Burst and coast use, swimming performance and metabolism of Atlantic cod *Gadus morhua* in sub-lethal hypoxic conditions

J.-D. Dutil*†, E.-L. Sylvestre*, L. Gamache*, R. Larocque*
and H. Guderley‡

**Ministère des Pêches et des Océans, Institut Maurice-Lamontagne, C. P. 1000,
Mont-Joli, Québec, G5H 3Z4 Canada and ‡Université Laval, Département de biologie,
Québec, Québec, G1K 7P4 Canada*

Prolonged swimming capacity (critical swimming speed, U_{crit}, protocol) and metabolism were measured for 14 Atlantic cod *Gadus morhua* exposed to seven oxygen levels within the non-lethal range normally encountered in the Gulf of St Lawrence (35 to 100% saturation). Burst-and-coast swimming was triggered earlier (at lower speeds) in hypoxia, and burst-and-coast movements were more frequent in hypoxia than in normoxia at low speeds. Furthermore, the metabolic scope beyond the metabolic rate at which Atlantic cod resorted to burst-and-coast movements decreased gradually as ambient oxygen concentration dropped. Overall, fewer burst-and-coast movements were observed in hypoxia while the distance swum in burst-and-coast mode remained *c.* 1% of the total distance swam in all tests. Oxygen availability had no effect on the rate of increase in metabolic rate with increasing velocity <50 cm s^{-1}, but limited swimming performances and metabolic rate at higher speeds. The prevailing low oxygen tensions on the bottom in the deep channels may impair the swimming capacity of Atlantic cod in the estuary and northern Gulf of St Lawrence. © 2007 Crown in Right of Canada

Key words: Atlantic cod; burst and coast; hypoxia; oxygen consumption; swimming.

INTRODUCTION

Due to rapid human population growth and climate-related factors, hypoxia is an expanding threat for marine animals (Wu, 2002). The St Lawrence River and Gulf are no exception. The slow landward advection velocity of the bottom waters in the Laurentian Channel (Gilbert *et al.*, 2005) combined with sedimentation of organic matter from the surface layer explain the low oxygen levels of the bottom layer and the decreasing oxygen availability towards the head of Laurentian Channel into the estuary (Sameoto & Herman, 1990; D'Amours, 1993). Dissolved oxygen levels in the deeper areas are now at an historical low (Gilbert *et al.*, 2005), exposing the demersal fauna to unfavourable oxygen conditions. In order to survive potentially limiting dissolved

†Author to whom correspondence should be addressed. Tel.: +1 418 775 0582; fax: +1 418 775 0740; email: dutiljd@dfo-mpo.gc.ca

oxygen tensions over most of their range in the deep waters of the St Lawrence, Atlantic cod *Gadus morhua* L. rely upon a variety of physiological and behavioural strategies. Many researchers are interested in these strategies (Kramer, 1987; Fritsche & Nilsson, 1993; Wu, 2002) and in understanding how they apply to fish populations facing hypoxia in their natural environment.

The ecological success of fishes under adverse dissolved oxygen conditions depends on their ability to sense variations in oxygen tension and to make quick adjustments in cardiovascular and respiratory activity (Fritsche & Nilsson, 1993). When exposed to hypoxia, fishes maintain oxygen delivery to the tissues while conserving energy through decreased activity (Wu, 2002). Most teleosts maintain oxygen delivery in hypoxia by increasing ventilation, thus maintaining a strong gradient in oxygen tension between water and blood (Holeton & Randall, 1967; Saunders & Sutterlin, 1971; Fritsche & Nilsson, 1989, 1990; Claireaux & Dutil, 1992; Wu, 2002; Lefrançois & Claireaux, 2003). Bradycardia generally follows the hyperventilatory response. In Atlantic cod, peripheral chemoreceptors located in the gill region monitor changes in both the internal and external environments (Burleson & Smatresk, 1990*a*,*b*; Fritsche & Nilsson, 1990) and physiological responses to changing oxygen tension are initiated within 2–8 min (Fritsche & Nilsson, 1989, 1990). Whether a fish remains motionless to minimize its energy expenditures or escapes to a different environment will depend on the species' relative tolerance to hypoxia. The most tolerant species appear to be the last to move out of the hypoxic area (Pihl *et al.*, 1991) and the most capable of limiting energy expenditures. Atlantic cod exposed to hypoxia decrease voluntary swimming activity and spontaneous active metabolic rate (Schurmann & Steffensen, 1994; Claireaux *et al.*, 1995, 2000; Schurmann & Steffensen, 1997) as well as feeding activity in hypoxia (Chabot & Dutil, 1999), possibly as an energy sparing and survival mechanism. Atlantic cod in the northern Gulf of St Lawrence avoid areas where oxygen saturation levels are low (D'Amours, 1993). Mortalities are observed at <28% saturation (LC_{05}) in the laboratory (Plante *et al.*, 1998). At this level, aerobic scope is reduced (Claireaux *et al.*, 2000) and further decreases with declining oxygen saturation. When saturation reaches 20%, which is close to LC_{50} values for the species at both 2 and 6° C (Plante *et al.*, 1998), scope for activity at any temperature (range 2–10° C) becomes zero (Claireaux *et al.*, 2000).

Two Atlantic cod stocks live in the Gulf of St Lawrence where they may encounter hypoxic waters during their seasonal migrations or during the growth season. The swimming capacity of Atlantic cod may be impaired in the moderately non-lethal hypoxic conditions in which they live. In challenging situations such as prolonged food deprivation (Martínez *et al.*, 2004) or exposure to colder temperatures (Sylvestre *et al.*, 2007), critical swimming speed (U_{crit}) performance is reduced and Atlantic cod resort to burst and coasts earlier to support aerobic swimming. In burst-and-coast swimming, fishes alternate active burst and passive coast movements (Blake, 1983). Based on calculations of a ratio of energy costs per unit distance travelled, biomechanical models suggest that an intermittent use of burst-and-coast swimming may confer energetic advantages over continuous swimming at cruising speeds and possibly at higher speeds (Weihs, 1974; Videler & Weihs, 1982). Whether the relative contribution of burst-and-coast swimming to U_{crit} increases with decreasing oxygen tension, given its

metabolic advantage, is unknown. This study examined swimming speed, oxygen consumption and use of burst and coast in Atlantic cod for a range of non-lethal oxygen tensions typically found in the deep channels of the St Lawrence.

MATERIAL AND METHODS

EXPERIMENTAL ANIMALS

Atlantic cod were captured by bottom trawl in NAFO area 4T in June 2002 and 2003. The animals were held in 7·5 m^3 tanks at the Maurice-Lamontagne Institute in Mont-Joli, Québec, Canada. They were fed a maintenance ration and maintained under natural photoperiod (48°38′ N) and at ambient salinity and temperature, except in the winter when seawater temperature was kept at >2° C. Prior to the experiment, 14 Atlantic cod were selected and transferred to smaller tanks (1·5 m^3). Fork length (L_F) and mass were mean ± s.d. 46·5 ± 3·2 cm (range: 39·5–51·0 cm) and 809·1 ± 212·5 g (range: 460–1315 g) respectively. The Atlantic cod were acclimated to the test temperature (7° C) for at least 6 weeks during which they were fed a ration of thawed capelin *Mallotus villosus* (Müller) amounting to 4% of the somatic mass per week (Jobling, 1988). They were food-deprived for 1 week before being tested. The 14 Atlantic cod were randomly associated to seven levels of dissolved oxygen, from normoxia to moderate hypoxia (35, 45, 55, 65, 75, 85 and 100% saturation). Two fish were tested at each level of hypoxia. The order of treatment testing was also random. Though size varied among individuals, L_F and mass were not correlated with oxygen level (linear least squares regression, $n = 14$ and $P > 0.05$); <1% of the variability in L_F and mass was explained by variations across oxygen levels.

EXPERIMENTAL SETUP

Metabolic rate and swimming performance at different oxygen levels were measured in a Blazka-type respirometer and swim tunnel (Martínez *et al.*, 2004). The swim tunnel was covered with black neoprene to isolate the fish from the surroundings and to maintain a constant temperature. The tunnel was lit with red LED lights and a video camera was used to record Atlantic cod behaviour during the swimming trial. A DC motor with a micrometric speed adjustment controlled current velocity in the respirometer. The correspondence between motor speed and current velocity was established for a range of velocities as follows: cross-sectional current velocities were measured at three longitudinal positions inside the swim tunnel, using a U-tube manometer and pitot tubes with one pitot facing the current while the other faced the opposite direction. Height differences were converted to velocity using Bernoulli's equation for ideal fluid flow. A grid was placed at the back of the tunnel to protect the fish from the propeller. The water flow to the respirometer passed through a column filled with Tri-Packs (FABCO Plastiques Inc., Montreal, Quebec, Canada). Dissolved oxygen content was adjusted by bubbling nitrogen into the column and monitoring oxygen tension at the outflow using a polarographic probe and a YSI-5000 oxymeter.

EXPERIMENTAL AND ANALYTICAL PROCEDURE

The U_{crit} test was based on Brett (1964) and many other subsequent studies (Beamish, 1978; Nelson *et al.*, 1994; Tang *et al.*, 1994; Schurmann & Steffensen, 1997; O'Steen & Bennett, 2003; Martínez *et al.*, 2004; Lapointe *et al.*, 2006). Prior to the test, the fish was caught, placed in a flexible stretcher, rapidly transferred to the swim tunnel and allowed to acclimate at 20 cm s^{-1} for 24 h in a normoxic and flow-through configuration. Then the oxygen tension was decreased within an hour to the target level, before the test was initiated. Each velocity stage lasted 32 min. Velocity was increased gradually over the

first 2 min of a stage; the current velocity remained constant for the next 30 min. Speed was changed in 10 cm s^{-1} increments (30, 40, 50, 60, 70 and 80). For each velocity stage, the system was opened during the first 5 min and then shut for 20 min while oxygen consumption was recorded. Then the system was reopened for 5 min to restore the target level of oxygen. The test ended following exhaustion of the fish. Exhaustion was defined as the third swimming interruption (30 s) for which the fish needed a decrease of velocity to start swimming again. After the test, the fish was transferred to a recuperation tank. Each test was monitored with a video camera and recorded for later examination.

A second and identical YSI-5000 oxymeter was linked to a computer and used to monitor dissolved oxygen in the tunnel. Water was pumped from the outer tunnel to a low-volume probe chamber and back to the outer tunnel. The probe was calibrated every day in air-saturated water. Probe data were validated using Winkler titration (Levy *et al.*, 1977). For each velocity stage, four 125 ml water samples were taken, two samples before closing the system, and two samples after opening the system 20 min later. A linear regression of the oxygen probe reading (O_{2P}) *v.* the Winkler titration value (O_{2W}) was calculated for all samples taken during the experiment and resulted in the following relationship: $O_{2P} = 1.024\ O_{2W} - 0.278$ ($r^2 = 0.995$). Probe readings for two individuals (45 and 65%) swimming at 20 cm s^{-1} did not fit the Winkler titration as they fell outside the 95% CI. The values, however, fitted within a 90% CI, and therefore were not excluded. Dissolved oxygen varied slightly during a trial and between replicate tests, but target values were used in the statistical analyses and figures.

Oxygen consumption (M_{O_2}) was calculated from the probe data for the 20 min period when the respirometer was used as a closed system. The following equation was used: $M_{O_2} = [(S_1 - S_2)(V_{resp} - V_{fish})](T_2 - T_1)^{-1}$, where S_1 and S_2 are the dissolved oxygen values before closing and after reopening the system, respectively, V_{resp} is the respirometer volume and V_{fish} is the fish volume. The value of ($V_{resp} - V_{fish}$) was estimated by subtracting the fish mass from the volume of the respirometer. $T_2 - T_1$ is the time during which the system was shut. M_{O_2} was calculated for each velocity level. M_{O_2} at U_{crit} is the mean M_{O_2} for the level at which U_{crit} was reached and M_{O_2} at first burst and coast is the mean M_{O_2} for the level at which burst and coast use began. To correct for mass differences, metabolic rates were standardized to a mass of 1 kg using an exponent of 0.8 (Reidy *et al.*, 1995; Hunt von Herbing & White, 2002).

Swimming speeds were determined as follows. The U_{crit} was determined following Brett (1964): $U_{crit} = u_i + [(t_i\ t_{ii}^{-1})u_{ii}]$, where u_i is the last level entirely swum by the fish (cm s^{-1}), t_i is the amount of time spent at the last swimming speed (min), t_{ii} is the prescribed swimming period and u_{ii} is the velocity increment (cm s^{-1}). The following correction was applied for solid blocking (Webb, 1975): $U_{crit\ corr} = U_{crit}\ [A_b\ (A_b - A_f)^{-1}]$, where U_{crit} is in cm s^{-1}, A_b represents the cross-sectional area of the inner tunnel (24 cm) and A_f the maximum cross-section area of the fish. The speed at first burst and coast (U_{b-c}) was determined using the same equations as for U_{crit}, with t_i being the time at which burst-and-coast movements began. The cost of transport was determined as the ratio of metabolic rate to swimming speed (Lee *et al.*, 2003).

Burst-and-coast movements were counted by examining the video records of each test. One third of the video record was examined at each velocity, *i.e.* the first 30 s of each 90 s period of recording at each velocity. The time at which burst-and-coast movements began was determined by plotting the cumulative number of burst and coasts (*y*-axis) against time (*x*-axis) and extrapolating this relationship to $y = 0$. Vertical and horizontal position in the swim tunnel and rest period on the back grid of the swim tunnel were also noted. The total distance swum in burst-and-coast mode during the test was estimated as the sum of distances covered during each burst-and-coast movement [<0.25, 0.50 and >0.75% of the length of the swim tunnel × (swim tunnel length − fish length)].

STATISTICS

Oxygen consumption data were analysed as follows: time (increasing velocity) and between-subject effects (oxygen) were tested with a repeated measure multivariate

analysis (GLM procedure, SAS Institute 8.2). This analysis rejects missing data and thus was restricted to velocities successfully completed by all fish (≤ 50 cm s^{-1}). The relationship between M_{O_2} at U_{crit} or M_{O_2} at first burst and coast and oxygen was described through linear least squares regression following Packard & Boardman (1988). The effects of oxygen level and L_F were analysed with ANCOVAs (GLM procedure, SAS Institute 8.2), with L_F as a covariate. Since only two fish were used at each level of oxygen, slopes in the relationship between swimming speed and L_F were assumed to be homogeneous across oxygen levels. The relationship between U_{crit} or U_{b-c} and oxygen was described through non-linear least squares regression, using a second-order polynomial model (U_{crit}) or a single-term two-parameter exponential rise to maximum model (U_{b-c}).

Burst and coast counts were analysed as follows: the last three velocity stages that each fish had completed were examined. Time (increasing velocity) and between-subject effects (oxygen) were tested with a repeated measure multivariate analysis (GLM procedure, SAS Institute 8.2). The relationship between number of burst-and-coast movements for the three velocity stages combined and oxygen level was tested through least squares regression. The correlation between rank number of the velocity stage at which burst and coast use was maximum and rank number of oxygen level was tested using Spearman's rank correlation coefficient. Counts were square-root transformed for the parametric tests.

Statistical analyses were conducted at the 5% significance level. Normality of distribution, homogeneity of variance and, where required, convergence criteria were all considered during data analysis.

RESULTS

Metabolic rate increased with increasing velocity and the rate of increase was not affected by oxygen availability (Fig. 1). For fish swimming ≤ 50 cm s^{-1}, velocity effect was significant (multivariate repeated analysis, $n = 14$ and $P < 0.001$), and this effect did not differ between levels of oxygen (same analysis, velocity–oxygen interaction term, $P > 0.05$). M_{O_2} at U_{crit} increased with increasing oxygen availability (linear least squares regression, $n = 14$, $P < 0.001$; Fig. 2), but there was no change in M_{O_2} at first burst and coast with increasing oxygen availability (linear least squares regression, $n = 14$, $P > 0.05$; Fig. 2). M_{O_2} at U_{crit} and M_{O_2} at first burst and coast coincided at low oxygen tensions.

U_{crit} and U_{b-c} were both lower at lower oxygen tensions (Fig. 2). Fish swimming under normoxia were the last to become exhausted: U_{crit} was reached at speeds $c.$ 80 cm s^{-1} and burst-and-coast swimming started at speeds >50 cm s^{-1}. In contrast, fish swimming at the lowest oxygen tensions reached U_{crit} at speeds <60 cm s^{-1} and used burst-and-coast swimming at lower speeds. The ANCOVA model with oxygen as the independent factor and L_F as the covariate ($n = 14$) explained 84% of the variance for both U_{crit} ($P < 0.05$) and U_{b-c} ($P < 0.05$). The effect of oxygen was significant in both cases ($P < 0.05$). Swimming velocities were not affected by L_F over the range used in the present study ($P > 0.05$ for both U_{crit} and U_{b-c}). The regression between U_{crit} and dissolved oxygen was significant, suggesting that U_{crit} decreased with decreasing oxygen tension but levelled off at $<70\%$ oxygen saturation (non-linear least squares regression, $n = 14$, $P < 0.001$, $r^2 = 0.82$; Fig. 2). In contrast, U_{b-c} decreased below 70% oxygen saturation, but was largely unaffected by oxygen tension $>70\%$ oxygen saturation (non-linear least squares regression, $n = 14$,
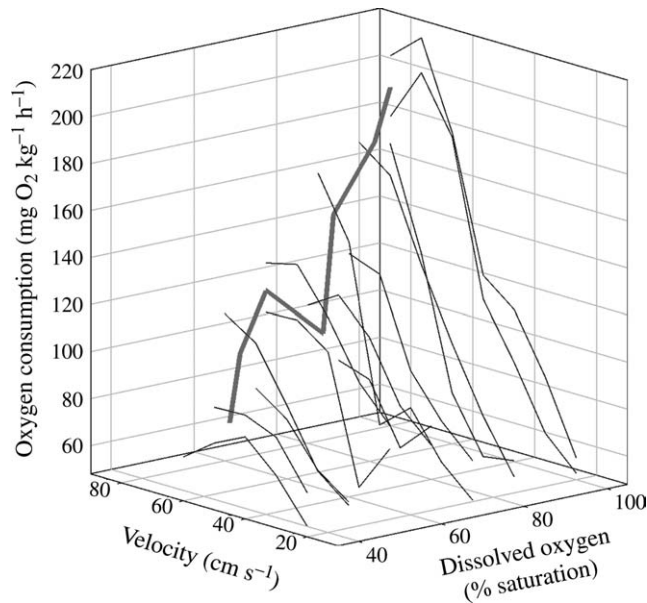
FIG. 1. Mass-specific oxygen consumption (M_{O_2}) in 14 Atlantic cod swimming at increasing velocities in normoxia and sub-lethal hypoxia. The bold line represents M_{O_2} at the critical swimming speed (U_{crit}).

$P < 0.01$, $r^2 = 0.54$; Fig. 2). For fish exposed to lower oxygen tensions, vertical and horizontal position in the tunnel were observed to be more variable, and swimming became unstable earlier in the test.

For fish swimming ≤ 50 cm s^{-1}, cost of transport decreased with increasing velocity. Velocity effect was significant (multivariate repeated analysis, $n = 14$, $P < 0.01$), but this change in the cost of transport did not differ between levels of oxygen (same analysis, velocity-oxygen interaction term, $P > 0.05$).

During the last three velocity stages swum, the number of burst-and-coast movements performed increased with increasing swimming velocity, but the onset of burst-and-coast swimming occurred at different velocities depending on oxygen tension (Fig. 3). The velocity effect was significant (multivariate repeated analysis, $n = 14$, $P < 0.001$) and this effect differed between levels of oxygen (same analysis, velocity-oxygen interaction term, $P < 0.05$), indicating that the rate of increase in the number of burst-and-coast movements with increasing velocity was affected by oxygen. The total number of burst-and-coast movements observed during the last three velocity stages swum decreased as oxygen tension decreased (linear least squares regression, $r^2 = 0.41$, $n = 14$, $P = 0.01$). The rank number of the velocity stage at which the peak number of burst-and-coast movements was observed correlated positively with oxygen tension (Spearman's rank correlation, $n = 12$, $P < 0.01$) indicating that burst-and-coast movements peaked at higher speeds in fish exposed to higher oxygen tensions. Atlantic cod exposed to higher oxygen levels swam longer distances, but overall a fish always swam c. 1% of the total distance in burst swimming mode.
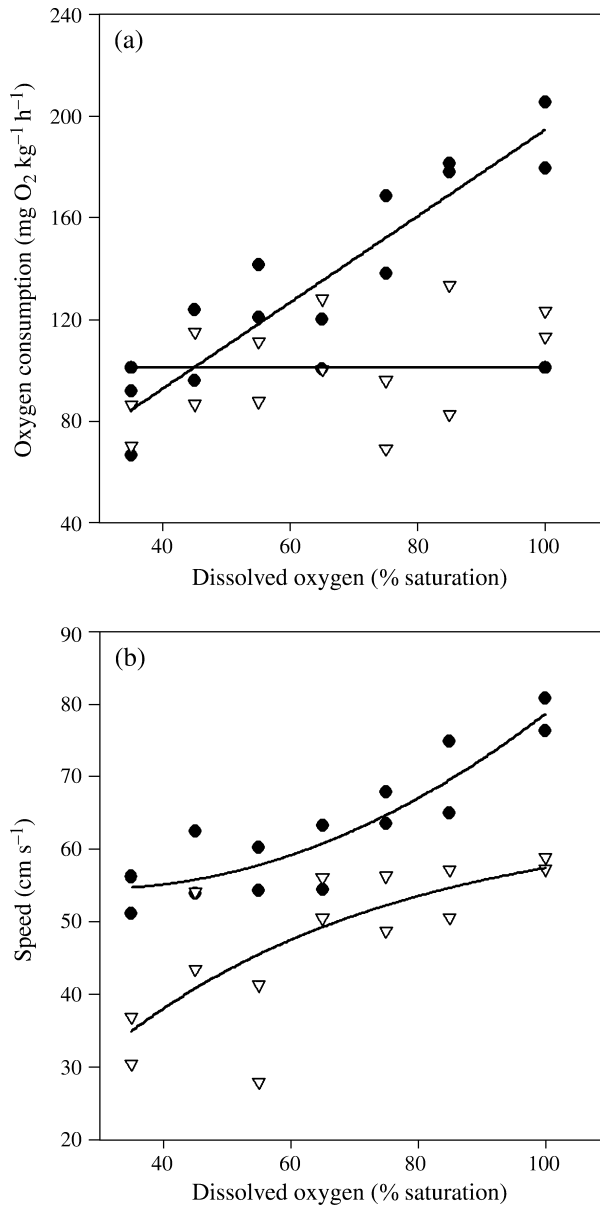
FIG. 2. A comparison of (a) oxygen consumption and (b) swimming speed at critical swimming speed (U_{crit}; ●) and speed at first burst and coast (U_{b-c}; ▽) of Atlantic cod in normoxia and sub-lethal hypoxia. Oxygen consumption data were fitted through linear least square regression: M_{O_2} at U_{crit}, $y = 25 \cdot 11 + 1 \cdot 69x$ ($r^2 = 0 \cdot 82$, $P < 0 \cdot 001$) and M_{O_2} at U_{b-c} non-significant ($r^2 = 0 \cdot 42$, $P > 0 \cdot 05$), so a straight line was drawn at mean value for M_{O_2} at U_{b-c}. Swimming speed data were fitted through non-linear least square regression: U_{crit} $y = 58 \cdot 37 - 0 \cdot 2706x + 0 \cdot 0047x^2$ ($r^2 = 0 \cdot 82$, $P < 0 \cdot 001$) and U_{b-c} $y = 64 \cdot 22 (1 - e^{-0 \cdot 022x})$ ($r^2 = 0 \cdot 54$, $P < 0 \cdot 01$).
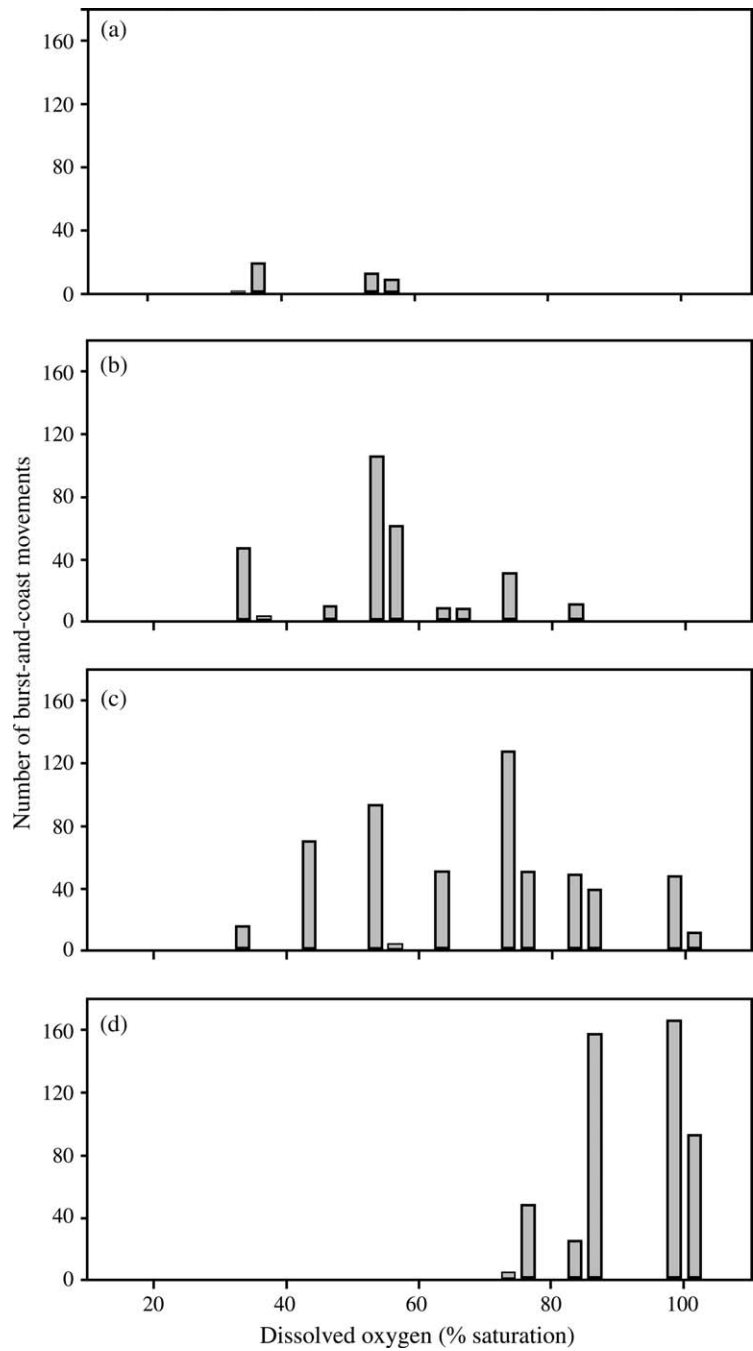
FIG. 3. Number of burst-and-coast movements performed by Atlantic cod at (a) 40, (b) 50, (c) 60 and (d) 70 cm s^{-1} during the critical swimming speed (U_{crit}) test in normoxia and sub-lethal hypoxia. The fish reached U_{crit} at velocities <70 cm s^{-1} in hypoxia (see Fig. 2).

DISCUSSION

BURST AND COAST USE IN HYPOXIA

Burst-and-coast propulsive movements are generated by axial white fibres to support and prolong swimming when current velocity gets stronger and oxygen consumption approaches its upper limit. Shifting from continuous swimming to burst-and-coast movements during a prolonged swimming activity may depend on the potential energy savings resulting from the combination of these swimming modes (Weihs, 1974; Videler & Weihs, 1982). In Atlantic cod, burst-and-coast support to swimming was observed at high cruising speeds close to U_{crit} (Videler, 1981; Martínez *et al.*, 2004; Lapointe *et al.*, 2006; Sylvestre *et al.*, 2007). Given their relationship to anaerobic processes, burst-and-coast movements were hypothesized to occur more frequently in hypoxia than in normoxia. Overall, fewer burst-and-coast movements were observed in hypoxia while the distance swum in burst-and-coast mode remained *c*. 1% of the total distance swum in all tests. This might be explained by shorter time periods to reach U_{crit} in hypoxia. Burst-and-coast swimming, however, was triggered earlier (at lower speeds) in hypoxia, and burst-and-coast movements were more frequent in hypoxia than in normoxia at low speeds. Furthermore, the metabolic scope beyond the metabolic rate at which Atlantic cod resorted to burst-and-coast movements decreased gradually as ambient oxygen concentration dropped. These observations point to a possible role of aerobic limitations as a trigger of burst and coast use. Atlantic cod may rely on burst-and-coast movements only as a last resort, *i.e.* when ambient oxygen limits aerobic swimming capacity. This may look paradoxical given that biochemical models suggest that burst-and-coast swimming is more efficient than steady swimming (Weihs, 1974; Videler & Weihs, 1982). Resorting to burst-and-coast movements, however, might add to the lactate accumulation and contribute to building an oxygen debt during exercise or have other unknown costs to the animal. Under normoxia, lactate is used by aerobic tissues as a fuel (Burgetz *et al.*, 1998; Richards *et al.*, 2002). This might not be the case in hypoxic conditions, unlike other environments where limits to aerobic swimming are imposed by other factors such as temperature and food deprivation (Martínez *et al.*, 2004; Lapointe *et al.*, 2006; Sylvestre *et al.*, 2007).

EFFECTS ON METABOLIC RATE AND SWIMMING CAPACITY: POTENTIAL IMPACTS ON WILD FISH

The present observations that maximal swimming speed and hence maximal metabolic rate decrease in fish exposed to hypoxia is consistent with other studies having used the U_{crit} protocol to assess prolonged swimming capacity (Basu, 1959; Dahlberg *et al.*, 1968). Previous studies on voluntary swimming in Atlantic cod exposed to gradual hypoxia have shown that mean swimming speed is reduced in hypoxic conditions presumably in order to limit energy expenditures and hence the demand for oxygen (Claireaux *et al.*, 1995, 2000; Herbert & Steffensen, 2005), whereas maximum voluntary swimming speed remains practically unchanged (Schurmann & Steffensen, 1994). It is unlikely, however, that Atlantic cod will

reach their U_{crit} voluntarily in an experimental context unless forced to swim against strong currents. Whilst the present results indicate that oxygen availability has no effect on the rate of increase in metabolic rate with increasing velocity <50 cm s^{-1}, suggesting that the contribution of aerobic metabolism to U_{crit} remained substantial at low oxygen tensions, forced swimming revealed the importance of oxygen availability in limiting U_{crit} performances in Atlantic cod at oxygen tensions representative of the fish habitat in the Gulf of St Lawrence.

The productivity of Atlantic cod stocks in the North Atlantic Ocean varies markedly, with the Gulf of St Lawrence stocks currently being the least productive on a *per capita* basis (Dutil & Brander, 2003). This situation has been ascribed to less productive habitats and in particular to lower temperatures in the St Lawrence (Dutil *et al.*, 2003). Bottom waters in the channels are warmer than in the overlying cold intermediate layer, with the disadvantage of a poor oxygen content (D'Amours, 1993). While Atlantic cod may avoid extreme temperature conditions (D'Amours, 1993; Castonguay *et al.*, 1999), particularly during summer, and may avoid lethal oxygen conditions (D'Amours, 1993; Plante *et al.*, 1998), low and continually decreasing oxygen levels on the bottom in the channels (Gilbert *et al.*, 2005) may limit their growth and survival capacity. A similar situation may prevail in the Baltic Sea (Matthäus & Franck, 1992; Neuenfeldt, 2002). Swimming speed studies yield information on the range of velocities fishes are able to support when carrying on their daily activities in the wild. Prolonged swimming speed, such as assessed through U_{crit} protocols, may be required for chasing schooling prey or when chased by fast predators, including mobile fishing gear such as bottom trawls (He, 1991; Reidy *et al.*, 1995). Trawls for Atlantic cod, for example, are towed at speeds ranging from 100 to 180 cm s^{-1} (2·2 to 3·9 body length s^{-1} for the present fish) (Winger *et al.*, 2000). Atlantic cod can hardly sustain such a speed for a prolonged period of time, even in normoxia. The altered swimming capacity of Atlantic cod in hypoxia further challenges its ability to obtain food and survive in its environment. Hence, reduced swimming capacities (present study) and reduced feeding and individual growth in hypoxia (Chabot & Dutil, 1999) combined with the negative impacts of cold temperature on growth and swimming performance may contribute to the lower productivity of Atlantic cod in the St Lawrence.

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