S. AND JOBLING, M. (1990). The behaviour and the relationship between food intake and growth of juvenile Arctic charr, *Salvelinus alpinus* L., subjected to sustained exercise. *Can. J. Zool.* **68**, 2185–2191.

DAVISON, W.(1989). Training and its effects on teleost fish. *Comp. Biochem. Physiol.* **94**A, 1–10.

DAXBOECK, C. (1981). A study of the cardiovascular system of fish (*Salmo gairdneri*) at rest and during swimming exercise. PhD thesis, University of British Columbia, Vancouver.

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

EDDY, F. B. (1982). Osmotic and ionic regulation in captive fish with particular reference to salmonids. *Comp. Biochem. Physiol.* **73**B, 125–141.

ERIKSEN, M., WAALER, B. A., WALLOE, L. AND WESCHE, J.(1990). Dynamics and dimensions of cardiac output changes in humans at the onset and at the end of moderate rhythmic exercise. *J. Physiol., Lond.* **426**, 423–437.

FARLINGER, S. AND BEAMISH, F. W. H. (1978). Changes in blood chemistry and critical swimming speed of largemouth bass, *Micropterus salmonides*, with physical conditioning. *Trans. Am. Fish. Soc.* **107**, 523–527.

FARRELL, A. P. (1991). Circulation of body fluids. In *Envrionmental and Metabolic Animal Physiology* (ed. C. L. Prosser), pp. 509–558. New York: John Wiley & Sons Inc.

FARRELL, A. P., JOHANSEN, J. A., STEFFENSEN, J. F., MOYES, C. D., WEST, T. G. AND SUAREZ, R. K. (1990). Effects of exercise training and coronary ablation on swimming performance, heart size and cardiac enzymes in rainbow trout, *Oncorhynchus mykiss. Can. J. Zool.* **68**, 1174–1179.

FARRELL, A. P., JOHANSEN, J. A. AND SUAREZ, R. K. (1991). Effects of exercise-training on cardiac performance and muscle enzymes in rainbow trout, *Oncorhynchus mykiss*. *Fish Physiol. Biochem.* **9**, 303–312.

FURNELL, D. J. (1987). Partitioning of locomotor and feeding metabolism in sablefish (*Anaplopoma fimbria*). *Can. J. Zool.* **65**, 486–489.

GALLAUGHER, P., AXELSSON, M. AND FARRELL, A. P. (1992). Swimming performance and haematological variables in splenectomized rainbow trout, *Oncorhynchus mykiss*. *J. exp. Biol.* **171**, 301–314.

GREER-WALKER, M. AND EMERSON, L. (1978). Sustained swimming speeds and myotomal muscle function in the trout, *Salmo gairdneri. J. Fish Biol.* **13**, 475–481.

HOCHACHKA, P. W. (1961). The effect of physical training on oxygen debt and glycogen reserves in trout. *Can. J. Zool.* **39**, 767–776.

HOLMGREN, S., AXELSSON, M. AND FARRELL, A. P.(1992). The effect of catecholamines, substance P and vasoactive intestinal polypeptide on blood flow to the gut in the dogfish *Squalus acanthias*. *J. exp. Biol.* **168**, 161–175.

HOLMGREN, S. AND NILSSON, S. (1974). Drug effects on isolated artery strips from two teleosts, *Gadus morhua* and *Salmo gairdneri*. *Acta physiol. scand.* **90**, 431–437.

JOBLING, M. (1981). The influences of feeding on the metabolic rate of fishes: a short review. *J. Fish Biol.* **18**, 385–400.

JONES, D. R. AND RANDALL, D. J. (1978). The respiratory and circulatory systems during exercise. In *Fish Physiology*, vol. VII (ed. W. S. Hoar and D. J. Randall), pp. 425–501. New York: Academic Press.

KAUFMANN, R. (1990). Respiratory cost of swimming in larval and juvenile cyprinids. *J. exp. Biol.* **150**, 343–366.

KAUFMANN, R. AND WIESER, W.(1992). Influence of temperature and ambient oxygen on the swimming energetics of cyprinid larvae and juveniles. *Envl Biol. Fishes* **33**, 87–95.

KICENIUK, J. W. AND JONES, D. R.(1977). The oxygen transport system in trout (*Salmo gairdneri*) during sustained exercise. *J. exp. Biol.* **69**, 247–260.

KIRSCH, R., HUMBERT, W. AND RODEAU, J. L. (1984). Control of the blood osmolarity in fishes with reference to the functional anatomy of the gut. In *Osmoregulation in Estuarine and Marine Animals* (ed. A. Pequeux, R. Gilles and L. Bolis), pp. 68–92. Berlin, Heidelberg, New York, Tokyo: Springer-Verlag.

LAUGHLIN, M. H. AND ARMSTRONG, R. B.(1982). Muscular blood flow distribution patterns as a function of running speed in rats. *Am. J. Physiol.* **243**, H296–H306.

LINDENFELD, J., WEIL, J. V., TRAVIS, V. AND HORWITZ, L. D. (1985). Hemodynamic response to normovolemic polycytemia at rest and during exercise in dogs. *Circulation Res.* **56**, 793–800.

MAILMAN, D. (1982). Blood flow and intestinal absorption. *Fedn Proc. Fedn Am. Socs exp. Biol.* **41**, 2096–2100.

MCKIRNAN, M. D., GRAY, C. G. AND WHITE, F. C. (1991). Effects of feeding on muscle blood flow during prolonged exercise in miniature swine. *J. appl. Physiol.* **70**, 1097–1104.

MCLEAN, E. AND ASH, R.(1989). Chronic cannulatinon of the hepatic portal vein in rainbow trout, *Salmo gairdneri*: A prerequisite to net absorption studies. *Aquaculture* **78**, 195–205.

OLSON, K. R. AND MEISHERI, K. D. (1989). Effects of atrial natriuretic factor on isolated arteries and perfused organs of trout. *Am. J. Physiol.* **256**, R10–R18.

RANDALL, D. J. AND DAXBOECK, C. (1982). Cardiovascular changes in the rainbow trout (*Salmo gairdneri* Richardson) during exercise. *Can. J. Zool.* **60**, 1135–1140.

THOMAS, S., POUPIN, J., LYKKEBOE, G. AND JOHANSEN, K.(1987). Effects of graded exercise on blood gas tensions and acid base characteristics of rainbow trout. *Respir. Physiol.* **68**, 85–97.

THORARENSEN, H., MCLEAN, E., DONALDSON, E. M. AND FARRELL, A. P. (1991). The blood vasculature of the gastrointestinal tract in chinook, *Oncorhynchus tshawytscha* (Walbaum) and coho, *O. kisutch* (Walbaum) salmon. *J. Fish Biol.* **38**, 525–531.

TUCKER, V. A. (1967). Method for oxygen content and dissociation curves on microliter blood samples. *J. appl. Physiol.* **23**, 410–414.

VATNER, S. F. (1978). Effects of exercise and excitement on mesenteric and renal dynamics in conscious, unrestrained baboons. *Am. J. Physiol.* **234**, H210–H214.

VATNER, S. F., HIGGINS, C. B. AND FRANKLIN, D. (1972). Regional circulatory adjustments to moderate and severe chronic anemia in conscious dogs at rest and during exercise. *Circulation Res.* **30**, 731–740.

VATNER, S. F., HIGGINS, C. B., MILLARD, R. W. AND FRANKLIN, D. (1974). Role of the spleen in the peripheral vascular response to severe exercise in untethered dogs. *Cardiac Res.* **8**, 276–282.

WIESER, W. (1989). Energy allocation by addition and by compensation: an old principle revisited. In *Energy Transformation in Cells and Animals* (ed. W. Wieser and E. Gnaiger), pp. 98–105. Stuttgart: Georg Thieme Verlag.