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# SEASONAL TEMPERATURE ACCLIMATISATION OF RAINBOW TROUT: CARDIOVASCULAR AND MORPHOMETRIC INFLUENCES ON MAXIMAL SUSTAINABLE EXERCISE LEVEL

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## Summary

**Adult rainbow trout (mass 600–1500 g; length 40–44 cm) were held in the laboratory for up to 28 days at seasonally appropriate temperatures. The maximal sustainable aerobic exercise level (involving slow-twitch muscle activity alone) was determined by following muscle recruitment patterns using electromyography. The mean (± S.E.M.) speeds recorded for maximal sustainable aerobic exercise were 0.52±0.02, 0.81±0.06 and 0.39±0.02 *BL* $s^{-1}$ (body lengths per second) for animals swimming at their acclimatisation temperatures of 4, 11 and 18 °C, respectively. Thus, fish acclimatised to 11 °C reached the highest maximal sustainable (purely aerobic) levels of exercise. They had similar stride lengths to the 4 °C animals, but generated less thrust per stride, as indicated by increased tail-beat frequency. Acclimatisation to 4 °C led to an increased mass of slow muscle and more effective tail beats (greater stride length at lower frequencies), relative to animals at higher temperatures. Fish acclimatised to 18 °C had the lowest stride length and a reduced aerobic swimming capacity.**

**Sustainable levels of aerobic exercise were reflected in unchanged values for mean heart rate and arterial blood pressure between rest and imposed, graded exercise. Radiolabelled microspheres were used to determine cardiac output ($\dot{V}$b) and regional blood flow distribution simultaneously in fish, both at rest and while swimming, for each acclimatisation temperature. Fish acclimatised to 11 °C had the greatest scope for increasing $\dot{V}$b. This resulted in a significant hyperaemia in slow muscle upon exercise (10-fold increase), without an active redistribution of flow from other tissues. Maximum $\dot{V}$b at 18 °C did not differ significantly from that at 11 °C but, because resting $\dot{V}$b was higher, the scope was reduced and was similar to that found at 4 °C. Specific blood flow to the active muscle was also reduced and this, together with decreased blood oxygen content and reduced slow muscle mass, may limit aerobic swimming performance at 18 °C.**

Key words: rainbow trout, *Oncorhynchus mykiss*, aerobic swimming performance, critical swimming speed, electromyography, acclimatisation.

## Introduction

Swimming activity in fishes has been categorised as: (1) ‘sustained’, defined as that maintained for longer than 200 min and not involving fatigue; (2) ‘prolonged’, maintained for between 15 s and 200 min and possibly ending in fatigue; or (3) ‘burst’, maintained for less than 20 s (Beamish, 1978). The standard laboratory technique for comparing the swimming performances of different fishes is to determine their critical swimming speed ($U_{crit}$; Brett, 1964). Determining $U_{crit}$ involves subjecting fish to stepwise increases in swimming speed until they fatigue and cease to hold station which, by definition, means they have then recruited some anaerobic (fast-twitch glycolytic or white) muscle fibres. Since sustainable swimming involves only the use of aerobic muscle (primarily slow-twitch oxidative or red fibres), this will represent a fraction of $U_{crit}$ that will reflect both lifestyle and individual body form. For example, maximum aerobic power output is sufficient to provide the thrust to power swimming at all speeds up to 80–90 %$U_{crit}$ in salmonids, but only up to 30–50 %$U_{crit}$ in cyprinids, where significant anaerobic metabolism is involved during steady performance (Jones, 1982).

The limits for sustainable (purely aerobic) exercise and the subsequent recruitment of anaerobic fibres are affected by laboratory acclimation to different temperatures (see reviews by Sidell and Moerland, 1989; Videler, 1993). The comprehensive study of Sisson and Sidell (1987) studying muscle fibre recruitment in striped bass acclimated to 25 and 9 °C showed

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that, at the lower temperature, fish had lower tail-beat frequencies but greater stride lengths. They concluded for cold-acclimated fish that either they were capable of transmitting more power into propulsion with each stroke or that less power was required to attain any given swimming speed. An increase in red muscle mass is a general response of temperate fishes to lower temperatures (Sidell and Moerland, 1989) which reduces the actual load borne by any individual slow fibre, shifting it to the left (higher contraction velocity) on its intrinsic force–velocity curve. This, in turn, contributes to an expansion of the limits of sustained swimming at cold environmental temperatures (Rome *et al.* 1985; Sisson and Sidell, 1987). In addition to these physiological factors, hydrodynamic analyses have shown that changes in the viscosity of water within the physiological temperature range may affect swimming performance. Briefly, frictional drag depends on kinematic viscosity arising in the velocity gradient of the boundary layer and increases with decreasing temperature owing to the greater water viscosity ($0.89\times10^{-6}\,m^2\,s^{-1}$ at 25 °C *versus* $1.52\times10^{-6}\,m^2\,s^{-1}$ at 5 °C). However, increased viscosity also dampens disturbances of flow at the boundary layer, delaying separation which, in turn, decreases the pressure drag (resulting from distortion of flow) experienced by the swimming fish at low temperatures (reviewed by Sidell and Moerland, 1989; Videler, 1993). However, these considerations are complicated by the probable existence of microturbulence in the swim tunnel. Additionally, since these data are based solely on laboratory acclimation studies, there is a clear need for comparative studies on fish seasonally acclimatised to various temperatures.

The physiological limits to sustainable exercise are most often discussed in terms of an adequate local provision of ATP for cellular respiration, especially regarding the adaptive changes in activity of enzymes from pathways of intermediary metabolism (Johnston, 1982). The role of the cardiovascular system may be equally important. In addition to its central role in oxygen transport, provision of adequate blood flow to working muscle is essential for effective mobilisation of fuels from remote depots and removal of metabolic waste products that may otherwise adversely affect force production by muscle fibres. The main aim of this study was therefore to determine how changes in sustainable exercise levels at different temperatures may be related to the performance of the cardiovascular system, for which few data are available. Direct measurements of cardiac stroke volume and cardiac output in fish have employed a variety of methods, including dye-dilution techniques, ultrasonic flow probes and labelled microspheres. The latter two techniques have been used to measure regional blood flow distribution and cardiac output simultaneously in three studies on intact rainbow trout (Stevens and Randall, 1967; Neumann *et al.* 1983; Wilson and Egginton, 1994). Unlike previous studies, where fish were only acclimated to different temperatures once they had been brought into the laboratory and kept under a fixed light regime, the present study investigated the response of swimming performance and associated cardiovascular changes to environmental temperature in rainbow trout seasonally acclimatised to summer (18 °C), autumn (11 °C) and winter (4 °C) temperatures when held under ambient photoperiod.

## Materials and methods

### *Experimental animals*

Adult rainbow trout, *Oncorhynchus mykiss* (Walbaum), were obtained sequentially from the same stock held at Leadmill Trout Farm, Hathersage, Derbyshire, during 1991–1993, when ambient temperatures (±2 °C) and experimental temperatures (±1 °C) were 18 °C (August to early September), 11 °C (late October to November) and 4 °C (January to early March). Fish mass (809.7±51.3 g, 18 °C group; 822.0±50.8 g, 11 °C group; 1118.4±119.6 g, 4 °C group; ANOVA, $P\geqslant0.05$) and total length (40.7±1.5 cm, 18 °C; 42.0±1.5 cm, 11 °C; 43.6±2.1 cm, 4 °C; $P\geqslant0.05$) followed the progression of one cohort through seasonal growth, although there was no systematic variation in any parameter with size over this relatively small range. Water temperature during transportation did not vary by more than 2 °C. The fish were held indoors for up to 28 days (minimum 14 days) to recover from transportation, in large circular tanks holding 600 l of continuously flowing aerated, dechlorinated tapwater maintained at the appropriate experimental temperature (±1 °C). Throughout this period, fish were exposed to natural photoperiods and fed a maintenance diet of commercial trout pellets. Feeding was stopped 2 days prior to surgical intervention to avoid a post-prandial elevation in metabolic rate and thereby aid subsequent recovery.

### *Surgical procedures*

Fish were anaesthetised by immersion in equimolar bicarbonate-buffered tricaine methanesulphonate (MS222: $100\,mg\,l^{-1}$). Each fish was then transferred to an operating table, where an oxygenated solution of buffered anaesthetic (MS222: $50\,mg\,l^{-1}$) was recirculated over the gills. The dorsal aorta was cannulated as described by Soivio *et al.* (1972). Electromyogram (EMG) electrodes (four per fish) were constructed from 1 m lengths of insulated copper wire (0.2 mm diameter); 2 mm of insulation was removed at one end, which was bent to form a hook and placed, with the aid of a hypodermic needle, into the relevant muscle block. Two electrodes were implanted into the superficial slow (red) muscle and two into the deep fast (white) muscle located 1 cm below the posterior edge of the dorsal fin, on the side contralateral to the red muscle EMG electrodes. Each pair of electrodes was sutured to one side of the body surface just posterior to the dorsal fin of the fish, allowing enough slack to enable free movement without pulling. Placement of the electrodes was checked at autopsy. A further group of animals from the same acclimation batch underwent a second cannulation. These fish, after the above surgical procedures (dorsal aorta cannulation and EMG electrode implantation), underwent cannulation of the caudal artery. A small hole was made using a hypodermic needle just posterior to the anal fin, and cannulae (Portex; i.d. 0.58 mm, o.d. 0.96 mm) with a sharpened metal insert (trocar) were inserted at an oblique angle

(approximately 45 °) and advanced to the spine. Again, the positions of cannulae were checked at autopsy. The cannula was flushed with heparinised saline and sutured in place, just ventral to the lateral line for easy access, and capped. Following surgery (30–40 min), the fish were allowed to recover for 48 h prior to experimentation in a Blazka-type water-channel maintained at the appropriate temperature (±0.5 °C) and at a velocity which enabled the fish to maintain position without prominent swimming movements. The total volume of the channel was 320 l and it was supplied with water from a thermostatted reservoir tank (200 l). The test channel was 115 cm in length and 19 cm in diameter with a current generated by a plastic propeller, belt-driven from a 1.1 kW speed-controlled motor (Brock Crompton Parkinson Motors). Actual water speed through the test section was calibrated against a Braystoke BFM 002 current flowmeter positioned in the centre of the water flow (accuracy, given as relative standard deviation of water velocity, was 2.2 % at 50 cm $s^{-1}$). Baffles (smaller plastic tubes) were placed in front of the propeller and at the end of the test channel to create approximately laminar, non-macroturbulent flow; this was confirmed by monitoring the path of dye introduced into the channel.

*Determination of maximal sustainable exercise levels*

The maximal sustainable exercise level was determined in a manner similar to that described by Wilson and Egginton (1994). Briefly, the EMG electrodes were connected to a pen recorder (George Washington Oscillograph 400 MD/2) with minimum disturbance to the fish, and then left for 1 h. The swimming protocol involved increasing the water speed in 5 cm $s^{-1}$ increments (approximately 0.1 body lengths per second, *BL* $s^{-1}$) every 30 min until bursts of EMG activity were recorded from the fast (white) muscle. As soon as this threshold ($U_{wm,crit}$) was established, the water velocity was reduced until all fast muscle activity disappeared. The limit for sustainable (aerobic) swimming was taken as one 5 cm $s^{-1}$ increment below that at which fast muscle recruitment was evident, and the fish was swum at this level for a further 30 min.

In a preliminary experiment to assess the effects of cannulation on swimming performance, four different fish were swum at 11 °C with only the EMG electrodes implanted. Fast muscle recruitment speeds, and subsequently the limits for sustainable (aerobic) exercise, were recorded (0.67±0.07 and 0.58±0.08 *BL* $s^{-1}$, respectively). After 48 h, these fish underwent a dorsal aortic cannulation and the procedure was repeated. Fast muscle recruitment occurred at 0.83±0.13 *BL* $s^{-1}$, and the limit for sustainable exercise was 0.64±0.09 *BL* $s^{-1}$ (values similar to those for fish with EMG electrodes alone, $P \geqslant 0.05$). Subsequent addition of a second (caudal artery) cannula to the same fish also produced no significant deleterious effect on the swimming performance (fast muscle recruitment speed 0.94±0.13 *BL* $s^{-1}$; maximal sustainable exercise level 0.80±0.09 *BL* $s^{-1}$, $P \geqslant 0.05$). Indeed, familiarity with the swimming tunnel would appear to overshadow any drag effect in determining the speed at which fast muscle is recruited.

*Swimming speed, heart rate and blood pressure*

Tail-beat frequency ($f_{TB}$) was measured by counting the bursts of EMG activity on the red muscle trace for 1 min in every alternate 2 min period and was initially confirmed by actual counts of tail beats (one beat is one cycle back and forth of the tail) visually recorded at the same time. Stride length ($L_S$) was calculated by dividing swimming speed (*BL* $s^{-1}$) by $f_{TB}$ (beats $s^{-1}$) to obtain distance (as a fraction of body length) travelled per tail beat. Heart rate and dorsal aortic blood pressure were continuously recorded during the exercise period *via* an external pressure transducer (Druck, model PDCR 75/2; S/N 107z) and data were collected for 1 min in every alternate 2 min period (as for $f_{TB}$). Mean arterial blood pressure ($\bar{P}a$) was calculated as (1×systolic + 2×diastolic)/3.

*Determination of regional blood flow and cardiac output*

Blood flow distribution and cardiac output were determined both at 'rest' and at the limit for sustainable exercise using the microsphere method. The protocol followed was similar to that described elsewhere (Neumann *et al.* 1983; Wilson and Egginton, 1994) and involved injecting $^{46}$Sc- and either $^{113}$Sn- or $^{57}$Co-radiolabelled microspheres (15±1.6 $\mu$m diameter, specific activity $3.05\times10^{7}$–$4.50\times10^{7}$ Bq $g^{-1}$; NEN-DuPont) *via* the dorsal aortic cannula, whilst a reference sample was simultaneously withdrawn from the caudal artery *via* a Braun syringe pump (Perfusor IV) at a rate of 0.65 ml $min^{-1}$ (to allow estimation of the cardiac output and absolute blood flows by the indicator dilution technique). Pilot experiments showed that a 1 min withdrawal was ample time to allow for complete microsphere trapping by the tissues (all microspheres should be trapped in the first pass of the circulation). The above procedure was repeated using microspheres with a different label once the fish had attained the maximal sustainable (aerobic) limit. The animal was then killed by an overdose of anaesthetic (MS222), injected *via* the dorsal aortic catheter, and the following organs were dissected out and weighed: ventricle, spleen, liver, gonad and intestine (stripped of fat and food). Samples of deep fast and lateral slow muscles were obtained from three positions along the length of the body and also from both sides of the body (the latter a test of adequate mixing of the microspheres in the blood). Gill samples were also taken to provide an estimate of non-trapping of the microspheres in the peripheral microcirculation and of the involvement of arterio-venous shunts (Buckberg *et al.* 1971; Heymann *et al.* 1977).

Microsphere activity in the above samples was determined using a multi-channel gamma counter (Packard Auto-Gamma 5650). All sample activity (cts $min^{-1}$) were corrected for background radioactivity and crossover between channels. Any samples with counts corresponding to less than 400 microspheres were discarded at this point to avoid the possibility of large errors in the estimation of blood flow (Buckberg *et al.* 1971). Blood flow rate $\dot{V}$ (ml $min^{-1}$ $g^{-1}$) in a particular tissue was calculated by scaling tissue to reference activities:

$$\dot{V} = \dot{V}p a_s / a_b , \quad (1)$$

where $\dot{V}$p is the speed of the pump (ml min$^{-1}$), $a_s$ is sample activity and $a_b$ is activity of withdrawn blood (both in cts min$^{-1}$).

Cardiac output ($\dot{V}$b; ml min$^{-1}$ g$^{-1}$) was calculated by dividing the total cts min$^{-1}$ injected by the withdrawn blood cts min$^{-1}$. Determination of the percentage activity in the gills reflects the extent of arterio-venous shunting or untrapping, and is determined by:

$$\text{percentage of total counts in gill} = \frac{\dot{V}_G}{\dot{V}b/M_g} \times 100, \qquad (2)$$

where $\dot{V}_G$ is gill blood flow (ml min$^{-1}$ g$^{-1}$), $\dot{V}$b is cardiac output (ml min$^{-1}$ g$^{-1}$) and $M_g$ is gill mass (g). The percentage of total counts in the gill never exceeded 10 %, and rarely 5 %. Cardiac minute work (J kg$^{-1}$ min$^{-1}$) was calculated by multiplying $\dot{V}$b by the mean arterial blood pressure ($\bar{P}$a), and stroke work (J kg$^{-1}$ beat$^{-1}$) was derived. Individual conductances for the various tissues were calculated by dividing blood flow rate to a particular tissue by the $\bar{P}$a at the moment of microsphere injection, in order to normalise data for inter-animal variation in $\bar{P}$a.

*Tissue masses*

At the end of the experiment, each fish was killed and the superficial slow muscle, which forms a triangular wedge along the side of the fish, was removed. Care was taken not to include superficial fast muscle or subcutaneous fat. The deep fast muscle was then dissected free of bone and weighed. This technique does not take into account the small (red) muscle fibres that occur in the bulk of the fast myotomes. However, since these only become active during bursts of activity at intermediate cruising speeds (Webb, 1971) and are a relatively small proportion of total muscle mass (around 5 %), no correction was made. Ventricle, gills and gonads were also removed and weighed, and scales removed from the anterior and mid regions of the fish above the lateral line were used for age determination.

*Statistical analyses*

Data were analysed using analysis of variance (ANOVA) for treatment effects, and Scheffe's *F*-test for *post-hoc* multiple comparisons. Differences were considered significant when $P \leqslant 0.05$. Tail-beat frequency and swimming speed data were related by least-squares linear regression; where appropriate,

Table 1. *Mean values for the gonosomatic index, white muscle mass, cardiac (ventricle) muscle mass and total gill mass, expressed as a percentage of body mass, and red muscle mass expressed as a percentage of total muscle mass, from rainbow trout acclimatised at seasonally appropriate temperatures*

| Acclimatisation temperature (°C) | GSI (%$M_b$) | $M_w$ (%$M_b$) | $M_c$ (%$M_b$) | $M_g$ (%$M_b$) | $M_r$ (%$M_w$) |
|---|---|---|---|---|---|
| 4 | 0.48±0.15* (5) | 45.12±3.25 (6) | 0.13±0.02 (7) | 1.58±0.19 (6) | 5.23±0.34* (6) |
| 11 | 0.10±0.02* (8) | 49.00±0.84 (9) | 0.10±0.01 (12) | 1.45±0.08 (12) | 4.28±0.33 (9) |
| 18 | 3.06±0.34 (5) | 45.91±0.74 (7) | 0.09±0.01 (5) | 1.55±0.08 (5) | 3.95±0.27 (7) |

Values are means ± S.E.M. (*N*).

* indicates a significant difference from the value for the 18 °C group; $P \leqslant 0.05$.

GSI, gonosomatic index [gonad mass (g)/body mass (g)]; $M_w$, white muscle mass; $M_c$, cardiac (ventricle) mass; $M_g$, total gill mass; $M_r$, red muscle mass; $M_b$, body mass.

Table 2. *Mean values for the mass-specific absolute blood flow rate and conductance of blood to a number of organs and tissues in the rainbow trout at rest*

| | 4 °C | | 11 °C | | 18 °C | |
|---|---|---|---|---|---|---|
| | Blood flow | Conductance | Blood flow | Conductance | Blood flow | Conductance |
| Ventricle | 1.2±0.7 (5) | 0.3±0.1 (3) | 2.5±2.1 (8) | 0.1±0.1 (6) | 3.3±2.8 (9) | 0.5±0.4 (9) |
| Spleen | 8.4±4.2 (7) | 1.7±0.8 (5) | 12.8±7.9 (8) | 0.5±0.3* (8) | 27.7±16.0 (5) | 3.7±2.3† (5) |
| Intestine | 8.4±6.5 (8) | 1.3±1.1 (8) | 10.2±4.5 (9) | 0.8±0.4 (8) | 25.5±5.4 (7) | 1.9±0.5 (7) |
| Gonad | 0.5±0.3 (7) | 0.1±0.1 (5) | 0.5±0.4 (8) | 0.03±0.01 (5) | 2.4±1.8 (7) | 0.4±0.3 (5) |
| Liver | 4.9±2.8 (6) | 1.1±0.7 (4) | 1.5±0.7 (7) | 0.1±0.1 (6) | 11.4±8.0 (6) | 2.4±1.9 (4) |
| Red muscle | 2.4±0.3 (7) | 0.3±0.04† (7) | 12.3±3.0 (8) | 1.1±0.3 (8) | 10.1±1.4 (9) | 0.8±0.5 (9) |
| White muscle | 0.6±0.08*,† (8) | 0.07±0.01* (7) | 1.7±0.3 (7) | 0.1±0.01* (7) | 2.3±0.3 (9) | 0.3±0.03† (8) |
| Skin | 0.5±0.1*,† (8) | 0.09±0.03* (5) | 1.7±0.3* (8) | 0.1±0.08* (7) | 3.9±0.5† (8) | 0.4±0.05† (8) |

Values are means ± S.E.M. (*N*).

* indicates a significant difference from the value for the 18 °C group; † indicates a significant difference from the value for the 11 °C group; $P \leqslant 0.05$.

Blood flow is given in ml min$^{-1}$ 100 g$^{-1}$ and conductance in ml min$^{-1}$ 100 g$^{-1}$ dPa$^{-1}$.

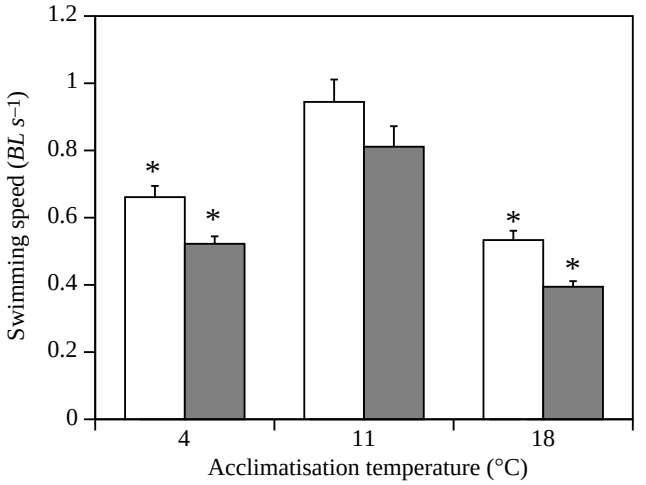


Fig. 1. The swimming speed (in $BL\,s^{-1}$) of white muscle recruitment (open bars) and the aerobic limit (maximum sustainable speed, filled bars) at three acclimatisation temperatures. Values are mean + S.E.M. For the 4 °C and 11 °C groups, $N$=17; for the 18 °C group, $N$=21. In all cases, an asterisk (*) indicates a significant difference from the value for the 11 °C group; $P \leqslant 0.05$.

exponential models were used. Values are reported as means ± S.E.M.

## Results

### *Experimental animals*

The ages of the fish changed throughout the experimental period (5.0±0.32 years for the 4 °C group; 4.1±0.14 years, 11 °C group; 4.2±0.2 years, 18 °C group; $P<0.05$). Cold-acclimatised animals were older because of sequential sampling of the same stock. At 18 °C, the gonosomatic index [gonad mass (g)/body mass (g), expressed as a percentage of body mass] significantly increased ($P \leqslant 0.0001$) from below 0.5 % (4 and 11 °C groups) to above 3 % (18 °C group), representing a sixfold increase in this index of reproductive investment at 18 °C (Table 1) when gonads filled the entire depth of the abdominal cavity. However, condition factor [(body mass/length$^3$)×100] remained similar (1.3±0.05, 4 °C group; 1.1±0.09, 11 °C group; 1.2±0.06, 18 °C group).

### *Tissue mass*

The mass of white muscle and gills, expressed as a percentage of body mass, did not differ with acclimatisation temperature (Table 1). The relative ventricular mass, however, showed a small but not statistically significant increase with decreasing temperature, while the relative proportion of slow (red) muscle increased significantly at 4 °C (Table 1).

### *Determination of maximal sustainable exercise levels*

The aerobic limit (maximal sustainable speed; $BL\,s^{-1}$) followed the same trend as that of white muscle recruitment (Fig. 1), peaking at 11 °C (0.81±0.06 $BL\,s^{-1}$), the middle of the temperature range examined. Sustained fast muscle recruitment was recorded, on average, at a swimming speed 0.14±0.02 $BL\,s^{-1}$ greater than the swimming velocity subsequently adopted for maximal aerobic swimming (i.e. confirming that maximal aerobic activity was taken just below the level that fast muscle becomes active).

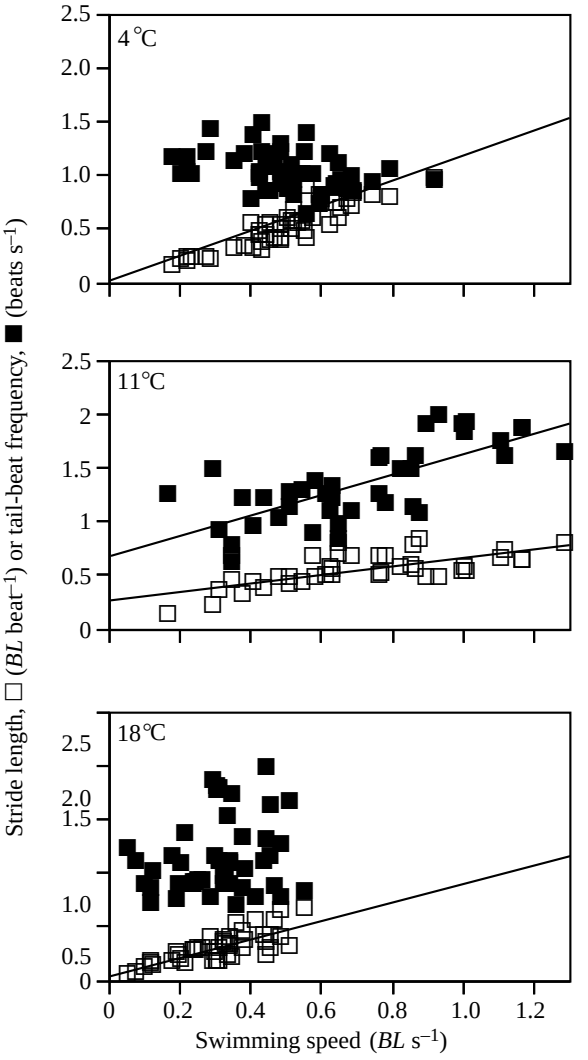


Fig. 2. Mean tail-beat frequency ($f_{TB}$, beats s$^{-1}$; filled squares) and mean stride length ($L_S$, $BL$ beat$^{-1}$; open squares) plotted against swimming speed ($U$; $BL\,s^{-1}$). Best-fitting linear regression lines are plotted where significant ($P \leqslant 0.05$). (A) 4 °C group ($N$=49); $L_S$=1.17$U$–0.0743; $r^2$=0.81. (B) 11 °C group ($N$=42); $f_{TB}$=0.945$U$+0.667; $r^2$=0.52 and $L_S$=0.353$U$+0.291; $r^2$=0.41. (C) 18 °C group ($N$=46); $L_S$=0.868$U$+0.024; $r^2$=0.58.

### *Electromyogram analysis*

At 4 °C, as swimming velocity increased, stride length ($L_S$) increased linearly ($r^2$=0.8), with no apparent change in tail-beat frequency ($f_{TB}$; Fig. 2A). Stride length declined as an exponential function of $f_{TB}$ ($L_S$=1.96$f_{TB}^{-1.41}$; $r^2$=0.5) such that at lower frequencies there was a clear increase in $L_S$. This latter relationship was not observed at higher temperatures, where $L_S$ remained constant over the range of $f_{TB}$ values recorded. At

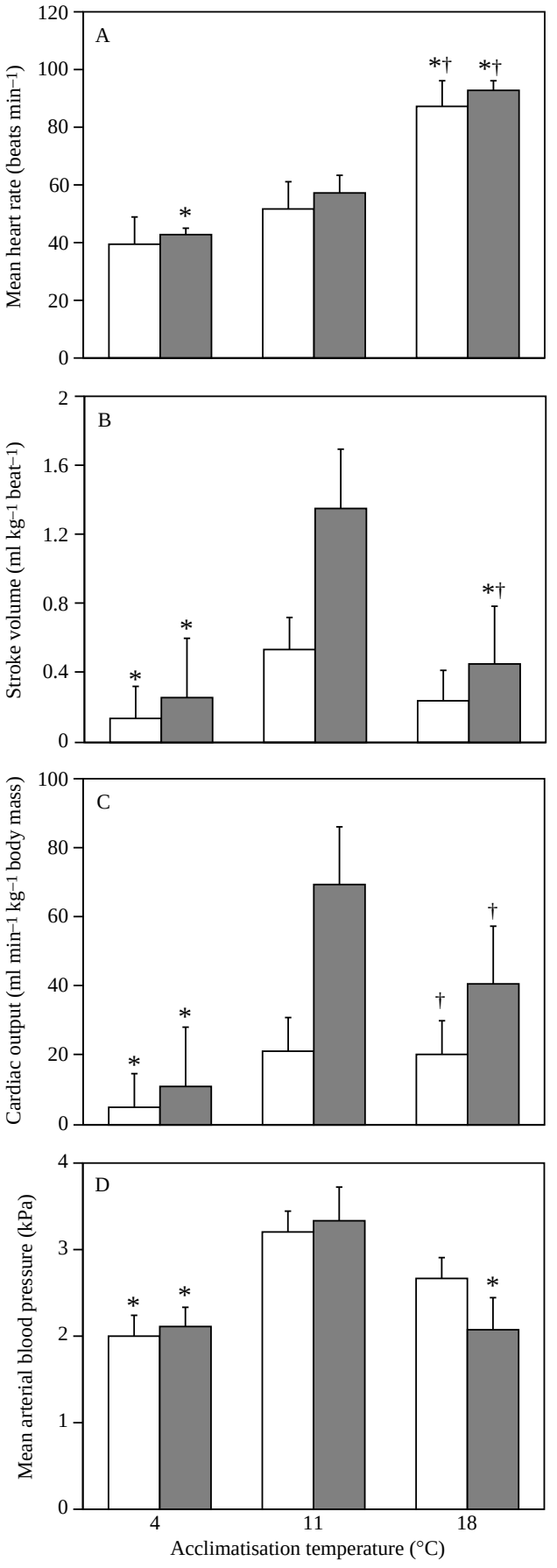


Fig. 3. Changes in cardiovascular performance at rest (open bars) and at the aerobic limit (filled bars) plotted at three acclimatisation temperature. (A) Mean heart rate ($f_H$; beats $min^{-1}$), (B) stroke volume ($V_S$; ml $kg^{-1}$ $beat^{-1}$), (C) cardiac output ($\dot{V}b$; ml $min^{-1}$ $kg^{-1}$ body mass) and (D) mean arterial blood pressure ($\bar{P}a$; kPa). Values are mean + S.E.M. For each temperature class, $N$=8. In all cases, an asterisk (*) indicates a significant difference from the value for the 11 °C group and a dagger (†) indicates a significant difference from the value for the 4 °C group; $P \leqslant 0.05$.

11 °C, both $f_{TB}$ and $L_S$ increased linearly with swimming velocity ($r^2$=0.5 and 0.4, respectively; Fig. 2B), although the regression slope of the latter was less steep than that at 4 °C. At 18 °C, $L_S$ again varied linearly with swimming speed ($r^2$=0.6), approaching a regression slope similar to that of the 4 °C-acclimatised animals (Fig. 2C) but, as with the 4 °C-acclimatised animals, the regression between swimming velocity and $f_{TB}$ was not significantly different from zero.

### *Cardiovascular responses*

#### *Resting fish*

There were no progressive changes in heart rate ($f$H) or mean arterial blood pressure ($\bar{P}a$) over each 30 min speed increment at any temperature, apart from a transient response to altered swimming speed which was not statistically significant. Repeated measurements were therefore combined to give averaged values for individual fish. Heart rate increased significantly with temperature from 37.8±1.4 beats $min^{-1}$ at 4 °C to 91±4.8 beats $min^{-1}$ at 18 °C ($P \leqslant 0.03$; Fig. 3A), with an apparent $Q_{10}$ of 1.5 between 4 and 11 °C, and 2.1 between 11 and 18 °C. Stroke volume ($V_S$; Fig. 3B) and cardiac output ($\dot{V}b$; Fig. 3C) showed a relative thermal independence between 11 and 18 °C (no significant differences), a pattern also seen for cardiac minute work (0.06 and 0.02 J $kg^{-1}$ $min^{-1}$ at 11 and 18 °C, respectively). Stroke work was highest at 11 °C (0.001 J $kg^{-1}$ $beat^{-1}$), but declined at 18 °C so that it was not significantly different from that at 4 °C, being only 20 % of the value at 11 °C. However, $\dot{V}b$ increased significantly from 4 to 11 °C (5.04±1.06 and 21.0±5.0 ml $min^{-1}$ $kg^{-1}$, respectively; $P \leqslant 0.05$). Compensatory changes in $\dot{V}b$ were modest at 18 °C (20.1±4.1 ml $min^{-1}$ $kg^{-1}$), leading to an apparent $Q_{10}$ for $\bar{P}a$ of 2.0 between 4 and 11 °C, and of 0.80 between 11 and 18 °C (Fig. 3D).

#### *Swimming fish*

Swimming at the limit for sustainable (aerobic) exercise did not significantly change heart rate or $\bar{P}a$ from resting values within thermal groups (Fig. 3A,D). Although $V_S$ increased 1.8-fold and 1.9-fold at 4 and 18 °C, respectively (Fig. 3B), the increase from 0.53±0.2 ml $kg^{-1}$ $beat^{-1}$ at rest to 1.35±0.4 ml $kg^{-1}$ $beat^{-1}$ upon exercise at 11 °C was most striking ($P \leqslant 0.06$), and the exercise value at 11 °C was significantly greater than the exercise values at 4 and 18 °C ($P \leqslant 0.05$). Cardiac minute work and stroke work followed a similar pattern to that of $V_S$, with the greatest increase with exercise occurring at 11 °C (3.5-fold and 2.9-fold increases,

respectively; $P<0.001$). Little change from resting values was found at either 4 or 18 °C, and although cardiac minute work at 18 °C was fivefold greater than that at 4 °C ($P=0.10$), the stroke work was similar. During maximal sustainable exercise, mean $\dot{V}$b increased 2.2-fold above resting levels at 4 °C and twofold at 18 °C, but $\dot{V}$b increased even more at 11 °C, from 21.0±5.0 ml min$^{-1}$ kg$^{-1}$ at rest to 69.3±18.8 ml min$^{-1}$ kg$^{-1}$ (Fig. 3C). The increase at 11 °C was significantly greater than that at 4 °C ($P\leqslant0.05$), but was not greater than that at 18 °C owing to the higher resting cardiac output.

### *Regional blood flow*

#### *Resting fish*

Blood flow distribution to all organs except slow (red) muscle appeared to be higher at 18 °C than at 4 °C (Table 2). These differences were only significant for fast (white) muscle and skin, where specific blood flow rates were 0.6±0.08 and 0.5±0.1 ml min$^{-1}$ 100 g$^{-1}$, respectively, at 4 °C and 2.3±0.3 and 3.9±0.5 ml min$^{-1}$ 100 g$^{-1}$, respectively, at 18 °C ($P\leqslant0.05$). These differences reflect those in $\bar{P}$a of individual fish, as conductance was similar at all three temperatures for most tissues examined (Table 2).

#### *Swimming fish*

At the limit of sustainable (aerobic) exercise, there was a significant increase in specific blood flow rate to slow (red) muscle at 11 °C (13.73±2.80 times resting blood flow), which was significantly greater than the increase at both 4 and 18 °C (4.57±0.58 times and 3.54±0.52 times resting levels, respectively; $P\leqslant0.05$). Exercise caused no significant changes to any other tissue examined at 11 °C (compare Tables 2 and 3), as blood flow rate to tissues simply increased in parallel with $\bar{P}$a (i.e. no change in conductance). At 4 °C, exercise caused no significant changes in specific blood flow when compared with resting values, although the increase in blood flow rate to liver (2.05±0.34 times resting blood flow) was somewhat greater ($P\leqslant0.06$) than that at 11 °C (0.61±0.29 times resting blood flow). At 18 °C, there was some increase ($P\leqslant0.07$) in both the specific blood flow and conductance to fast muscle (2.27±0.30 times resting blood flow; 2.15±0.26 times resting conductance); these increases were slightly greater than those at 11 °C (1.46±0.19 times and 1.44±0.20 times, respectively; $P\leqslant0.05$). Conductance of the skin also increased upon exercise at 18 °C, relative to values at 4 and 11 °C, though not significantly so (Table 3).

## Discussion

The present values obtained for fast muscle recruitment speed ($U_{wm,crit}$) were within the range reported in a previous study on similarly operated, untrained rainbow trout (0.5–1.5 *BL* s$^{-1}$; Kiceniuk and Jones, 1977), where the effects of surgery and the combined drag of catheters and electrodes were considered to be significant. In the present study, there was no significant increase in drag (effect on swimming performance) when the fish were cannulated in addition to having EMG electrodes implanted, indicating that the EMG electrodes were the major source of drag. Despite this, the range for the maximal sustainable (aerobic) limit obtained in the present study (0.39–0.81 *BL* s$^{-1}$; 74–86 %$U_{wm,crit}$) compares well with values for unrestrained swimming obtained from continuous monitoring of lake-dwelling brown trout *Salmo trutta* using ultrasonic tags, which gave a range of 0.15–1.0 *BL* s$^{-1}$ during sustained swimming (Ross *et al.* 1980; Schulz and Berg, 1992). Solomon (cited in Butler, 1985) recorded speeds of 0.5–1.0 *BL* s$^{-1}$ for 3–4 h periods in sea trout migrating up rivers. Sustained swimming in salmonids is predominantly (up to 80 %$U_{crit}$) effected by aerobic slow muscle, with no electrical activity recorded from mosaic fast muscle (Webb, 1971; Jones, 1982; Randall and Daxboeck, 1982). Kiceniuk and Jones (1977) recorded little change in blood pH at swimming speeds below 91 %$U_{crit}$, indicating that exercise was predominantly aerobic.

Table 3. *Mean values for the mass-specific absolute blood flow rate and conductance of blood to a number of organs and tissues in the rainbow trout at the limit for sustainable (aerobic) exercise*

| | 4 °C | | 11 °C | | 18 °C | |
|---|---|---|---|---|---|---|
| | Blood flow | Conductance | Blood flow | Conductance | Blood flow | Conductance |
| Ventricle | 2.6±1.9 (5) | 0.3±0.3 (4) | 6.8±5.2 (8) | 0.7±0.5 (5) | 10.9±6.8 (9) | 1.6±1.3 (9) |
| Spleen | 7.6±3.9 (7) | 1.2±0.5 (6) | 1.5±0.4* (8) | 0.1±0.03 (6) | 36.1±24.3† (6) | 5.7±3.6 (5) |
| Intestine | 15.0±6.9* (8) | 1.5±0.7 (8) | 21.1±9.2 (9) | 2.9±2.0 (8) | 40.3±10.4 (7) | 5.5±2.4 (7) |
| Gonad | 0.2±0.1 (7) | 0.04±0.01 (5) | 0.5±0.4 (8) | 0.03±0.01 (5) | 4.1±3.5 (7) | 0.8±0.7 (5) |
| Liver | 3.7±1.7 (7) | 0.7±0.1 (5) | 0.9±0.7 (7) | 0.1±0.07 (5) | 16.8±10.0 (6) | 3.5±2.0 (4) |
| Red muscle | **8.9±2.0**† (6) | **0.9±0.1**† (6) | **120.8±27.4*** (8) | **7.3±1.6*** (8) | **22.4±4.4**† (9) | **2.5±0.5**† (9) |
| White muscle | 0.9±0.1*,† (8) | 0.09±0.01* (7) | 2.2±0.4 (7) | 0.3±0.04 (7) | 3.0±0.4 (9) | 0.4±0.07 (8) |
| Skin | 0.6±0.1* (8) | 0.09±0.01* (5) | 1.3±0.2* (8) | 0.1±0.03* (7) | 4.1±0.5† (8) | 0.5±0.1† (8) |

Values are means ± S.E.M. (*N*).

* indicates a significant difference from the value for the 18 °C group; † indicates a significant difference from the value for the 11 °C group; $P\leqslant0.05$.

Values highlighted in bold type are significantly greater than the corresponding values at rest; $P\leqslant0.05$.

Blood flow is given in ml min$^{-1}$ 100 g$^{-1}$ and conductance in ml min$^{-1}$ 100 g$^{-1}$ dPa$^{-1}$.

The preferred swimming speeds of rainbow trout estimated in the present study are therefore well within their aerobic scope. In addition, the aerobic limit was greatest at 11 °C, the middle of the acclimatisation temperature range examined. Brett (1964) also found the greatest aerobic limit at the middle of the temperature range studied (approximately 13 °C), for the sockeye salmon *Oncorhynchus nerka*, over a range of 5–24 °C. Aerobic metabolic rate may be limited at higher temperatures by a number of factors, such as (a) decreased oxygen solubility in water and plasma; (b) decreased total arterial oxygen content and haematocrit at 18 °C (Taylor *et al.* 1993), which would limit peripheral oxygen supply; (c) increased energy expenditure on seasonal reproductive effort (Table 1), which would reduce the overall energy available for swimming; (d) reduced slow muscle mass at 18 °C (Table 1), which would lead to fast (anaerobic) muscle being recruited at a relatively slower swimming velocity. Sisson and Sidell (1987) showed that these and other factors culminate in fish acclimated to higher temperatures being less capable of transmitting power into propulsion with each fin stroke.

At lower temperatures, the decrease in mechanical power output of aerobic muscle forces the recruitment of anaerobic fibres at a relatively slower swimming speed, and swimming endurance decreases. This has been referred to as compression of recruitment order (Rome *et al.* 1985). A possible compensatory mechanism would be to increase the volume of aerobic muscle at lower temperatures, and an increase in red muscle mass was, in fact, recorded (1.33-fold increase between 18 and 4 °C; Table 1). Egginton and Sidell (1989) showed that a similar increase in striped bass was due to fibre proliferation (hyperplasia) as well as to an enlargement of existing cells (hypertrophy). As a consequence, when red muscle is maximally recruited, the load is distributed over a greater number of red muscle fibres in cold-acclimated animals. The result is an expansion of the limits of tail-beat frequency and/or swimming speed for a cold-acclimated animal compared with that of a warm-acclimated animal subjected to acute cooling (Rome *et al.* 1985; Sisson and Sidell, 1987). Similar results were obtained with brown trout (*S. trutta*) that had undergone a period of training (subjected to a water velocity of 0.25 m $s^{-1}$, equivalent to 0.54–0.83 *BL* $s^{-1}$) in holding tanks prior to experimentation where $U_{crit}$ values were 1.85–2.18 *BL* $s^{-1}$ over the temperature range 5–15 °C (Butler *et al.* 1992). However, by studying untrained rainbow trout that have been seasonally acclimatised, and not just temperature-acclimated, we have demonstrated that this compensation is incomplete as the aerobic limit was reduced at both 4 and 18 °C.

### *Kinematics of swimming*

Fish from this population vary their stride length (*L*S) in a regular manner in response to increasing water velocity at all temperatures, albeit with different slopes (Fig. 2). At 4 °C, the greatest *L*S values were generated by low tail-beat frequencies ($f_{TB}$), which may reflect a greater effectiveness of swimming, possibly achieved by increases in tail-beat amplitude (Bainbridge, 1958) or height of the trailing edge of the caudal fin (Sidell and Moerland, 1989). While the mean $f_{TB}$ range (1.03–1.35 beats $s^{-1}$) is in agreement with that of Ross *et al.* (1980), who found that the preferred rate in free-swimming brown trout *S. trutta* of similar size was 1–2 beats $s^{-1}$, only at 11 °C (close to their optimal temperature) does this translate into a predictable contraction frequency among individuals at a given speed (Fig. 2B), as found by other workers (Hunter and Zweifel, 1971; Sisson and Sidell, 1987). The absence of such a relationship at either 4 or 18 °C suggests that not all individuals acclimatise to non-optimal temperatures in an identical manner and, therefore, that individuals require different contractile frequencies to meet the demands of increasing water velocity. However, Bainbridge (1958) described the frequency–speed relationship below 5 beats $s^{-1}$ as curvilinear, possibly because the fish were modulating their tail-beat amplitude at low frequencies, which makes discussion of stride length problematic. For example, over this restricted range of swimming speeds, it is difficult to distinguish statistically between a truly linear change in *L*S and the near-linear portion of a monotonic curve, such that *L*S may eventually reach an asymptote if swimming speed were increased beyond that appropriate for sustainable activity.

Although the pressure drag acting upon the fish will decrease at lower temperatures, the limit for sustainable (aerobic) exercise was also low at 4 °C, despite any increase in swimming effectiveness and slow muscle mass. J. D. Altringham, C. I. Smith and C. S. Wardle (unpublished results) found that steady swimming speeds increased with an increase of $f_{TB}$ and recruitment of more lateral muscle. Since an increase of $f_{TB}$ did not occur with swimming speed for the 4 °C trout, they would experience a compression of recruitment order, thereby limiting aerobic performance. In addition, the frictional component of the total drag would still be high compared with that of warm-acclimatised animals, which may also have an impact on the power requirements of swimming. Fish acclimatised to 11 °C attained the highest sustainable aerobic limit but, although the average *L*S was not significantly different from that of the 4 °C animals, they achieved a higher aerobic swimming capacity through increasing $f_{TB}$, reflecting a declining effectiveness of swimming as water velocity increased. Stride length at 18 °C appeared to be reduced (58 %, $P>0.05$) compared with that of the 4 °C-acclimatised animals. This agrees with work on striped bass (Sisson and Sidell, 1987) and indicates that fish at higher temperatures are less capable of transmitting power into propulsion with each stroke, resulting in 18 °C-acclimatised trout having a lowered aerobic limit compared with those acclimatised to 11 °C, in principle showing a similar compression of recruitment order as for the 4 °C group.

### *Cardiovascular responses*

Stable values for heart rate and mean arterial blood pressure indicated that sustainable levels of aerobic exercise were being studied at all temperatures. Both variables have been shown to increase up to 15 min after an increase in swimming velocity, returning to pre-exercise levels within 30 min, in a variety of

fish species (Stevens and Randall, 1967; Kiceniuk and Jones, 1977; Randall and Daxboeck, 1982; Butler, 1985). Heart rate increased with acclimatisation temperature, reflecting the intrinsic thermal sensitivity of biological systems and possibly also compensating for a reduced oxygen-carrying capacity in the blood (Taylor *et al.* 1993). Apparent $Q_{10}$ values were within the ranges previously reported for intact fish (1.3–1.6 for winter flounder *Pseudopleuronectes americanus* from 5 to 15 °C, Cech *et al.* 1976; 2.6 for rainbow trout from 5 to 12 °C and 1.5 from 12 to 20 °C, Wood *et al.* 1979; and 1.8 for the largescale sucker *Catostomus macrocheilus* from 5 to 16 °C, Kolok *et al.* 1993). At all temperatures examined, cardiac output increased upon maximal sustainable exercise from resting levels, as found for most fish, with the exception of short-finned eels *Anguilla australis schmidtii* (Davie and Forster, 1980). The greatest increase in cardiac performance above resting levels occurs at 11 °C, and this may be more than sufficient to compensate for an apparent reduction in swimming effectiveness compared with 4 °C-acclimatised animals. The increased cardiac output (3.3-fold) was mainly due to a significant increase in stroke volume (2.5-fold) above resting levels but did not, however, lead to an increase in mean arterial blood pressure, implying a decrease in total peripheral resistance at 11 °C. Fish at 18 °C are clearly unable to maintain high stroke volumes and, therefore, despite a further tachycardia, $\dot{V}$b and $\bar{P}$a are severely compromised. The lack of a chronotropic response with exercise implies that an increase in cardiac output upon exercise was met in all groups by an inotropic response and, because this did not lead to an increase in $\bar{P}$a, it follows that an increased $\dot{V}$b was matched by a decrease in the total peripheral resistance. Our values for resting cardiac output in rainbow trout above 4 °C are within the range reported for other resting fish, measured by a variety of techniques (11.4–62.5 ml $min^{-1}$ $kg^{-1}$; 5–18 °C). The strong thermal dependence of $\dot{V}$b was illustrated by Johansen *et al.* (1968), who recorded values of 40–70 ml $min^{-1}$ $kg^{-1}$ in the electric eel *Electrophorus electricus* at a temperature of 28–30 °C, which approaches those of resting mammals at 37 °C. Resting $\dot{V}$b increased with water temperature in the winter flounder *P. americanus* over the temperature range 5–15 °C ($Q_{10}$=2.70; Cech *et al.* 1976), in the rainbow trout over the temperature range 6–18 °C ($Q_{10}$=2.60; Barron *et al.* 1987) and in the largescale sucker *C. macrocheilus* over the temperature range 10–16 °C ($Q_{10}$=2.01; Kolok *et al.* 1993). We did not observe the linear relationship between $\dot{V}$b and temperature found by Barron *et al.* (1987), working on rainbow trout held at 6, 12 or 18 °C, although the latter study used the indicator dilution method with Evan's Blue dye, which is a less accurate method for determining $\dot{V}$b (Heymann *et al.* 1977). Kolok *et al.* (1993) determined $\dot{V}$b over a range of seasonal acclimatisation temperatures using ultrasonic flow probes on the largescale sucker *C. macrocheilus* and suggested that, rather than a linear relationship, 'pockets of thermal insensitivity' may occur in some species. This was found to be the case for stroke volume in the present study: while the apparent $Q_{10}$ was 1.5 between 4 and 11 °C, it was 0.3 between 11 and 18 °C. In contrast, Kolok *et al.* (1993) found the region of thermal insensitivity to occur between the two lower temperatures (5 and 10 °C, apparent $Q_{10}$=0.4).

The scopes of cardiac output and cardiac minute work were significantly reduced at 4 °C compared with values at 11 and 18 °C, reflecting the temperature-dependent decrease in heart and metabolic rates. A possible compensatory mechanism would be to increase inotropism by ventricular hypertrophy (Graham and Farrell, 1990), which may result in greater ventricular filling and hence stroke volume. Although a small increase in cardiac muscle mass was recorded at 4 °C compared with 18 °C (Table 1; $P$>0.05), there was no significant increase in stroke volume upon exercise at 4 °C, indicating that increased wall thickness had failed to compensate for reduced contractility. At 18 °C, scope for cardiac output and cardiac minute work were also reduced compared with the 11 °C values. This may, in part, reflect a significant decrease in total arterial oxygen content at 18 °C (Taylor *et al.* 1993) and contribute to a reduced aerobic limit.

The present values for absolute blood flow to various tissues at rest are similar to those obtained previously using radiolabelled microspheres (Cameron, 1974; Neumann *et al.* 1983; Wilson and Egginton, 1994). The only other study to examine the effects of seasonal acclimatisation temperature on relative blood flow distribution (Barron *et al.* 1987) reported that perfusion of fast muscle alone was influenced (increased with temperature). They suggested that such an increase in relative flow to fast muscle maintained blood pressure during temperature-dependent changes in cardiac output by decreasing peripheral resistance. Unlike Barron *et al.* (1987), we found no temperature-dependent increase in cardiac output between 11 and 18 °C, and so increases in flow to skin and fast muscle (Table 3) may represent active regulation to accommodate an increased metabolic rate at higher temperatures. Increased blood flow to fast muscle at 18 °C would also facilitate mobilisation of protein from degenerating muscle fibres to the hypertrophying gonads, a mechanism described by Weatherley and Gill (1987). Blood flow to the gonads was increased at 18 °C, presumably reflecting greater amounts of energy expended on reproductive effort. This may limit the scope for activity at these submaximal swimming speeds, although some additional increase in intestinal blood flow with exercise confirms the lack of major adrenergic tone and suggests that hypoperfusion of muscle is unlikely to be the sole cause of reduced swimming performance. However, reduced swimming velocities *per se* may not be maladaptive. Hoar and Randall (1978) concluded that a low mean swimming speed may have ecological significance by increasing the energetic effectiveness of migration owing to a decrease in the cumulative cost of transport.

All tissues examined showed an active increase in blood flow with maximal sustainable (aerobic) exercise apart from the gonads, whose blood flow simply reflected mean arterial blood pressure (similar conductance). At 11 °C, there was a 10-fold increase in flow to slow muscle upon maximal sustainable exercise, and a decrease in total peripheral resistance (see above), which was sustained without any major redistribution

of blood from other tissues. Increased stroke volume can be achieved by greater cardiac filling (Frank–Starling mechanism), by increasing the muscle-pump effect *via* increased tail-beat frequencies and/or by redistribution of blood *via* sympathetic vasoconstriction to increase venous pressure (Stevens and Randall, 1967; Randall and Daxboeck, 1982). There is evidence of some vasoconstriction occurring at the level of the spleen and liver (conductance decreased upon exercise), suggesting that blood may be being actively diverted from these organs, although splenic contraction did not occur as haematocrit was unchanged (data not shown). Animals acclimatised to 4 °C had an attenuated slow muscle hyperaemia, reflecting the reduced scope of cardiac output, although the increased volume of slow muscle may offer partial compensation. Also, arterial blood had an increased oxygen content and haematocrit (Taylor *et al.* 1993); therefore, restricted blood flow to working muscle is unlikely to be the only factor limiting aerobic activity at low temperatures, which also reflects swimming effectiveness. 18 °C-acclimatised fish also had a significantly lower blood flow to slow muscle during sustained exercise than did those at 11 °C. A reduced volume of slow muscle and total arterial oxygen content coupled with higher metabolic rate suggests that, in this case, limited perfusion may be a significant factor in determining swimming capacity at higher temperatures.

In conclusion, this study demonstrates that swimming and cardiovascular performance are not linearly related to seasonal temperature. Trout alter their gross swimming characteristics at different temperatures, possibly ameliorating cardiovascular limitations to sustained activity in the cold. Although the fish acclimatised to 4 °C were apparently more efficient at sustainable aerobic swimming than those acclimatised to higher temperatures, fish acclimatised to 11 °C achieved the highest aerobic limit by increasing tail-beat frequency and cardiac output. This allowed a 10-fold increase in blood flow to the working muscle, achieved mainly through passive redistribution of blood flow (steal effect) and an increased tail-beat frequency (muscle-pump effect). 18 °C-acclimatised fish had the least effective swimming performance and a reduced aerobic limit due to physiological constraints (impaired cardiac output). Thus, trout appear to be optimised for sustained swimming performance in the middle of their thermal range which may, in part, explain why migration is rarely observed at the seasonal extremes.

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