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EFFECTS OF HIGH INTENSITY EXERCISE TRAINING ON CARDIOVASCULAR FUNCTION, OXYGEN UPTAKE, INTERNAL OXYGEN TRANSPORT AND OSMOTIC BALANCE IN CHINOOK SALMON (*ONCORHYNCHUS TSHAWYTSCHA*) DURING CRITICAL SPEED SWIMMING

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Summary

To examine cardiorespiratory plasticity, cardiovascular function, oxygen consumption, oxygen delivery and osmotic balance were measured at velocities up to critical swimming speed (U_{crit}) in seawater-adapted chinook salmon. We used two groups of fish. The control group had swum continuously for 4 months at a low intensity ($0.5\,BL\,s^{-1}$) and the other was given a high-intensity training regimen (a U_{crit} swim test on alternate days) over the same period of time. Compared with available data for other salmonid species, the control group had a higher maximum oxygen consumption ($\dot{M}_{O_2max}$; $244\,\mu mol\,O_2\,min^{-1}\,kg^{-1}$), cardiac output ($\dot{Q}_{max}$; $65\,ml\,min^{-1}\,kg^{-1}$) and blood oxygen content (Ca_{O_2}; $15\,ml\,O_2\,dl^{-1}$). Exercise training caused a 50 % increase in $\dot{M}_{O_2max}$ without changing either U_{crit} or Ca_{O_2}, even though there were small but significant increases in hematocrit, hemoglobin concentration and relative ventricular mass. During swimming, however, exercise-trained fish experienced a smaller decrease in body mass and muscle moisture, a smaller increase in plasma osmolality, and reduced venous oxygen stores compared with control fish. Consequently, exercise training apparently diminished the osmo–respiratory compromise, but improved oxygen extraction at the tissues. We conclude that the training-induced increase in $\dot{M}_{O_2max}$ provided benefits to systems other than the locomotory system, such as osmoregulation, enabling trained fish to better multitask physiological functions while swimming. Furthermore, because a good interspecific correlation exists between $\dot{M}_{O_2max}$ and arterial oxygen supply ($\dot{T}_{O_2max}$; $r^2=0.99$) among temperate fish species, it is likely that Ca_{O_2} and $\dot{Q}_{max}$ are principal loci for cardiorespiratory evolutionary adaptation but not for intraspecific cardiorepiratory plasticity as revealed by high intensity exercise training.

Key words: salmon, cardiac output, heart rate, oxygen consumption, plasma osmolality, oxygen transport, swimming, exercise training, osmo–respiratory compromise, *Oncorhynchus tshawytscha*.

Introduction

Maximum oxygen consumption ($\dot{M}_{O_2max}$) of rainbow trout (*Oncorhynchus mykiss*) is thought to be closely related to the capacity of the cardiovascular system to transport oxygen (Gallaugher et al., 1995). However, the extent to which internal oxygen transport in fish responds to exercise training, i.e. cardiovascular plasticity, is unresolved because comprehensive *in vivo* studies are lacking. Aerobic exercise training can affect various components of the salmonid cardiovascular system, causing cardiac hypertrophy (Hochachka, 1961; Farrell et al., 1990) and increasing *in vitro* maximum cardiac output ($\dot{Q}_{max}$) (Farrell et al., 1991), hematocrit (Hct; Hochachka, 1961; Zbanyszek and Smith, 1984; Thorarensen et al., 1993), arterial oxygen content (Ca_{O_2}; Thorarensen et al., 1993) and muscle capillarity (Davie et al., 1986; Sänger, 1992). In other words, plasticity has been shown to exist in many of the individual components responsible for internal oxygen convection. These data suggest that, in theory, both arterial oxygen supply to the tissues ($\dot{T}_{O_2}= \dot{Q}\times Ca_{O_2}$, where $\dot{Q}$ is cardiac output) and the arterio–venous oxygen difference (E_{O_2}) could increase with exercise training. However, part of this prediction was not borne out when many of the variables were measured simultaneously for the first time *in vivo* (Thorarensen et al., 1993). After a low intensity aerobic training regimen, chinook salmon (*Oncorhynchus tshawytscha*) responded with only a small improvement in $\dot{T}_{O_2}$ and no effect on either $\dot{M}_{O_2max}$ or critical swimming speed (U_{crit}).

Earlier, Davison (Davison, 1989) concluded that the evidence for training-induced improvements in internal oxygen transport capacity and swimming performance is equivocal, largely because the magnitude of many of the reported changes was small. It is also possible that the exercise training regimens used in previous studies were not always of a sufficient intensity or duration to elicit cardiovascular change. Consequently, we used a high-intensity exercise-training regimen over a 4-month period in an attempt to elicit a maximum cardiorespiratory response before measuring cardiorespiratory performance during a critical swimming speed test.

We also explored the possibility that exercise training can provide benefits beyond those that directly benefit locomotory performance. During swimming in sea water (SW), ionic and osmotic balance are disrupted (Rao, 1968; Rao, 1969; Farmer and Beamish, 1969; Byrne et al., 1972; Wood and Randall, 1973a; Wood and Randall, 1973b; Webb, 1975; Febry and Lutz, 1987) because the functional surface area of the gills (Booth, 1979; see also Wood and Perry, 1985) and the permeability of the gills to ions (Gonzalez and McDonald, 1992; Gonzalez and McDonald, 1994) both increase. This enhanced diffusional exchange of gases, ions and water with the environment is the so-called 'osmo-respiratory compromise' (Randall et al., 1972; Nilsson, 1986), which has been well-studied in freshwater (FW) fish (Gonzalez and MacDonald, 1992; Gonzalez and MacDonald, 1994). In resting rainbow trout, for example, the estimate is that one sodium ion is lost across the gills for every eight molecules of oxygen taken up. However, when $\dot{M}_{O_2max}$ increases during exercise, sodium loss is enhanced more than $\dot{M}_{O_2}$ such that one sodium ion is lost for every five molecules of oxygen taken up (Gonzalez and MacDonald, 1992). In contrast to FW fish, the relationships between osmoregulatory capacity, swimming performance and $\dot{M}_{O_2max}$ have not been well investigated in SW salmon, especially with respect to training effects. Ionic/osmotic imbalances have been linked, however, to reductions in aerobic swimming performance in juvenile salmonids (Houston, 1959; Brauner et al., 1992). Our working hypothesis, given the osmo–respiratory compromise, was that exercise-trained fish with a higher aerobic capacity would be better able to manage the metabolic costs of ionic and osmotic regulation while swimming.

Materials and methods

Experimental animals and training protocol

Fish were derived from a stock of chinook salmon (*Oncorhynchus tshawytscha* Walbaum) that we had studied previously and control fish had been held for 4 months while swimming continuously at a low speed of 0.5 body lengths per second ($BL\,s^{-1}$) (Thorarensen et al., 1993). A full description of the training tanks and fish husbandry is given in Kiessling et al. (Kiessling et al., 1994b). A second group was subjected to a high intensity exercise training protocol as follows. On alternate days during the first month of training, the fish performed a U_{crit} swimming challenge. For this challenge, the fish swam initially at $1\,BL\,s^{-1}$ for 20 min, and then swimming velocity was subsequently increased in steps of $0.5\,BL\,s^{-1}$, each 10 min in duration, until either U_{crit} or $2.5\,BL\,s^{-1}$ was reached. The intensity of the training was then increased during the next 3 months; the fish swam for 20 min at each velocity up to U_{crit}. The exercise training procedure lasted approximately 2 h. In between training sessions, the fish swam at the same speed as the controls. Both groups of fish were fed satiation levels of dry pellets *via* an automatic food dispenser. Based on a sub-sample of 20 fish for each group, significant growth (approximately 15 %; $P<0.05$) occurred in both the control and exercise-trained groups of fish during the 4-month period (Kiessling et al., 1994a). For the control fish, the average initial and final body mass was 343 g and 387 g, respectively, whereas the average initial and final body mass for the exercise-trained fish was 338 g and 387 g, respectively. Fish length at the end of the experiment was 31–33 cm. Water temperature range was 8–10 °C during the training period (November through February).

Surgical procedures

On the day before surgery, fish were individually transferred to a 20 l indoor holding tank continuously flushed with SW at 9–10 °C. Fish were anaesthetised in a chilled solution of 2-pheoxyethanol in SW (1:2,000) and placed supine on an operating sling. Anaesthesia was maintained by continuously irrigating the gills with a solution of 2-phenoxyethanol in chilled SW (1:4,000). A cannula (PE50, Clay Adams, Parsippany, NJ, USA) was inserted into the dorsal aorta (DA) as described by Thorarensen et al. (Thorarensen et al., 1993) and modified from Soivio (Soivio, 1975). The cannula was externalised through the thin skin membrane under the maxillary and filled with heparinised ($150\,i.u.\,ml^{-1}$) saline (0.9 % NaCl). This cannula was used for sampling arterial blood and measuring arterial blood pressure (P_{DA}). A pulsed Doppler flow probe (TMI, Iowa City, IA, USA) was placed around the ventral aorta, just distal to the bulbus arteriosus, to provide a continuous measurement of $\dot{Q}$ (cardiac output; total blood flow in the ventral aorta). The flow probes were made with rigid plastic collars and selected to fit snugly around the vessel. The ventral aorta was accessed via the opercular cavity (Steffensen and Farrell, 1998). A 3–5 mm segment of the vessel was teased free from the surrounding tissue without rupturing the pericardium or obstructing the coronary artery. Silk thread (3–0) was used to suture the leads from the probe to the isthmus and to the side of the fish, behind the cleithrum and just under the lateral line. The leads and the cannula were also anchored in front of the dorsal fin. The entire operation lasted less than 20 min. The fish were allowed to recover for 4–5 h in a 20 l tank and then overnight in a swim tunnel. The total recovery time before experimentation commenced was 24 h. Body mass was measured immediately before transfer to the swim tunnel.

Experimental protocol

The critical swimming test involved incremental velocity

steps in a Brett-type swim tunnel. U_{crit} (cm s^{-1} or BL s^{-1}) and $\dot{M}_{O_2}$ were measured using methods described previously (Thorarensen et al., 1993; Gallaugher et al., 1995). Before the fish started swimming, routine $\dot{Q}$, P_{DA} and $\dot{M}_{O_2}$ values were recorded and an arterial blood sample was taken. Swimming speed was then increased to 1 BL s^{-1} and the sampling procedure was repeated. Subsequently, swimming velocity was increased in steps of 0.25 BL s^{-1}, each step being maintained for 20 min. $\dot{Q}$, P_{DA} and $\dot{M}_{O_2}$ were recorded at each velocity increment after the fish reached a steady state, i.e. approximately 8–10 min after the velocity was increased. As the fish approached U_{crit} (as indicated by 'burst and coast' swimming behaviours in the otherwise steady swimming pattern), a blood sample was taken at each water velocity step. Blood samples at U_{crit} were always drawn while the fish was swimming, which in some cases required a reduction of water velocity by one step (see Gallaugher et al., 1992). After fatigue, $\dot{Q}$, P_{DA} and $\dot{M}_{O_2}$ were recorded following a 1 h recovery period. Consequently, all cardiorespiratory variables were measured under resting conditions, while the fish swam at 1 BL s^{-1}, at 80 % U_{crit}, at 100 % U_{crit} and after a 1 h recovery period. Under resting conditions and during recovery, the water velocity was just sufficient to keep the fish orientated into the water current but stationary on the bottom of the swim tube. After the experiment, fish were anaesthetised to calibrate the flow probe and then sacrificed with a blow to the head prior to body mass and heart mass being measured. Relative ventricle mass (RVM) was calculated as 100×ventricle mass/body mass. All procedures were in accordance with the Canadian Council on Animal Care and approved by Simon Fraser University.

Blood sampling and analytical techniques

For each 1.0 ml arterial blood sample, arterial O_2 tension (P_{aO_2}) and arterial O_2 content (C_{aO_2}), arterial pH (pHa), hematocrit (Hct), hemoglobin concentration ([Hb]) and plasma osmolality were determined. Plasma lactate concentration [La] was measured only at rest, at U_{crit} and during recovery. To prevent anemia as a result of the repetitive blood sampling, 1.0 ml of blood, made up from blood used to measure P_{aO_2} and pHa, any remaining blood from the sample and blood from a normocythemic donor fish, was returned to the experimental fish via the DA cannula.

Measurements of P_{aO_2} were made using a Radiometer (Copenhagen) E5046 P_{O_2} electrode in a D616 cell and whole blood pHa was determined on samples injected into a Radiometer pH microelectrode (type E5021). Both electrodes were regulated at the experimental water temperature and linked to a Radiometer PHM71 acid–base analyzer. A second oxygen electrode system was used to measure water P_{O_2}. C_{aO_2} was measured in 30 μl blood samples using the method of Tucker (Tucker, 1967). Hct was measured in triplicate (20 μl samples drawn into microcapillary tubes) using a Haemofuge (Heraeus Sepatech, Netherlands) centrifuge (10,000 ***g*** for 3 min). Sigma diagnostic kits (Sigma Chemical Co., St Louis, MO, USA) were used to measure blood [Hb] (no. 525A) in 20 μl blood samples and [La] (no. 826-UV) in 100 μl plasma samples. Mean cell hemoglobin concentration (MCHC) was calculated as [Hb]/Hct. Plasma osmolality was measured in triplicate on 10 μl samples using a Wescor (5100) Vapour Pressure Osmometer (Wescor, Logan, UT, USA).

Measurements of muscle dry matter

Analyses of muscle dry matter and ash content were performed on separate samples of control (N=20) and exercise-trained fish (N=20) at the end of their exercise training. Similar analyses were performed for control (N=8) and exercise-trained (N=8) fish after the 1 h recovery from the U_{crit} swim test. These analyses involved drying tissue at 100 °C for 16–18 h (muscle dry matter, % of tissue wet mass) or 3 h at 600 °C (ash content, % of tissue wet mass), as described by Kiessling et al. (Kiessling et al., 1994a).

Possible effects of surgery on oxygen consumption and critical swimming performance

To determine if osmotic disruption during swimming was in some way influenced by tissue damage associated with the placement of the Doppler flow probes, a separate group of exercise-trained fish (N=6) received only a DA cannula for the U_{crit} test in the swim tunnel. In addition, $\dot{M}_{O_2max}$ was measured in separate groups of exercise-trained and control fish that had not been cannulated, and this allowed us to assess the effects of the Doppler flow probe and cannulation procedures.

Calibration of flow probes

Doppler flow probes measure relative changes in $\dot{Q}$. Therefore, each probe was calibrated *in situ* at the end of the U_{crit} experiment with the fish re-anaesthetised. To do this, a Transonic flow probe (Transonic Inc., Ithaca, NY, USA), which measures absolute blood flow, was placed around the bulbus and ventral aortic flow was recorded simultaneously from the Doppler and Transonic flow probes. (Transonic flow probes were not used for the experiments because of their rather larger size. The smaller Doppler flow probes were less likely impair swimming in these fish weighing 300–400 g.) Doppler flow probes were successfully calibrated in six fish from each group. Experiments were attempted on 14 fish for each group, but in some cases the flow probe was not successfully calibrated and in others either blood pressure or hematology measurements were missing. For statistical purposes, only fish that had all variables measured successfully were included in the cardiovascular data analysis. Variables that were measured and not included below were in general agreement with the overall findings.

Data acquisition and measurements of cardiorespiratory variables

P_{DA} was measured with a LDI5 pressure transducer (Narco, Houston, TX, USA) connected to a Grass preamplifier (Model 791J, Grass Instruments, Quincy, MA, USA). The pressure transducer was calibrated daily and regularly referenced to the water level in the swim tunnel during the experiment. The signals from the flow meter, pressure transducer and the

oxygen meter were amplified by a Grass chart recorder (Model 7PCP B, Grass Instruments Quincy, MA, USA) and stored by a computer. The computer sampled signals for blood flow and blood pressure at a rate of 5 Hz. Variables were measured for 6 min and then averaged. Labtech Notebook software (Laboratory Technology Corp., Wilmington, MA, USA) was used to process the signals and to calculate heart rate, f_H.

Calculations of oxygen extraction and systemic vascular resistance

Compared with our experience with rainbow trout, chinook salmon were less tolerant of extensive surgery. Therefore, rather than adding a second cannula to sample venous blood for direct measurements of E_{O_2}, we decided to calculate E_{O_2} as a percentage using the Fick equation ($E_{O_2}=100[\dot{M}_{O_2}/(\dot{Q}\times Ca_{O_2})]$). To preclude possible errors associated with tissue utilisation of oxygen directly from the water, this calculation was only performed at $\dot{M}_{O_2max}$. While the net amount of oxygen delivered to the tissues by the cardiovascular system is equal to $\dot{T}_{O_2}\times E_{O_2}$, tissues such as the skin and the gill epithelia can utilise oxygen directly from the water (Kirsch and Nonnotte, 1977; Daxboeck et al., 1982). Even so, the contribution of this form of oxygen delivery is considered to be minimal when salmonids approach $\dot{M}_{O_2max}$ (Neuman et al., 1983; Thorarensen et al., 1996; Brauner et al., 2000a). Systemic vascular resistance (R_{sys}) was calculated from $R_{sys}=P_{DA}/\dot{Q}$ and the small effect of venous blood pressure on R_{sys} was disregarded.

Statistics

Mean values ± S.E.M. are presented throughout the text and figures and the fiducial limit for accepting significance was $P<0.05$. There was variability in individual swimming performance and therefore swimming speed was normalised to $\%U_{crit}$ to assist in some comparisons. All variables were compared with a three-way ANOVA with individuals, swimming velocity and training level as factors. Mean levels at each swimming speed were compared with a least-square estimate. Statistical comparisons of hematological variables at rest, 1 *BL* s^{-1}, approx. 80 % U_{crit}, U_{crit} and during recovery were made between control and exercise-trained groups using a repeated-measures ANOVA. Changes in hematological variables within each group were analyzed by a paired *t*-test for means. The other variables reported here were statistically analysed using the GLM procedure in SAS (Version 6, SAS Institute Inc., Cary, NC, USA). A significant difference between the control and exercise-trained fish was regarded as a training effect.

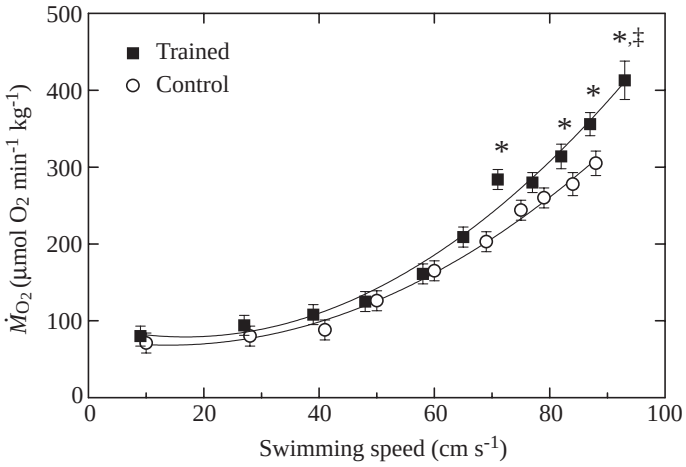

Fig. 1. Changes in $\dot{M}_{O_2}$ as a function of swimming velocity in exercise-trained (N=9) and (N=7) control groups of uncannulated fish. *Significant ($P<0.05$) difference from control fish; ‡significant difference from exercise-trained cannulated fish.

Results

Swimming performance and oxygen uptake

The changes in $\dot{M}_{O_2}$ during swimming are presented for uncannulated fish in Fig. 1. U_{crit} values were not statistically different for control and exercise-trained fish. However, at the higher swimming velocities, $\dot{M}_{O_2}$ for exercise-trained fish was significantly greater than that for control fish. Similarly for cannulated fish, $\dot{M}_{O_2}$ was 50 % higher in exercise-trained

Table 1. *Cardiorespiratory variables from control and exercise-trained chinook salmon before and during critical speed swimming (U_{crit})*

	Control fish		Exercise-trained fish	
Variable	Rest	At U_{crit}	Routine	At U_{crit}
$\dot{M}_{O_2}$ (μmol O_2 min^{-1} kg^{-1})	63±13	244±28*,‡	74±13	366±28*,‡
$\dot{Q}$ (ml min^{-1} kg^{-1})	35.8±4.5	65.6±7.3*	33.6±3.6	65.1±7.9*
V_{SH} (ml)	0.63±0.07	1.04±0.11*	0.63±0.04	0.97±0.13*
f_H (beats min^{-1})	57±4	63±2*	53±2	64±2*
P_{DA} (kPa)	3.2±0.2	4.0±0.2*	3.5±0.3	3.6±0.3‡
R_{sys} (kPa min^{-1} kg^{-1} ml^{-1})	0.095±0.010	0.071±0.015	0.112±0.016	0.058±0.014*
$\dot{T}_{O_2}$ (μmol O_2 min^{-1} kg^{-1})	217±20	393±39*	213±30.9	408±50*
Ca_{O_2} (ml O_2 dl^{-1})	13.8±0.6	13.5±0.6	13.9±0.5	14.1±0.5

Values are means ± S.E.M. (N=6).
*Significant difference from rest value ($P<0.05$); ‡significant difference between control and exercise-trained fish ($P<0.05$).

(366 μmol O_2 kg^{-1} min^{-1}) compared with control fish (244 μmol O_2 kg^{-1} min^{-1}, Table 1), and again U_{crit} values were not significantly different (control, 2.31±0.06 *BL* s^{-1}, *N*=9; exercise-trained, 2.13±0.08 *BL* s^{-1}, *N*=7). Cannulated and uncannulated fish had the same U_{crit} value, but $\dot{M}_{O_2max}$ was significantly higher in uncannulated (413 μmol O_2 kg^{-1} min^{-1}) compared with cannulated (366 μmol O_2 kg^{-1} min^{-1}), exercise-trained fish.

Effect of swimming and training on heart mass and cardiovascular variables

Relative ventricular mass was significantly larger in the exercise-trained fish (0.114±0.003, *N*=12) compared with control fish (0.101±0.003, *N*=12). Although the heart mass of exercise-trained fish was larger, $\dot{Q}$ was not significantly different between exercise-trained and control fish at any swimming velocity (Table 1, Fig. 2A). Routine $\dot{Q}$ was 35.8 ml min^{-1} kg^{-1} and 33.6 ml min^{-1} kg^{-1} for the control and exercise-trained groups, respectively, and with swimming, $\dot{Q}$ increased by 94 % and 83 % to maximum values of 65.5 and 65.1 ml min^{-1} kg^{-1}, respectively (Table 1). $\dot{Q}_{max}$ was recorded at swimming velocities of 90±6 % and 94±1 % of U_{crit} for control and exercise-trained fish, respectively. While routine $\dot{Q}$ and $\dot{Q}_{max}$ were the same in both groups of fish, V_{SH} and f_H increased somewhat differently. At the lower swimming speeds, exercise-trained fish increased V_{SH} (Fig. 2E) to a

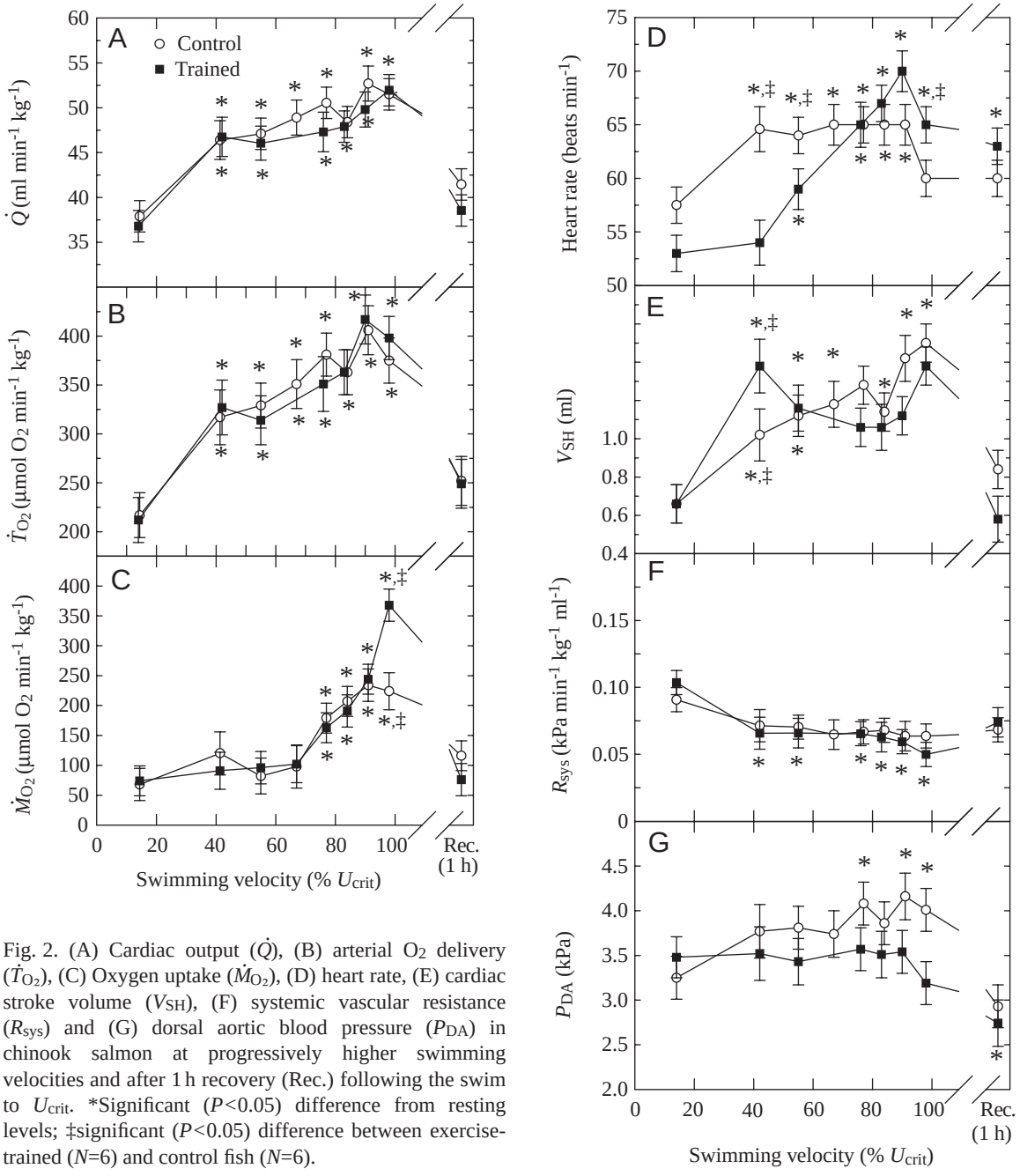

Fig. 2. (A) Cardiac output ($\dot{Q}$), (B) arterial O_2 delivery ($\dot{T}_{O_2}$), (C) Oxygen uptake ($\dot{M}_{O_2}$), (D) heart rate, (E) cardiac stroke volume (V_{SH}), (F) systemic vascular resistance (R_{sys}) and (G) dorsal aortic blood pressure (P_{DA}) in chinook salmon at progressively higher swimming velocities and after 1 h recovery (Rec.) following the swim to U_{crit}. *Significant (P<0.05) difference from resting levels; ‡significant (P<0.05) difference between exercise-trained (*N*=6) and control fish (*N*=6).

Table 2. *Hematological variables for control and exercise-trained groups of chinook salmon*

		Swimming speed				
	Fish	Rest	Approximately 80 % U_{crit}	U_{crit}	Recovery	Mean
Hct (%)	Control	29.7±0.8 (9)	30.6±1.6 (9)	29.1±1.9 (9)	28.4±1.7 (9)	29.4±0.7 (9)
	Exercise-trained	31.8±1.6 (10)	32.9±1.3 (10)	33.1±1.4‡ (10)	31.4±1.3 (10)	32.3±0.7‡ (10)
[Hb] (g dl^{-1})	Control	11.4±0.3 (9)	11.4±0.5 (9)	11.5±0.6 (9)	11.0±0.5 (9)	11.4±0.2 (9)
	Exercise-trained	13.2±0.8‡ (10)	13.7±0.9‡ (10)	12.8±0.6 (10)	12.8±0.7‡ (10)	13.1±0.4‡ (10)
MCHC (g l^{-1})	Control	385±14 (9)	376±11 (9)	399±13 (9)	390±15 (9)	388±7 (9)
	Exercise-trained	413±12 (10)	418±18* (10)	390±17 (10)	384±19 (10)	408±10‡ (10)
Ca_{O_2} (% vol.)	Control	13.7±0.5 (9)	14.3±0.6 (7)	14.0±0.6 (7)	13.8±1.1 (7)	13.9±0.3 (7)
	Exercise-trained	14.9±1.2 (9)	14.9±0.7 (9)	14.4±1.1 (9)	14.7±0.8 (9)	14.7±0.5 (9)
Pa_{O_2} (kPa)	Control	13.0±0.63 (9)	10.9±0.46* (8)	9.72±0.53* (9)	11.2±0.76* (8)	
	Exercise-trained	13.9±0.91 (10)	10.5±0.65*(9)	7.24±0.9*,‡ (7)	11.1±1.23* (7)	
Plasma pHa	Control	7.93±0.03 (9)	7.85±0.01* (8)	7.83±0.05* (8)	7.8±0.03* (8)	
	Exercise-trained	7.91±0.02 (10)	7.86±0.02* (9)	7.77±0.04* (8)	7.82±0.03* (7)	

Salmon were tested at rest, approximately 80 % U_{crit}, U_{crit} and after a 1 h recovery period from U_{crit}.
*Significant difference from values at rest (P<0.05); ‡significant difference between control and exercise-trained values (P<0.05).
Values are means ± S.E.M. (N).

greater degree than control fish (Fig. 2D). Even so, >60 % of the total increase in $\dot{Q}$ associated with critical swimming had occurred at a velocity of 1 BL s^{-1} in both groups of fish (Fig. 2A). At U_{crit}, V_{SH} increased by 61–65 % and f_H by 10–22 % (Table 1).

There was no significant change in P_{DA} with increased swimming velocity in exercise-trained fish, but P_{DA} increased significantly for control fish (Fig. 2G). The significantly lower P_{DA} for exercise-trained fish came about because R_{sys} decreased significantly, whereas R_{sys} did not change significantly in control fish.

Effect of swimming and training on hematological variables

Hematological variables are compared in Table 2. Hct, [Hb], MCHC and Ca_{O_2} did not change significantly with swimming velocity in either group, nor were they significantly different following the 1 h recovery (Table 2). However, swimming induced an arterial hypoxemia in both groups of fish because Pa_{O_2} was significantly reduced at all swimming velocities and during recovery.

Small, but statistically significant training effects were observed for some hematological variables (Table 2). The overall mean values for Hct, [Hb], and MCHC were significantly higher in exercise-trained fish (Table 2). Also, the extent of the arterial hypoxemia at U_{crit} was significantly greater for exercise-trained fish (Table 2). Nevertheless, Ca_{O_2} was unaffected by high-intensity exercise training.

Changes in plasma [La] and pHa during swimming were similar for control and exercise-trained fish. Plasma pHa was decreased significantly at all swimming velocities and during recovery (Table 2). Plasma [La] increased significantly at U_{crit} and increased further still during recovery. However, plasma [La] values were not significantly different between control and exercise-trained fish and were, respectively, at rest:

Table 3. *Body mass and plasma osmolality at rest, at* U_{crit}*, and after recovery for 1 h from* U_{crit} *in control and exercise-trained groups of chinook salmon, and in exercise-trained chinook salmon with only a dorsal aorta (DA) cannula*

		Swimming speed		
	Fish	Rest	At U_{crit}	Recovery
Body mass (g)	Control	372±26 (7)		342±24* (7)
	Exercise-trained	390±17 (8)		369±17* (8)
	Exercise-trained (DA cannula only)	335±22 (7)		319±21* (7)
Plasma osmolality (mosmol kg^{-1})	Control	330±12 (9)	396±12* (9)	370±31 (7)
	Exercise-trained	317±12 (9)	353±17*,‡ (9)	374±20* (9)
	Exercise-trained (DA cannula only)	302±7 (6)	339±11* (6)	

Control and exercise-trained fish received both a DA cannula and a Doppler flow probe implanted on the ventral aorta (VA).
Values are means ± S.E.M. (N).
*Significant difference from rest (P<0.01); ‡significant difference between control and exercise-trained values (P<0.01).

0.4±0.1 mmol l^{-1} (N=5) and 0.8±0.2 mmol l^{-1} (N=9); at U_{crit}: 3.6±0.4 mmol l^{-1} (N=6) and 3.0±0.4 mmol l^{-1} (N=8): after the 1 h recovery: 4.6±0.7 mmol l^{-1} (N=6) and 5.1±0.8 mmol l^{-1} (N=8).

Effect of swimming and training on arterial oxygen transport.

The measured variables associated with arterial oxygen convection are summarised in Table 1. $\dot{T}_{O_2}$ increased during swimming because $\dot{Q}$ increased, while Ca_{O_2} was unchanged. High-intensity exercise training did not have a significant effect on $\dot{T}_{O_2}$ (Fig. 2B); neither $\dot{Q}_{max}$ nor Ca_{O_2} was significantly different between control and exercise-trained fish. The recovery from fatigue did not differ between control and exercise-trained fish in that the recovery values for $\dot{M}_{O_2}$, $\dot{Q}$, R_{sys} and $\dot{T}_{O_2}$ were not significantly different from routine values (Fig. 2). However, f_H was significantly higher and P_{DA} was significantly lower than the pre-exercise values in the exercise-trained, but not the control group.

Given that exercise training increased $\dot{M}_{O_2max}$ and not $\dot{T}_{O_2}$, the improvement in oxygen delivery to tissues came about through an increase in E_{O_2}. At $\dot{M}_{O_2max}$ the calculated E_{O_2} for exercise-trained fish (90 %) was significantly greater than for the control group (62 %) (Table 1). Furthermore, because $\dot{Q}$ and $\dot{T}_{O_2}$ had increase by 50 % at low swimming velocities and there was little change in $\dot{M}_{O_2}$ until fish were swimming at a velocity close to 60–80 % of U_{crit} (Fig. 2), it is likely that E_{O_2} decreased at low swimming velocities.

Effect of swimming and training on water balance

Control and exercise-trained fish lost a similar amount of body water while swimming to the same U_{crit}. Body mass decreased significantly by 8 % and 5 % in control and exercise-trained fish, respectively (Table 3). A similar amount (5 %) of water loss occurred in the fish that had received only a DA cannula (Table 3) and so water loss was not significantly affected by implanting a Doppler flow probe. The loss of body water was reflected in an increase in plasma osmolality. Compared with routine values, plasma osmolality increased significantly at approx. 80 % U_{crit}, U_{crit} and after the 1 h recovery period in both groups of fish (Fig. 4). In addition, both muscle dry matter (Fig. 3) and ash content (1.75±0.03 %, N=8) increased significantly following exercise, compared with fish sampled directly from the training tanks.

Exercise-trained fish had a significantly greater muscle dry matter (Fig. 3) and ash (1.56±0.03 %, N=20 *versus* 1.51±0.03 %, N=20) compared with control fish. In addition,

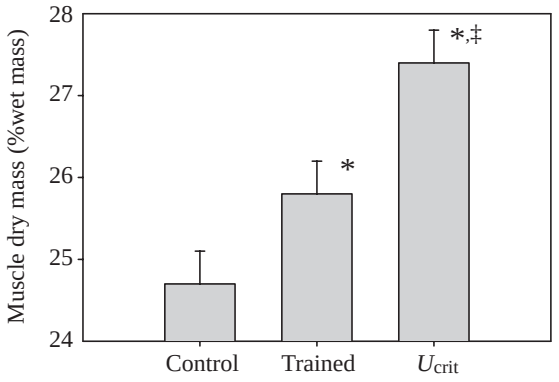

Fig. 3. Muscle dry mass of control and exercise-trained chinook salmon. Fish were sampled either directly from the training tanks (Control and Trained), or 1 h after swimming to U_{crit} in a swim tunnel (U_{crit}). Muscle dry mass for the U_{crit} value represents the combined value for samples from control and exercise-trained chinook salmon, since individually they were not significantly (P>0.05) different from each other. *Significant (P<0.05) difference from control; ‡significant (P<0.05) difference after a swim at U_{crit} (N=8).

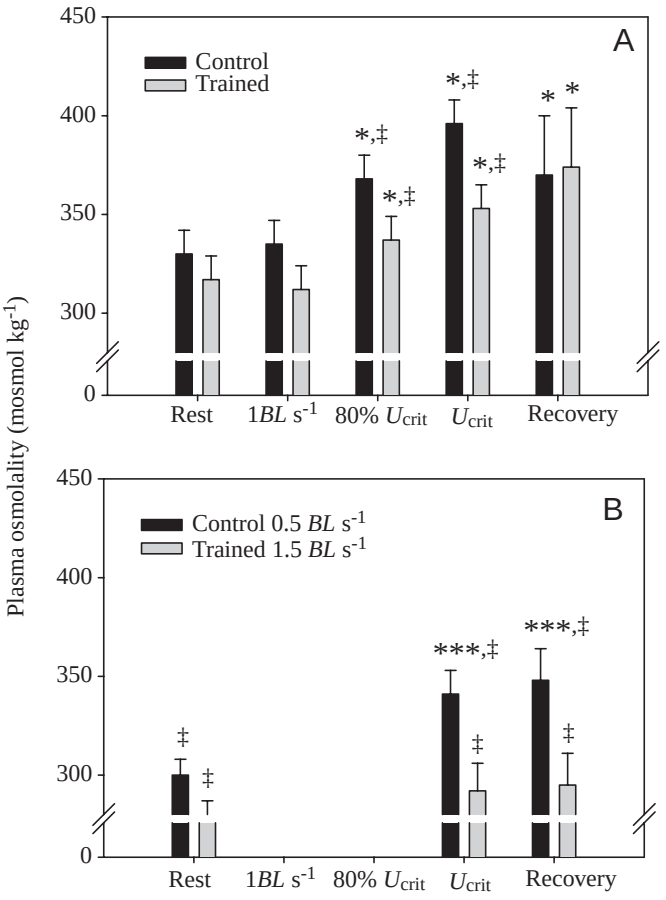

Fig. 4. (A) Changes in plasma osmolality in control (black bars) and exercise-trained (grey bars) chinook salmon at 1 *BL* s^{-1}, at 80 % U_{crit}, at U_{crit}, and after a 1 h recovery following U_{crit}. *Significant (P<0.01) difference compared with the rest value; ‡significant (P<0.01) difference between control and exercise-trained fish. (B) Changes in plasma osmolality in control (black bars; 0.5 *BL* s^{-1}) and exercise-trained (grey bars; 1.5 *BL* s^{-1}) groups of chinook salmon at U_{crit}, and after a 1 h recovery following U_{crit} compared with rest. These fish were exercise-trained at a low intensity in a previous study (Thorarensen et al., 1993) and these new data are included to illustrate that both high intensity and low intensity training regimens can influence the ability of chinook salmon to osmoregulate during activity. ***Significant (P<0.001) difference compared with the rest values; ‡significant (P<0.001) difference between control (N=6–10) and exercise-trained fish (N=6–9).

exercise-trained fish were significantly better at defending their plasma osmolality during exercise (Fig. 4A). Plasma osmolality was significantly lower in exercise-trained fish compared with control fish at approx. 80 % U_{crit} and at U_{crit}. Plasma osmolality at rest and after a 1 h recovery period, however, was not significantly different between control and exercise-trained fish (Fig. 4A). In view of this finding, we measured plasma omolality in stored samples from fish used in our earlier study that employed a less intense training regimen with chinook salmon (Thorarensen et al., 1993). As in the present study, trained fish were better at defending plasma osmolality during swimming (Fig. 4B).

Discussion

Interspecific comparison of internal oxygen convection

Few studies have comprehensively measured cardiovascular status in swimming fish (see reviews by Farrell and Jones, 1992; Bushnell et al., 1992). The present study represents the first such measurements for chinook salmon. Many of the routine cardiovascular variables ($\dot{Q}$, f_H, V_{SH} and P_{DA}) measured at U_{crit} are similar to those measured for other salmonid species. However, $\dot{Q}_{max}$ measured *in vivo* for chinook salmon (65 ml min^{-1} kg^{-1}) was 22 % greater than that measured *in vivo* for rainbow trout (53 ml min^{-1} kg^{-1}; Kiceniuk and Jones, 1977). Chinook salmon also have an elevated value for Ca_{O_2} compared with other salmonids. As a result, $\dot{T}_{O_2max}$ stands out as the highest salmonid value reported to date (Fig. 5). In fact, a good correlation (r^2=0.99) exists between $\dot{T}_{O_2max}$ and $\dot{M}_{O_2max}$ among the relatively few studies with temperate fish species (Fig. 5) and this confirms that Ca_{O_2} and $\dot{Q}_{max}$ can vary significantly and in parallel among fish species (Farrell, 1991; Brill, 1996). Therefore, Ca_{O_2} and $\dot{Q}_{max}$ clearly represent primary loci for the evolutionary adaptations that accompany interspecific differences in $\dot{M}_{O_2max}$. Indeed, tunas are characterised by having exceptionally high values for $\dot{M}_{O_2max}$, Ca_{O_2} and $\dot{Q}_{max}$ compared with other teleosts. $\dot{M}_{O_2max}$ for skipjack tuna may be more than fourfold higher than for chinook salmon (Gooding et al., 1981), while $\dot{Q}_{max}$ is 150–200 ml min^{-1} kg^{-1} (Brill and Bushnell, 1991b) and routine Ca_{O_2} is 19 ml dl^{-1} (Brill and Bushnell, 1991a), increasing to perhaps 25 ml dl^{-1} during exercise (Brill and Bushnell, 1991b). Similar correlations between $\dot{T}_{O_2max}$ and $\dot{M}_{O_2max}$ exist among mammals, and this is taken as evidence that changes in $\dot{T}_{O_2max}$ are required to change $\dot{M}_{O_2max}$ (di Pamprero 1985; Wagner 1993). Thus, selection for a high $\dot{T}_{O_2max}$ among teleost species appears to involve a concurrent expansion of both $\dot{Q}_{max}$ and Ca_{O_2}. This type of evolutionary adaptability in cardiovascular design among fish clearly contrasts with the rather limited cardiovascular plasticity induced by chronic exercise training, as observed here and in earlier studies with salmonids (see Introduction for references). Consequently, the constraints on cardiorespiratory design in fish at the evolutionary and acclimation levels may be qualitatively different.

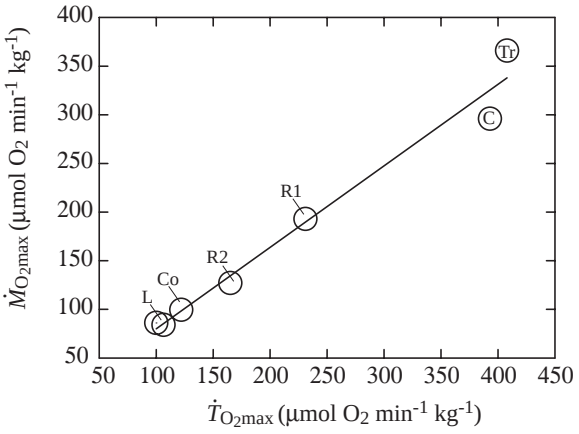

Fig. 5. Maximum oxygen uptake ($\dot{M}_{O_2max}$) as a function of maximum arterial oxygen transport ($\dot{T}_{O_2max}$) measured *in vivo* with swimming fish. Values were obtained from the following sources: chinook salmon from control (C) and exercise-trained (TR) groups, this study; dogfish (D), Piiper et al., 1977; leopard shark (L), Lai et al., 1990; rainbow trout (R1), Kiceniuk and Jones, 1977, and (R2), Thorarensen et al., 1996; and values for Atlantic cod *Gadus morhua* (Co), are based on those from Axelsson, 1988, for $\dot{Q}_{max}$; Soofiani and Priede, 1985, for $\dot{M}_{O_2max}$ and Gallaugher, 1994, for Ca_{O_2}. The equation describing the linear regression is $\dot{M}_{O_2max}$=0.84 $\dot{T}_{O_2max}$ –4.03 (r^2=0.99).

Effects of exercise training on internal oxygen convection

We are the first to comprehensively measure the cardiorespiratory changes associated with chronic, high-intensity exercise training in fish and to delineate the resultant cardiorespiratory benefits. The exercise-training regimen used here produced a clear improvement in $\dot{M}_{O_2max}$. However, this change did not directly benefit locomotory performance in terms of improving U_{crit} and there was no training effect on $\dot{T}_{O_2max}$. Given the increase in $\dot{M}_{O_2max}$, we anticipated that Ca_{O_2} would increase beyond the level observed previously with a lower intensity exercise regimen (continuous exercise training at 1.5 BL s^{-1}, which represented about 60 % U_{crit} or 40 % $\dot{M}_{O_2max}$, increased Hct, [Hb] and Ca_{O_2}; Thorarensen et al., 1993). Instead, we observed smaller training effects on Hct and [Hb] and no training effect on Ca_{O_2}. This result, coupled with the fact that $\dot{T}_{O_2max}$, U_{crit} and $\dot{M}_{O_2max}$ can all be altered with experimental blood doping in SW rainbow trout (Gallaugher et al., 1995), suggests that a routine Hct (32.3 %) may be near an upper limit for chinook salmon under these environmental conditions. As in previous studies (Gallaugher et al., 1992; Thorarensen et al., 1993; Gallaugher et al., 1995), we also observed arterial hypoxemia at U_{crit}. However, the extent of this arterial hypoxemia was greater in the exercise-trained group. This training effect might be related either to the somewhat higher Hct in exercise-trained fish (arterial hypoxemia in rainbow trout was reported to be Hct-dependent; Gallaugher and Farrell, 1998), or to a lower venous oxygen content in exercise-trained fish. Despite an intensified arterial hypoxemia with swimming, Ca_{O_2} was unaffected in exercise-trained fish.

Like Ca_{O_2}, $\dot{Q}_{max}$ was unaffected by intense exercise training.

Therefore, $\dot{M}_{O_2max}$ improved because exercise training improved E_{O_2} rather than $\dot{T}_{O_2max}$. The calculated E_{O_2max} increased from 65 % in control fish to 90 % in exercise-trained fish. Although E_{O_2max} was calculated and probably should be confirmed with direct measurements in future work, our calculations are in line with values reported for other fish species and mammals. For example, E_{O_2max} in exercising rainbow trout was between 65 % and 85 % at U_{crit} (Kiceniuk and Jones, 1977; Brauner et al., 2000a; Brauner et al., 2000b). Similarly, in exercising mammals E_{O_2max} is typically 60–80 % (Taylor et al., 1987; Jones et al., 1989; Longworth et al., 1989; Piiper, 1990), but can reach 80–90 % in muscles during relatively short periods of intense exercise (Richardson et al., 1993). That E_{O_2max} is plastic and can respond to training is a novel finding for fish. Thus, intraspecific cardiovascular plasticity that enhances $\dot{M}_{O_2max}$ in response to training clearly contrasts with the interspecific adaptations in $\dot{Q}_{max}$ and Ca_{O_2} that produce species differences in $\dot{M}_{O_2max}$.

Greater oxygen extraction at the tissues is perhaps not entirely unexpected as a training response, given that exercise training is known to improve capillarity in fish muscles (Davie et al., 1986; Sänger, 1992). Although capillary density was not measured in our fish, two lines of indirect evidence suggest that muscle capillarity could have increased with exercise training. First, the cross-sectional area of the red locomotory muscles, which have a better capillary supply compared to white muscle (Egginton, 1992), was shown to increase relative to that of white muscles in fish with the same training regimen (Kiessling et al., 1994b). Second, the lower R_{sys} at U_{crit} in the exercise-trained group is consistent with more capillary beds being perfused simultaneously. Some of these could be in the skeletal muscle, although a higher intestinal blood flow during swimming (see below) also could contribute to a lower R_{sys}. Increased capillarity increases the diffusional surface area for oxygen and reduces the mean distance between capillaries and mitochondria, both of which would increase oxygen conductance (Weibel et al., 1992). Red skeletal muscle in skipjack tuna is characterised by a high capillary density, a small fibre size and a high mitochondrial volume density (Mathieu-Costello et al., 1992; Mathieu-Costello et al., 1996). In addition, capillary manifolds are present in tuna red muscle and these manifolds increase venular capillary surface area, favouring increased oxygen extraction by the muscle. Increased capillarity also increases the mean capillary transit time of red blood cells, even if $\dot{Q}_{max}$ is unchanged, and so more time is available for the unloading of oxygen. Transit time has been implicated as one of the limitations to oxygen extraction from blood in mammals (Saltin, 1985). In addition to capillary changes, an increase in muscle myoglobin concentration could increase E_{O_2max}. Exercise training is known to increase muscle myoglobin concentrations (Love et al., 1977) and myoglobin is also known to facilitate oxygen transport within the muscle fibres (Gayeski et al., 1985; Bailey and Driedzic, 1986).

The above findings all point to oxygen diffusion between the capillaries and the mitochondria being a significant limiting factor, i.e. the cardiorespiratory system in salmonids may be diffusion-limited rather than perfusion-limited during exercise. This would then explain why exercise training affected E_{O_2max} rather than $\dot{T}_{O_2max}$ in chinook salmon. A training-induced increase in the diffusive surface area of capillaries, the residence time of blood in capillaries, or a myoglobin-mediated facilitated diffusion of oxygen in muscle cells could all have contributed to a higher E_{O_2max} during swimming. This suggestion that oxygen transfer to the tissues is diffusion-limited during exercise in salmon is consistent with the results of blood-doping experiments in rainbow trout. Gallaugher et al. (Gallaugher et al., 1995) found that while blood doping could be used experimentally to improve $\dot{T}_{O_2max}$, the benefits to either $\dot{M}_{O_2max}$ or U_{crit} were rather small whenever Hct was artificially increased above its routine level. Diffusion limitations for oxygen transfer at the gills, however, do not appear to be as severe as at the tissues because Ca_{O_2} was maintained in spite of the swimming-induced arterial hypoxemia, and oxygen transport to the tissues was not adversely affected.

A large proportion of the salmonid heart muscle relies on venous blood for its oxygen supply (Farrell, 1992; Steffensen and Farrell, 1998). Therefore, a potential problem associated with an increase in E_{O_2max} is that the reduction in the amount of oxygen in venous blood might impair myocardial oxygen supply during swimming. Even so, this problem may have been ameliorated in trained chinook salmon because $\dot{Q}_{max}$ was unchanged and P_{DA} was lower in exercise-trained fish. Hence, myocardial oxygen demand, which is directly related to myocardial power output, may have been lower in trained fish. Furthermore, a training effect on the coronary supply to the heart could help alleviate the problem of lower venous oxygen content. We found that relative ventricular mass was plastic and responded to exercise training, albeit in a limited manner. The 10 % increase in relative ventricular mass is consistent with the 12 % observed earlier by Hochachka (Hochachka, 1961), but lower than the unusual 46 % increase observed by Greer Walker and Emerson (Greer Walker and Emerson, 1978). Nevertheless, several studies report only isometric cardiac growth with exercise training (see Farrell et al., 1990 for references). How cardiac remodelling might relate to changes in myocardial oxygen supply and the coronary circulation is unclear.

Effects of exercise training on swimming performance and osmotic balance

Exercise-training effects on U_{crit} are equivocal. For example, several authors have observed positive training effects on U_{crit} (Nahhas et al., 1982; Besner and Smith, 1983; Farrell et al., 1990), but in all cases the improvement in U_{crit} was rather small (<20 %). In contrast, no training effect on U_{crit} was observed in either rainbow trout (Farrell et al., 1991) or chinook salmon (Thorarensen et al., 1993; this study). Undoubtedly, the different responses among training studies reflect, in part, important differences in the intensity and duration of the exercise-training regimens that have been used in the past, as well as in the level of exercise that the control

fish were subjected to. We found no effect of cannulation on U_{crit}, while others have reported that cannulae reduce U_{crit} in rainbow trout (e.g. Kiceniuk and Jones, 1977). We have no explanation for this but there are a number of possibilities. Firstly, our fish were not held stationary in the holding tanks and this may have increased the overall exercise capabilities of the fish. Secondly, chinook salmon have not been held under culture conditions that select for growth rather than athleticism for as many generations as have rainbow trout. Also, surgical techniques have improved over time and this may have minimized the impact of cannulation procedures in more recent studies.

If salmon do not swim much faster when exercise-trained, even when the training regimen is high intensity and for long periods, what then are the benefits of exercise training? Below, we present the idea that exercise-training lessens the osmo–respiratory compromise during swimming.

With swimming and the attendant improvement in gas exchange at the gills, it is well established that there is a somewhat greater and disruptive effect on passive ion movements across the gills of FW fish (Gonzalez and MacDonald, 1992; Gonzalez and MacDonald, 1994). Numerous studies have shown that, as a result of swimming, teleosts dehydrate in SW and hydrate in FW (e.g. Rao, 1969; Farmer and Beamish, 1969; Byrne et al., 1972; Wood and Randall, 1973a; Wood and Randall, 1973b). We used plasma osmolality and tissue water content as measures of osmoregulatory performance during swimming and the changes we observed in SW chinook salmon are consistent with progressive dehydration. Besides the gills, the gut is an important osmoregulatory organ in SW in that it is responsible for the water uptake that counteracts the passive water loss occurring across the gills. Therefore, for dehydration to occur during swimming, water loss *via* the gills must exceed water absorption *via* the intestine. The exact mechanisms by which this imbalance comes about are unknown, but a decrease in gut blood flow could certainly play a role by impairing intestinal water absorption, adding to the problem of increased diffusional losses at the gills. Normally, when fish swim or struggle, gut blood flow decreases (Thorarensen et al., 1993; Farrell et al., 2001), presumably as a mechanism to divert blood flow to locomotory muscles (Randall and Daxboeck, 1982; Thorarensen et al., 1993). Nevertheless, exercise-trained chinook salmon are better able to defend intestinal blood flow during swimming (Thorarensen et al., 1993). Consequently, the finding here, as well as in our earlier study (Thorarensen et al., 1993), that exercise-trained chinook could defend their plasma osmolality while swimming better than control fish, might be explained, in part, by better gut blood flow and water uptake during exercise.

We propose that the higher $\dot{M}_{O_2max}$ values of the exercise-trained fish, in part, reflect an osmoregulatory cost that enabled plasma osmolality to be better maintained despite elevated water loss across the gills. However, exactly what this osmoregulatory cost might be in active fish is difficult to ascertain because estimates are highly variable (see Morgan and Iwama, 1991). Using data from Rao (Rao, 1968) and Farmer and Beamish (Farmer and Beamish, 1969), Webb (Webb, 1975) estimated an osmoregulatory cost of approx. 16 % of the net cost of swimming at U_{crit} for SW-adapted adult rainbow trout and tilapia. A similar osmoregulatory cost of 20 % of the net cost of swimming was reported by Febry and Lutz (Febry and Lutz, 1987) for exercise-trained ($1\,BL\,s^{-1}$ for 3 weeks), SW-adapted hybrid tilapia during prolonged swimming (approximately $2.5\,BL\,s^{-1}$). If we accept these estimates as reasonable for chinook salmon, then it would appear that the 50 % higher $\dot{M}_{O_2max}$ in trained fish would be more than adequate for partially defending plasma osmolality. Consequently, it is likely that functions in addition to osmoregulation also benefited from the training-induced increase in $\dot{M}_{O_2max}$. Other possibilities should include protein synthesis and digestion because exercise-trained chinook salmon can maintain their growth rate despite a higher energy expenditure (Thorarensen et al., 1993; present study). It is also possible that exercise-trained fish had better stamina and could recover from exercise faster because there was less of an oxygen debt, but further experiments would be needed to test these ideas.

Throughout the discussion we have assumed that exercise training was the sole contributor to the observed differences in the trained and control fish. However, this may not be the case. The trained fish were captured by dipnet every other day and this in itself could have contributed to the observed responses. Repeated stress (i.e. the struggling in the dipnet) could have had an additional training effect on the cardiorespiratory system. Similarly, the repeated stress may have desensitized the fish in some way that they were able to perform better in the swim test. Alternatively, the training regime may have reduced the stress response associated with the swim test. Gonzales and MacDonald (Gonzales and MacDonald, 1992) examined the potential effect of acute stress on the osmo-respiratory compromise in FW rainbow trout by injecting adrenaline. They found a short-lived (60 min) but dramatic increase in sodium loss without any change in oxygen uptake, such that one sodium was lost at the gills for every 0.9 oxygen molecules taken up, i.e. a tenfold change compared to resting fish. In the same study, rainbow trout were also shown to be able to physiological adjust to these acute effects on gill ion permeability. For example, after approx. 3 h of continuous swimming at 85 % U_{crit} and 2-6 h after exhaustive exercise, sodium losses were reduced relative to oxygen uptake. Unfortunately the present data cannot be used to resolve the concern about the role stress may have played in the chronic training effects, but future experiments in which stress hormones are measured might be useful in this respect.

To conclude, our observations on training effects suggest that it is perhaps time to present a more integrated perspective of the potential benefits of exercise training to fish. Foremost, an intense and chronic exercise training regimen was needed to elicit a 50 % improvement in $\dot{M}_{O_2max}$. While this in itself is not large, the resultant benefits to critical swimming speed and arterial oxygen transport were smaller still. Direct benefits to

swimming performance may be limited to increases in muscle mass, capillary density and associated cellular changes, and these contribute to a lower vascular resistance and, more importantly, an increase in oxygen extraction by tissues. At the same time, exercise-trained fish apparently are better able to multitask other physiological processes (e.g. osmoregulation, ionic balance, digestion, growth, etc.) during locomotion and may not sustain as high an oxygen debt when swimming. An obvious benefit of better multitasking abilities for exercise-trained fish would be a more rapid recovery from the physiological disruptions associated with swimming. A more rapid recovery could have important ecological and survival benefits for fish that are athletically more fit. Multitasking of physiological functions is not a new idea in fish physiology. Brill (Brill, 1996) and Korsmeyer et al. (Korsmeyer et al., 1996) suggested the cardiorespiratory adaptations seen in tuna, which support a heightened $\dot{M}_{O_2max}$, not only permit continuous swimming, but also permit oxygen supply to other metabolic functions, three of which are known to have especially high rates (somatic and gonadal growth, digestion and recovery from exercise). However, we suggest that, whereas in tuna evolutionary adaptations have improved $\dot{T}_{O_2max}$, improved $\dot{M}_{O_2max}$ and multitasking as a result of training reflects an improvement in tissue oxygen extraction. This difference could arise because oxygen delivery may be diffusion-limited during exercise. Consequently, cardiorespiratory plasticity primarily alters the diffusion conditions for oxygen transfer, while evolutionary adaptation involves perfusion factors that determine $\dot{T}_{O_2max}$. Ultimately, substantial increases in $\dot{T}_{O_2max}$ cannot occur without also increasing the workload of the heart through increases in blood viscosity and $\dot{Q}_{max}$. Yet, cardiac plasticity is apparently quite limited, with typically only a 10 % increase in ventricular mass in response to exercise training. In contrast, intraspecific variation in ventricular mass and the coronary circulation is extensive among fishes (Farrell, 1991).

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