



Full length article

Effects of exhaustive swimming and subsequent recuperation on flesh quality in unstressed Atlantic cod (*Gadus morhua*)



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ABSTRACT

Wild Atlantic cod of commercial size (1.9 ± 0.5 kg) were swum to exhaustion in a large swim tunnel in an attempt to mimic the initial stage of trawling, when fish swim at the trawl mouth until they fatigue or lose interest and subsequently drop into the trawl. The objective of the study was to investigate if exhaustive swimming in unstressed cod had any negative effects on fillet quality, and if post-exercise recuperation for 2, 4, 6, or 10 h could reverse such potential effects. Exhaustive swimming increased muscle stiffness during *rigor mortis*, but did not change time to maximum stiffness or cause any marked increase in fillet redness. Plasma lactate and cortisol were elevated after exercise, but recovered steadily during the recuperation period. Swimming to exhaustion also caused a drop in muscle pH, which returned to pre-exercise level within four hours of recuperation. We conclude that exhaustive swimming in unstressed Atlantic cod only has a moderate and reversible short lasting negative effect on flesh quality, which is less severe than the poor quality frequently observed in commercially caught cod.

1. Introduction

In the Northeast Atlantic, the majority of Atlantic cod (*Gadus morhua*) is caught by demersal trawls (ICES, 2015). Fishing by trawling is highly efficient with the potential of large catches. However stress associated with large sized catches and lengthy hauls can give poor or variable quality of the fillet, including reduction in muscle pH, fillet gaping and presence of residual blood in the white muscle (Margeirsson et al., 2007; Olsen et al., 2013). A recent study suggest that the reduction in fillet quality associated with trawling can be reversed if the fish are allowed to recover in holding tanks for a minimum of six hours after they are taken on board (Olsen et al., 2013).

Fishing by trawling is an active process in which shoals of fish are herded in front of an approaching net (Winger et al., 2010). Typically, the fish swim at the trawl mouth with the same speed as the moving net until they fatigue or lose interest and subsequently drop into the trawl and get retained in the cod end (Maine and Sangster, 1981; Wardle, 1993).

Exhaustive swimming is expected to be the first of several potential

causes of poor flesh quality of trawled fish, because it depletes the energy stores of the red and white muscle of fish (Lurman et al., 2007). The energy status of the white muscle, which constitutes the majority of the muscles in fish, is of great importance to the quality, as low energy following pre-mortem activity is associated with a reduction of muscle pH, fillet gaping and a faster onset of *rigor mortis* (Borderías and Sánchez-Alonso, 2011). Other factors, such as rapid thermal changes, confinement in the codend, barotrauma (Rummer and Bennett, 2005) and burst swimming (Maine and Sangster, 1981; Wardle, 1993), also represent potential stressors during trawling operations, which could contribute to the deterioration of the fillet quality.

The physiological responses to exhaustive exercise have been well documented (reviewed by Kieffer, 2000) and several studies suggests that strenuous activity induced experimentally in tank systems (Erikson et al., 2011; Donaldson et al., 2014) or associated with commercial capture by trawl (Olsen et al., 2013; Digre et al., 2017) or gillnet (Farrell et al., 2001), may have severe negative effects on fillet (i.e. muscle) quality parameters. However, most of these studies include elements of “fright” or “stress” in addition to muscle activity, and there

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is little information about exercise-induced effects on fillet quality in unstressed fish. Here we examine if exhaustive swimming in unstressed Atlantic cod has any negative effects on fillet quality, and if post swimming recuperation can reverse such potential effects. An important part of the objective was to mimic the initial “swimming stage” during trawling operations in an attempt to compare potential negative effects of exhaustive swimming *per se* with the overall fillet quality seen after commercial capture of cod by trawl. We have addressed these questions by measurements of muscle pH, time to full muscle stiffness and fillet redness during recuperation in cod subjected to an experimental swimming regime, which ultimately led to exhaustion. In addition, blood lactate, plasma glucose and plasma cortisol were measured to provide some insight into the metabolic perturbations associated with recovery after swimming to exhaustion.

2. Materials and methods

2.1. Animals and husbandry

A total of 110 wild Atlantic cod (body mass: 1.9 ± 0.5 kg, body length: 64 ± 6 cm, mean \pm SD) were captured by Danish seine in May and June 2013 on the fishing ground “Furholmen” outside Ingøy in Finnmark, Norway. Immediately after capture, the fish were placed in a holding tank supplied with running seawater on board the fishing vessel and transported approximately 290 km to the Aquaculture research station in Tromsø, Norway. At the research station, the fish were held under natural photoperiod (69°N) in an outdoor tank (3 m diameter, 7000 l) supplied with filtered seawater, until the start of the experiment in mid-September 2013. The fish were fed to satiation three times a week with thawed capelin (*Mallotus villosus*), until 48 h prior to transfer of fish into a large-scale outdoor swim tunnel (see Section 2.2). There were no differences in sex distribution for the whole batch (N = 58 females and N = 52 males), but there was a significant difference in gender distribution for the 10-h recuperation group (13 females and 5 males).

2.2. Swim tunnel

The experiment took place in a large swim tunnel submerged in an 11 m diameter circular tank. The volume of the swim chamber was ca 1400 l and measured ca 2.8 m in length and 0.8 m in height and width. A flow director consisting of 5×5 cm tubes of 1 m length was placed in front of the swim chamber to minimize turbulence. Grids of plastic coated stainless steel mesh were placed in the front and rear ends of the fish chamber to prevent fish from escaping the tunnel. The rear grid could be opened to remove exhausted fish. A three-bladed propeller with an integrated 5.5 kW electrical motor (Flygt PP4650.410 with 11° blade angles, Xylem Water Solutions, Charlotte, NC, USA) mounted in a 0.8 m diameter aluminum tube housing propelled the water flow to a maximum speed of 1.2 m s^{-1} .

2.3. Experimental set-up

2.3.1. Control fish

Three days before the swimming trial (see Section 2.3.3), 15 fish were collected randomly from the holding tank and sampled (see Section 2.4) to establish baseline levels of the measured parameters in rested unstressed fish (control). The procedure was completed in less than 10 min. Two days later, the remaining 95 fish were transferred to the swim tunnel and acclimated for 24 h at a water speed of 0.12 m s^{-1} (water temperature: $10.3 \pm 0.2^\circ \text{C}$) prior to the swimming trial.

2.3.2. Exclusion of non-swimmers

Previous pilot studies have shown that 5–10% of gadoid fish will cease swimming in the swim tunnel before they are physically exhausted (Svalheim and Karlsson-Drangsholt, unpublished data).

Therefore, non-swimmers were excluded from the trial, as the aim of the present study was to investigate effects of exhaustive swimming on flesh quality and recuperation. Non-swimmers were identified by initially increasing the water velocity from 0.12 to 0.24 m s^{-1} in two increments of 0.06 m s^{-1} during 10 min, whereafter velocity was maintained at 0.24 m s^{-1} for 30 min. Fish that refused to swim at this speed were pinched gently in the tail three times with use of fingers. The ones that remained unwilling to swim (8 fish) were subsequently taken out and excluded from the experiment. Velocity was then reduced to 0.12 m s^{-1} and the remaining fish were left in the chamber for another 24 h, until the swimming trial commenced.

2.3.3. Swimming trial

Following the exclusion of non-swimmers, the fish density in the swim tunnel was 118 kg m^{-3} . All the remaining 87 fish were swum together in the swimming trial. During the trial, the water velocity was increased by 0.001 m s^{-1} every second for 20 min, until maximum speed of 1.2 m s^{-1} was reached. This water velocity corresponds to a swimming velocity of about 2 body lengths per second, which is twice the critical velocity for sustained aerobic swimming (U_{crit}) in Atlantic cod (~ 1.0 body lengths per second; Lurman et al., 2007). Hence, swimming at velocities above U_{crit} should therefore induce a switch from aerobic to anaerobic swimming and ultimately result in exhaustion (see Reidy et al., 2000).

When fish ceased swimming and laid back on the grid, they were pinched gently in the tail up to three times (with use of fingers) to check if they were willing to swim further. If they did not respond to the third pinch, they were subsequently taken out of the tunnel by a dip net and either sampled directly (0 h recuperation) or assigned randomly to one of the other recuperation groups (2, 4, 6, and 10 h).

2.3.4. Recuperation

The recuperation groups (2, 4, 6, and 10 h) were kept in lid-covered steel mesh (4×4 cm) cages ($1 \times 1 \times 1$ m) placed in a large fibreglass tank (11 m diameter) supplied with running seawater to ensure flow-through of oxygen saturated water.

2.4. Sampling procedure

All fish were euthanized by a blow to the head and blood was collected from the caudal vessels within 1 min, using 6 ml heparinized vacutainers with 4×0.9 mm needles (BD Diagnostics, Franklin Lakes, NJ, USA). Measurements of pH were then obtained from the epaxial part of the white muscle tissue, rostrally to the dorsal fin on the left side of the fish, using a WTW330/set-1 pH-metre (Wissenschaftliche-Technische Werkstätten, Weilheim, Germany) equipped with a Hamilton double pore glass electrode (Hamilton Bonaduz AG, Bonaduz, Switzerland). The instrument was calibrated frequently using pH 4.01 and 7.00 buffers at 10°C , and the electrode was cleaned with demineralized water between each measurement. Concentrations of lactate and glucose were obtained from samples of whole blood, using the hand-held meters Lactate Scout+ (SensLab GmbH, Germany) and FreeStyle Lite (Abbott Diabetes Care, Inc., Alameda, CA), respectively. The remaining blood was then centrifuged at $2700 \times g$ for 5 min at 4°C , and plasma was transferred to cryo tubes, frozen in liquid nitrogen and stored at -80°C until further analysis of plasma cortisol was undertaken (see Section 2.7).

Immediately after blood collection and pH-measurements, all the fish were exsanguinated by cutting the *Bulbus arteriosus* and *Vena cardinalis communis* on both sides. The fish were then bled for 30 min in a tank supplied with running seawater. Afterwards, weight (g), length (cm) and gender of each fish were registered. The liver and gonads were then taken out and weighed (g) to determine hepatosomatic (HSI) and gonadosomatic indices (GSI) by tissue weight $\times 100/\text{total weight}$. The fish were then gutted, covered with plastic film and placed on ice in standard plastic fish boxes.

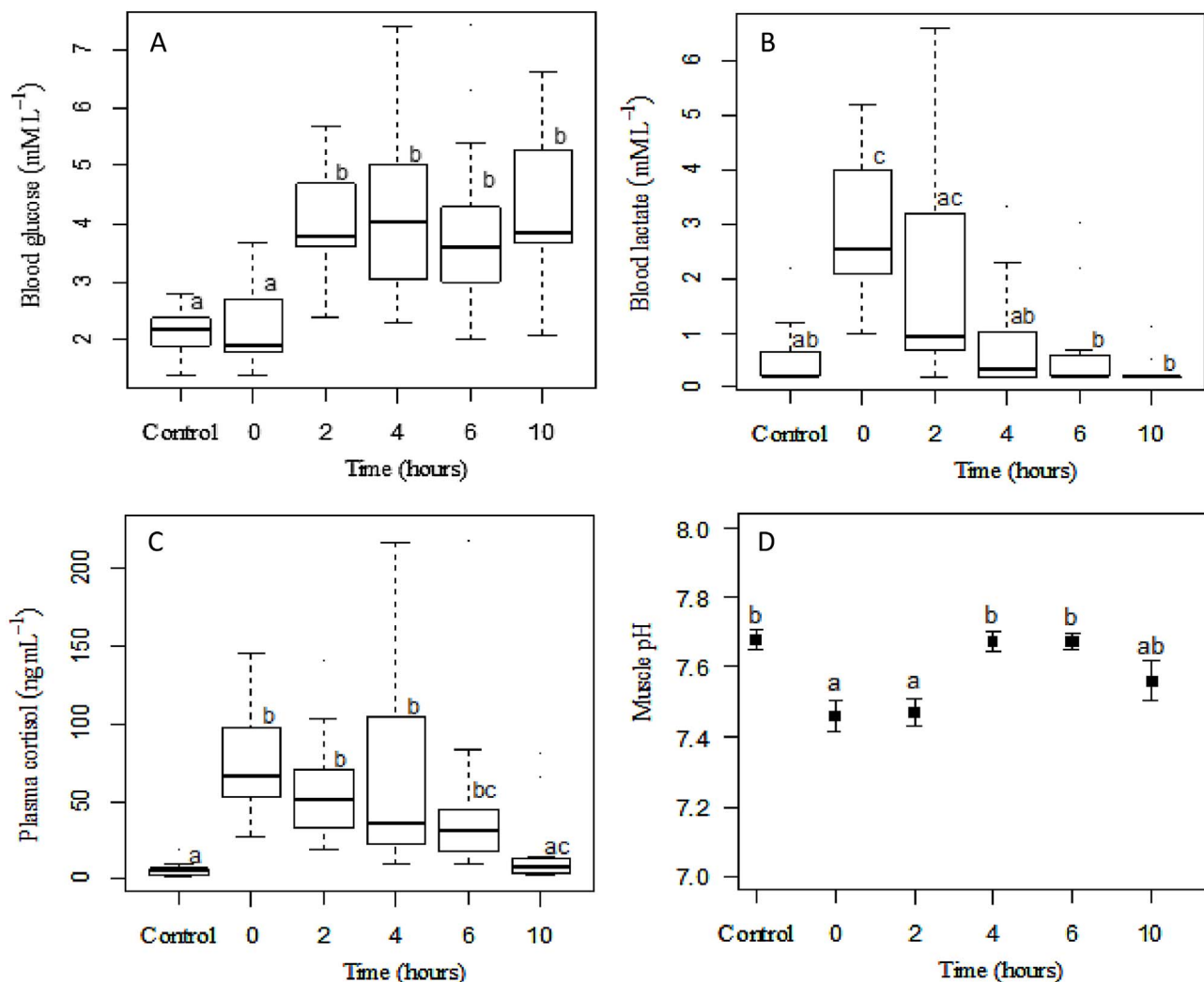


Fig. 1. Blood glucose (A), blood lactate (B), plasma cortisol (C) and muscle pH (D) in Atlantic cod during recuperation (0, 2, 4, 6, and 10 h), following swimming to exhaustion in an experimental swim tunnel. Control; non-exercised fish.

2.5. Evaluation of muscle stiffness

Muscle stiffness, used as an indirect measure of *rigor mortis*, was measured by use of an Effegi penetrometer (FT 02 Penetrometer, QA Supplies LLC, Virginia, USA), according to Berg et al. (1997). Briefly, the maximum force (kPa) required to push a 8 mm diameter stainless steel plunger 5 mm into the flesh was recorded. Measurements were made on 4 different locations (spaced approximately 50 mm apart) along the loin. Over the first 12 h, muscle stiffness was recorded approximately every 4 h. Thereafter, measurements were made, approximately every 8 h, up to 72 h *post mortem*.

2.6. Evaluation of fillet redness

All fish were filleted 72 h *post mortem* and the fillet opposite to that used for measurements of pH and sampling of muscle tissue was used for evaluation of fillet redness. The fillets were not skinned, but the black lining of the peritoneum was removed in order to evaluate the belly flap. Each fillet was evaluated by a sensory panel of 5 trained/experienced persons, according to Olsen et al. (2013), and the fillets were given a score from 0 to 2, where 0 was a white fillet, 1 was a pinkish fillet and 2 was a clearly red fillet.

2.7. Cortisol analysis

Plasma concentrations of cortisol were analyzed by use of radioimmunoassay (RIA), according to Schulz (1985) and Tveiten et al. (2010). Briefly, cortisol was extracted from 300 μ l plasma with 4 ml diethyl ether under vigorous shaking for 4 min. The aqueous phase was frozen in liquid nitrogen, and the organic phase was transferred to a glass tube, evaporated in a water bath at 45 °C and then reconstituted by addition of 600 μ l assay buffer prior to assaying by RIA. The antiserum used was raised in New Zealand white (NZW) rabbits and the detection limit for the assay was 0.6 ng ml⁻¹ (Tveiten et al., 2010).

2.8. Data analysis

Statistical analysis was performed using the statistical software R, version 3.1.3 (R Core Team, 2013), with the packages LearnBayes (Albert, 2014) and pgirmess (Giraudeau, 2014). For each treatment group, all response variables were evaluated for normality and equal variance, using Shapiro Wilkins normality test and density plots. For normally distributed data, one-way ANOVA with type III sum of squares (F-test) was used for testing the effect of recuperation on the various parameters in question. Multiple comparisons were made using a post hoc Tukey Honest Significant Differences Test (Tukey HSD). These data are presented as means \pm standard errors (SE). For the non-normally distributed data, a Kruskal Wallis rank-sum test was used to test for the

effect of recovery on non-parametric response variables, followed by a Multiple-comparison Kruskal-Wallis “kruskalmc” post-hoc test in the package pgrmss. These data are presented as medians in the text and graphically with Tukey box-plot. Outliers are defined as any value lower than 1.5 x Interquartile range (IQR) than the lower quartile and 1.5xIQR higher than the upper quartile of the boxplot. Outliers are not shown graphically.

Some fish had blood lactate levels below the detection limit (0.4 mM) of the instrument. These observations were replaced with a set value of 0.2 mM under the assumption that the unknown values is somewhere between 0 and 0.4 mM. Spearman rank-order correlation analysis was used to examine possible significant correlations between blood glucose, blood lactate, fillet redness, maximum muscle stiffness, time to maximum muscle stiffness, cortisol, condition factor (C), HSI, GSI, and muscle pH. Correlations are presented as Spearman's rank correlation coefficient rho (ρ).

3. Results

3.1. Morphological and physiological assessments

There were no significant differences in HSI (2.99 ± 1.53), GSI (0.57 ± 0.44) or C (0.72 ± 0.11) between the recuperation groups, and there were no correlations between HSI, GSI or C and the other response variables, except for a possible effect of HSI and C on the time to reach maximum muscle stiffness.

Blood glucose did not change during exercise, but increased significantly ($p < 0.001$) within two hours of recuperation from about $2\text{--}4 \text{ mmol L}^{-1}$ and remained at this level throughout the 10-h recuperation period (Fig. 1A). There were significant correlations between blood glucose and fillet redness ($p = 0.043$), maximum muscle stiffness ($p = 0.035$) and muscle pH ($p = 0.034$) (Table 1).

Blood lactate, unlike blood glucose, increased during exercise from about 0.2 mmol L^{-1} in the control group to 2.55 mmol L^{-1} in the group sampled immediately after exercise ($p < 0.001$) (Fig. 1B). Blood lactate then decreased gradually and returned to control level within the first 4 h of recuperation. There were significant correlations between blood lactate and maximum muscle stiffness ($p = 0.030$),

cortisol ($p < 0.001$) and muscle pH ($p < 0.001$) (Table 1).

Plasma cortisol rose significantly during exercise from 4 ng ml^{-1} in the control fish to 66 ng ml^{-1} in the fish sampled immediately after exercise. Plasma cortisol then decreased gradually during recuperation to reach pre-exercise level by the end of the recuperation period (Fig. 1C). There was a significant ($p < 0.001$) correlation between plasma cortisol and blood lactate (Table 1).

Muscle pH dropped significantly ($p < 0.001$) during exercise from 7.68 ± 0.11 (control) to 7.46 ± 0.19 immediately after exercise and 7.47 ± 0.18 after 2 h of recuperation. Muscle pH then returned to pre-exercise level during the next 2 h of recuperation and remained at this level during the remainder of the recuperation period (Fig. 1D). There was a significant correlation ($p = 0.005$) between muscle pH and maximum muscle stiffness.

3.2. Fillet redness and post-mortem development of muscle stiffness

The post mortem muscle stiffness was significantly ($p < 0.001$) higher ($884 \pm 63 \text{ kPa}$) for the fish sampled immediately after exercise than both the controls ($665 \pm 113 \text{ kPa}$) and the recuperated fish (Fig. 2A). The 6- hour recuperation group took significantly longer time to reach maximum muscle stiffness than both the control group and the 10-h recuperation group, but not the other recuperation groups (Fig. 2B). There was a significant correlation between time to maximum rigor and HSI ($p = 0.002$) and C ($p < 0.001$) (Table 1).

There was a trend, although not significant, towards increased redness of the fillet among the fish recuperated for 2 and 6 h (Fig. 2C), but the variation in fillet redness within the groups were substantially. There was a significant correlation between blood glucose and fillet redness ($p = 0.043$) (Table 1).

4. Discussion

The present study suggests that swimming to exhaustion may deteriorate post mortem flesh quality of Atlantic cod by decreasing muscle pH and increasing muscle stiffness during *rigor mortis*. These effects were temporary and muscle pH and stiffness returned to pre-exercise levels when the fish were allowed to recuperate for at least 4 h

Table 1

Spearman rank correlation (ρ) and p-values for pooled cortisol, lactate, muscle pH, glucose, hepatosomatic index (HSI), gonadosomatic index (GSI), condition factor (C), fillet redness, time to maximum muscle stiffness and maximum muscle stiffness.

		Cortisol	Lactate	Muscle pH	Glucose	HSI	GSI	C	Fillet redness	Time to maximum muscle stiffness	Maximum muscle stiffness
Cortisol	ρ	1	0.46	-0.17	0.13	-0.11	0	-0.12	0.08	0.17	0.26
	p-value		> 0.001	0.091	0.197	0.285	0.976	0.239	0.435	0.098	0.009
Lactate	ρ		1	-0.35	-0.08	-0.09	-0.17	0.06	0.02	0.04	0.21
	p-value			> 0.001	0.407	0.354	0.095	0.573	0.867	0.701	0.030
Muscle pH	ρ			1	0.08	-0.14	0.11	-0.17	0.08	0.14	-0.27
	p-value				0.430	0.158	0.263	0.080	0.397	0.175	0.005
Glucose	ρ				1	0.21	0.18	0.02	0.2	0.03	-0.21
	p-value					0.036	0.076	0.853	0.043	0.741	0.035
HSI	ρ					1	0.13	0.55	0	0.32	0.12
	p-value						0.196	> 0.001	0.963	> 0.001	0.244
GSI	ρ						1	0.05	0.08	-0.03	0.01
	p-value							0.634	0.396	0.764	0.894
C	ρ							1	-0.11	0.35	0.35
	p-value								0.290	> 0.001	> 0.001
Fillet redness	ρ								1	0.02	-0.09
	p-value									0.822	0.387
Time to maximum muscle stiffness	ρ									1	0.11
	p-value										0.269
Maximum muscle stiffness	ρ										1
	p-value										

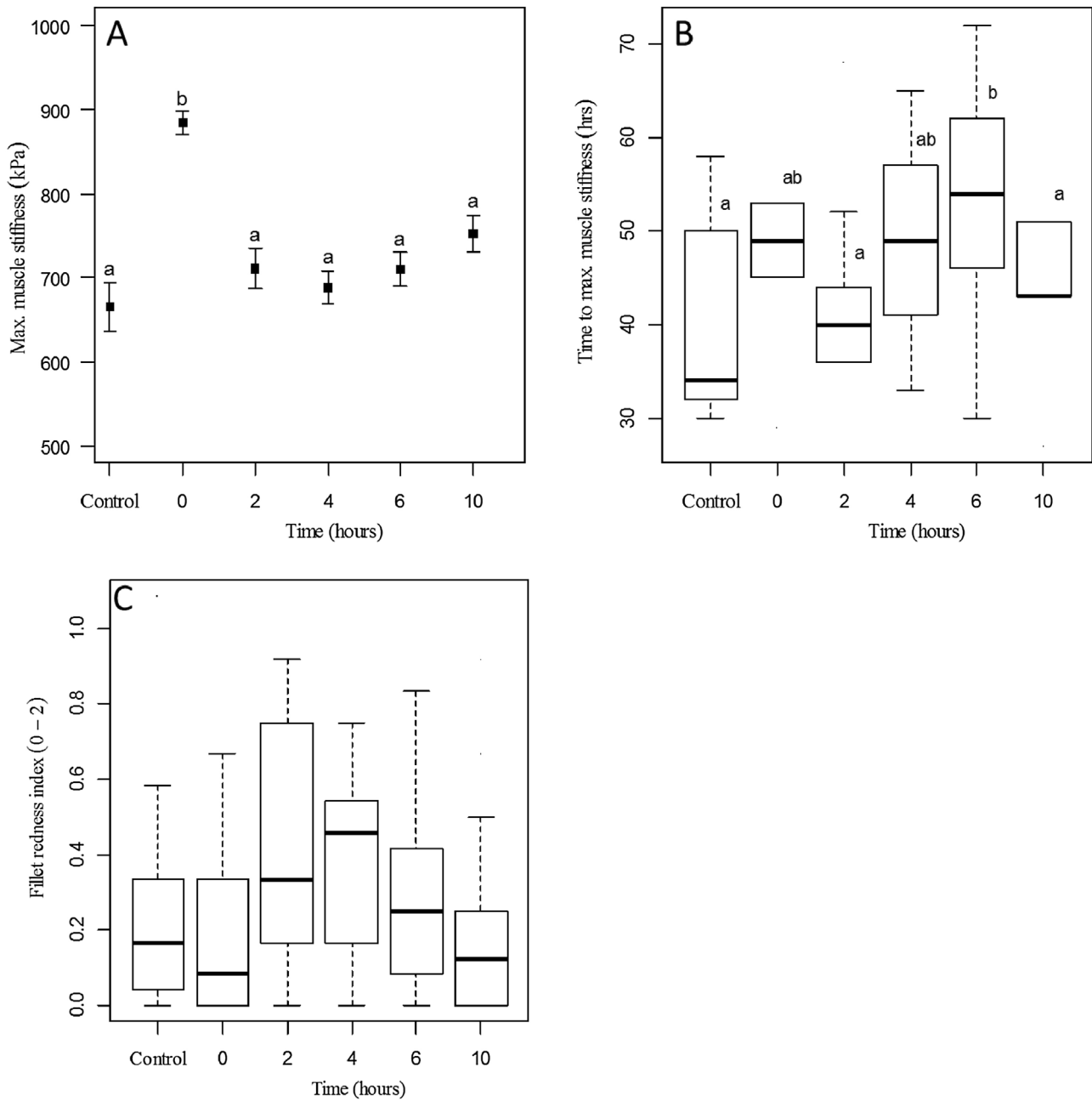


Fig. 2. Maximum muscle stiffness (A), time to maximum muscle stiffness (B) and fillet redness (C) of the white muscle of Atlantic cod slaughtered during recuperation (0, 2, 4, 6, and 10 h) after swimming to exhaustion in an experimental swim tunnel. Control; non-exercised fish.

after the swimming trial. Our results partly corroborate previous studies on Atlantic cod caught by trawl (Olsen et al., 2013; Digre et al., 2017), suggesting that recuperation for 6 h or more after catch may improve the fillet quality substantially. However, unlike the latter studies, which revealed substantial changes in muscle pH, blood lactate and fillet redness, only minor changes in these quality parameters were observed in the present study. Also, the increase in plasma cortisol observed during exercise in our study (from 4 to 66 ng ml⁻¹) was modest and the level observed 2 h into the recuperation period (~50 ng ml⁻¹) was considerably lower than that seen during recovery in both cod caught by trawl (~120 ng ml⁻¹ 3 h after catch; Digre et al., 2017) and pacific salmon caught by gillnet (~1200 ng ml⁻¹ 2 h after catch; Farrell et al., 2001). It seems therefore that exhaustive swimming in unstressed Atlantic cod only has a moderate effect on metabolic stress parameters and fillet quality, and that other stress factors (i.e. crowding in the codend, barotrauma, etc.) probably contribute to the poor fillet quality

frequently observed in cod caught by trawl.

Presence of residual blood in the white muscle of cod caught by trawl (Olsen et al., 2013), causing the flesh to appear red or brown, is considered a quality defect (Margeirsson et al., 2007). This could be due to insufficient exsanguination after catch or increased blood flow to the white muscle during the first few hours after exercise, as suggested by Olsen et al. (2013). Studies on rainbow trout (*Oncorhynchus mykiss*) suggest that up to 80% of the cardiac output is routed to the white muscle during recovery from exhaustive swimming (Neumann et al., 1983). A transient increase in blood flow to the white muscle is probably therefore the most likely explanation for the increase in fillet redness observed during recovery of cod caught by trawl (Olsen et al., 2013).

The increase in blood glucose (0–2 h after exercise) and circulating cortisol is most likely part of the recovery process to restore metabolic status after exhaustion. However, the exact role of cortisol in post-

exercise recovery is not clear. Some evidence suggest that cortisol is primarily associated with mobilization of energy (Gamperl et al., 1994), while other studies indicate that cortisol may be involved in the glycogen recovery metabolism (Eros and Milligan, 1996; Milligan, 2003; Mommsen et al., 1999). There is also some evidence suggesting that cortisol may even prolong the metabolic recovery following exercise, although the mechanisms of its negative effect is not clear (Gamperl et al., 1994). Nevertheless, the plasma values of cortisol observed in the present study were moderate (Gamperl et al., 1994), indicating that the level of exhaustion was not severe.

Swimming to exhaustion increased both maximum muscle stiffness and the time to reach maximum muscle stiffness, and both parameters returned to pre-exercise levels during the recuperation period. There were however no clear pattern with respect to the time to reach maximum muscle stiffness, due to large individual variations within the recuperation groups. Onset and peak level of muscle stiffness are important indicators of fillet quality in fish. A rapidly increasing, and/or high peak of muscle stiffness, indicates low muscle energy status at slaughter and increases the potential for muscle segment gaping and dryness, flesh softening, and colorimetric changes, resulting in reduced shelf-life of the fillets (Bahuaud et al., 2010; Borderías and Sánchez-Alonso, 2011; Erikson and Misimi, 2008; Sigholt et al., 1997; Skjervold et al., 2001).

Swimming to exhaustion resulted in a significant drop in muscle pH, which recovered 2–4 h into the recuperation period. The drop in pH is likely the result of anaerobic activity and production of lactic acid during exercise, as indicated by the elevation of blood lactate during the same period. Most of the lactate produced in the white muscle is resynthesized to glycogen within the muscle itself, but 10–20% of it is released slowly into the blood (Milligan, 1996) with a peak level occurring 2–4 h post-exercise. This pattern has been observed for rainbow trout chased around in a tank (Milligan and Girard, 1993), haddock swum in a large swim tunnel (A. Karlsson-Drangsholt unpublished) and in cod caught by trawl (Olsen et al., 2013). We observed a slightly different pattern, with a peak level of blood lactate occurring immediately after exercise followed by a gradual decrease to almost full recovery 4 h into the recuperation period. It should be noted though that the removal of muscle lactate and resynthesis of glycogen is usually slower, and may require up to 12 h in some species (Kieffer, 2000). Nevertheless, all these studies, including our study, resulted in similar types of metabolic and acid/base disturbances.

We conclude that exhaustive swimming in cod only has a moderate and reversible short lasting negative effect on flesh quality, which is less severe than the poor quality frequently observed in commercially caught cod.

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