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Does condition of Atlantic cod (*Gadus morhua*) have a greater impact upon swimming performance at U_{crit} or sprint speeds?

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Summary

To compare the sensitivity of sprint and critical (U_{crit}) swimming speeds to the condition of Atlantic cod (*Gadus morhua*) and to identify the best anatomic, behavioural and biochemical correlates of these types of swimming, we established two groups of cod that were fed or starved for 12 weeks. We evaluated sprint swimming and U_{crit} performance as well as the speed at which repeated burst–coast movements began in the U_{crit} test before measuring the metabolic capacities of red and white muscle sampled caudally, centrally and rostrally and the anatomic characteristics of the cod. White muscle lactate was measured directly after the U_{crit} test. As expected, the twofold difference in Fulton's condition factor (0.5±0.04 for starved and 1.0±0.1 for fed cod) was accompanied by large differences in the anatomic and biochemical parameters measured. Despite the relative sparing of muscle aerobic capacity during starvation and despite the greater use of oxidative fibres during U_{crit} compared with sprint swimming, these types of swimming differed by much the same extent between starved and fed cod. In the U_{crit} tests, white muscle lactate levels and lactate accumulation per burst–coast movement were considerably higher in fed than starved cod, suggesting more intensive use of fast muscle fibres in cod in good condition. Multiple regression analysis indicated strong correlations between U_{crit}, the speed at which regular burst–coasting began and the activity of pyruvate dehydrogenase (PDH) in red muscle (both caudal and central positions). PDH activity may limit the rate of oxidative ATP production by red muscle. The activity of cytochrome *c* oxidase in rostral white muscle was the strongest correlate of sprint swimming, suggesting that aerobic preparation of white muscle facilitates rapid contraction. The correlation between U_{crit} and sprint swimming was weak, perhaps due to inter-individual differences in sensitivity during sprint tests.

Key words: white muscle, red muscle, condition, U_{crit} swimming, sprint swimming, aerobic metabolism, anaerobic metabolism, Atlantic cod, *Gadus morhua*.

Introduction

For active predatory fish, swimming capacity is a critical determinant of feeding success and survival. Sprint swimming influences a fish's capacity to escape predators and to capture prey. Fast-starts, or startle responses, are some of the most rapid locomotory behaviours of vertebrates (Jayne and Lauder, 1993). Burst swimming, i.e. swimming activity lasting less than 20 s, also includes sprints that are slower and last longer than fast-start responses. Sprint swimming is powered by the white glycolytic fibres that constitute the bulk of the myotomal mass (Domenici and Blake, 1997; Franklin and Johnston, 1997). By contrast, sustained swimming allows fish to migrate long distances, maintain position or explore the environment in search of food or more favourable living conditions. Sustained swimming principally relies upon red oxidative fibres, as they are resistant to fatigue and have a slow shortening speed (Bone et al., 1978). Prolonged swimming is intermediate between sprint and sustained swimming. The U_{crit} protocol, in which fish swim at gradually increasing speeds, is frequently used to evaluate prolonged swimming performance. In Atlantic cod, *Gadus morhua*, glycolytic fibres are recruited during unsteady, burst–coast swimming towards the end of U_{crit} tests (Reidy et al., 2000). Electromyography shows only red muscle activity during steady swimming, activation of red and white muscle during unsteady swimming and decreased red muscle activity with increasing speed (Jayne and Lauder, 1994). Whereas electromyographic studies are rarely coupled with U_{crit} tests, electromyographic data suggest that white muscle becomes active only at speeds near U_{crit}. However, in salmonids, white muscle is metabolically activated during swimming at speeds above 70% U_{crit}, with levels of phosphocreatine, glycogen and even ATP decreasing markedly (Burgetz et al., 1998; Richards et al., 2002). Prolonged swimming, as measured by the U_{crit} protocol, relies upon both oxidative and glycolytic fibres.

In temperate habitats, fish frequently encounter marked seasonal variations in food availability that may modify their

performance. When reductions in prey abundance are coupled with seasonal decreases in temperature, fish may survive using the reserves accumulated during periods of high productivity. However, generalised decreases in prey availability can cause extended periods of starvation from which fish have difficulty recovering. In Atlantic cod, hepatic lipid reserves are the first to be mobilised during starvation, followed by glycogen from liver and muscle and finally by muscle protein (Black and Love, 1986). Such decreases in macromolecular content lead to marked increases in tissue water, from 15 to 80% in the liver and from 77 to 92% in white muscle (Dutil et al., 1995; Lambert and Dutil, 1997a,b). Accordingly, prolonged starvation markedly modifies the metabolic capacities of cod tissues (Guderley et al., 1996; Dutil et al., 1998). Just as cod white muscle is more affected by starvation than red muscle, starvation has a lesser impact upon the aerobic than the glycolytic capacity in both fibre types (Martínez et al., 2003). The activities of four glycolytic enzymes [phosphofructokinase (PFK), lactate dehydrogenase (LDH), pyruvate kinase (PK) and creatine kinase (CK)] decline markedly in red and white muscle with starvation, while the activities of two mitochondrial enzymes [cytochrome *c* oxidase (CCO) and citrate synthase (CS)] are less affected. These changes in muscle metabolic capacity are likely to reduce swimming capacity, in particular all behaviours relying upon glycolytic capacity.

The sparing of muscle aerobic capacity during starvation (Johnston and Goldspink, 1973; Beardall and Johnston, 1983; Loughna and Goldspink, 1984) should favour the maintenance of routine locomotor activities at the detriment of sprint swimming. In cod, endurance at speeds above U_{crit} is reduced 70% by a decrease in condition. As the number of burst–coast movements is the best predictor of endurance at these speeds, glycolytic fibres provide much, if not most, of the needed power (Martínez et al., 2003). U_{crit} tests assess the speed a fish can sustain during the experimental swimming period (typically 20–40 min). While swimming near U_{crit} is supported both by oxidative and glycolytic fibres, sprint swimming is only powered by glycolytic fibres. Given these differences in the muscles participating in sprint and U_{crit} swimming, the loss of glycolytic capacity occurring with a decrease in condition should have a stronger impact upon sprint than U_{crit} swimming.

Biomechanical studies of fish swimming clearly show that muscle fibres work differently according to their position along the trunk (van Leeuwen et al., 1990; Altringham et al., 1993; Rome et al., 1993; Wardle and Videler, 1993; Jayne and Lauder 1994; Thys, 1997). In Atlantic cod, caudal white muscle fibres transmit the force generated in the rostral and middle myotomes towards the caudal fin (Altringham et al., 1993; Davies et al., 1995; Johnston et al., 1995). Caudal fibres are primarily active during lengthening and only produce positive work towards the end of their activity. In Atlantic cod, enzymatic activities in white and red fibres change longitudinally (Martínez et al., 2000, 2003), with the highest mass-specific activities being found caudally. While these longitudinal patterns are intriguing, particularly in light of the longitudinal differences in performance during swimming, it is unknown whether the metabolic capacities of the muscle fibres at different positions in the cod are correlated with the capacities for sprint and U_{crit} swimming.

In the present study, we established two groups of Atlantic cod, differing widely in condition, to evaluate (1) whether sprint or U_{crit} swimming is more sensitive to differences in condition and (2) to obtain a wide range of muscle metabolic capacities to evaluate metabolic correlates of sprint and U_{crit} swimming performance. During 12 weeks, the cod in one group were fed every second day whereas those in the other group were starved. We then determined the sprint and U_{crit} swimming performance of the cod. Sprint swimming was measured as described by Martínez et al. (2002). The U_{crit} was measured using a Blazka swimming respirometer following Reidy et al. (2000). As cod can differ in the extent to which they rely upon unsteady swimming while reaching U_{crit} (Nelson et al., 1994, 1996), we filmed the fish to quantify burst–coast movements. A white muscle biopsy was taken directly after the U_{crit} test to determine lactate levels. We assessed the metabolic capacities of white and red muscle sampled at three locations along the body (behind the head, at the middle of the body and at the caudal peduncle). We estimated the total amount of red and white muscle by dissection. In white muscle, we measured glycolytic enzymes (PFK, PK, CK and LDH), mitochondrial enzymes (CCO and CS) and a biosynthetic enzyme [nucleoside diphosphate kinase (NDPK)]. In red muscle, we measured PFK, LDH, CCO, CS and pyruvate dehydrogenase (PDH), a mitochondrial enzyme controlling rates of pyruvate oxidation (Richards et al., 2002). These data were used to examine the links between sprint and U_{crit} swimming, the speed at repeated burst–coasting, condition, anatomic characteristics, enzyme, protein and water levels in white and red muscles along the cod body.

Materials and methods

Experimental animals

Atlantic cod, *Gadus morhua* L., were captured by trawling in the St Lawrence Estuary near Matane (Québec, NAFO area 4TVn) in June (17–23), 1999. The fish were kept in circular tanks (13 m^3) at Institut Maurice-Lamontagne. These tanks were kept at ambient seawater temperatures (6.9±0.5°C) and salinity (≈28.6±0.3‰) and under natural photoperiod. For three weeks, fish were fed a maintenance ration of capelin, *Mallosus villosus.* Then, 60 healthy fish were treated with metomidate (3 mg l^{-1}) in seawater at 10°C and tagged with a Visual Implant Tag (Northwest Marine Technology, Shaw Island, WA, USA) in the first dorsal fin to follow individual performance. They were weighed and measured for fork length and, finally, randomly placed into four circular tanks (1.5 m^3; N=15 each, 2 each for the starved and fed groups). The tanks were kept at constant temperature (9.9±0.4°C) and salinity (≈28.7±1.1‰) and under natural daylengths. During the two weeks following tagging, fish were fed the maintenance ration

to allow them to recover from the stress of tagging. Then, during the next 12 weeks, the fed group was fed to satiation with capelin every second day, while the other group received nothing. No mortality occurred during this period. Swim tests were conducted at the holding temperature, first on the starved fish and then the fed fish. Food was withheld from the fed fish 48 h before the sprint test. After the sprint test, the fish were given 24 h in a quiet tank near the respirometer before the U_{crit} test. The fish were sacrificed by a blow to the head immediately after the U_{crit} swimming test, measured, weighed and dissected (see below).

Sprint and U_{crit} *swimming protocols*

Swim performance was measured at Institut Maurice-Lamontagne. We used a swimming flume based on Nelson et al. (2002), modified and used as described by Martínez et al. (2002) to measure sprint performance. Statistical analysis used the maximum velocity recorded in the first six sections of the tunnel as a measure of a fish's sprint performance. U_{crit} swimming tests were performed in a Blazka-type respirometer (BII Consulting, Burlington, ON, Canada) equipped with an electrical grid at the downstream end. The respirometer had a total volume of 109 litres, a swimming section of 94 cm length and 24 cm in diameter, and was powered by a motor with micrometric adjustment of the power. U_{crit} tests were based on Brett (1964) as modified by Nelson et al. (1994) without the overnight acclimation. First, the fish had a 30 min acclimation in the swim tunnel at a speed of 15 cm s^{-1}. The speed was then gradually increased to 25 cm s^{-1} (an increase of 1 cm s^{-1} min^{-1} over 10 min), the speed at which the test started. Fish swam for 20 min intervals, between which the speed was increased by 10 cm s^{-1} in 10 min (1 cm s^{-1} min^{-1} increment) until the fish was exhausted and the test terminated. The fish was considered exhausted when it would stay against the back grid and would not react to electrical stimuli. An electrical stimulus was given to a fish only if it remained against the back grid for at least 5 s. The exact swimming speeds were established using a speed–position relationship determined in preliminary tests. The U_{crit} speed was calculated as described by Brett (1964), and a solid blocking effect correction was performed according to equation 94 of Webb (1978). For the starved cod, this correction was between 4 and 6% whereas for fed cod it ranged between 7 and 11.5%. Fish were filmed during the U_{crit} swimming test to establish their position in the chamber (for application of the speed–position relationship) as well as the number of burst–coast movements.

Dissection and tissue sampling

Hematocrit was measured immediately after death according to Klawe et al. (1963). A sample of white muscle (approximately 1 g) was rapidly removed from below the dorsal fin for lactate measurements. Muscle samples for enzyme measurements (3–5 g and 0.5–2 g for white and red muscle, respectively) were taken at three sites along the cod body: (1) behind the head (rostral), (2) at the middle of the body and (3) in the caudal region. White muscle samples were taken above the lateral line, while red muscle was dissected just under the lateral line. Care was taken during these dissections to avoid contamination by pink fibres. All tissues sampled were immediately frozen in liquid nitrogen, then stored at –70°C before transport on dry ice to Université Laval, where they were stored at –70°C until biochemical assay. We quantified the total mass of white and red muscle by dissection. Intermediate (pink) fibres were included with red muscle in this dissection. All tissues (±0.1 g) and gonads were weighed to calculate the hepatosomatic index (liver mass × somatic $mass^{-1}$) and the gonadosomatic index (gonad mass × somatic $mass^{-1}$), respectively. Somatic mass was calculated by subtracting the mass of the gonad and stomach contents from the total mass of the fish.

Tissue extraction

Extracts were prepared by homogenising samples in 10 volumes of 50 mmol l^{-1} imidazole HCl, 2 mmol l^{-1} $MgCl_2$, 5 mmol l^{-1} ethylene diamine tetraacetic acid (EDTA), 1 mmol l^{-1} reduced glutathione, 0.1% Triton X-100, pH 7.5, using a Polytron (Brinkman Instruments, Rexdale, Ontario, Canada) for three 20-s periods. The samples were maintained on ice during and between the periods of homogenisation.

Enzyme activity assays

CCO, CS, NDPK, PFK, PK and LDH were measured according to Couture et al. (1998). For CK (E.C. 2.7.3.2), the assay was as follows: 75 mmol l^{-1} Tris-HCl, 5 mmol l^{-1} $MgCl_2.6H_2O$, 4 mmol l^{-1} glucose, 0.75 mmol l^{-1} ADP, 5 mmol l^{-1} AMP, 0.3 mmol l^{-1} NADP, 24 mmol l^{-1} phosphocreatine (omitted for control), hexokinase and glucose-6-phosphate dehydrogenase in excess, pH 7.6. For PDH (E.C. 1.2.4.1), the assay followed Thibault et al. (1997) as follows: 50 mmol l^{-1} Tris-HCl, 0.5 mmol l^{-1} EDTA, 0.2% Triton X-100, 2.5 mmol l^{-1} NAD, 0.1 mmol l^{-1} coenzyme A, 1 mmol l^{-1} $MgCl_2$, 0.1 mmol l^{-1} oxalate, 1 mg ml^{-1} bovine serum albumin, 0.6 mmol l^{-1} p-iodonitrotetrazolium violet (INT), 6 U lipoamide dehydrogenase, 0.2 mmol l^{-1} thiamine pyrophosphate, 5 mmol l^{-1} pyruvate (omitted for control), pH 7.8. Enzyme activities were expressed in international units (μmol substrate transformed to product min^{-1}) per gram tissue wet mass. Muscle enzyme activities were measured in the order: PFK, PK, CK, LDH, CS, CCO and NDPK in white muscle, and PFK, PDH, CS, CCO, LDH in red muscle. All assays were run in duplicate.

Muscle protein, lactate and water contents

Protein concentration was measured using bicinchoninic acid (Smith et al., 1985) in white and red muscle. Muscle water content was determined after drying 2 g of muscle for 48 h at 60°C. Lactate content was determined using the method of Gutmann and Wahlefeld (1974). Red muscle water content was measured in the rostral sample.

Chemicals and biochemicals were purchased from Sigma Chemical Co. (St Louis, MO, USA), Boehringer Mannheim

Co. (Montréal, Canada), Fisher Scientific Co. (Montréal, Canada) or ICN Pharmaceuticals Inc. (Montréal, Canada).

Calculated parameters and statistical analyses

Individual growth rates were calculated using the following equation:

$$\text{Growth rate} = [(\ln W_2 - \ln W_1)/t] \times 100 ,$$

where growth rate is measured in % day^{-1}, W_1 represents the initial mass in g or fork length in cm, W_2 represents the final mass in g or fork length in cm, and t is the number of days of the experimental period.

We used JMP 3.2.1 (SAS Institute Inc., Duxbury Press, Belmont, CA, USA) to examine the effect of feeding treatment (starvation and feeding) upon anatomic parameters and general characteristics. We further examined the impact of feeding treatment and site of sampling (with repeated measures in individual fish) on enzyme activity, protein and water content. In these models, feeding treatment was a factor, sampling site was nested within fish and fish was nested within feeding treatment. An interaction term between feeding treatment and sampling site was also included. Enzyme activity, total protein and water content were the variables. Data met the criteria of normality and homogeneity of variances. Differences were considered significant at α=0.05. The *a posteriori* 'add contrasts' function was used to establish specific differences due to feeding treatment at each sampling site. Longitudinal differences in enzyme activity were also examined with separate models for each feeding group. In these models, site of sampling (repeated measures in individual fish) was the factor, and *a posteriori* application of the 'add contrasts' function was used to establish specific differences.

A forward stepwise analysis was used to determine which variables best explain the variability of sprint and U_{crit} performance as well as the speed at the start of repeated burst–coast movements. In addition to the biochemical variables, the following morphological variables were included in the analysis: somatic mass (g), length (cm), somatic condition factor, sex, gonadosomatic index and hepatosomatic index, as well as the mass (g) of gonads, liver, stomach, intestine, heart, gill arches, carcass, total white muscle and total red muscle. The criterion of entry and removal of a variable into the regression was based on the level of significance of the R^2 (P<0.05 to stay in the model). Once the forward stepwise analysis was finished, we plotted the significant variables from the forward stepwise model *versus* the predicted performance variable (sprint, U_{crit} or speed at repeated burst–coasts) to see how they correlated. As length is one of the major factors determining maximum speed in fish, it was added to the last model. Once the final models were built, we checked the collinearity between the remaining variables using the VIF (variance inflation factor). When a variable showed marked collinearity with the others, it was removed from the model (statistical VIF index >15). Models used the forward stepwise analysis and the VIF option in JMP 3.2.1 (SAS Institute Inc.).

Table 1. *General characteristics of Atlantic cod before and after 12 weeks of feeding or starvation at 9.9±0.4°C and a salinity of 28.7±1.1‰*

Parameter	Starved cod	Fed cod
Initial mass (g)	754±136	799±166
Initial length (cm)	46.1±2.8	45.6±3.0
Initial condition factor (initial mass × length^3)×100	0.76±0.03	0.84±0.10
Final mass (g)	509±112	1457±356
Final length (cm)	45.8±2.8	52.3±3.2
Final length range (cm)	40.3–50.3	46.2–58.3
Final condition factor	0.53±0.04	1.0±0.1
White muscle mass (g)	143±46	732±178
Red muscle mass (g)	10.4±3.4	23.4±6.0
Growth rate in mass (% day^{-1})	–0.49±0.11	0.78±0.20
Growth rate in length (% day^{-1})	–0.01±0.01	0.18±0.06
Gonadosomatic index	1.0±0.8	0.8±0.5
Hepatosomatic index	0.6±0.1	7.9±2.1
Hematocrit (%)	32.55±10.27	34.24±5.29
Male/female	15/15	15/14

Data are shown as means ± S.D.

Results

After 12 weeks, cod from the fed and starved groups differed considerably. The condition factor and hepatosomatic index were much lower for the starved cod compared with the fed cod (P<0.0001), but the gonadosomatic index did not differ between the groups (Table 1). The cod in the fed and starved groups differed much less in the amount of red muscle than in the amount of white muscle. Starved cod had 81% less white muscle but only 56% less red muscle mass than fed cod (Table 1). Thus starved cod had a greater relative mass of red muscle in the body (by 22%; P=0.002), a higher percentage of red muscle in the total muscle mass (by 56%; P<0.0001) and a greater ratio of red to white muscle (by 58%; P<0.0001). Growth rates based on total mass or on fork length were negative for starved cod (Table 1). These differences in growth and condition were not caused by tank effects since we observed the same patterns in the two tanks for each group and previous evidence clearly demonstrates an impact of feeding treatment on these variables (Dutil et al., 1998).

Effects of feeding regime on biochemical parameters

As expected for such a large difference in condition factor, starvation significantly decreased the activity of all enzymes measured in white muscle (PFK, PK, CK, LDH, CS, CCO and NDPK; P<0.0001; Fig. 1). The activity of the glycolytic enzymes increased in the rostral-caudal direction (PFK, PK and LDH, P<0.0001; CK, P=0.046), as did the activity of CS and CCO (P=0.007). NDPK levels decreased in the rostral-caudal direction (P<0.0001). At each site of sampling, starved cod had significantly lower levels of all enzymes measured (P<0.0001), with the greatest differences being apparent for PFK, PK and LDH.

In red muscle, the activity of all of the enzymes measured

was significantly lower in starved than fed cod at each site of sampling (P<0.0001, except for PDH in the middle of the body where P=0.0003; Fig. 2). As in white muscle, the glycolytic enzymes were more affected than the mitochondrial enzymes, with the exception of CCO behind the head. Enzyme activity in red muscle showed longitudinal variation within each group. Mitochondrial enzymes increased in the rostral-caudal direction in both fed and starved cod. A rostral-caudal increase in glycolytic enzyme activities was also apparent in starved cod. However, in fed cod, glycolytic enzyme activities in red muscle were higher in rostral than caudal samples.

While starved cod had a lower protein content in white and red muscle than fed cod (P<0.001; Figs 1, 2), site of sampling did not systematically affect this parameter. Only in starved cod did sampling site affect protein contents: in red muscle, the rostral sample had the lowest protein content, whereas in white muscle the rostral sample had the highest protein content. Starved cod had water contents of approximately 85% in red and white muscle; these were 5–6% higher than in fed cod. In white muscle, water content showed a significant interaction between feeding regime and the site of sampling (P<0.0001): rostral samples had the lowest water content in fed cod while in starved cod, the caudal samples were lowest. Starvation did not significantly change the hematocrit (P=0.38; Table 1).

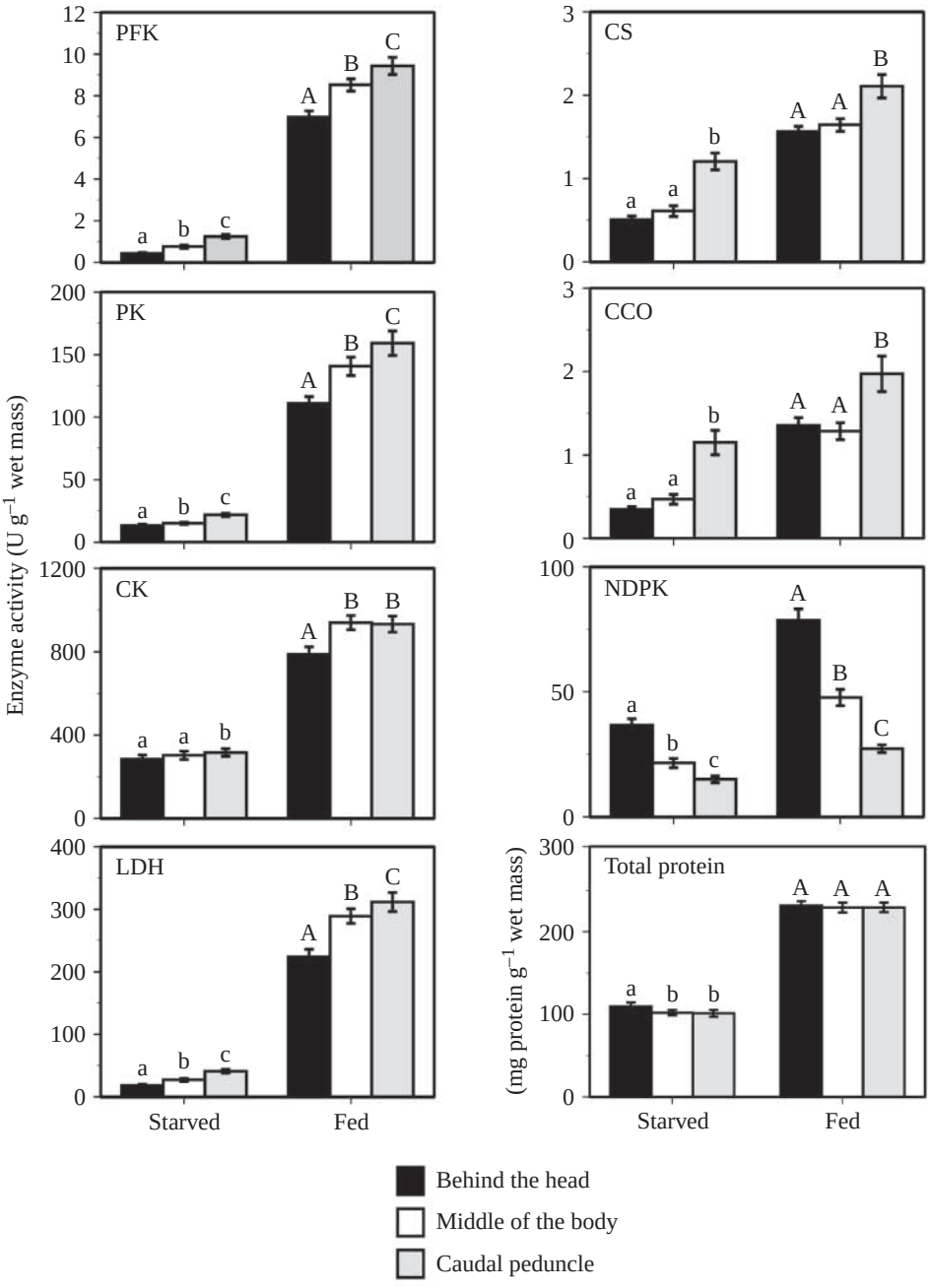

Fig. 1. Activities of glycolytic, mitochondrial and biosynthetic enzymes and total protein in white muscle, sampled at three sites along the length of the body (behind the head, in the middle of the body and at the caudal peduncle) in starved and fed cod. Data are shown as means ± S.E.M. Letters above the error bars for each feeding treatment (starved/fed) indicate statistical differences; when two columns have the same letter, they do not differ (*a posteriori* comparisons, P>0.05). Lowercase letters are for starved cod while uppercase letters are for fed cod. Abbreviations: CCO, cytochrome *c* oxidase; CK, creatine kinase; CS, citrate synthase; LDH, lactate dehydrogenase; NDPK, nucleoside diphosphate kinase; PFK, phosphofructokinase; PK, pyruvate kinase.

Sprint swimming performance and its physiological correlates

The sprint performance of cod expressed as absolute (cm s^{-1}) or relative speeds [body length s^{-1} (bl s^{-1})] was significantly affected by starvation (P<0.0001 and P<0.05, respectively; Fig. 3). The maximal burst swimming speed expressed as cm s^{-1} decreased by 30% whereas the speed expressed in bl s^{-1} decreased by 21.5%. Most cod traversed the tunnel in less than 2 s and reached their maximal speed early in the tunnel. Sex did not affect the sprint swimming performance in either group (P>0.05).

Of all anatomic and biochemical variables measured, the activities of CCO and NDPK in white muscle behind the head were the strongest correlates of sprint performance expressed

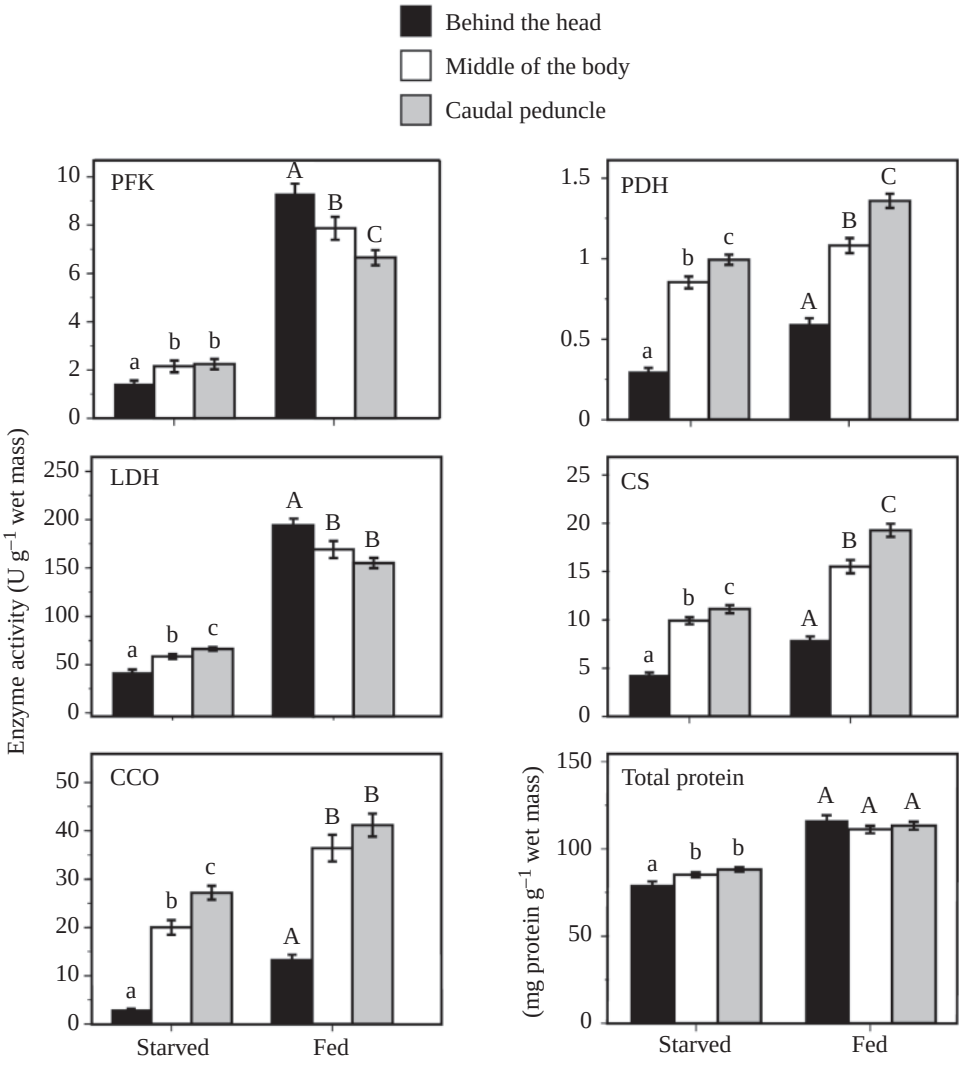

Fig. 2. Activities of total protein, glycolytic and mitochondrial enzymes in red muscle, sampled at three sites along the length of the body (behind the head, in the middle of the body and at the caudal peduncle) in starved and fed cod. Data are shown as means ± S.E.M. Letters above the error bars for each feeding treatment (starved/fed) indicate statistical differences; when two columns have the same letter, they do not differ (*a posteriori* comparisons, $P>0.05$). Abbreviations: CCO, cytochrome *c* oxidase; CS, citrate synthase; LDH, lactate dehydrogenase; PDH, pyruvate dehydrogenase; PFK, phosphofructokinase.

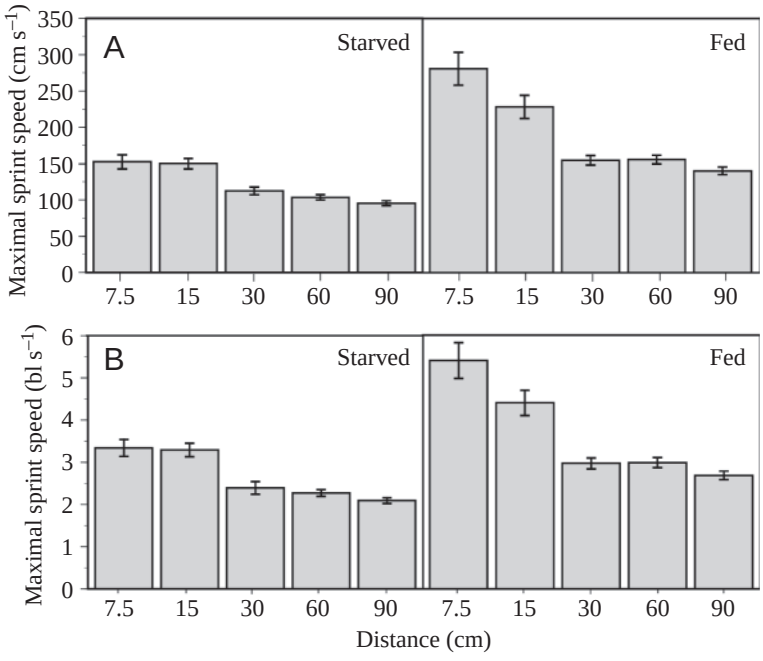

Fig. 3. Absolute (cm s^{-1}) and relative (bl s^{-1}) sprint swimming speed of cod as they pass through the swimming apparatus, after 12 weeks of feeding treatment. Sprint speeds were measured as explained in Martínez et al. (2002). We show the fastest speed between the beginning of the tunnel and the lasers located at the indicated distances from the first laser in the tunnel. Data are shown as means ± S.E.M.

Table 2. *Multiple regression models that best explain the variability of sprint swimming in Atlantic cod*

			Without fork length	With fork length
Absolute sprint speed (cm s^{-1})				
Variables in the model			*P*-value	*P*-value
CCO activity in rostral white muscle			<0.0001	<0.0001
NDPK activity in rostral white muscle			0.0051	0.0035
Total protein in central white muscle			Not in model	0.0411
Fork length (cm)			Not in model	0.0407
Model	R^2	d.f.	*F*-ratio	*P*-value
	0.51	2	29.40	<0.0001
With fork length	0.56	4	17.47	<0.0001
Relative sprint speed (bl s^{-1})				
Variables in the model				
CCO activity in rostral white muscle			<0.0001	<0.0001
PDH activity in rostral red muscle				0.043
Fork length (cm)				0.041
Model	R^2	d.f.	*F*-ratio	*P*-value
	0.29	1	22.37	<0.0001
With fork length	0.44	3	13.34	<0.0001

Variables entered into the model using forward stepwise selection.

as cm s^{-1} (Table 2). Both variables were positively correlated with the absolute speed. Because length markedly affects swimming speeds and is positively correlated with sprint swimming, we constructed a model in which length was forced to stay. In this model, the same variables (CCO and NDPK in the rostral white muscle) and the protein level in white muscle at the middle of the body were correlated with absolute sprint swimming (Table 2). The activity of CCO in rostral white muscle was the primary correlate of relative sprint swimming speed (Table 2). When length was forced to stay in the model, both the activity of CCO in white muscle and PDH in rostral red muscle were correlated with sprint performance (Table 2). Overall, the activity of CCO in rostral white muscle was the strongest correlate of sprint swimming performance of cod.

U_{crit} *swimming and its physiological correlates*

At the end of the U_{crit} test, lactate concentration in white muscle was 13-fold higher in fed than starved cod (Fig. 4). Starvation markedly decreased U_{crit} in absolute or relative speeds (P<0.0001 and P=0.003, respectively). The U_{crit} expressed in cm s^{-1} decreased by 38% whereas the relative speed decreased by 28%. Cod changed their swimming pattern towards the end of the tests, going from steady swimming to burst–coast movements. Although some cod used occasional burst–coast movements earlier, it was only near the end of the test that repeated burst–coasting occurred. Accordingly, the speed at which repeated burst–coast movements appeared was similar to the U_{crit}, for absolute as well as for relative speeds (Fig. 4). The total number of burst–coast movements made during a test was decreased by starvation (Wilcoxon test, P=0.02) but was quite variable, with burst–coasts ranging from 1 to 83 in the starved group and from 1 to 187 in the fed group. Lactate generation during burst–coasts was considerably lower in starved than fed cod (0.62±0.2 *vs* 2.9±1.2 μmol lactate g^{-1} burst–$coast^{-1}$; Wilcoxon test, P=0.001).

Of all the anatomic, biochemical and behavioural parameters we measured, the same four variables were consistently retained as correlates of U_{crit} performance and explained as much as 80% of variance (P<0.0001; Table 3). The strongest correlate was always the speed at which regular burst–coast movements started (P<0.0001); the other three variables were CK activity in rostral white muscle, NDPK activity in the white muscle sampled in the middle of the body and hematocrit.

To identify the biochemical or anatomic correlates of U_{crit} swimming, we omitted the speed at which regular burst–coasting started, given its proximity to the U_{crit}. For U_{crit} expressed as absolute speed, this model explained 56% of the variability (P<0.0001; Table 4). PDH activity in central red muscle and water content in central white muscle were the strongest correlates (P=0.0004), followed by CS activity in caudal red muscle (P=0.01) and CS activity in central white muscle (P=0.025). When performance was expressed as relative speeds, these variables explained 41% of the variability.

Given that speed at which regular burst–coasting begins is an externally visible estimator of U_{crit}, we also sought its biochemical and anatomic correlates. When this speed was expressed in absolute speeds, PDH level in red muscle and total protein concentration in white muscle sampled at the caudal peduncle were significant correlates and explained 52% of the variability (Table 5). When this speed was expressed as bl s^{-1}, these variables explained 35% of the variability.

Good athlete/bad athlete vs *specialised swimming styles*

To evaluate which of these patterns better describes the swimming capacities of the cod we used, we examined the correlations between individual sprint and U_{crit} performance, using both relative and absolute speeds. Weak positive correlations were found between the sprint and U_{crit} speeds expressed in cm s^{-1} (R^2_{adj}=0.11; P=0.005) as well as with the speed at which repeated burst–coast movements started in cm s^{-1} (R^2_{adj}=0.14; P=0.002). Analysis of the correlations of residuals from the length–speed relationships did not reveal stronger relationships.

Discussion

As prolonged periods of food limitation have a stronger impact upon white than red muscle in many fish species, swimming behaviours that rely only upon glycolytic fibres should be more affected by starvation than those that rely both upon aerobic and glycolytic fibres. We tested this prediction

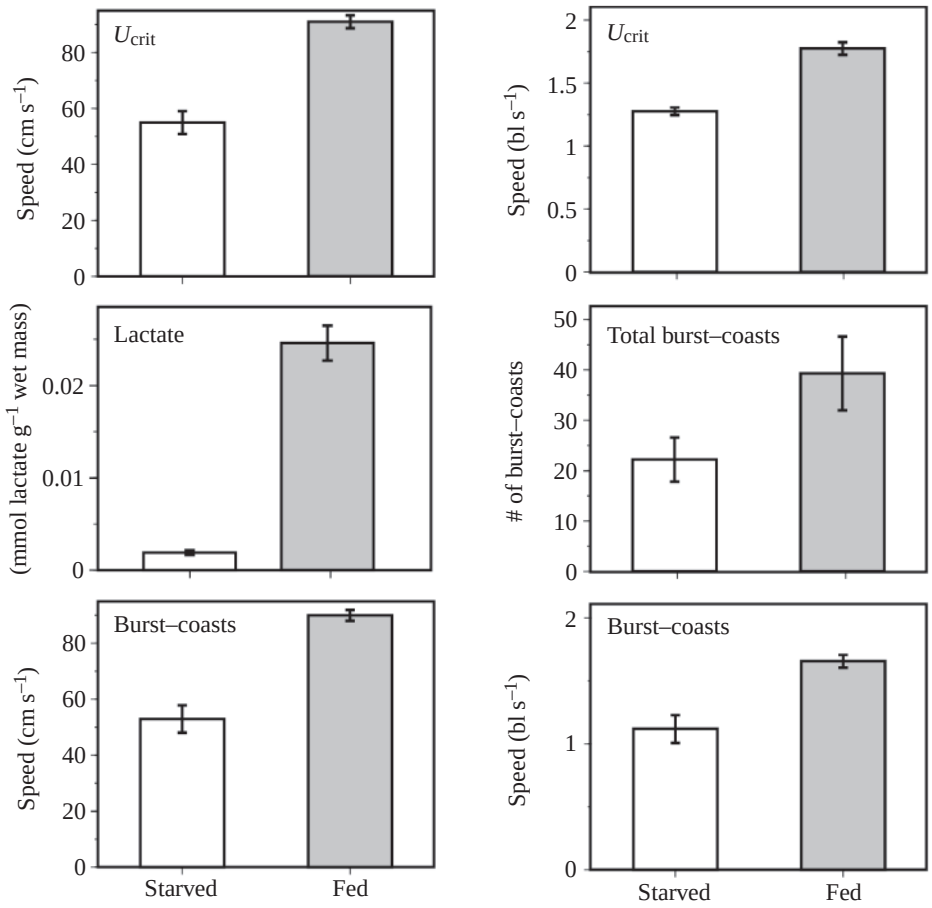

Fig. 4. U_{crit} swimming performance (in absolute cm s^{-1} and relative bl s^{-1} speeds) of cod after 12 weeks of feeding or starvation, white muscle lactate accumulation, the number of burst–coast movements during the U_{crit} tests and the speed at which regular burst–coast movements started (in cm s^{-1} and bl s^{-1}). Data are shown as means ± S.E.M.

by examining the U_{crit} and sprint swimming capacities of cod that had either been fed or starved for 12 weeks. Our biochemical and anatomic results confirm the greater breakdown of white compared with red muscle as well as the relative conservation of mitochondrial enzyme activities in both red and white muscle during starvation. Nonetheless, the decline in performance with starvation was slightly greater for U_{crit} than for sprint swimming. When expressed as absolute speeds, both swimming speeds were decreased 30–38% by starvation, and when expressed as relative speeds, the decrease was 21–28%. We are confident that the similar impact of starvation on sprint and critical swimming speed is not due to inaccuracy in these performance estimates. Given our need to characterise 59 cod in a fairly short period, we reduced the acclimation period in the tunnel prior to the U_{crit} tests. Longer acclimation periods are essential when U_{crit} tests are used to establish basal metabolic rates but seem less critical in establishing critical swimming speeds. Effectively, salmonids are able to repeat U_{crit} performance with as little as 40–78 min of recuperation between tests (Lee et al., 2003; Jain and Farrell, 2003). Furthermore, the U_{crit} results we obtained are similar to those obtained for cod with similar condition factors after a 24 h acclimation to the swim tunnel (D. Lapointe, H. Guderley and J.-D. Dutil, personal communication).

The similar impact of starvation on sprint and critical swimming speeds suggests that, particularly in cod, with its paucity of oxidative muscle, glycolytic muscle makes an important contribution to U_{crit} swimming. Starvation decreases the distance and time swum by Atlantic cod during prolonged swimming above U_{crit} by 70% (Martínez et al., 2003). This greater impact of starvation on endurance above U_{crit} was not due to a greater difference in condition factors. The starved fish had a mean condition of 0.5 in both studies, and the fed fish had a condition of 0.8 in Martínez et al. (2003) compared with 1.0 in the present study. Rather, we suggest that prolonged swimming at speeds above U_{crit} makes greater demands on white muscle than sprint or U_{crit} swimming. The very brief nature of sprint swimming does not exhaust white muscle. Repeated burst–coast movements provide a clear signal that endurance limits are being reached (Martínez et al., 2003; Peake and Farrell, 2004). The similarity between the U_{crit} and the speed at which cod began repeated burst–coasting suggests that the contractile contribution of white muscle to U_{crit} swimming is limited. While the metabolic activation of glycolytic fibres at speeds >70% U_{crit} in rainbow trout (Burgetz et al., 1998; Richards et al., 2002), and in smallmouth bass during their transition between steady and unsteady swimming (Peake and Farrell, 2004), suggests a contribution of glycolytic fibres at speeds before repeated burst–coasting, clearly this contribution will be greater at higher speeds. Thus, when starvation of Atlantic cod leads to marked loss of white muscle, the sprint and U_{crit} swimming capacities are reduced, but

Table 3. *Multiple regression models using behavioural, anatomic and biochemical variables that best explain the variability of swimming performance at* U_{crit} *for Atlantic cod, and models in which fork length or mass were forced to stay in the model*

			With fork length	With mass
U_{crit} (cm s^{-1})				
Variables in models		*P*-value	*P*-value	*P*-value
Speed at repeated burst–coasts (cm s^{-1})		<0.0001	<0.0001	<0.0001
CK activity in rostral white muscle		0.0004	0.002	0.005
NDPK activity in central white muscle		0.02	0.02	0.03
Hematocrit		0.04	0.04	0.04
Fork length			0.96	
Somatic mass				0.89
Model	R^2	d.f.	*F*-ratio	*P*-value
	0.82	4	67.97	<0.0001
With fork length	0.82	5	53.35	<0.0001
With mass	0.82	5	53.38	<0.0001
U_{crit} (bl s^{-1})				
Variables in models				
Speed at repeated burst–coasts (bl s^{-1})		<0.001	<0.001	<0.001
CK activity in rostral white muscle		0.003	0.003	0.007
NDPK in central white muscle		0.02	0.02	0.04
Hematocrit		0.04	0.03	0.04
Fork length			0.40	
Mass				0.54
Model	R^2	d.f.	*F*-ratio	*P*-value
	0.79	4	53.69	<0.0001
With fork length	0.79	5	42.86	<0.0001
With mass	0.78	5	42.54	<0.0001

endurance at speeds above U_{crit} falls to a much greater extent. Given the exclusive contribution of aerobic metabolism to sustained swimming, comparison of sprint and sustained swimming in starved and fed cod would be useful.

In contrast to our demonstration of an effect of condition on sprint performance of cod, previous studies have not always found an effect of condition (Reidy et al., 2000; Martínez et al., 2002). This contradiction can be explained by the differentiation of the condition of the experimental groups and by non-linear effects of condition. In the current study, the condition factor of fed cod was twice as high as that of starved cod (1.0 *versus* 0.5). A smaller range in condition factors was used in previous studies (0.7–1.1, Reidy et al., 2000; 0.6–0.8, Martínez et al., 2002). Condition factors between 0.6 and 0.9 are not unusual for cod from northern Norway and from the northern Gulf of St Lawrence (Eliassen and Vahl, 1982; Lambert and Dutil, 1997a). A mean condition near 0.7 is normal for cod from the Gulf of St Lawrence during the spring, just after spawning (Lambert and Dutil, 1997a). However, cod

Table 4. *Multiple regression models obtained using only anatomic and biochemical variables to explain the variability of swimming performance at* U_{crit} *of Atlantic cod*

			cm s^{-1}	bl s^{-1}
Variables in the model			*P*-value	*P*-value
PDH activity in central red muscle			0.0004	0.0007
Water content in central white muscle			0.0004	0.005
CS activity in central white muscle			0.025	0.03
CS activity in caudal red muscle			0.011	0.10
Model	R^2	d.f.	*F*-ratio	*P*-value
cm s^{-1}	0.56	4	19.81	<0.0001
bl s^{-1}	0.41	4	11.05	<0.0001

Table 5. *Multiple regression models that best explain the variability of the speed at the start of repeated burst–coast movements of Atlantic cod in absolute (cm* s^{-1}*) and relative (bl* s^{-1}*) speeds*

			cm s^{-1}	bl s^{-1}
Variables in the model			*P*-value	*P*-value
PDH activity in caudal red muscle			0.0015	0.006
Total protein in caudal white muscle			0.0004	0.03
Model	R^2	d.f.	*F*-ratio	*P*-value
cm s^{-1}	0.52	2	32.66	<0.0001
bl s^{-1}	0.35	1	16.68	<0.0001

with condition factors between 0.5 and 0.6 have a great risk of mortality (Dutil and Lambert, 2000). Nonetheless, even short-term (5 day) starvation of trout depletes muscle glycogen, thereby reducing glycogen mobilisation and lactate accumulation during exhaustive exercise, as well as accelerating the post-exercise recuperation of phosphocreatine levels (Scarabello et al., 1991). Clearly, many aspects of the physiology and motivation of fish are affected by starvation, and the effects of condition are unlikely to be linear.

During extensive starvation, the ultrastructure of white muscle is more affected than that of red muscle (Johnston and Goldspink, 1973; Patterson et al., 1974; Beardall and Johnston, 1983, 1985; Black and Love, 1986). At the ultrastructural level, volume fractions of mitochondria and myofibrils, fibre size and capillary supply are reduced. The increased water content in white muscle reduces the total volume of muscle fibres occupied by myofibrils and mitochondria and dilutes cytosolic enzymes (Beardall and Johnston, 1985). We found that starvation led to similar increases (5–6%) in the water content of red and white muscle but reduced the total volume of white muscle much more (by 81%) than that of red muscle (up to 56%). Nonetheless, white muscle mass was not a correlate of sprint or U_{crit} swimming in our multiple regression models, even though a significant partial correlation exists between white muscle mass and sprint swimming performance.

In white muscle, contractile activity during burst swimming primarily depends on ATP generation from phosphocreatine and from anaerobic glycolysis (Bone, 1966; Bone et al., 1978; Dobson et al., 1987; Altringham and Ellerby, 1999). During the first few seconds of intense muscular activity, such as sprint swimming, ATP is maintained at a relatively constant level, but the PCr level declines steadily as the CK reaction replenishes the depleted ATP. Sprint swimming can be maintained only for a short duration, just enough for the activation of anaerobic glycolysis in muscle fibres.

How then could the mitochondrial capacity of white muscle play a role in setting sprint swimming performance, as the strong correlation between CCO activity in white muscle and sprint swimming might suggest? This strong relationship may reflect a role of oxidative phosphorylation in minimising intracellular gradients in ATP and PCr before exercise. This suggestion is supported by the study of Hubley et al. (1997), who examined the influence of mitochondrial volume density upon spatial gradients in PCr and ATP in muscle fibres. An increase in the mitochondrial volume density in the range found in white fibres reduces the severity of temporal-spatial gradients in PCr and ATP during burst activity. In this fashion, the higher oxidative capacity in white muscle of fed cod could increase their sprint swimming performance by making ATP available to a greater proportion of the myofibrils than in starved cod. The deterioration of white fibres in starved cod would slow oxidative ATP production, lead to greater intracellular gradients in ATP and PCr, thus compromising the already diminished contractile function. Furthermore, higher oxidative capacities would facilitate return to resting PCr and ATP levels between sprints.

The longitudinal variation in catabolic capacities of white muscle may reflect the longitudinal variation of muscle work during swimming. Rostrally, muscle fibres generate power while shortening and then transfer this power towards the caudal peduncle, while caudally active fibres resist stretching (negative work) (van Leeuwen et al., 1990; Rome, 1992; Wardle and Videler, 1993; Jayne and Lauder, 1995). Davies et al. (1995) found that rostral white fibres of cod have faster relaxation times than caudal fibres. Furthermore, these rostral fibres show a faster activation phase than the caudal fibres. Therefore, maximum power will be produced by the rostral white fibres, given their relatively short contraction time (Thys et al., 2001). Our results suggest that the positive work by rostral white muscle may be limited by its oxidative capacities. This may explain, in part, why sprint swimming performance was positively related with the levels of CCO in the rostral muscle.

Fed and starved cod did not differ in the speed increment that was supported by burst–coasting (i.e. the maximal speed attained minus the speed at which repeated burst–coasting began). This was surprising since fed cod with higher anaerobic capacity and functionally intact white muscle were expected to maintain this anaerobically fuelled swimming for longer, thus leading to a greater separation between U_{crit} and the speed at which repeated burst–coasting began. Examination of the videotapes of the U_{crit} tests showed that even though both fed and starved cod made extensive use of burst–coast movements (Fig. 4), starved cod made weaker burst–coast movements than fed cod. In agreement with this observation, white muscle lactate levels at U_{crit} were about 13-fold higher in fed than in starved cod (Fig. 4), and lactate generation per burst–coast was 5-fold higher in fed cod. Glycogen depletion and weak burst–coast movements are typical of starved fish (Black and Love, 1986; Scarabello et al., 1991; Martínez et al., 2003) and agree with the low lactate levels in starved cod. When swimming could no longer be maintained aerobically in the U_{crit} test, starved and fed cod responded differently, but the end result was the same, virtual exhaustion. In starved cod, the highly degraded white muscle only had a weak glycolytic swimming capacity (inability to make strong burst–coast movements), bringing rapid exhaustion, while in fed cod, the intensity and high energetic cost of burst–coasting with its concomitant accumulation of lactate also led to rapid exhaustion. So, no matter what the nutritional status was, the speed at which cod started burst–coasting repeatedly was similar to their critical swimming speed. The variability in the number of burst–coast movements used by the cod may reflect variability in swimming strategies. Some cod may start sporadic use of burst–coast movements at slower speeds as a way to save energy. Biomechanical models show that burst–coasting may be 4–6 times more efficient than steady swimming (Videler and Weihs, 1982; Winger et al., 2000). We examined whether the number of burst–coast movements could explain variability in U_{crit}, as found by Winger et al. (1999). Such a relationship was only apparent for the starved cod taken alone.

PDH activity in red muscle at the caudal peduncle was the strongest correlate of the speed at which repeated burst–coasting started. Glycogen is utilised at virtually all sustained swimming speeds (Richards et al., 2002). By its transformation of pyruvate to acetyl-CoA, PDH transfers substrates from glycolysis to the Krebs cycle. In the initial phases of prolonged swimming tests such as the U_{crit}, glycogen is slowly depleted during its oxidation to CO_2 and H_2O. Our data indicate that cod with higher PDH activity in caudal red muscle can swim faster aerobically, delaying use of burst–coast movements and attaining a better performance in the U_{crit} tests. Thus, PDH activity in red muscle may set the rate at which oxidative muscle can break down pyruvate. The caudal musculature, both red and white, consistently showed the highest levels of mitochondrial enzymes (present study; Martínez et al., 2003), suggesting its force transmission is critical during aerobic activity. PDH activity was not only a good indicator of the speed at which repeated burst–coasting started, but, given the limited extension provided by burst–coasting, it also influenced the U_{crit} of cod. In fact, when the speed at first burst–coast was omitted in our stepwise analysis, PDH activity in red muscle (this time in the middle of the body) was again the variable that best explained variability in U_{crit} (Table 4).

Whereas U_{crit} and sprint swimming were affected to a similar extent by starvation, cod with high U_{crit} values were not consistently strong sprinters. At first glance, the use of white muscle during the U_{crit} protocol would suggest that such fish have a greater glycolytic capacity and should be stronger sprinters. While this tendency is apparent in comparison of the mean performances of the fed and starved cod, analysis of the inter-individual variability in these swimming behaviours does not reveal similar hierarchies of U_{crit} and sprint performance. We have previously reported that inter-individual differences in sprint performance were maintained despite considerable changes in condition index (Martínez et al., 2002). Given the short duration of sprints, inter-individual differences in the sensitivity to the stimulus may condition sprint swimming as much as muscle metabolic status, explaining the weak correlation between sprint and U_{crit} swimming performance.

The energetic condition of wild cod in some populations is rather poor in the spring after prolonged starvation during winter. The present study and Martínez et al. (2003) indicate that winter starvation will decrease sprint, U_{crit} and endurance during swimming at speeds above U_{crit} through its effects on muscle mass and metabolic capacities. Sprint and U_{crit} swimming speeds were reduced by 21–38% by starvation but, to exceed their U_{crit}, food-deprived cod were limited to burst–coast movements that seemed less powerful than those of well-fed cod, leading to a greater reduction in performance. In the wild, seasonal and inter-individual variation in swimming capacity are likely to occur as cod vary in condition. Cod weakened by a winter fast may be at risk in terms of natural selection just when they must carry out long migrations to replenish their reserves during the short summer period.

In summary, we used cod in a range of conditions and examined their swimming performance and its potential metabolic determinants. Multiple regression analysis allowed us to look at the inter-individual variation in U_{crit}, the speed at which repeated burst–coast movements began and sprint swimming and to pinpoint the physiological and biochemical parameters most closely linked to this inter-individual variation. The speed at which regular burst–coasting began was closely related to U_{crit}. The strong link between PDH activity in red muscle (both caudal and central positions) and the speed at the start of repeated burst–coasting shows that muscle mitochondrial capacity is an interesting correlate of whole animal performance. Of the various parameters we measured, both anatomic and biochemical, this best predicted U_{crit} performance. Sprint swimming was also best predicted by white muscle activity of a mitochondrial enzyme, CCO, suggesting that aerobic preparation/recuperation of muscle for rapid contraction is central for this brief activity. Finally, despite the considerably greater loss of white than red muscle during starvation, sprint and U_{crit} swimming differed by virtually the same extent between starved and fed cod.

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