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Source: *Physiological and Biochemical Zoology: Ecological and Evolutionary Approaches*, Vol. 80, No. 2 (March/April 2007), pp. 178-185

Published by: The University of Chicago Press. Sponsored by the Division of Comparative Physiology and Biochemistry, Society for Integrative and Comparative Biology

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Effects of Ration Size and Hypoxia on Specific Dynamic Action in the Cod

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Accepted 10/13/2006; Electronically Published 1/12/2007

ABSTRACT

We present the first data on the effect of hypoxia on the specific dynamic action (SDA) in a teleost fish. Juvenile cod (*Gadus morhua*) were fed meals of 2.5% and 5% of their wet body mass (BM) in normoxia (19.8 kPa P_{O_2}) and 5% BM in hypoxia (6.3 kPa P_{O_2}). Reduced O_2 availability depressed the postprandial peaks of oxygen consumption, and to compensate for this, the total SDA duration lasted 212.0 ± 20 h in hypoxia, compared with 95.1 ± 25 h in normoxia. The percentage of energy associated with the meal digestion and assimilation (SDA coefficient) was equivalent between the different feeding rations but higher for fish exposed to hypoxia. Comparing peak oxygen consumption during the SDA course with maximum metabolic rates showed that food rations of 2.5% and 5% BM reduced the scope for activity by 40% and 55%, while ingestion of 5% BM in hypoxia occupied 69% of the aerobic scope, leaving little energy for other activities.

Introduction

Feeding is followed by a marked increase in the metabolic rate, termed the specific dynamic action (SDA). The phenomenon of SDA integrates the sum of all energetic expenditures involved in feeding, and it thus includes both a muscular mechanical component (the prey capture and subsequent gut motility) and the endogenous postabsorption of nutrients and digestion. It is, however, generally agreed that the major contributor for the incremental oxygen consumption results from the energy requirements of biochemical transformation of food and de novo protein synthesis occurring in the postabsorptive state, leading

to the deposition and turnover of tissue components (Brown and Cameron 1991a, 1991b; Bureau et al. 2002).

Measurements of oxygen consumption ($\dot{M}O_2$) as a proxy for metabolism are commonly used to quantify the energetic loss associated with SDA in aquatic breathers (Brett and Groves 1979). Past work has shown that the SDA energy demand in fish depends on meal size (Fu et al. 2005b), composition (Jobling and Davies 1980), ambient temperature (Soofiani and Hawkins 1982), body size (Hunt von Herbing and White 2002), and swimming activity (Blaikie and Kerr 1996). The energy demand associated with SDA in fish normally ranges between 5% and 20% of the ingested prey energy (Jobling 1983; Priede 1985), and SDA is therefore regarded as a significant bioenergetic "cost of growth" (Jobling 1981). However, given that SDA originates from energy requirements for food assimilation by tissue components, it cannot be regarded as a simple metabolic loss (Mallekh and Lagardere 2002).

Fluctuations in oxygen saturation occur more often in the aquatic milieu than the terrestrial, and although O_2 availability is central for fueling the anabolic and catabolic processes in the postabsorptive state in fish, the potential effects of hypoxia on SDA have received only little attention (Axelsson and Fritsche 1991; Chabot and Dutil 1999; Axelsson et al. 2002).

The Atlantic cod (*Gadus morhua* L.) is an economically important round fish that has recently begun to be cultured because of declines in wild stock caught during the past decades. Controlled-growth trials of cod kept in prolonged hypoxia have shown lower feed intake and reduced feed efficiency resulting in an overall negative effect on fish performance (Chabot and Dutil 1999). Hypoxic events commonly occur in coastal areas (Johannessen and Dahl 1996) as well as in intense aquaculture, so we examined the effect of hypoxia on SDA.

The aim of this study was to study the SDA profile in cod exposed to two levels of oxygen saturation: normoxia (>19 kPa) and moderate hypoxia (6–7 kPa). The hypoxia levels chosen for these experiments resemble levels commonly observed in the benthic habitats of the cod (Neuenfeldt 2002). It was hypothesized a priori that reduced O_2 availability would lower the amplitude of SDA but prolong its duration.

Material and Methods

Fish and Maintenance

Atlantic cod *Gadus morhua* were collected by trawling in the northern part of Øresund (east Denmark). All cod were immediately transferred to 450-L holding tanks at the Marine

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Biological Laboratory and acclimated to 10°C at a light regime of 12L : 12D. All animals were kept in accordance with the Danish institutional guidelines for animal research (permission 2004/561-894). The fish were held for at least 3 wk before experimentation and fed a restricted diet of herring fillets. The energy content of herring fillets was determined using adiabatic bomb calorimetry (Parr 1271; Parr Instrument, Moline, IL). The mean energy content of four random herring samples was 9.92 kJ g⁻¹ wet weight.

Respirometer and Measure of Metabolic Rate

Measurements of $\dot{M}O_2$ were carried out with computerized intermittent-flow respirometry, as described by Steffensen (1989). A 50-L darkened tank filled with filtered seawater was kept at a constant temperature of 10.0° ± 0.1°C using a Hetofrig cooler. The water was in constant circulation and aerated. A specially designed cylindrical Plexiglas respirometer (3.3 L) equipped with Perspex tubes facilitated feeding within the chamber (Fig. 1). The respirometer was immersed in the 50-L tank. Water was continuously circulated through a UV filter (11 W) to reduce microbial growth. Laboratory activity was limited, and the water surface was shielded with polystyrene to prevent visually disturbing the fish during inspections of the

experiment. At the end of each experiment, all equipment was carefully cleaned.

Oxygen tension was measured continuously using a WTW 340i oxymeter at frequency of 1 Hz, and the oxygen consumption of fish was determined for a 5-min interval every 10 min. The control of the opening-closing mechanism of the respirometer and calculations of $\dot{M}O_2$ were done with Loli-Resp software (LoligoSystems). The electrode was mounted directly into one end of the respirometer chamber, where a Plexiglas plate prevented contact with the fish and propagated water mixing. A ± 1-V signal from the oxymeter was transmitted through a galvanic isolation amplifier and collected directly by a PC. Ambient oxygen tension for the hypoxic trials was measured using an OxyGuard Handy electrode (Birkerød, Denmark). The electrode was connected to a relay controlling the oxygen saturation in the tank via a solenoid valve that regulated nitrogen gas delivery to the tank. Oxygen electrodes were calibrated twice a week to adjust for changes in atmospheric pressure.

Oxygen decrease was calculated as the slope (α , $\Delta O_{2, \text{sat}} \Delta t^{-1}$), and oxygen consumption ($\dot{M}O_2$, mg O₂ kg⁻¹ h⁻¹) was calculated by the standard formula $\dot{M}O_2 = \alpha V_{\text{resp}} \beta M_b^{-1}$, where V_{resp} is the volume of the respirometer minus the volume of the fish (L) and body mass is equalized body volume, β is oxygen solubility, and M_b is the body mass of the fish (kg).

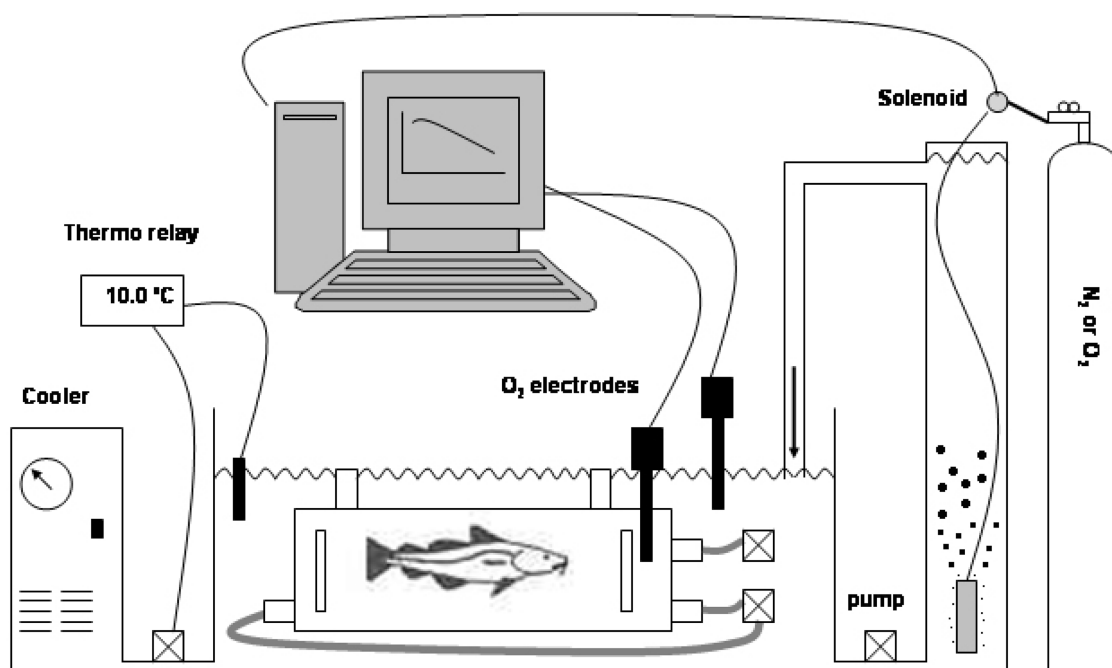


Figure 1. Illustration of the equipment used to measure the oxygen consumption of cod at 10°C. Each measuring period consisted of 240 s flushing followed by a 60-s wait period, and the decline in Po₂ was measured over the last 300 s. Feed was provided through either one of the mounted tubes. The oxygen saturation of the ambient water was measured with a second electrode and regulated via a relay box by bubbling nitrogen or air into the water. See text for further details.

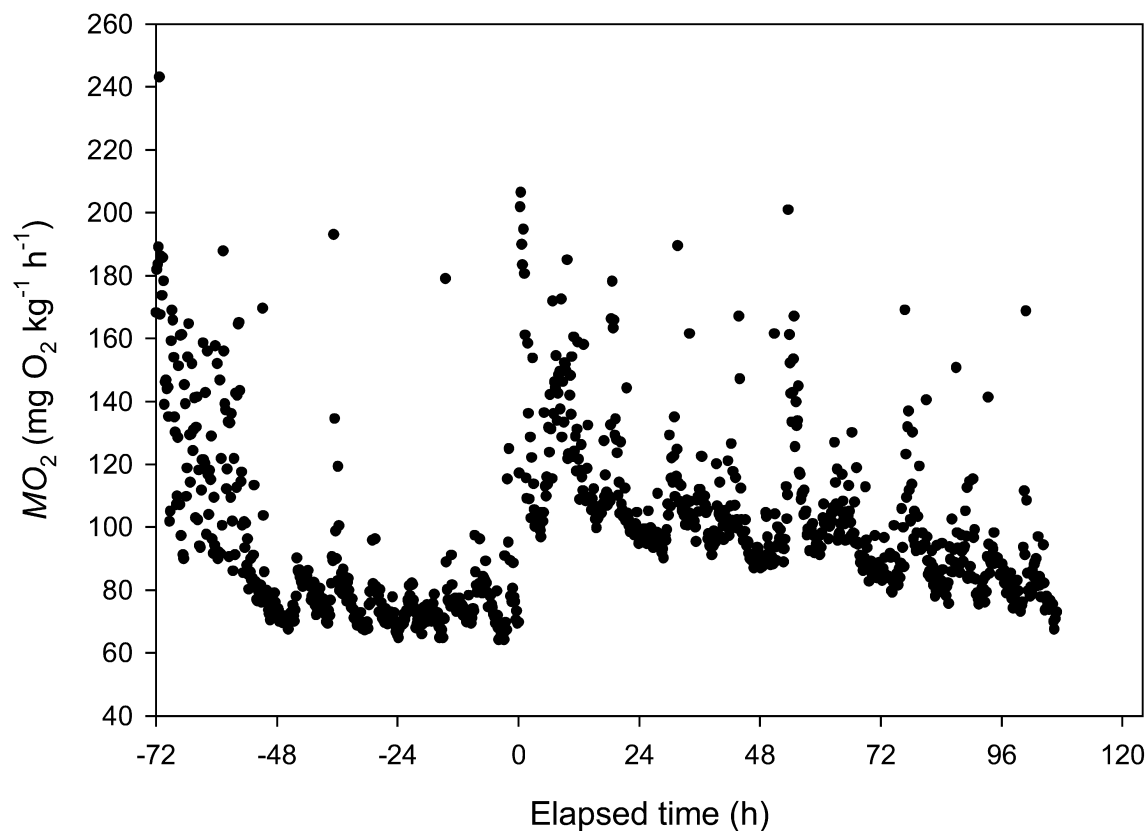


Figure 2. Example of raw data on the specific dynamic action (SDA) effect of an 8.7-g herring ration (5% body mass) fed to a 174-g cod at time 0. The total SDA duration was 107 h. The initial $\dot{M}O_2$ values are elevated due to handling, while subsequent spikes are a result of spontaneous activity.

Experimental Protocol

Experiment 1: Metabolic Scope at Decreasing Oxygen Saturations.

Atlantic cod (126.2 ± 4.5 g, mean \pm SEM, $N = 29$) were used to measure maximum metabolic rates (MMR) at various levels of oxygen saturation. Fish were exercised by continuously chasing them for 5 min in a nearby 50-L tank supplied with aerated seawater (10°C). A chasing procedure was chosen over critical swimming tests because past studies show that this species poorly maintains stationary positions in swim flumes (Hunt von Herbing and White 2002; Fu et al. 2005a). The placement of the exercise tank ensured minimum transportation time before measuring oxygen consumption rates. The experimental setup and equipment used to measure $\dot{M}O_2$ were nearly identical to those used by Schurmann and Steffensen (1997), modified with the use of Loli-Resp for $\dot{M}O_2$ calculations. The MMR was measured at five different oxygen levels, approximately 25%, 35%, 50%, 75%, and 100% saturation. Oxygen consumption data were collected over 10-min periods, and the MMR usually occurred within the

first or second period after the fish were placed in the respirometer.

Experiment 2: SDA. Cod (155.5 ± 6.7 g, $N = 18$) were starved for a minimum of 3 d before insertion in the respirometer (protocol adopted from Jobling 1994). The fish were then left for between 24 and 72 h to settle, during which $\dot{M}O_2$ measurements became stable (Fig. 2). The $\dot{M}O_2$ data 6 h before feeding were used to produce an unfed reference value. Minor elevations in $\dot{M}O_2$ occurring within this period were eliminated by fitting the data set to a bimodal normal distribution according to Steffensen (1989). Feeding was facilitated through one of two Perspex tubes mounted on the respirometer, depending on the orientation of the fish. Meals of herring fillet were prepared as a single ration, equivalent to 2.5% or 5% wet body mass (BM), measured with an accuracy of ± 0.1 g using a Sartorius BP 3100S scale. After acceptance of food, $\dot{M}O_2$ was measured until consumption rates returned to $\pm 5\%$ of the unfed reference value. A similar procedure was applied for the hypoxic trials. In brief, after settlement (24–72 h), the cod was

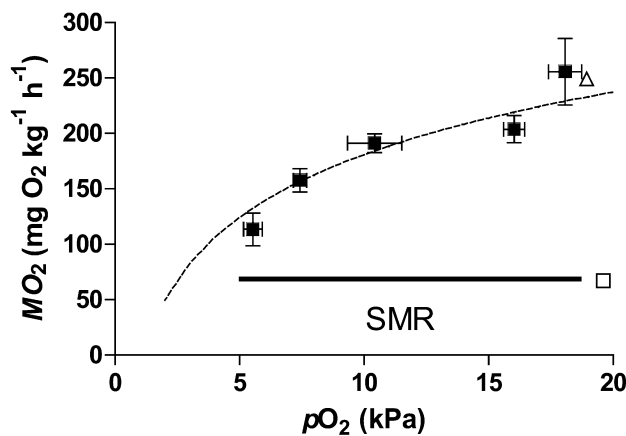


Figure 3. Maximum metabolic rates (MMR) of cod at different oxygen saturations (10°C). Solid squares indicate MMR at five different O_2 levels; the open square denotes the prefeeding metabolic rate at normoxia (SMR), and the triangle indicates the MMR of cod determined by Soofiani and Priede (1985). Error bars are SEM.

exposed stepwise to hypoxia over several hours (3–6 h) using a feedback-controlled solenoid valve to inject nitrogen. The final level of hypoxia was set at approximately 30% saturation (range 5.9–7.1 kPa). Once oxygen consumption rates became stable, food was offered. If food was not accepted immediately, the ambient Po_2 was briefly adjusted toward higher O_2 saturation and then decreased after ingestion.

Data Handling and Analysis

The SDA variables were calculated by subtracting the $\dot{M}O_2$ from an unfed reference (specific for each fish tested), obtained using all $\dot{M}O_2$ data from the 6 h before feeding. Oxygen consumption data were averaged to 1-h intervals. Five variables of the SDA response were quantified: (1) peak metabolic rate ($\dot{M}O_{2peak}$), the maximum oxygen consumption observed averaged over 1 h during the SDA course; (2) the percentage reduction of the aerobic scope for activity, calculated as $100(\dot{M}O_{2peak} - \text{prefeeding } \dot{M}O_2) / (\text{MMR} - \text{prefeeding } \dot{M}O_2)$; (3) the time required to reach $\dot{M}O_{2peak}$ (t_{peak}); (4) SDA duration, from time of feeding until the $\dot{M}O_2$ returned to $\pm 5\%$ of prefeeding status; and (5) the SDA magnitude, expressed as the total energy used as a percentage of energetic content of the meal. Oxygen consumption was converted to energy using an oxycaloric coefficient of 14.06 kJ g O_2^{-1} (Gnaiger 1983). Sigma Stat (ver. 2.03; SPSS) was used for data analysis. SDA variables were grouped according to food ration or Po_2 treatment and tested for differences with one-way ANOVA. Significance level was accepted at $P < 0.05$. All data are presented as means \pm SEM.

Results

Experiment 1 and Prefeeding Metabolic Rate

The MMR was measured in six cod (except for 49% O_2 level; $N = 5$) at each of five oxygen saturations, 27%, 37%, 49%, 77%, and 86%, and was 112.7 ± 4.7 , 159.9 ± 5.8 , 192.4 ± 5.8 , 203.3 ± 6.8 , and 247.7 ± 8.6 mg O_2 kg^{-1} h^{-1} , respectively (Fig. 3). Log transformation of oxygen saturation (kPa) resulted in a linear model of MMR at different O_2 saturations given by

$$\text{MMR (mg } O_2 \text{ kg}^{-1} \text{ h}^{-1}) = 185(\log_{10} \text{ kPa}) - 6.86, \quad (1)$$

with $r^2 = 0.97$. Body mass differed significantly between the experimental groups fed 2.5% and 5% BM ($P < 0.05$), but there were no significant differences in the prefeeding metabolic rates (Table 1).

SDA

The $\dot{M}O_2$ was collected each 10 min, but SDA variables were quantified using 1-h averages. Mean values of $\dot{M}O_2$ at 6-h intervals are shown for feeding rations of 2.5% and 5% BM at normoxia and for 5% BM at hypoxia (Fig. 4). The absolute $\dot{M}O_{2peak}$ values differed significantly between the experiments, but expressing limitation of $\dot{M}O_{2peak}$ relative to scope for activity showed no significant difference between the rations (2.5% vs. 5% BM) or between the oxygen treatments.

There was no effect of ration size in t_{peak} under normoxic conditions, but t_{peak} occurred significantly later for the 5% BM ration in hypoxia compared with normoxia (29 vs. 10 h, respectively; $P < 0.01$). The duration of the SDA response lasted significantly longer in fish fed 5% BM than in fish fed 2.5% BM, which again was significantly longer in hypoxia compared with fish fed 5% BM in normoxia.

The total energy expended on SDA (integration of postprandial $\dot{M}O_2 - \text{prefeeding } \dot{M}O_2$) was not significantly different between the ration sizes or the oxygen treatments. Expressing the energy expended relative to the energy ingested (the SDA coefficient) showed no significant difference between the feeding rations in normoxia, but the coefficient was significantly higher for fish exposed to hypoxia.

Discussion

Prefeeding Metabolic Rates

The prefeeding values were higher (Table 1) than previous values for standard metabolism of cod at 10°C (Schurmann and Steffensen 1997) but lower than those presented by Soofiani and Priede (1985). Resting metabolism, however, is affected by several factors such as prolonged fast (Fu et al. 2005a), hormonal balance (McKenzie et al. 2003), sex, and seasonality (Karamushko and Christiansen 2002), and hence the metabolic

Table 1: Specific dynamic action (SDA) of cod at 10°C during digestion of 2.5% and 5% body mass (BM) herring diet and 5% BM in hypoxia

SDA Parameters	Normoxia (19.8 kPa)		Hypoxia (6.3 kPa)	ANOVA	
	2.5% BM	5% BM	5% BM	2.5% vs. 5% BM	Normoxia vs. Hypoxia
Mass (g)	188.4 ± 12.2	147.1 ± 5.6	137.0 ± 7.7	*	NS
Prefeeding metabolic rate	59.7 ± 9.1	71.3 ± 2.0	70.2 ± 5.2	NS	NS
MMR (mg O ₂ kg ⁻¹ h ⁻¹) ^a	235.6	235.6	142.6	NS	*
$\dot{M}O_{2peak}$ (mg O ₂ kg ⁻¹ h ⁻¹) ^b	128.2 ± 13.3	160.6 ± 12.6	121.3 ± 5.3	*	*
Limitation of aerobic scope for activity (%)	39.8 ± 8.0	54.5 ± 7.0	69.4 ± 7.0	NS	NS
t_{peak} (h)	6 (–40)	10 (1–34)	29 (11–128)	NS	*
Duration (h)	48.6 ± 13.1	95.1 ± 5.6	212.2 ± 19.8	*	*
SDA energy (kJ)	3.9 ± 1.3	7.1 ± .9	11.7 ± 2.3	NS	NS
SDA coefficient (%)	8.1 ± 2.8	9.7 ± 1.3	17.9 ± 4.3	NS	*
N	5	8	5		

Note. Variables are defined in text; values are means ± SEM, except for t_{peak} , which is displayed as mean, with range in parentheses.
% BM = percentage of body mass used as ration.

^a Maximum metabolic rate (MMR) derived from 29 cod; see “Material and Methods.”

^b $\dot{M}O_{2peak}$ (averaged for 1 h) relative to prefeeding metabolic rate.

* Significant difference between treatments ($P < 0.05$).

rate may vary between studies, even if measured at similar temperatures.

Effects of Hypoxia on SDA

The major finding of this work is that hypoxia does influence the SDA response of cod. The metabolic profile of SDA in hypoxia was more depressed and prolonged (Fig. 4) compared with that observed in normoxia. Accordingly, quantification of SDA in hypoxia resulted in significantly lower $\dot{M}O_{2peak}$, delayed t_{peak} , and prolonged duration (Table 1).

To our knowledge, there is no previous study of the effect of hypoxia on SDA in fish, using $\dot{M}O_2$ as a proxy for metabolic rates. A somewhat similar SDA response, however, has been reported in shore crab *Carcinus maenas*, which exhibited a lower transient $\dot{M}O_{2peak}$ in hypoxia ($PO_2 < 4$ kPa), though the SDA duration was unchanged, compared with normoxia (Mente et al. 2003). Herein the SDA duration was more than doubled in hypoxia. In addition, the absolute increase in oxygen consumption was lower in hypoxia compared with that in normoxia. Thus, our results suggest that hypoxia limits the $\dot{M}O_{2peak}$ level of the SDA and, as a result, prolongs the postprandial condition.

Several factors besides a respiration limitation may be involved in the shift toward a lower $\dot{M}O_{2peak}$ in hypoxia. Axelsson and Fritzsche (1991) showed that hypoxia imposed an increased visceral vascular resistance in the blood flow in cod, leading to a reduction of the celiac and mesenteric artery blood supply. It may be expected that a reduction in blood flow can depress

the levels of expensive cellular processes, such as protein synthesis, due to decreased uptake of nutrients. Downregulation to complete abolition of protein synthesis has been demonstrated in crucian carp exposed to anoxia (Smith et al. 1996), supporting the concept that the SDA response is compromised below a certain lower level of O₂ saturation.

Comparison of MMR at 19.8 and 6.3 kPa with the corresponding $\dot{M}O_{2peak}$ during feeding provided an opportunity to evaluate the limitation on the scope for activity during feeding. In normoxia, the ingestion of 2.5% and 5% BM herring rations led to limitations of the scope for activity by 40% and 55%, respectively, results in line with past work on SDA in adult cod (Saunders 1963). In hypoxia, however, the postprandial $\dot{M}O_{2peak}$ occupied 69% of the aerobic scope for activity at t_{peak} , leaving little energy available for other metabolic expenditures, such as locomotory activities. This implies a trade-off for fish in hypoxic conditions—on the one hand, they are left with little excess energy for any activity after feeding, but on the other hand, they risk reduced growth as a consequence of eating less. Both scenarios are further limited by any increase in activity. Unfortunately, little is known about the energy allocation for activity in fish after feeding in the wild, but it may be expected that cod have higher costs in the wild (compared with laboratory results) in order to forage and migrate or for predator avoidance. This led Chabot and Dutil (1999, p. 488) to conclude that “any increase in activity would exacerbate further the effect of hypoxia on the amount of food that can be processed, and would result ultimately in slower growth rates.”

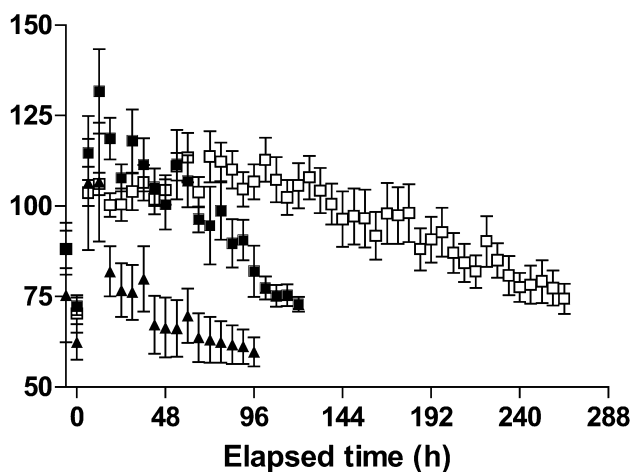


Figure 4. SDA effect on mean oxygen consumption ($\text{mg O}_2 \text{ kg}^{-1} \text{ h}^{-1}$) of cod after ingestion of 2.5% body mass (BM) ration (triangles, $N = 5$), 5% BM ration in normoxia (solid squares, $N = 8$), and 5% BM ration in hypoxia (open squares, $N = 5$). Feeding was completed at time 0 min. Error bars are SEM.

The Effect of Ration Size on SDA

For most animals, it has been shown that increasing meal size is followed by a larger SDA response, including heightened $\dot{M}\text{O}_{2\text{peak}}$ and longer SDA durations (Secor and Faulkner 2002). Correspondingly, cod fed a half-ration (2.5% BM) had a lower $\dot{M}\text{O}_{2\text{peak}}$ and a significantly shorter SDA duration than cod fed the 5% ration. An SDA duration of 48.6 h when fed 2.5% BM is in accord with the durations reported by Blaikie and Kerr (1996), who fed adult cod (1.3–1.5 kg) a similar ration of fresh capelin while keeping the fish active at low speed (~ 0.4 body lengths s^{-1}). Our results confirm a positive correlation between meal size and SDA duration as described by Jobling and Davies (1980) and Fu et al. (2005b), and the durations were in accord with results on larger conspecifics fed similar rations (Blaikie and Kerr 1996). Our results of the SDA durations are also in line with estimates of the time required for cod to evacuate herring rations at 10°C. Andersen (2001) provided a square root model to describe the gastric evacuation rates of cod, accounting for meal size, energetic density of prey, water temperature, and predator size. Accordingly, the gastric evacuation of a 5% BM ration of herring meal (mean ration 7.75 g wet weight) was calculated to last 121 h. Conversely, after 93.5 h (the mean SDA duration for 5% BM ration in normoxia), the stomach content of a fed cod (25 cm, 155 g) would correspond to 6.3% of the original ingested meal (or 0.5 g), a ration too insignificant to cause heightened protein synthesis demand to exceed the $\pm 5\%$ prefeeding $\dot{M}\text{O}_2$ criteria used in this study.

In contrast to the long SDA durations found herein, Hunt von Herbing and White (2002) demonstrated that very small cod (<8 g) have much shorter durations, less than 10 h, when

fed relatively large meals of up to 7.5% BM. These short durations were, however, explained by a higher mass-specific metabolism and the growth rate characteristics of very small fish. Furthermore, the $\dot{M}\text{O}_{2\text{peak}}$ for small cod reached more than 98% of the aerobic scope during normoxia (Hunt von Herbing and White 2002), a result similar to that of Soofiani and Hawkins (1982), who showed that young cod (30–83 g) fed 3.7% BM raised their metabolic rates to levels similar to those found by U_{crit} (maximum sustainable swimming speed) tests. The findings in the latter two studies were not identical, but both studies indicate that body size has a large influence on SDA variables and possibly that small fish (<80 g) have larger potential to direct their energy flux into growth. In contrast, our $\dot{M}\text{O}_{2\text{peak}}$ values were not close to MMR in any of the normoxic feeding trials. This suggests that the respiratory capability was not limiting in any of the normoxic trials and supports the view that a cellular metabolism involved in SDA reached a saturation point (Jobling 1981; Boyce and Clarke 1997).

SDA Coefficients

A series of studies have expressed the summed energetic cost (kJ) associated with the digestion and meal assimilation, the SDA coefficient. In our study, the total cost associated with digestion was not significantly affected by ration size in normoxia, but the SDA coefficient was significantly higher for fish exposed to hypoxia (Table 1). This result must, however, be interpreted with caution since the prolonged experimental conditions in hypoxia may lead to overestimation of the SDA magnitude due to episodic occurrences of spontaneous activity. This bias was minimized by using 1-h mean values of $\dot{M}\text{O}_2$ in all calculations for the SDA variables. Overall, an energetic loss of 8%–18% is well in accordance with a previous estimate of the SDA coefficient of cod (14%; Karamushko 1993) but also representative for a range of other teleosts (Jobling 1981).

Boyce and Clarke (1997) reported high SDA coefficients when feeding smaller rations compared to satiation levels, suggesting that the cost of digestion is fixed regardless of meal size. In our study, variable ration sizes (2.5% vs. 5% of isonitrogenous and isoenergetic content) did not influence the SDA coefficient significantly.

In conclusion, the results show that hypoxic conditions prolong the postabsorptive state of fish by limiting the peak oxygen consumption levels. In this way, less food is assimilated over time, and this may have negative impacts on the growth performance.

Acknowledgments

The authors wish to thank the crew of R/V *Ophelia* for supplying the cod. Barbara Grisdale-Helland (Institute of Aquaculture Research [AKVAFORSK], Sunndalsøra, Norway) is

greatly acknowledged for comments on the manuscript and analysis of herring energy content. We also thank Niels G. Andersen (Danish Institute for Fisheries Research, Denmark) and Denis Chabot (Maurice Lamontagne Institute, Fisheries and Ocean Canada) for comments on the manuscript. Two anonymous reviewers are also acknowledged for constructive comments on the manuscript. The Norwegian Research Council is gratefully acknowledged for funding (project 146469/140).

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