Differences in Exercise Physiology between Two Atlantic Cod (*Gadus morhua*) Populations from Different Environments

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Accepted 10/27/93

Abstract

*Little attention has been given to the study of physiological variation within species of fish. This study examines whether a population of Atlantic cod (*Gadus morhua*) found in the brackish Bras d'Or Lakes of Nova Scotia, Canada, has different physiological characteristics than a nearby Atlantic ocean cod population. Chronically cannulated cod from both populations were exercised to exhaustion in a swim-tunnel respirometer. Although exercise performance was identical between populations, the physiological bases of that performance differed substantially. Cod from the Bras d'Or system had greater metabolic, ventilatory, and cardiac rates as well as a greater estimated resting metabolic rate. The Bras d'Or population used a greater proportion of anaerobic metabolism in attaining its maximal performance level as surmised from a relatively greater metabolic acid-base disturbance and higher lactate levels during recovery from exercise in the Bras d'Or population. Bras d'Or cod also had generally more dilute plasma and lower levels of hemoglobin in its erythrocytes than conspecifics from the Atlantic Ocean. These results are discussed as possible clues to the physiological adjustments necessary for cod to succeed in the Bras d'Or system.*

Introduction

Evolutionary biologists and population geneticists have long held an appreciation for the considerable degree of variation within a species (e.g., Clarke 1979). Yet, despite the close ties between organismal performance

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Physiological Zoology 67(2):330–354. 1994. 0031-935X/94/6702-9302$02.00

physiology and one component of natural selection, mortality selection (Arnold 1983; Endler 1986), few physiological studies on natural populations have focused on intraspecific variation (Burggren and Bemis 1990). Nelson (1990) and Nelson and Mitchell (1992) have recently demonstrated substantial intraspecific variation in the exercise physiology of feral, freshwater fish populations. These studies and others (e.g., Thomas and Donahoo 1977; Giles 1991) suggest that, for freshwater fish, like some invertebrates (see, e.g., Pierce, Rowland-Faux, and O'Brien 1992; Singh and Long 1992), "population" or "strain" may be a more appropriate unit of physiological definition than "species." An interesting question is whether this finding applies to fish in general or to populations of a wide-ranging marine fish like Atlantic cod.

The Atlantic cod (*Gadus morhua*) is an interesting species in which to look for intraspecific variation in physiological performance. Although genetic analyses have suggested small variability throughout its broad North Atlantic range (Mork et al. 1985), people familiar with the cod fishery have long recognized discrete stocks over regional scales based on morphological differences. For example, "The cod-fish which occupy the banks lying between the latitudes of 41 and 45, are very different on the different banks, and are kept so distinct, and are so similar on the respective banks that a man acquainted with the fishing business will separate those caught on one bank from those caught on another with as much ease as we separate the apple from the pear" (Lincoln 1791). This perception was generally verified by analyses of meristic characters and, to a lesser degree, biochemical characters (see discussions in Love [1970, 1980]); whether these intraspecific differences have a genetic basis or whether they have any implications for differential survival in the respective regions is unknown. Since Atlantic cod habitats can vary considerably over regional scales, we were interested in determining whether populations of cod occupying these disparate habitats differed in their exercise physiology.

The Bras d'Or Lakes in Nova Scotia, Canada, are an excellent system in which to look for population-level physiological divergence in cod populations. Faunal interchange between the Bras d'Or Lakes and the Atlantic Ocean proper is probably limited by the constrictions in available depth strata, the narrowness of the channels, and the strong tidal currents through the Great Bras d'Or and the Little Bras d'Or (Krauel 1975; fig. 1). Finally, the Bras d'Or system has a lower salinity and is characterized by a more heterogenous and stochastic environment in terms of temperature and salinity when compared with nearby regions of the Atlantic Ocean such as the Scotian Shelf (fig. 2; Krauel 1975).

The purpose of this study, then, was (1) to test whether significant variation exists in the exercise physiology of two Atlantic cod populations occupying

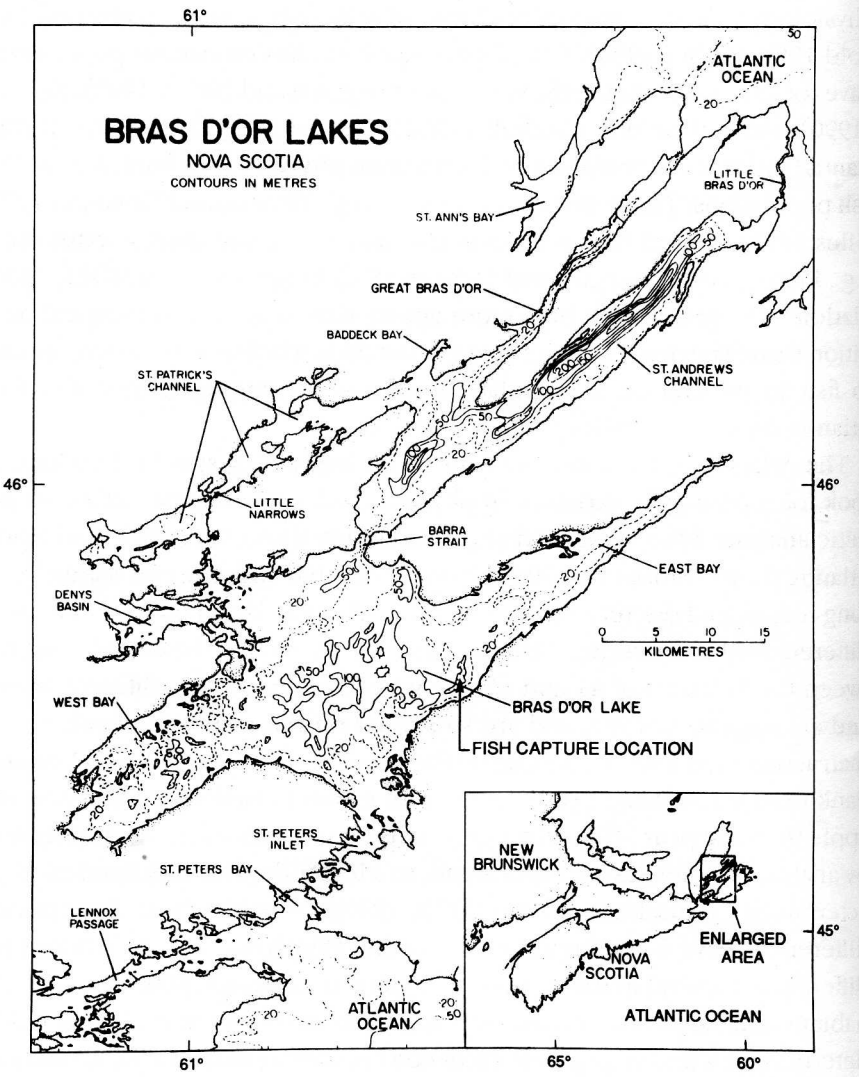

Fig. 1. Map of the Bras d'Or Lakes showing bathymetry and the site of fish capture.

geographically close, environmentally disparate environments and, if so, (2) to deduce the underlying processes that account for any differences.

Material and Methods

Population Origins and Handling

Atlantic cod (*Gadus morhua*) were collected from two locations with different environmental characteristics: (1) Scotian Shelf cod (SSC; 0.93–

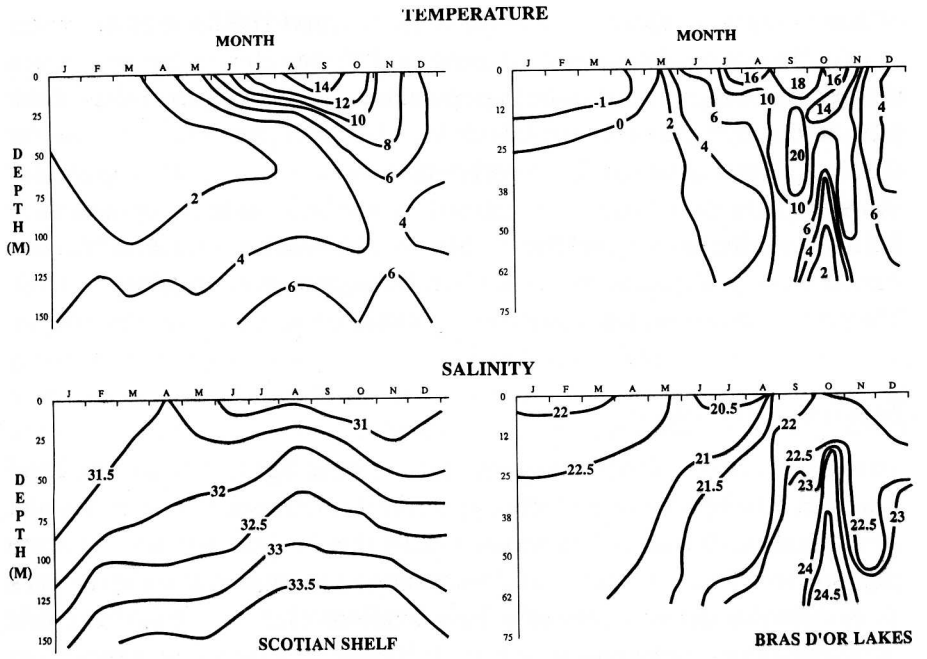

Fig. 2. Ten-year average temperature and salinity profiles for the Scotian Shelf in the vicinity of where SSC were captured, taken from Drinkwater and Taylor (1982). Temperature and salinity profiles for the Bras d'Or Lakes were constructed from data from Krauel (1975) for 1972–1974 in the vicinity of where BDC were captured. Salinity is given in parts per thousand, while temperature is reported in °C.

1.22 kg; $\bar{X}$ = 1.10 ± 0.11 kg [± SD]; all data are presented hereafter as ± SD) were captured by angling off the Nova Scotian coast at 64°25′W, 44°33′N, during November of 1991; (2) Bras d'Or cod (BDC; 0.85–1.12 kg; $\bar{X}$ = 1.00 ± 0.11 kg) were also captured by angling during November of 1991 but from the brackish Bras d'Or Lakes in Cape Breton, Nova Scotia, 60°8′W, 45°85′N (fig. 1). Captured fish were transported to Dalhousie University in 1,500-L aerated tanks containing water from their native environment. Scotian Shelf cod were captured when the surface temperature was 10.5°C and were gradually lowered to 2°C over a period of 53 d. They were maintained at this temperature for 30 d before experimentation. Bras d'Or cod were captured when the surface temperature was 11.2°C and the surface salinity was 18.2‰; BDC were gradually lowered to 2°C over a period of 64 d and were maintained at that temperature and a nominal 20‰ salinity until experimentation began 75 d later.

Animals were held at Dalhousie University in 6,000-L circular tanks supplied with either 2°C filtered Atlantic Ocean water (SSC) or a 2:1 mixture

of filtered Atlantic Ocean water and dechlorinated Halifax City tap water (BDC). The experimental temperature of 2°C was chosen because it is a temperature experienced by both populations for a large proportion of the year (4–5 mo; fig. 2) and because it was the appropriate temperature for the time of year in which the experiments were conducted. Both groups of fish were held in a current maintained by a submersible pump and were fed a mixed diet of chopped squid (*Illex illecebrosus*) and mackerel (*Scomber scombrus*). Experiments took place from January 22 to April 10, 1992. The photoperiod was maintained on a natural cycle.

Experimental Protocol

Twenty-four hours prior to surgery, animals were lightly anesthetized and placed in a 19-cm-diameter, 1-m-long acclimation tube designed to acquaint the animals with a tunnel environment. During acclimation, the fish were exposed to a 15-cm/s current and were trained to avoid resting at the back of the tube by the presence of a light gradient (1,000 lx maximum; 0 lx minimum) from back to front. At least 24 h before the start of an experiment the animals were gently slid from their acclimation tube into an anesthetic bath containing their holding water and ethyl *m*-amino benzoate methane sulfonate salt (MS-222; Sigma; 100 mg/L initially, 25 mg/L during the surgery). Anesthetized cod were cannulated in the afferent branchial artery by a variation of a method originally described by Soivio, Nyholm, and Westman (1975). After the 15–25-min operation, cod were placed in the swim-tunnel respirometer regulated at 2°C and artificially ventilated until spontaneous breathing resumed (usually within 2 min). On recovery, the current speed in the swim-tunnel respirometer was turned up to the acclimation speed of 15 cm/s.

The 19.2-cm-diameter, 85-L swim-tunnel respirometer was designed by the Department of Engineering at Guelph University (Farmer and Beamish 1969). It was powered by an adjustable-speed hydraulic drive and had collimators placed at strategic locations to make the flow as laminar as possible. Calibration of the swim tunnel was by cinematography; dye suspension released at the upstream end of the tank was videotaped and film frames counted to calculate water velocity. Water velocity was calibrated to revolutions of the impeller shaft measured with a tachometer. Flow appeared laminar at all speeds encompassing the range over which cod swam and increased linearly with motor revolutions.

The cannula was led out of the respirometer through a small hole, which was sealed with grease during respirometry. Adequacy of sealing was routinely checked by filling the respirometer with hypoxic water ($P_{O_2} < 50$ Torr; no fish present); intrusion of atmospheric O_2 was not detected at any

speed. Bacterial O_2 consumption ($\dot{M}O_2$) was not measurable over a period comparable to an entire protocol for critical swimming speed (U_{crit}). The swimming section of the swim-tunnel respirometer was surrounded by two-way mirrors, allowing observation of the fish by investigators while the fish saw only a reflection of itself. Animals were motivated to swim by a light gradient; most fish swam into the current so as to remain at low light levels. This was sufficient for fish at low to intermediate current speeds, but a 12-V electrified grid with manually activated switch was located on the downstream retaining screen to prevent uncooperative fish from resting and to provide a mechanism for uniformly exhausting each fish. A pulse of current was sent through the grid only when a fish was actually attempting to rest there. Exhaustion was defined by the inability to respond to the electrical stimulus.

An experiment began by taking an initial measurement of $\dot{M}O_2$ at the 15-cm/s acclimation speed. Immediately afterward, preexercise blood samples (1 mL) were taken, and the velocity of the swim tunnel was subsequently increased to 20 cm/s. Cod were swum until exhausted by increasing the water velocity 10 cm/s every 30 min (critical swimming speed protocol, $U_{crit\ 10,30}$; Beamish 1978). Blood was sampled at the end of each 30-min swimming period, whereas $\dot{M}O_2$ was measured during the middle 20-min period of each velocity increment. Surplus blood was always returned to the fish along with a volume of seawater teleost Ringer's solution (Hoar and Hickman 1963) sufficient to replace the blood volume sampled. The respirometer was flushed with fresh experimental water during the last 5 min of a fish's 30 min at a given velocity and during the first 5 min at the next velocity before being sealed for the subsequent $\dot{M}O_2$ measurement. The O_2 tension was not allowed to fall below 85% saturation. The water temperature in the respirometer was maintained at $2.0 \pm 0.1°C$ and regulated with a computer-controlled relay system.

On exhaustion, the flow in the swim-tunnel respirometer was reduced to 12 cm/s and remained that way throughout recovery. The fish were not forced to swim during recovery, although many did. Blood was sampled directly after exhaustion and at 0.5, 2, 4, and 8 h after exhaustion. Oxygen consumption was also measured at all of these times except directly at exhaustion.

Measurements and Calculations

Critical swimming speed was calculated according to the method of Brett (1964). The water velocity was corrected for the acceleration of the water around the fish (solid blocking) according to equations (105) and (106) of Webb (1974; note the error in eq. [105]: the denominator should be $S_t^{3/2}$),

with empirically measured maximum cross-sectional areas used to calculate the fish's mean diameter, the fish's mass and unit density used to approximate volume, and interpolated body-shape factor values derived from the graphs found in Pope and Harper (1966). A fish was approximated as a continuum of elliptical cross sections with their maximum value occurring at one-third of body length (starting at the anterior end of the fish). Summary statistics for the blocking corrections and their effects on U_{crit} are presented in table 1. For clarity, all data presented as a function of swimming speed are shown as if the fish were swimming at a nominal, uncorrected water velocity.

Blood pressure was monitored with a Gould model P23 transducer; the signal was recorded on a Georz SE 120 chart recorder and heart rate calculated therefrom. Ventilation frequency was measured by timing 20 ventilatory cycles with a stopwatch.

Oxygen partial pressures were measured with a Radiometer 5046 P_{O_2} electrode regulated at the experimental temperature. The signal from the electrode was amplified with electronics supplied by the Cameron Instrument Company and recorded on either an ABB Georz SE 120 chart recorder (blood and water P_{O_2}) or an Apple II microcomputer (water P_{O_2}) using data capture software described in Webber (1985). Oxygen consumption was calculated according to the following equation:

$$\dot{M}_{O_2} = ((\Delta P_{O_2}/\Delta T) \times (V - M) \times \alpha)/M,$$

where ΔP_{O_2} is the change in partial pressure of O_2 in the water (mmHg), ΔT is the measurement interval (min), V is the respirometer volume (L), M is the animals' mass (kg), and α is the O_2 solubility coefficient at the experimental temperature and salinity interpolated from tables found in Boutilier, Heming, and Iwama (1984).

Blood was sampled anaerobically into a cooled "gastight" Hamilton syringe. A 50-μL subsample was immediately dispensed into 500 μL of ice-cold 0.6 N $HClO_4$ and centrifuged for 0.5 min at 4°C. The supernatant was decanted, neutralized with KOH, recentrifuged, and frozen at −80°C for later measurement of glucose and lactate. A second subsample was centrifuged and the plasma withdrawn and frozen at −80°C in sealed, humidified chambers for the later measurement of plasma protein and ion concentrations.

Whole-blood pH was determined with a microcapillary pH electrode (Radiometer G279/G2) connected to a Radiometer PHM84 pH meter. Total plasma CO_2 was measured with a Corning model 965 CO_2 analyzer. Venous plasma bicarbonate concentration and partial pressure of CO_2 (P_{CO_2}) were calculated via a rearrangement of the Henderson-Hasselbalch equation using an apparent pK (pK_{app}) derived from Severinghaus (1965) and adjusted for

TABLE 1

TABLE 1

Summary of calculated shape factors and their use in correcting the swimming speeds of the 11 Atlantic cod used in this study

	Bras d'Or Fish					Scotian Shelf Fish				
Fish	Length (cm)	XSA (cm^2)	K_3	$U_{crit(u)}$ (cm/s)	$U_{crit(c)}$ (cm/s)	Length (cm)	XSA (cm^2)	K_3	$U_{crit(u)}$ (cm/s)	$U_{crit(c)}$ (cm/s)
1	43.8	42.16	.936	47.5	50.5	48.5	51.24	.936	46.0	50.7
2	46.5	46.60	.935	44.7	48.6	51.5	57.0	.935	45.7	50.5
3	46.5	37.13	.928	51.7	54.9	47.5	40.0	.929	45.0	48.1
4	48.0	44.00	.932	50.0	54.1	47.0	42.44	.932	50.0	53.8
5	45.8	44.43	.935	42.8	46.1	47.5	43.89	.932	45.2	49.0
6						50.5	52.05	.934	47.5	51.9
$\bar{X}$	46.1	42.86	.933	47.3	50.8	48.8	47.77	.933	46.6	50.7
SD . . .	1.5	3.57	.003	3.6	3.7	1.8	6.62	.002	1.9	2.1

Note. XSA, Empirically measured maximal cross sectional area; $U_{crit(u)}$, critical swimming speed, uncorrected for the local acceleration of water around the animal (solid blocking); $U_{crit(c)}$, critical swimming speed, corrected for solid blocking effects; and K_3, shape factor calculated from graphs found in Pope and Harper (1966).

pH with the coefficients and solubility of CO_2 (α CO_2) values for rainbow trout supplied by Boutilier et al. (1984) extrapolated to 2°C.

Hemoglobin concentration was determined colorimetrically with a Gilford Response spectrophotometer and Sigma reagents (Sigma bulletin 525). Hematocrit was measured by centrifugation of blood contained in microcapillary tubes.

Whole-blood glucose and lactate concentrations were determined enzymatically by measuring changes in the absorbance of nicotinamide coenzymes at 339 nm in a Gilford Response spectrophotometer according to procedures derived from Bergmeyer (1984; see Nelson [1990] for further detail). Plasma protein was measured using the bicinchoninic method described by Smith et al. (1985). Plasma [Na^+] was measured with a Corning model 410 flame photometer, while plasma [Cl^-] was measured colorimetrically by a mercuric thiocyanate method (Zall, Fisher, and Garner 1956).

Statistics

Data were analyzed according to the general linear models (GLM) procedure of the Statistical Analysis System (SAS Institute 1989). The general model was an unbalanced, two-way, repeated-measures ANOVA with population and time (either specific swimming speed or time after exhaustion) as the class variables. Tukey's multiple comparison test was used to compare main effect means. The hypothesis that the populations were not distinct for a given variable was also tested with a specific *F*-test using individuals as the error term. The significance of differences among interaction means (i.e., specific combinations of time and population) was determined by comparison of least-squares confidence intervals (SAS Institute 1989).

Results

Swimming Performance

We had previously reported that critical swimming performance in cod was highly repeatable (Nelson, Tang, and Boutilier 1992*a*). In this preliminary experiment at 15°C, cod were swum at least twice and up to four times with a minimum of 2 wk between swimming trials. Subsequent swimming performance was strongly correlated with the initial U_{crit} ($r^2 = 0.60$, $P < 0.01$, *F*-test, $n = 10$), which suggests that one-time measurements of U_{crit} are reasonable gauges of swimming performance in Atlantic cod.

Size had a small influence on the U_{crit} for cod of the size range used in these experiments. The best-fit function that describes the relationship be-

tween fish size and U_{crit} (corrected) for fish swum at 2°C in our laboratory is linear and is

$$U_{crit} = 0.95\ L + 4.7, \quad r = 0.458,$$

where L is fish length and $n = 23$ (fish ranged in size from 43.8 to 51.5 cm, $\bar{X} = 47.4 \pm 2.11$). This relationship suggests that the size-attributable variance in U_{crit} between the smallest and largest fish in our data set is on the order of 14%. The narrow size range was by design; this attempt to limit scaling effects resulted in different age structures between the two experimental groups. Cod from the Bras d'Or system averaged 6.33 ± 0.52 yr old, whereas the SSC averaged only 3.33 ± 0.82 yr old (E. Trippel, personal communication). Despite these age differences, U_{crit}'s were virtually identical between SSC and BDC when swum in their native salinities (50.67 ± 2.06, $n = 6$, and 50.85 ± 3.69, $n = 5$, respectively; table 1).

Metabolic Rate

The populations had statistically distinct aerobic metabolic rates, as evidenced by a significant population effect in the general model (ANOVA, $P < 0.001$) and a rejection of the hypothesis that the populations were the same by Tukey's multiple comparison test ($P < 0.05$). However, a specific F-test did not support the hypothesis that these populations had distinct metabolic rates ($P = 0.06$). Although standard metabolic rate was not measured in these animals, linear extrapolation of the first four points from the line relating mean $\dot{M}O_2$ as a function of nominal swimming speed to zero swimming speed produced "resting" metabolic rate estimates of 16.97 μmol/(kg × min) ($r = 0.96$) for BDC vs. 9.6 μmol/(kg × min) ($r = 0.94$) for SSC (fig. 3). Extrapolation of individual values for $\dot{M}O_2$ at each swimming speed, after correction for solid blocking, to a swimming speed of zero produced mean estimates for resting metabolic rate of 19.12 ± 24.58 μmol/(kg × min) and 14.13 ± 17.56 μmol/(kg × min) for BDC and SSC, respectively. These estimates of resting $\dot{M}O_2$ compare favorably with the mean value of 21.49 μmol/(kg × min) ($n = 19$) for "routine" $\dot{M}O_2$ of similarly sized Atlantic cod at 3°C reported by Saunders (1963).

The greater aerobic cost of being a cod from the Bras d'Or Lakes was evident throughout swimming and recovery, suggesting that the aerobic metabolism available for exercise or "scope for activity" (Fry 1978) was similar between populations (fig. 3). The lack of a significant interaction term between time and population in the general ANOVA model also supports the conclusion that change in metabolic rate with increased swimming speed and subsequent recovery was not significantly different between pop-

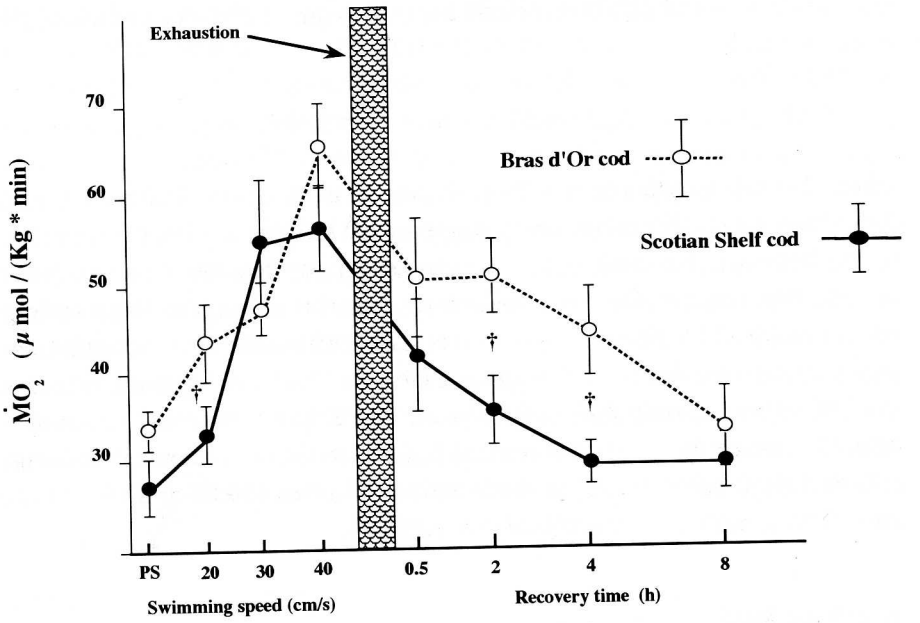

Fig. 3. Whole-animal $\dot{M}O_2$ *in SSC (●) and BDC (○) during swimming to their* U_{crit} *and at various times during recovery from exhaustion. The scaled bar separates the swimming and recovery portions of the experiment. Means and standard errors are presented for six (SSC) and five (BDC) animals. The preswim measurement (PS) was taken at 15 cm/s current velocity, but the animals were not necessarily swimming; daggers (†) represent time points where the populations differed at the* $P < 0.05$ *level or greater. Times where* $\dot{M}O_2$ *differed significantly from the preswim value were 30 cm/s, 40 cm/s, and 0.5 h of recovery for both populations and also 2 and 4 h of recovery for BDC.*

ulations. We estimate "actual" scope for activity to be 32.67 ± 7.11 μmol/(kg × min) for SSC versus 38.22 ± 13.07 μmol/(kg × min) for BDC, calculated by subtracting minimal measured $\dot{M}O_2$ from maximal measured $\dot{M}O_2$, and "absolute" scope for activity to be 50.74 μmol/(kg × min) for SSC versus 51.88 μmol/(kg × min) for BDC, calculated by subtracting the extrapolated values for resting $\dot{M}O_2$ from the maximal measured $\dot{M}O_2$. If we adopt a recovery criterion for $\dot{M}O_2$ of not being different at the $P < 0.05$ level from the "preswim" value, then it took BDC 8 h to recover from exhaustive exercise whereas SSC recovered within 2 h (fig. 3). The populations had significantly distinct rates of $\dot{M}O_2$ at the 2- and 4-h postswimming measurements ($P < 0.01$). Osmoregulatory costs also do not appear to be involved in the generally higher metabolic rate observed in BDC, because these are predicted to be less at 20‰ (Febry and Lutz 1987).

Acid-Base Balance

Cod that underwent a critical swimming protocol experienced a significant 0.35-pH-unit reduction of venous pH regardless of population origin; because the response of venous blood pH differed between the two populations during recovery from exercise, the interaction term between time and population origin was significant (ANOVA, $P < 0.001$; fig. 4, top). Four hours was sufficient to return venous pH to preexercise levels in recovering SSC, whereas venous pH had not returned to preexercise levels in BDC even after 8 h of recovery. Venous pH levels differed between populations by both statistical tests ($P < 0.05$).

Venous plasma [HCO_3^-] was also determined by an interaction between the population origin of the cod and time during the experimental procedure. Both cod populations had a preexercise [HCO_3^-] of about 8.5 mM; bicarbonate levels climbed throughout exercise to 10.3 mM in SSC, whereas venous [HCO_3^-] in BDC remained stable during exercise with a noticeable decline evident at exhaustion (fig. 4, center). Recovery was characterized by a return of venous blood [HCO_3^-] to preexercise levels in SSC, whereas bicarbonate never returned to preexercise levels in BDC. The populations differed from each other with respect to venous plasma [HCO_3^-] for the entire experiment according to both statistical tests ($P < 0.05$; fig. 4, center).

A significant interaction between population and time was also apparent in the venous P_{CO_2} results (ANOVA, $P < 0.001$; fig. 4, bottom), but interpopulation P_{CO_2} was not different by either direct statistical test of this hypothesis. The critical swimming speed protocol elicited a significant increase in P_{CO_2} that was identical between populations up until the 40-cm/s swimming speed (fig. 4, bottom). Venous blood P_{CO_2} was significantly higher at exhaustion and after 0.5 h of recovery in SSC ($P < 0.01$) but significantly lower than BDC during the last two recovery times ($P < 0.05$). The elevated venous P_{CO_2} levels during the late stages of recovery in BDC are consistent with the higher aerobic metabolic rate in this population at that time (fig. 3).

Metabolites

Different levels of blood lactate following 2 h of recovery from exhaustive exercise accounted for the significant interaction between populations and sampling time for blood [lactate] ($P < 0.001$; fig. 5, top). Lactate levels rose significantly in fish from both populations after they had swum at 40 cm/s ($P < 0.05$; fig. 5, top) and remained significantly higher than the preswim values throughout recovery. Blood lactate levels had started to decline in SSC after 4 h of recovery but had only reached their zenith in BDC at this

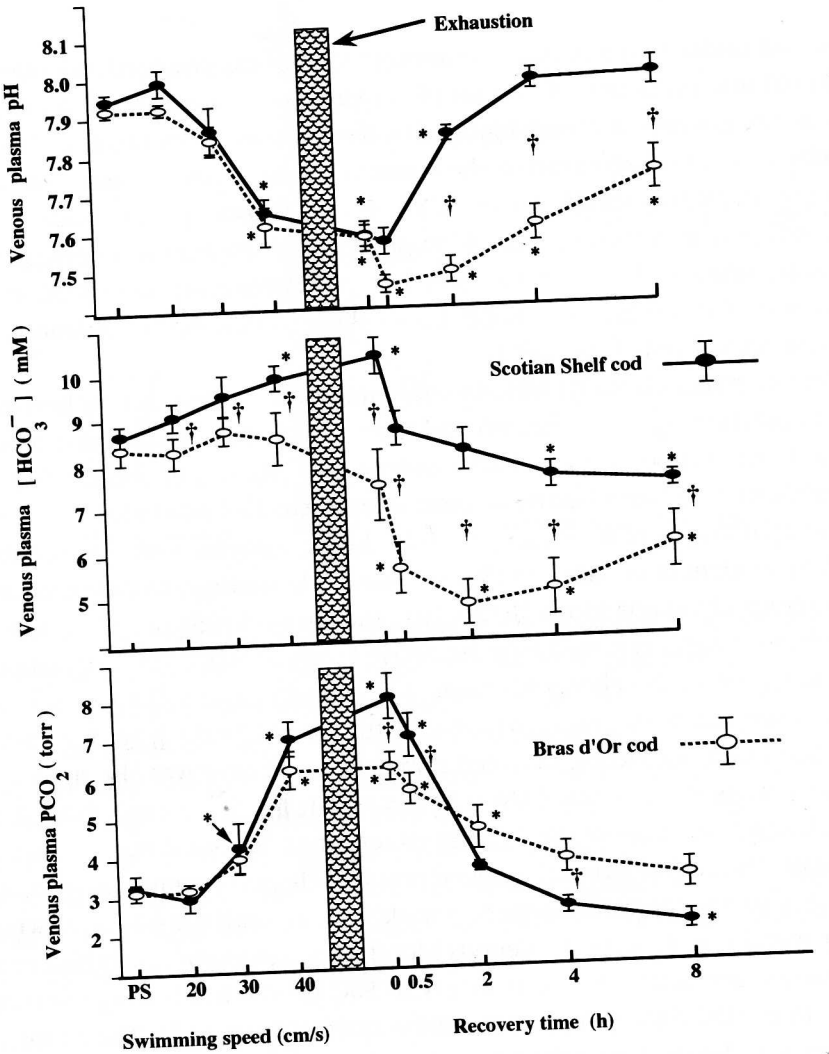

Fig. 4. Blood acid-base status in SSC (●) and BDC (○) during swimming to their U_{crit} *and at various times during recovery from exhaustion. Top, venous blood pH. Details are as in fig. 3; asterisks indicate samples that differed from the preswim sample (*PS*) at the* $P < 0.05$ *level or greater. Center, venous plasma* $[HCO_3^-]$ *calculated from the total plasma* CO_2 *content of venous plasma. Details are as in fig. 3 and fig. 4, top. Bottom, venous plasma* P_{CO_2} *as calculated from venous blood pH and total venous* CO_2*. Details are as in fig. 3 and fig. 4, top.*

time. Blood [lactate] started to decline in BDC after 8 h of recovery from exhaustive exercise, but on a trajectory that would not return it to preexercise levels until 45 h after exhaustion; the homologous trajectory had SSC re-

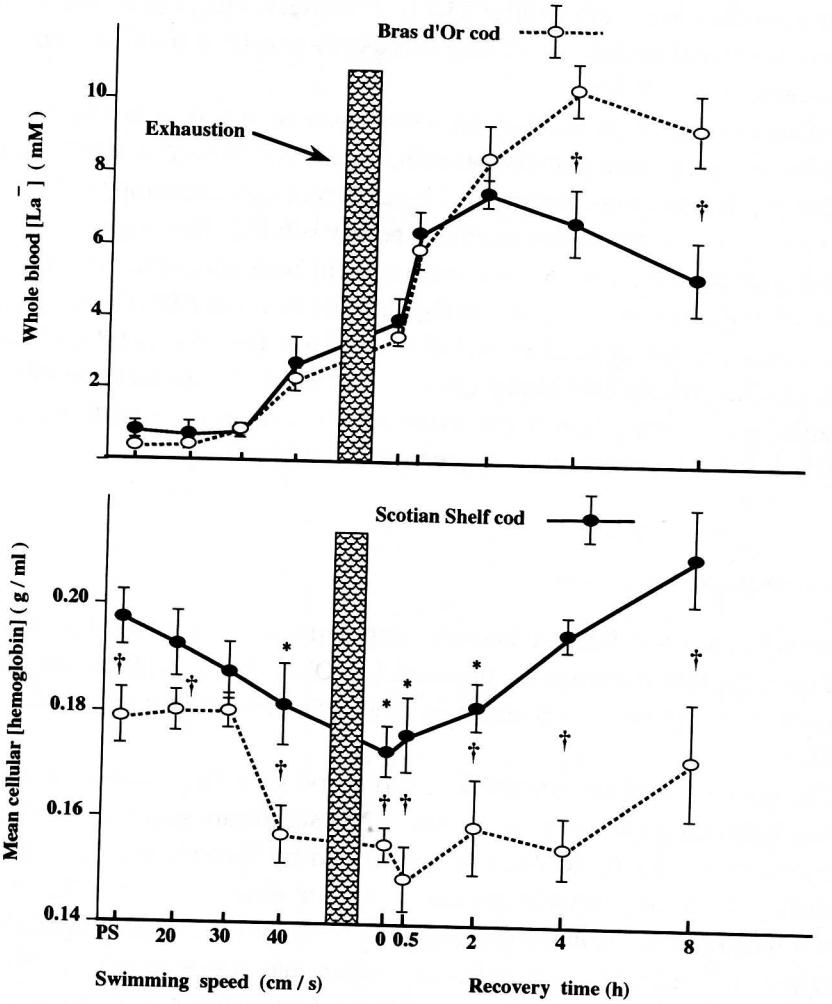

Fig. 5. Whole-blood lactate concentration and mean cellular hemoglobin concentration in SSC (●) and BDC (○) during swimming to their U_{crit} *and at various times during recovery from exhaustion. Details are as in fig. 3. Top, whole-blood lactate concentration. All samples, except those for 20- and 30-cm/s swimming speeds, differed from the preswim sample (PS) at the* $P < 0.05$ *level or greater. Bottom, mean cellular hemoglobin concentration. For BDC, all samples after the 30 cm/s swimming speed differed from the preswim sample (PS) at the* $P < 0.05$ *level or greater. For SSC, samples significantly different from the preswim sample are indicated by asterisks.*

turning to preexercise values after 23.7 h of recovery. The populations were distinct for blood [lactate] according to Tukey's test ($P < 0.05$) but not by the specific F-test ($P = 0.19$).

Venous blood glucose concentration was lower in BDC than in SSC, which resulted in a significant population effect in the ANOVA ($P < 0.001$; table 2). The populations were distinct for blood glucose concentration by Tukey's test ($P < 0.05$) but not by the specific F-test ($P = 0.11$). Blood glucose rose significantly during recovery from swimming in both populations (ANOVA, $P < 0.001$). Plasma protein was also significantly lower in BDC for the entire experiment according to all statistical tests of that hypothesis ($P < 0.001$). Both plasma protein and blood glucose exhibited significant time effects because of the contraction of the extracellular compartment with exercise and their resultant increase in concentration (table 2).

Cardiorespiratory Factors

Venous P_{O_2} was not different between populations (table 2) but did change significantly with the exercise protocol (ANOVA, $P < 0.01$). The only significant interpopulation difference in venous P_{O_2} occurred at exhaustion ($P < 0.05$).

The volume of blood occupied by red blood cells (hematocrit) was different between populations according to the significant population term in the general model (ANOVA, $P < 0.001$) and by Tukey's test ($P < 0.05$). Hematocrit was significantly greater in BDC at exhaustion and after 0.5 h and 4 h of recovery from swimming ($P < 0.05$; table 2). Both populations had hematocrits that were significantly higher than preswim values after 0.5 h and 2 h of recovery and were significantly lower after 8 h of recovery.

Blood hemoglobin concentration was marginally different between populations; the population term in the general model was equivocal ($P = 0.05$) and the populations had similar [Hb] by the two specific tests of that hypothesis. Cod from the Bras d'Or Lakes had significantly lower blood [Hb] at three of the nine individual sampling periods (0.5 h, 2 h, and 8 h of recovery; $P < 0.05$; table 2).

The quotient of these two previous measurements, mean cellular hemoglobin concentration ([MCHC]), responded to the exercise and recovery protocol differently in the two populations, as indicated by a significant interaction between population and sampling time ($P < 0.001$; fig. 5, bottom). This was the result of a smaller decrease in [MCHC] with exercise in SSC. The [MCHC] was also different between the populations according to both statistical tests of this hypothesis (Tukey's, $P < 0.05$; specific F-test, $P < 0.01$), being lower in BDC in all but the 20 cm/s sample (fig. 5, bottom).

TABLE 2

Blood chemistry measurements in SSC (n = 6) and BDC (n = 5) before forced swimming, at various nominal swimming speeds, and at various times during recovery from exhaustive exercise

	Swimming Speed (cm/s)								Time after Exhaustion (h)									
	PS		20		30		40		Exhaustion		0.5		2		4		8	
	BDC	SSC	BDC	SSC	BDC	SSC	BDC	SSC	BDC	SSC	BDC	SSC	BDC	SSC	BDC	SSC	BDC	SSC
Plasma Cl^- (mM)	159.2 (5.02)	170.5 (3.83)	159.6 (4.67)	171.3 (4.23)	161 (7.31)	171.5 (2.59)	167.2 (3.40)	175 (4.69)	171.2 (4.32)	180.8 (3.19)	171.3 (4.72)	181.2 (2.99)	169.8 (6.18)	179 (4.65)	168 (2.83)	178.2 (5.78)	168.3 (1.53)	177.6 (5.64)
Protein ($mg \cdot dL^{-1}$)	2.97 (.49)	6.22 (.76)	2.95 (.58)	6.23 (.93)	3.01 (.81)	6.37 (.98)	3.45 (.74)	6.20 (1.00)	3.29 (.65)	6.45 (.94)	3.29 (.87)	6.97 (1.00)	3.69 (.74)	6.92 (1.21)	3.48 (.97)	6.38 (1.46)	3.10 (.99)	5.72 (.41)
Venous P_{CO_2} (Torr)	28.6 (6.44)	23.3 (6.36)					16.8 (2.40)	15.6 (1.65)	25.8 (4.67)	20.21 (2.64)			18.9 (5.34)	22.0 (7.99)	21.1 (9.74)	22.3 (6.07)	22.4 (6.08)	25.6 (4.04)
Hematocrit (%)	25.6 (2.71)	23.2 (3.94)	25.8 (3.07)	23.5 (4.57)	25.5 (2.82)	22.8 (3.41)	25.8 (3.51)	23.8 (2.88)	27.1 (2.77)	24.9 (3.14)	30.0 (2.67)	26.9 (4.08)	29.1 (1.83)	27.0 (3.87)	25.8 (3.84)	23.5 (3.52)	21.9 (3.24)	20.7 (2.20)
Hemoglobin ($mg \cdot dL^{-1}$)	4.57 (.55)	4.57 (.95)	4.64 (.54)	4.53 (1.05)	4.59 (.47)	4.30 (.87)	4.03 (.50)	4.34 (.79)	4.22 (.55)	4.32 (.64)	4.45 (.44)	4.73 (.77)	4.62 (.40)	4.92 (.83)	4.15 (.51)	4.60 (.83)	3.71 (.28)	4.35 (.60)
Glucose (mM)	4.85 (1.29)	6.31 (2.18)	4.88 (1.29)	5.94 (1.71)	4.99 (1.40)	6.07 (1.58)	4.95 (1.45)	6.52 (1.55)	4.72 (1.79)	6.10 (1.66)	5.80 (1.16)	7.31 (1.60)	6.89 (1.43)	9.24 (1.89)	8.87 (1.37)	8.57 (1.92)	8.52 (.71)	10.6 (2.75)

Note. PS, Preswim sample. Means of six fish with the standard deviation shown in parentheses underneath are presented for SSC, while the numbers for BDC represent means of five animals with the standard deviation underneath. Significance levels are not presented in the table for clarity but are, at the $P < 0.05$ level for populations differing from each other and times differing from the preswim sample, as follows: Plasma Cl^-: for populations, all points; for time, all points except 20 and 30 cm/s; plasma protein: for populations, all points; for time, BDC—2 and 4 h of recovery; SSC—0.5 and 2 h of recovery; Venous P_{O_2}: for populations, exhaustion; for time, BDC—40 cm/s, 2 and 8 h of recovery, SSC—none; hematocrit: for populations, exhaustion, 0.5 and 4 h of recovery; for time, both populations—0.5, 2, and 8 h of recovery; hemoglobin: for populations, 0.5, 2, and 8 h of recovery; for time, BDC—40 cm/s, exhaustion and 8 h of recovery, SSC—8 h of recovery; plasma glucose: for populations, all points except 4 h of recovery; for time, both populations—2, 4, and 8 h of recovery.

Cardiac rate was different between the two populations and also changed differently with the exercise and recovery protocol. This was shown by a significant interaction term between time and population in the general model (ANOVA, $P < 0.001$) and by the populations' having distinct cardiac rates by both Tukey's and the specific F-tests ($P < 0.05$ and $P < 0.01$, respectively; fig. 6, top). Heart rate was significantly higher in BDC in each individual time period with the exception of 2 h after exhaustion (fig. 6, top). The higher initial heart rate in BDC seems to have compromised some of their flexibility to respond to increased cardiac demand, as heart rate only rose an average of 2 beats/min during forced swimming to exhaustion. In contrast, heart rate rose an average of 5 beats/min during exhaustive exercise in SSC. It is interesting that maximal cardiac rates were registered during recovery in both groups.

The ventilation frequency results largely paralleled those of $\dot{M}O_2$ (see above), which suggests that ventilation volume is similar between populations (fig. 6, bottom). Ventilatory rate was significantly greater in BDC for the whole experiment according to the significant population term in the general model ($P = 0.001$) and by Tukey's test ($P < 0.05$). However, the specific ANOVA F-test did not support the hypothesis that the populations were distinct ($P = 0.23$). The interpopulation differences were most noticeable during the last 6 h of recovery, when ventilation in BDC remained fairly stable around 22 breaths/min while ventilation in SSC declined toward the initial value of 16 breaths/min (fig. 6, bottom).

Plasma Ion Status

Plasma sodium was significantly lower in BDC than in SSC throughout the entire experiment by all statistical measures (general model population term, $P < 0.001$; Tukey's test, $P < 0.05$; specific ANOVA, F-test, $P < 0.05$; fig. 7). Plasma sodium also increased significantly during recovery from exhaustive exercise in both populations as shown by a time effect in the general model ($P < 0.001$). Every sample after the 40-cm/s swimming speed had a higher [Na^+] than the preswim sample ($P < 0.01$). Plasma chloride changes paralleled those of sodium in every respect (table 2); the statistical analysis was virtually identical to that for sodium, reaching the same conclusions.

Discussion

Cod from the Bras d'Or Lakes achieved the same level of performance as SSC, appearing to take advantage of more anaerobically derived energy pro-

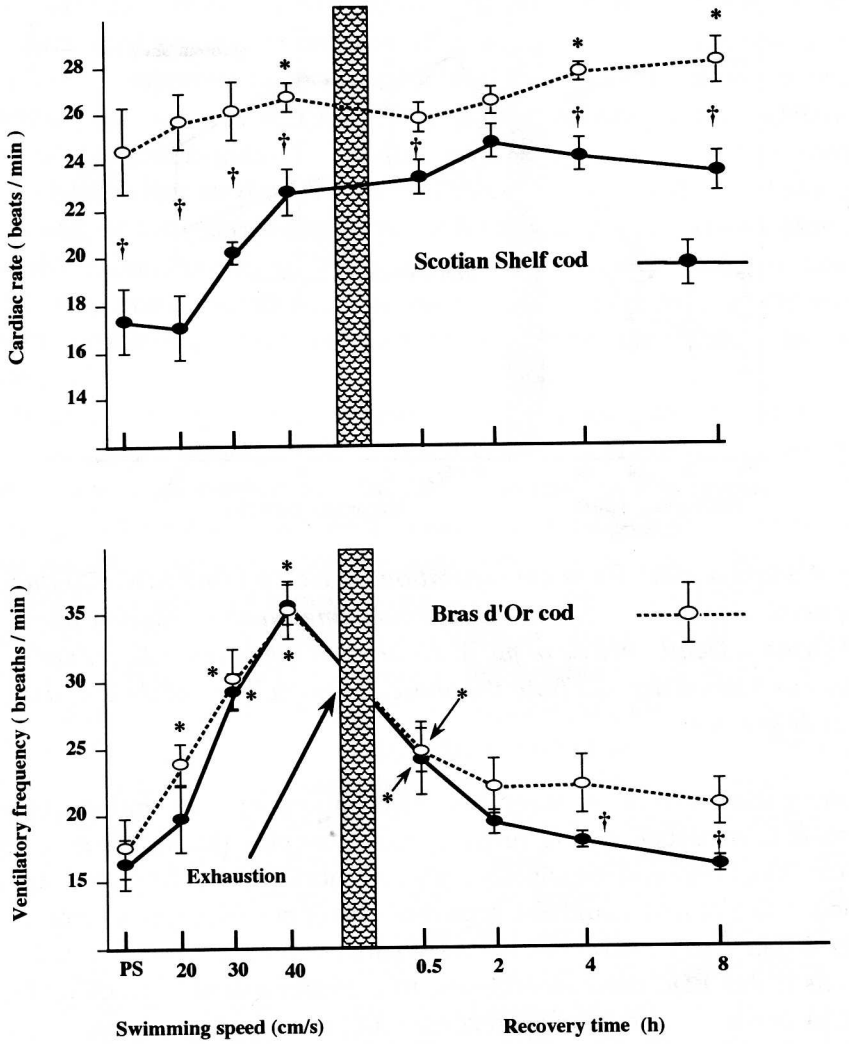

Fig. 6. Cardiac rate and ventilation frequency in SSC (●) and BDC (○) during swimming to their U_{crit} *and at various times during recovery from exhaustion. Details are as in fig. 3 and fig. 4, top. Top, cardiac rate. For SSC, all samples after the 20 cm/s swimming speed differed from the preswim sample (PS) at the* $P < 0.05$ *level or greater. For BDC, samples significantly different from the preswim sample are indicated by asterisks. Bottom, ventilation frequency.*

duction in the process. We draw this conclusion from (1) the greater and earlier metabolic acidosis in BDC, evidenced by an earlier depletion of plasma bicarbonate while pH was similar and P_{CO_2} lower than in SSC, and the severe [HCO_3^-] and pH depressions during recovery in BDC, and (2), the higher blood [lactate] in BDC during the late phases of recovery. Con-

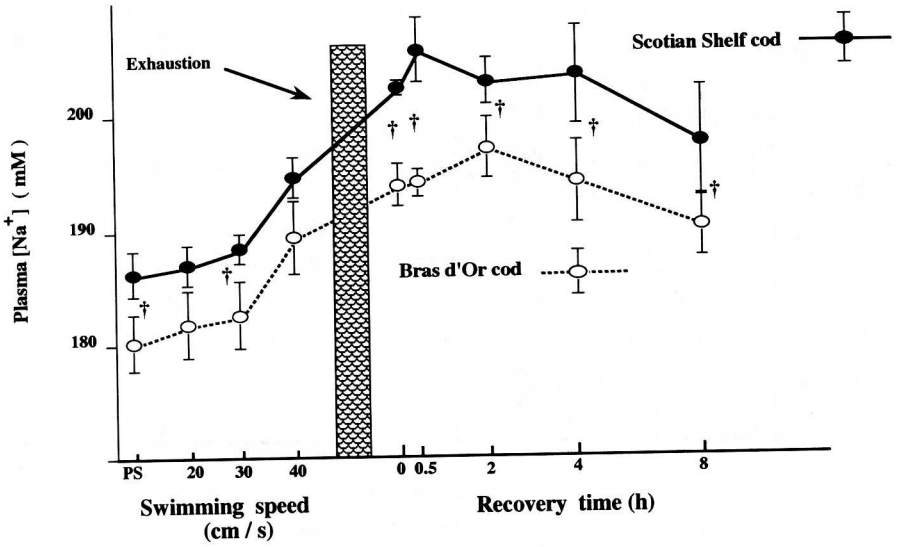

Fig. 7. Plasma sodium ion concentration in SSC (●) and BDC (○) during swimming to their U_{crit} *and at various times during recovery from exhaustion. Details are as in fig. 3. All samples taken after the 30 cm/s swimming speed differed from the preswim sample (*PS*) at the* $P < 0.05$ *level or greater.*

sidering the trade-offs between oxidative phosphorylation and glyconeogenesis from lactate during recovery from exercise (Shulte, Moyes, and Hochachka 1992) and the slightly higher metabolic rate of BDC, the higher blood lactate in BDC could also represent slower processing of accumulated lactate (fig. 5, top). However, the most parsimonious explanation of these results is that BDC used anaerobiosis to a greater extent in reaching U_{crit}. The prolonged elevation of postexercise $\dot{M}O_2$ and ventilation (figs. 3, right; fig. 6, bottom) in BDC is also consistent with a greater use of anaerobic metabolism during exercise.

An important message from this result is that intraspecific differences in physiology need not translate into performance differentials. Thus, if there is strong stabilizing selection to maintain performance within a population, environmental evocation of physiological responses that compromise performance will be balanced by responses in other systems that help maintain exercise performance. One interpretation of our results is that BDC have been selected or acclimatized to maintain exercise performance by expanding their use of anaerobic metabolism. Alternatively, if BDC are less able to process the end products of anaerobic metabolism in their lower salinity environment, results similar to ours could be expected.

Since our estimates of scope for activity were roughly equal between populations, the question arises as to why BDC would need to rely more

on anaerobic metabolism to achieve an identical performance level. Work on other cod in our laboratory at 15°C has shown that an animal's morphology is important in determining its U_{crit} (Nelson, Tang, and Boutilier 1992*b*); it is likely that morphological differences between the populations result in relatively greater drag profiles for similarly sized BDC. A second likelihood is that we did not effectively measure the true maximal $\dot{M}O_2$ and that BDC actually have a smaller scope for activity and need to supplement aerobic metabolism to achieve a maximal performance similar to that of SSC ($\dot{M}O_2$ measurements near exhaustion [50–60 cm/s] are not very robust owing to unavoidable electrolysis of the water when the shocking grid was activated).

Some of our physiological measurements may also give us clues to how any increased reliance on the relatively inefficient anaerobic powering of exercise may have arisen in BDC. We think that the differential levels of hemoglobin within the red cells of each population could be a key clue. Because of the seasonally warmer temperatures experienced by BDC (fig. 2) and the presence of hypoxic strata in this system (Krauel 1975), considering hemoglobin's role only as a respiratory protein might lead one to postulate either unchanged or higher hemoglobin levels in BDC (Grigg 1974; Weber and Jensen 1988). Thus, if consistently lower concentrations of hemoglobin per erythrocyte in BDC arose through selective or acclimatory forces, these forces are presumably operating on physiological systems other than respiration (see below). Whatever the source of the larger erythrocytes with lower [Hb] in BDC, it could explain some of the differences observed between the two populations. Specifically, the higher cardiac and ventilatory rates in BDC could accrue in part from differences in O_2 uptake kinetics of their larger erythrocytes with a lower [Hb]. Similarly, rheological differences between the populations owing to the different sized erythrocytes could be responsible for the earlier entry into anaerobiosis by BDC. By carrying a similar amount of hemoglobin around in larger cells, the BDC have theoretically reduced viscous drag in the larger blood vessels but are at increased risk of suffering capillary blockage (Fung 1984).

Although the ability of individual fish populations to have unique patterns of use of anaerobic metabolism has been described before (DiMichele and Powers 1982; Nelson 1990), this is an important finding for an exploited species like cod. Since using relatively inefficient anaerobic metabolism involves the allocation of energy resources for immediate needs versus the conservation of resources for growth and gamete production, finding populations that differ in this regard may be the first step toward isolating factors that control such allocation processes.

A second pertinent finding of this study is the general dilution of plasma in BDC compared to SSC. All measured osmotically active substances (he-

moglobin, glucose, protein, sodium, and chloride) were lower in BDC. The lower ion levels are probably a reflection of some degree of osmoconformity in cod (a common observation in euryhaline fishes; see table in Bentley [1971]), but the lower levels of glucose and protein probably require different explanations. Since cod exercised at 15°C and 11‰ salinity undergo a massive hemoconcentration during recovery from exercise, which probably contributes to their eventual demise (Y. Tang, J. A. Nelson, and R. G. Boutilier, unpublished manuscript), one possibility is that lower levels of osmotically active substances in BDC plasma are the result of selective or acclimatory pressures to limit hemoconcentration. Cod from the Bras d'Or Lakes are more likely to encounter warm temperatures and low salinities than are SSC (fig. 2). Lower plasma osmolyte levels in resting animals would have the effect of ameliorating postexercise blood concentrations, resulting in a lower postexercise blood viscosity. However, without concomitant reductions of intracellular osmolytes, the lower ECF concentrations could contribute to hemoconcentration by encouraging water loss to the cells. This second possibility does not appear true, as, judging from the changes in plasma protein and glucose concentrations, contraction of the extracellular fluid compartment was basically uniform between populations (table 2). By similar logic, the lower hemoglobin concentration in each BDC erythrocyte (fig. 5, bottom) may simply reflect the general reduction in osmotic activity in BDC.

Exercise can initiate a cascade of adrenergic and direct responses within the red cell that cause swelling (Primmett et al. 1986). Since hemoglobin is an osmolyte within the red cell, any reduction in its concentration should result in proportionately less swelling. Evolutionary or acclimatory forces acting to minimize postexercise blood viscosity in BDC would do well to minimize the concentrations of intracellular, nondiffusible osmolytes in red cells, of which hemoglobin is one. Despite this inferred lower osmotic activity of the larger BDC red cells, erythrocytic swelling was actually greater in this population (fig. 6, bottom). Scotian Shelf cod had an average 0.022 ± 0.015 g/mL postexercise reduction in intraerythrocytic hemoglobin concentration, whereas BDC suffered an average 0.039 ± 0.012 g/mL reduction, or 1.77 times that of SSC. This is probably due to more adrenergic swelling in BDC because of the lower plasma pH (Tang and Boutilier 1988). Presumably swelling would have been even worse if BDC had had smaller, more concentrated erythrocytes. An interesting further test of this idea would be to compare the degree of adrenergic swelling between these two populations (cf. Butler et al. 1989) while controlling pH. If the BDC population has been selected for minimized postexercise hemoconcentration, we would predict a blunting of the adrenergic response in this population.

A third revealing feature of our data is the identical rise in plasma ion concentrations in animals exercised at different salinities. This implies that the exercise-induced rise in extracellular fluid ion levels is the result of exchanges with the intracellular compartment and not the environment and contradicts previous assumptions that postexercise increases in plasma ion concentrations in marine fish were the product of ionoregulatory systems not keeping pace with demand (Byrne, Beamish, and Saunders 1972). This finding also suggests that postexercise, intercompartmental fluid movements are similar between sea- and freshwater vertebrates (Boutilier, Emilio, and Shelton 1986).

The major finding of this study is that physiologically distinct populations of Atlantic cod coexist over scales that are spatially small compared with their geographic range. While this is not a unique finding for marine organisms (Hilbish and Koehn 1985) nor even for marine fishes (DiMichele and Powers 1982), this is an unusual finding for a species such as Atlantic cod that can have home ranges in the thousands of kilometers, with isolated individuals that have been documented to move over most of the species' North Atlantic range (Harden-Jones 1968). The generality of this finding and its applicability to other fishes, cod populations, and physiological measurements is unknown.

Acknowledgments

We would like to thank Dale Webber, Todd Bishop, and Shannon Reidy for their excellent technical support of this project. We would also like to thank Dr. Ed Trippel, Dr. Louis Fortier, and Jim Fennel for the use of their unpublished information. Helpful comments on the manuscript were made by Drs. G. H. Pogson and S. R. Kerr. Funding for this research has been provided by the Ocean Production Enhancement Network (OPEN), one of the 15 Networks of Centre of Excellence supported by the government of Canada and by a North Atlantic Treaty Organization (NATO) International Research Fellowship (J.A.N.). Funds were also provided by Dalhousie Graduate School Research Development Grants to J.A.N. and Y.T.

Literature Cited

Arnold, S. J. 1983. Morphology, performance and fitness. Am. Zool. 23:347–361.

Beamish, F. W. H. 1978. Swimming capacity. Pages 101–187 in W. S. Hoar and D. J. Randall, eds. Fish physiology. Vol. 7. Academic Press, New York.

Bentley, P. J. 1971. Endocrines and osmoregulation. Springer, New York. 300 pp.

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Literature Cited

ARNOLD, S. J. 1983. Morphology, performance and fitness. Am. Zool. 23:347–361.

BEAMISH, F. W. H. 1978. Swimming capacity. Pages 101–187 in W. S. HOAR and D. J. RANDALL, eds. Fish physiology. Vol. 7. Academic Press, New York.

BENTLEY, P. J. 1971. Endocrines and osmoregulation. Springer, New York. 300 pp.

BERGMEYER, H. U. 1984. Methods of enzymatic analysis. Vol. 6. Metabolites. Pt. 1. Carbohydrates. Chemie, Weinheim. 701 pp.

BOUTILIER, R. G., M. G. EMILIO, and G. SHELTON. 1986. The effects of mechanical work on the electrolyte and water distribution in amphibian skeletal muscle. J. Exp. Biol. 120:330–350.

BOUTILIER, R. G., T. H. HEMING, and G. K. IWAMA. 1984. Physicochemical parameters for use in fish respiratory physiology. Pages 403–430 in W. S. HOAR and D. J. RANDALL, eds. Fish physiology. Vol. 10a. Gills. Academic Press, New York.

BRETT, J. R. 1964. The respiratory metabolism and swimming performance of young sockeye salmon. J. Fisheries Res. Board Can. 21:1183–1226.

BURGGREN, W. W., and W. E. BEMIS. 1990. Studying physiological evolution: paradigms and pitfalls. Pages 191–228 in M. H. NITECKI, ed. Evolutionary innovations. University of Chicago Press, Chicago.

BUTLER, P. J., M. AXELSSON, F. EHRENSTRÖM, J. D. METCALF, and S. NILSSON. 1989. Circulating catecholamines and swimming performance in the Atlantic cod, *Gadus morhua*. J. Exp. Biol. 141:377–387.

BYRNE, J. M., F. W. H. BEAMISH, and R. L. SAUNDERS. 1972. Influence of salinity, temperature, and exercise on plasma osmolality and ionic concentration in Atlantic salmon (*Salmo salar*). J. Fisheries Res. Board Can. 29:1217–1220.

CLARKE, B. C. 1979. The evolution of genetic diversity. Proc. R. Soc. Lond. 205B: 453–474.

DIMICHELE, L., and D. A. POWERS. 1982. Physiological basis for swimming endurance differences between LDH-B genotypes of *Fundulus heteroclitus*. Science 216:1014–1016.

DRINKWATER, K., and G. TAYLOR. 1982. Monthly means of the temperature, salinity, and density along the Halifax section. Can. Tech. Rep. Fisheries Aquatic Sci., no. 1093. 67 pp.

ENDLER, J. A. 1986. Natural selection in the wild. Princeton University Press, Princeton, N.J.

FARMER, G. J., and F. W. H. BEAMISH. 1969. Oxygen consumption of *Tilapia nilotica* in relation to swimming speed and salinity. J. Fisheries Res. Board Can. 26:2807–2821.

FEBRY R., and P. LUTZ. 1987. Energy partitioning in fish: the activity-related cost of osmoregulation in a euryhaline cichlid. J. Exp. Biol. 128:63–85.

FRY, F. E. J. 1978. Effects of environmental factors on the physiology of fish. Pages 1–98 in W. S. HOAR and D. J. RANDALL, eds. Fish physiology. Vol. 3. Academic Press, New York.

FUNG, Y. C. 1984. Biodynamics: circulation. Springer, New York. 404 pp.

GILES, M. A. 1991. Strain differences in hemoglobin polymorphism, oxygen consumption, and blood oxygen equilibria in three hatchery broodstocks of Arctic charr, *Salvelinus alpinus*. Fish Physiol. Biochem. 9:291–301.

GRIGG, G. C. 1974. Respiratory function of blood: fishes. Pages 331–368 in M. FLORKIN and B. T. SHEER, eds. Chemical zoology. Vol. 8. Academic Press, New York.

HARDEN-JONES, F. R. 1968. Fish migration. Arnold, London. 325 pp.

HILBISH, T. J., and R. K. KOEHN. 1985. The physiological basis of natural selection at the *Lap* locus. Evolution 39:1302–1317.

HOAR, W. S., and C. P. HICKMAN, JR. 1963. A laboratory companion for general and comparative physiology. 3d ed. Prentice-Hall, Englewood Cliffs, N.J. 307 pp.

KRAUEL, D. P. 1975. The physical oceanography of the Bras d'Or Lakes 1972–1974. Environ. Can. Fisheries Mar. Serv. Tech. Rep. 570. 357 pp.

LINCOLN, H. R. 1791. Letter. In J. Bellknapp. 1813. History of N. Hampshire. Bradford & Read, Boston. Reprinted as pages 217–221 in J. C. Pearson. 1972. Fish and fisheries of colonial North America: a documentary history of the fishing resources of the United States and Canada. Pt. 2. New England states. National Technical Information Service, Springfield, Va.

LOVE, R. M. 1970. The chemical biology of fishes. Academic Press, London. 547 pp.

———. 1980. The chemical biology of fishes. Vol. 2. Advances 1968–1977. Academic Press, London. 943 pp.

MORK, J., N. RYMAN, G. STÅHL, F. UTTER, and G. SUNDNES. 1985. Genetic variation in atlantic cod (*Gadus morhua*) throughout its range. Can. J. Fisheries Aquatic Sci. 42:1580–1587.

NELSON, J. A. 1990. Muscle metabolite response to exercise and recovery in yellow perch (*Perca flavescens*): comparison of populations from naturally acidic and neutral waters. Physiol. Zool. 63:886–908.

NELSON, J. A., and G. S. MITCHELL 1992. Blood chemistry response to acid exposure in yellow perch (*Perca flavescens*): comparison of populations from naturally acidic and neutral environments. Physiol. Zool. 65:493–514.

NELSON, J. A., Y. TANG, and R. G. BOUTILIER. 1992*a*. Effects of environmental factors on the energy metabolism and swimming performance of fish. Canadian Conference for Fisheries Research annual meeting, Jan. 3–5, 1992, abstracts.

———. 1992*b*. Physiological and morphological constraints on locomotor capacity in Atlantic cod. Can. Soc. Zool. Bull. 23(2):93.

PIERCE, S. K., L. M. ROWLAND-FAUX, and S. M. O'BRIEN. 1992. Different salinity tolerance mechanisms in Atlantic and Chesapeake Bay conspecific oysters: glycine betaine and amino acid pool variations. Mar. Biol. 113:107–115.

POPE, A., and J. J. HARPER. 1966. Low-speed wind tunnel testing. Wiley, New York. 457 pp.

PRIMMETT, D. R. N., D. J. RANDALL, M. MAZEAUD, and R. G. BOUTILIER. 1986. The role of catecholamines in erythrocytic pH regulation and oxygen transport in rainbow trout (*Salmo gairdneri*) during exercise. J. Exp. Biol. 122:139–148.

SAS INSTITUTE. 1989. SAS/STAT user's guide. Version 6. 4th edition. Vol. 1. Cary, N.C., SAS Institute. 943 pp.

SAUNDERS R. L. 1963. Respiration of the Atlantic cod. J. Fisheries Res. Board Can. 20: 373–386.

SCHULTE, P. M., C. D. MOYES, and P. W. HOCHACHKA. 1992. Integrating metabolic pathways in post-exercise recovery of white muscle. J. Exp. Biol. 166:181–195.

SEVERINGHAUS, J. W. 1965. Blood gas concentrations. Pages 1475–1487 in W. O. FENN and H. RAHN, eds. Handbook of physiology. Vol. 2. American Physiological Society, Washington, D.C.

SINGH R. S., and A. D. LONG. 1992. Geographic variation in *Drosophila:* From molecules to morphology and back. Trends Ecol. Evol. 7:340–345.

SMITH, P. K., R. I. KROHN, G. T. HERMANSON, A. K. MALLIA, F. H. GARTNER, M. D. PROVENZANO, E. K. FUJIMOTO, N. M. GOEKE, B. J. OLSON, and D. C. KLENK. 1985. Measurement of protein using bicinchoninic acid. Anal. Biochem. 150:76–85.

SOIVIO, A., K. NYHOLM, and K. WESTMAN. 1975. A technique for repeated sampling of the blood of individual resting fish. J. Exp. Biol. 62:207–217.

TANG, Y., and R. G. BOUTILIER. 1988. Correlation between catecholamine release and degree of acidotic stress in trout. Am. J. Physiol. 255:R395–R399.

THOMAS, A. E., and M. J. DONAHOO. 1977. Differences in swimming performance among strains of rainbow trout (*Salmo gairdneri*). J. Fisheries Res. Board Can. 34:304–306.

WEBB, P. W. 1974. Hydrodynamics and energetics of fish propulsion. Bull. Fisheries Res. Board Can., no. 190. 159 pp.

WEBBER, D. M. 1985. Monitoring the metabolic rate and activity of (*Illex illecebrosus*) with telemetered jet pressures. M.S. thesis. Dalhousie University. 123 pp.

WEBER, R. E., and F. B. JENSEN. 1988. Functional adaptations in hemoglobins from ectothermic vertebrates. Annu. Rev. Physiol. 50:161–179.

ZALL, D. M., D. FISHER, and M. Q. GARNER. 1956. Photometric determination of chloride in water. Anal. Chem. 28:1665–1668.