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The effect of energetic condition on growth dynamics and health of Atlantic cod (*Gadus morhua*)

By J.-D. Dutil[1], G. Godbout[1,2], P. U. Blier[2] and D. Groman[3]

[1]*Ministère des Pêches et des Océans, Institut Maurice-Lamontagne, Mont-Joli, Québec, Canada;* [2]*Laboratoire de biologie évolutive, Université du Québec à Rimouski, Rimouski, Québec, Canada;* [3]*Aquatic Diagnostic Services, Atlantic Veterinary College, University of PEI, Charlottetown, Prince Edward Island, Canada*

Summary

Prolonged starvation resulting in sublethal condition factor values was hypothesized to have a detrimental effect on short-term growth capacity upon refeeding. Cod *(Gadus morhua*) were food-deprived and their length and mass measured before refeeding and after 3, 6, 9 and 12 weeks of *ad libitum* feeding. Total mass increase during the first 3 weeks of feeding was greatest in fish with a higher initial condition factor. The reverse situation was observed during the last 3 weeks of feeding. Specific growth rate peaked in the period from week 4 to week 6, except in cod with the highest condition factor for which a steady decline in specific growth rate was observed, and was not influenced by the condition factor at the start of the feeding period. Total mass increase over 12 weeks was also not influenced by initial condition factor. Thus by the end of the experiment, condition factors were lowest in fish with initially low condition factors. The hepatosomatic index and gonadosomatic index did not differ at the end of the experiment, but the proportion of mature cod increased with increasing initial condition factor. A disease outbreak caused significant mortalities among fish shortly after the start of the feeding period. Forty-one percent of the fish had died after 84 days. No mortality was observed among fish that had started the experiment with the highest condition factor. Mortality was inversely related to initial condition factor. Growth was examined for survivors exclusively. Poor condition in wild fish may increase vulnerability to diseases and compensatory growth may not allow cod with low condition factors to fully recover unless food availability remains high over a long period of time.

Introduction

When refed after a period of slow or negative growth caused by moderate or severe starvation, fish will restore their energy reserves. They may exhibit a short period of exceptionally faster growth known as compensatory or 'catch-up' growth by which food-deprived fish will reach the size of continuously fed conspecifics. During early life stages food-deprived fish may even grow to larger sizes than continuously fed fish (Hayward et al., 1997; Hayward and Wang, 2001). Catch-up growth is well documented in fishes, particularly in freshwater species and salmonids, including Atlantic salmon *Salmo salar* (Nicieza and Metcalfe, 1997), rainbow trout *Oncorhynchus mykiss* (Dobson and Holmes, 1984), and arctic charr *Salvelinus alpinus* (Miglavs and Jobling, 1989). Previous studies on catch-up growth have focused on its impacts on life-history strategies (Nicieza and Metcalfe, 1997) and feeding hierarchies (Jobling and Koskela, 1996) as well as on the underlying physiological processes (Bélanger et al., 2002). Hayward et al. (1997) examined compensatory growth in view of determining whether restricted rations elicited hyperphagia and could be used advantageously to increase production in aquaculture facilities by using an optimal feeding regime which would bank on catch-up growth mechanisms. In most studies, short periods of food deprivation were considered, from a few days to 1 or 2 weeks (Dobson and Holmes, 1984; Ali and Wootton, 2001; Zhu et al., 2001, 2003), but others used periods as long as 100–120 days (Hayward et al., 1997; Hayward and Wang, 2001).

Wild fish may experience prolonged periods of food-deprivation. For instance, Atlantic cod (*Gadus morhua*) cease feeding for as much as 70 days during prespawning and much of the spawning period (Fordham and Trippel, 1999). They also have to cope with periodic food shortages, particularly in winter (Dutil et al., 2003). When shortages occur, cod must draw energy from body reserves accumulated during periods when food was available (Black and Love, 1986; Lambert and Dutil, 1997b). As a result, cod exhibit marked seasonal and annual variations in energy reserves (Lambert and Dutil, 1997a; Schwalme and Chouinard, 1999), and growth rate (Chouinard and Fréchet, 1994; Dutil et al., 1999), with faster growth occurring in summer and autumn and peak somatic condition factors being reached in autumn. These variations are particularly pronounced in cold water stocks (Dutil et al., 1999; Dutil and Brander, 2003). Low condition factors reached following a prolonged period of fasting are expected to promote catch-up growth when food becomes more abundant. The success of the cod growout industry in eastern Canada may build on this increased capacity for growth in post-spawning cod caught inshore in early summer; cod more than double their body weight in 4–6 months when provided with a nearly unlimited source of raw wild mackerel and capelin (Murphy, 2002). On the other hand, metabolic capacities are depressed and some tissues are significantly atrophied following an extended period of starvation (Dutil et al., 1998; Bélanger et al., 2002; Martínez et al., 2003) which may potentially impair catch-up growth in more emaciated individuals. Few studies have considered long periods of food deprivation in catch-up growth studies (Weatherley and Gill, 1981; Pedersen and Jobling, 1989; Paul et al., 1995) and none has examined whether poor condition affected the time course of catch-up growth upon refeeding.

The present study assesses whether poor condition resulting from prolonged food deprivation may prevent catch-up growth to occur. Prolonged starvation resulting in sublethal

condition factor values was hypothesized to have a detrimental effect on short-term growth capacity upon refeeding. The experimental protocol and laboratory observations are compared with the situation prevailing in the Gulf of St Lawrence in view of understanding how poor condition may lower survival, growth and production in cold water stocks.

Materials and methods

Atlantic cod were obtained from the St Lawrence estuary in June 1996 using a bottom trawl with a small mesh liner and a protective basket in the codend. The catch was released directly into a tank on the deck from which very healthy cod were selected and transferred to holding tanks. The fish were shipped daily from the trawler to our institute and were held under local natural photoperiod in 13-m^3 circular tanks with a 30–50 L min^{-1} flow of oxygenated water until used for the experiment. During acclimation, fish were treated for external parasites with formalin (0.02%, 1-h bath, once a week for two consecutive weeks) and were fed frozen capelin (*Mallotus villosus*) above maintenance needs two or three times weekly. Salinity averaged 27.4 ± 1.3 PSU (mean ± SD) and temperature gradually increased from 6°C in June to 10°C in late August and then gradually decreased to 6°C by mid-October.

The experiment was conducted in a flow-through system of eight 1.5 m^3 circular tanks under the natural photoperiod for the area. Water temperature was controlled and increased from ambient (6°C) to experimental (10°C) over a period of 1 week. Temperature and salinity averaged 10.0 ± 0.3°C and 27.8 ± 1.3 PSU, respectively, and did not vary with time or among tanks during the experiment. Dissolved oxygen was maintained above 80% saturation.

The experiment was done in two stages, with fish being initially food-deprived and then fed to satiation, and started in the second week of October for all treatments. Cod ranging in size from 40 to 50 cm fork length were selected and allocated to the different tanks on the basis of their relative condition factor. They were anaesthetized in a solution of metomidate hydrochloride (4 mg L^{-1}), received two Visual Implant Tags (Northwest Marine Technology, Shaw Island, WA, USA) in the first dorsal fin and were measured (fork length, ±1 mm) and weighed (±1 g). Food deprivation occurred over either 7 weeks (second week of October to first week of December) or 12 weeks (first week of October to first week of January). At the end of the starvation period, the experimental design was represented by the following (Table 1): one tank contained fish with lower condition factors (group 1; 12 weeks of starvation), 3 pairs of tanks (six tanks in all) contained fish with intermediate condition factors (groups 2, 3, 4; 12 weeks of starvation); and one tank contained fish with higher condition factors (group 5; 7 weeks of starvation). Group 5 was starved for a shorter period of time to achieve a significant difference in initial condition factor from group 4. During the following 12 weeks, fish were fed frozen capelin three times a week and were measured at 3-week intervals. They received in each meal all the food they could ingest within 1 h. Food consumption was determined for each meal by weighing the distributed food and what was left over.

Fish were killed after 12 weeks of feeding (group 5, last week of February; other groups, first week of April) and their liver (±0.1 g), gonads (±0.1 g), head-on carcass mass (±1 g) were measured. Condition factor (K), hepatosomatic (HSI) and gonadosomatic (GSI) index, liver and white muscle energy content, and liver, gonads and white muscle moisture content

Table 1
Size and condition factor of Atlantic cod (mean ± SD, number of observations) after starvation and before (subscript i) and after (subscript f) 12 weeks of feeding. The condition factor after feeding was calculated using somatic (Ks_f) and total fish mass (K_f). Groups are defined on the basis of condition factor at the start of the feeding experiment: group 1, fish with lower condition factors; group 5, fish with higher condition factors; groups 2–4, fish with intermediate condition factors. Differences among groups were tested with ANOVAs assuming a one-way treatment structure; the last column shows the *F* ratio and associated probability

	K group									
	1	2		3		4		5		
Tank	1	2	3	4	5	6	7	8	Probability
$Length_i$(mm)	462 ± 26 (20)	443 ± 27 (20)	456 ± 26 (20)	460 ± 30 (20)	459 ± 26 (20)	464 ± 24 (20)	462 ± 21 (20)	467 ± 23 (21)	$F = 1.59$ $P = 0.142$
$Mass_i$(g)	505 ± 93 (20)	511 ± 88 (20)	560 ± 84 (20)	652 ± 130 (20)	639 ± 105 (20)	748 ± 122 (20)	729 ± 91 (20)	863 ± 129 (21)	$F = 28.05$ $P < 0.001$
K_i	0.51 ± 0.04 (20)	0.58 ± 0.02 (20)	0.59 ± 0.03 (20)	0.66 ± 0.02 (20)	0.65 ± 0.02 (20)	0.74 ± 0.02 (20)	0.74 ± 0.03 (20)	0.84 ± 0.03 (21)	$F = 290.2$ $P < 0.001$
$Length_f$(mm)		481 ± 32 (10)	512 ± 29 (8)	502 ± 35 (15)	509 ± 31 (11)	505 ± 28 (17)	515 ± 17 (13)	511 ± 26 (21)	$F = 1.66$ $P = 0.140$
$Mass_f$(g)		1060 ± 239 (10)	1082 ± 186 (8)	1191 ± 374 (15)	1357 ± 270 (11)	1384 ± 287 (17)	1450 ± 177 (13)	1420 ± 280 (21)	$F = 4.11$ $P < 0.01$
K_f		0.94 ± 0.06 (10)	0.82 ± 0.17 (8)	0.91 ± 0.13 (15)	1.02 ± 0.09 (11)	1.06 ± 0.12 (17)	1.06 ± 0.07 (13)	1.06 ± 0.10 (21)	$F = 8.39$ $P < 0.001$
Ks_f		0.90 ± 0.06 (10)	0.79 ± 0.16 (8)	0.88 ± 0.13 (15)	0.97 ± 0.11 (11)	1.00 ± 0.10 (17)	0.99 ± 0.06 (13)	0.99 ± 0.09 (21)	$F = 60.12$ $P < 0.001$

were determined as described in Dutil and Lambert (2000). Mortalities were recorded daily. Gross signs of potential diseases were noted on all fish, being characterized as either patches of red/erythemic skin, bleeding or non-bleeding ulcers on the body and one or more fins. Fish were classified into two categories: category 1, no wound or only patches of red/erythemic skin; category 2, bleeding or non-bleeding ulcers on the body and one or more fins. The heart and spleen of most survivors in groups 2, 3 and 4 were preserved in 10% formalin. Samples were embedded in paraffin and slides were prepared and stained with eosin and hematoxylin at the Centre for Bone & Periodontal Research, Royal Victoria Hospital, Montréal. One or two sections of each tissue were examined for significant morphological changes and the samples classified by either presence or absence of lesions. When lesions were present, each tissue with lesions was ranked based on severity and complexity: *incidental* – one histologic lesion or focus of inflammation per tissue section; *mild* – between 1–10% of tissue section was qualitatively estimated to be impacted by lesion or inflammation; *moderate* – 10–30% of the tissue section was qualitatively estimated to be impacted by lesion or inflammation; *severe* – >30% of the tissue section was qualitatively estimated to be impacted by lesion or inflammation.

Statistical analyses

Statistical analyses were conducted with SAS Release 8.02. Differences were considered significant at $\alpha = 0.05$. Differences in size, condition factor, GSI and HSI among groups of fish at the start and end of the feeding period were tested assuming a one-way treatment structure. Homogeneity of variance was tested with the Brown–Forsythe's test. Differences were tested with ANOVAs when variances were homogeneous. Tukey's test was used for pairwise comparisons. Differences in GSI were tested for mature and immature fish separately (Dutil et al., 2003). Heterogeneity of variances precluded ANOVAs on HSI. Thus differences in HSI at the end of the feeding period were tested with the Kruskal–Wallis chi-square statistic on rank scores. Differences in mean length and condition factor at the start of the feeding period between survivors and deceased cod were tested for each group separately (groups 2, 3 and 4), using a two-sample *t*-test (variances were equal in all cases).

The association between survival and initial condition factor and between presence and absence of morphological damages based on microscopic examinations of the heart and spleen and initial condition factor were tested by combining the results from replicate tanks and computing the Mantel–Haenszel statistic controlling for survival. The association between maturity (number of fish with GSI below and above 3%) and survival was assessed while controlling for the effects of sex, as sex might have had an impact on survival during maturation. The association between sex and survival (maturing and immature fish combined) and between maturity and survival for each sex separately were tested using the chi-square test for independent samples. The association between maturity and survival controlling for sex was tested with the Mantel–Haenszel statistic. The odds ratio, i.e. the relative risk of not surviving given the maturity status, was calculated for the two sexes combined using the logit estimator for the common odds ratio, and the assumption of homogeneous odds ratios for males and females was tested with the Breslow-Day test.

The proportions of fish that survived or died during the feeding period and the number of fish with GSI below and above 3% at the end of the feeding period were calculated for each tank, and differences in their proportions between tanks were tested within each group using the chi-square test for independent samples. There was no tank effect for any of the groups ($P > 0.05$) and thus we pooled the data to examine differences among groups in the number of fish that survived or died and in the proportion of fish with GSI below and above 3%.

Growth was examined for survivors exclusively. The effects of the initial condition factor on total mass increment and specific growth rate were tested by ANOVA. Specific growth rate was calculated as the difference between final and initial mass ($\log_e$-transformed) divided by the number of days for the period examined. Two tests were performed, one considering tank as a nested factor in the analysis and individual fish within the tanks as replicates, and a second one considering tanks as replicates ($n = 2$ observations).

Results

There was no difference in the mean length of cod between tanks at the start of the feeding period, but the mean mass and condition factor differed significantly ($P < 0.05$, Table 1). Pairwise comparisons indicated significant differences in condition factor between groups, but not between replicate tanks within groups ($P < 0.05$). Variances were found to be homogeneous for length, mass and condition factor ($P > 0.05$).

Disease

During the feeding period, a disease outbreak caused significant mortalities among fish (Fig. 1; Table 2). Mortality was 41% overall after 84 days (66/161). There was a strong association between initial condition factor (K group) and mortality (Mantel–Haenszel $\chi^2 = 46.98$, $n = 1$; $P < 0.001$), with no significant difference between replicate tanks ($P > 0.05$ in all three cases). No mortality was observed among fish that started the experiment with the highest condition factor (K group 5, 0% mortality) whereas no survival was observed among fish that started the experiment with the lowest condition factor (K group 1, 100% mortality). Mortality was highest early in the experiment: 44 and 22 fish over the first and last 42 days of the experiment, respectively.

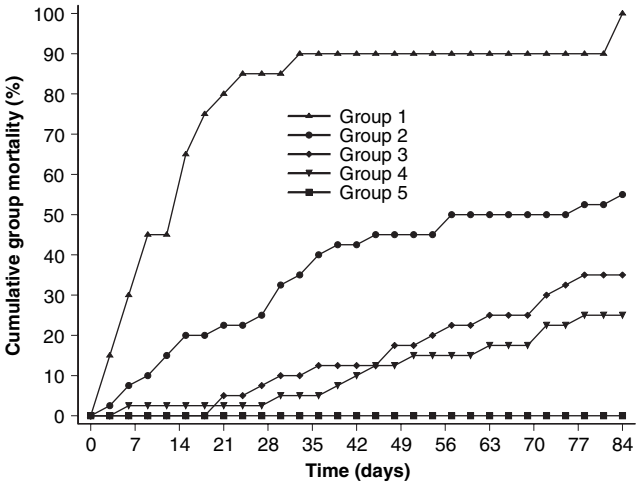

Fig. 1. Cumulative mortality (%) over a 12-week period for different groups of Atlantic cod defined on the basis of condition factor at the start of the feeding period (Table 1). Differences between replicate tanks at the end of the experiment are shown in Table 2

Table 2
Numbers of fish that survived or died during the 12-week feeding period. Groups are defined on the basis of condition factor at the start of the feeding experiment (Table 1). Total counts are shown as well as counts for males and females separately

	K group							
	1	2		3		4		5
Tank	1	2	3	4	5	6	7	8
Total								
Survived	0	10	8	15	11	17	13	21
Died	20	10	12	5	9	3	7	0
Males								
Survived	0	9	6	10	9	11	10	18
Died	15	7	10	5	7	2	7	0
Females								
Survived	0	1	2	5	2	6	3	3
Died	5	3	2	0	2	1	0	0

The majority of fish had few external wounds, i.e. were classified into category 1 (56% overall); this percentage did not vary among groups with intermediate condition factors (K groups 2, 3 and 4; $P > 0.05$). There was no correlation between initial condition factor and relative abundance of external wounds when controlling for survival (Mantel–Haenszel $\chi^2 = 2.039$, $n = 2$; $P > 0.05$) in groups with intermediate condition factors (K groups 2, 3 and 4). Similarly, no correlation was found between initial condition factor and presence of morphological changes as based on microscopic examinations of the heart and spleen (Mantel–Haenszel $\chi^2 = 0.011$ and 0.620 for heart and spleen, respectively, $n = 1$; $P > 0.05$) of 70 fish having survived the experiment in groups with intermediate condition factors (K groups 2, 3 and 4). A slight majority of survivors exhibited no morphological changes to the heart and spleen (56% overall). When significant morphological changes were observed, lesions were characterized by inflammation of the heart muscle (myocarditis) and spleen (splenitis) with or without multifocal microsporean-induced xenoma formation, and in very few cases bacterial, granuloma formation. The microsporean and bacterial agents could not be identified by microscopic examination alone, but distribution and morphology of the lesions were consistent with infection by *Loma branchialis* and *Aeromonas salmonicida*. Most of the fish were classified as having incidental or mild lesions (75%). Others were classified as having moderate (16%) and in fewer cases severe (8%) lesions.

There was no association between sex and survival ($\chi^2 = 1.12$, $n = 1$; $P > 0.05$; Table 2). While in females survival was not associated with maturity status ($\chi^2 = 1.74$, $n = 1$; $P > 0.05$), there was a strong association in males ($\chi^2 = 19.02$, $n = 1$; $P < 0.001$) with a greater proportion of maturing males having survived (Table 3). The sex ratio was imbalanced (35 females and 126 males) and low counts for females may explain this outcome. When controlling for sex, survival and maturity were strongly associated (Mantel–Haenszel $\chi^2 = 19.79$, $n = 1$; $P < 0.001$) with an odds ratio of 7.1 (95% confidence interval, 2.8–18.0). The ratios for males and females were homogeneous ($P > 0.05$).

There were no significant differences in mean length at the start of the feeding period between survivors and deceased cod of groups 2 and 4 ($P > 0.05$). There was also no difference in mean length at the start of the feeding period between survivors and deceased cod of group 3 ($P > 0.05$), but condition factor differed significantly ($P = 0.01$), being slightly lower in deceased fish (0.648 ± 0.019, range 0.629–0.687, $n = 14$) than in survivors (0.665 ± 0.019, range 0.621–0.693, $n = 26$).

Table 3
Numbers of fish with GSI < 3% (I) and GSI > 3% (M) at the end of the 12-week feeding period. Column percentages are shown in parentheses. Groups are defined on the basis of condition factor at the start of the feeding experiment (Table 1). Total counts are shown as well as counts for males and females separately

	K group			
	2	3	4	5
Total				
I	11 (61.1)	12 (46.1)	9 (30.0)	3 (14.3)
M	7 (38.9)	14 (53.9)	21 (70.0)	18 (85.7)
Males				
I	9	8	6	3
M	6	11	15	15
Females				
I	2	4	3	0
M	1	3	6	3

Growth of survivors

Mass increase was influenced by the condition factor at the start of the feeding period and varied among time intervals (Fig. 2). Mass increase in groups with intermediate condition factors was lowest in the first and final 3-week intervals, and highest in the second and third 3-week intervals. Thus mass increase started slowly, increased in the mid-period and then declined in all three groups. In groups with intermediate condition factors, mass increase during the first 3 weeks of feeding was lowest in fish with a lower initial condition factor (group 2: tank 1, 75 ± 42 g, $n = 10$; tank 2, 88 ± 16 g, $n = 8$) and greatest in fish with a higher initial condition factor (group 4: tank 1, 138 ± 44 g, $n = 17$; tank 2, 163 ± 39 g, $n = 13$). The effect of the initial condition factor was significant ($P < 0.05$), but not that of the tank ($P = 0.43$) when individual fish were considered as replicates. A different analysis in which tanks were considered as replicates ($n = 2$ observations) also indicated a significant effect of initial condition factor ($P < 0.05$). Mass increase during the first

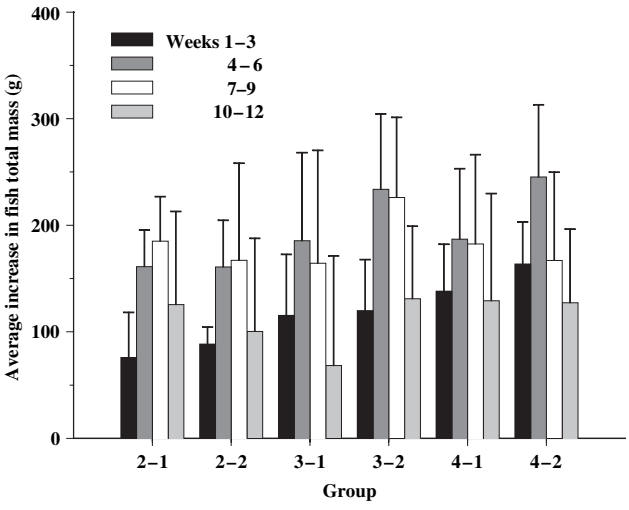

Fig. 2. Increase in fish total mass (g) at 3-week intervals for different groups (first digit) of Atlantic cod defined on the basis of condition factor at the start of the feeding period (Table 1). Results for two replicate tanks are shown (second digit)

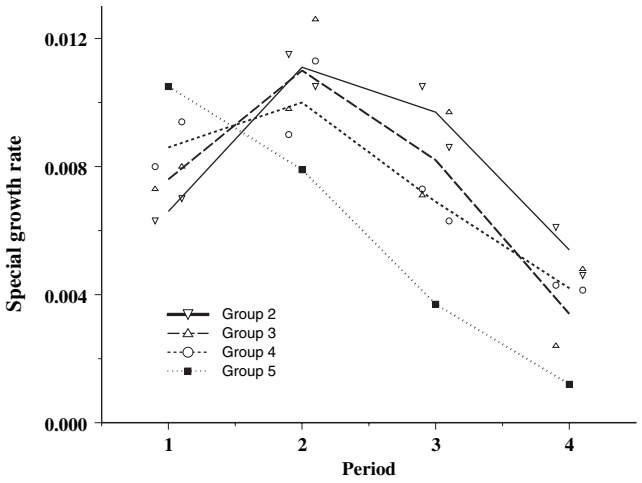

Fig. 3. Specific growth rate in fresh mass at 3-week intervals for different groups of Atlantic cod defined on the basis of condition factor at the start of the feeding period (Table 1). Period 1, weeks 1–3; period 2, weeks 4–6; period 3, weeks 7–9; period 4, weeks 10–12. Results for replicate tanks are shown as open symbols and average values are represented by lines

3 weeks was 213 ± 44 g (n = 21) in fish with the highest initial condition factor (group 5). During the last 3 weeks of feeding, mass increase was not influenced by initial condition factor whether individual fish (P = 0.60; no tank effect P = 0.32) or tanks (P = 0.63; n = 2 observations) were considered as replicates. Mass increase during the last 3 weeks was 38 ± 78 g (n = 21) in fish with the highest initial condition factor (group 5), which was much less than in fish with intermediate condition factors (Fig. 2).

Specific growth rate of intermediate K-groups (groups 2–4) was not influenced by the condition factor at the start of the feeding period (Fig. 3). Growth rate was highest in the period from week 4 to 6 and lowest in the period from week 10 to 12, with intermediate values observed at the start of the feeding period. Growth rate of cod with the highest initial condition factor (group 5) was higher than in cod with intermediate condition factors from week 1 to 3, but then was consistently less and declined throughout the remainder of the feeding period. The effect of initial condition factor for weeks 1 to 3 and weeks 10 to 12 was not significant (groups 2–4; P = 0.14 and P = 0.46, respectively), nor was the tank effect (P = 0.33 and P = 0.26, respectively) when individual fish were considered as replicates. Similar results were obtained when tanks were used as replicates (P = 0.13 and P = 0.41, respectively).

Initial condition factor had no significant effect on total mass increase over 12 weeks, whether individual fish (groups 2–4, P = 0.38; tank effect not significant, P = 0.18) or tanks (P = 0.34, n = 2 observations) were considered as replicates. Total mass increase averaged 613 ± 224 g in cod with intermediate condition factors (groups 2–4, n = 74) and 557 ± 236 g in cod with the highest initial condition factor (group 5, n = 21).

By the end of the feeding period the mean length of surviving cod did not differ between tanks but significant differences were found for the somatic and total mass, and for the condition factor based on both somatic and total mass (P < 0.05, Table 1). Pairwise comparisons indicated no significant differences between replicate tanks within groups for length, mass and condition factor (P > 0.05). Variances were found to be homogeneous for length, mass and condition factor (P > 0.05). HSI and GSI did not differ between tanks at the end of the feeding period (P = 0.33; Table 4). The proportion of cod with GSI > 3% increased with increasing initial condition factor (χ^2 = 10.72, P < 0.05; Table 3).

Discussion

This study clearly established an inverse relationship between condition factor and disease prevalence. No mortality or sign of disease was observed among fish with the highest condition factors whereas all fish with the lowest condition factors died within a short period of time when refed following a food-deprivation period. Emaciation is often interpreted as resulting from a disease (Rand and Cone, 1990; Mellergaard and Nielsen, 1996; Overton et al., 2003) rather than being interpreted as a contributing factor to disease (Vethaak and Jol, 1996). Because all fish originated from the wild, it is most likely that all of the agents which caused lesions noted on histology also originated from infection in the wild. It is worth noting, however, that when fish are held in captivity (especially in recirculating systems) for any length of time, the combination of captivity stress on the immune system and the presence of infectious agents in the captive population may cause horizontal transfer of disease. Horizontal transfer of disease may be further enhanced through aggressive behaviours. Cod may exhibit aggressivity in the form of fast approaches, grunting and proding; actual biting is rarely observed however (Brawn, 1961). Ultimately, we have no way of quantifying or confirming that this happened in the experiment. The only concrete information we have regarding the disease conditions recognized by histology is an estimate of severity or chronicity. Clearly lesions that are chronic (and therefore likely present when the fish were captured) will show more granulomatous

Table 4
Liver-somatic index (HSI) and gonads-somatic index (GSI) of Atlantic cod (mean ± SD, number of observations) after starvation and after 12 weeks of feeding. Groups are defined on the basis of condition factor at the start of the feeding experiment (Table 1). GSI-I, GSI index for immature individuals, GSI-M, GSI index for mature individuals. CMH, Mantel–Haenszel chi-square on ranks

	K group							
	2		3		4		5	
Tank	2	3	4	5	6	7	8	Probability
HSI	7.2 ± 1.2 (10)	5.7 ± 2.1 (8)	6.2 ± 2.5 (15)	6.6 ± 1.8 (11)	7.0 ± 1.5 (17)	7.2 ± 0.9 (13)	6.5 ± 1.2 (21)	CMH = 6.85 P = 0.33
GSI-I	1.1 ± 1.0 (5)	1.0 ± 0.6 (6)	1.4 ± 0.7 (7)	0.9 ± 1.0 (5)	1.7 ± 0.8 (4)	1.7 ± 0.5 (5)	2.1 ± 0.8 (3)	F = 1.37 P = 0.26
GSI-M	7.1 ± 2.3 (5)	9.4 ± 1.2 (2)	6.5 ± 3.5 (8)	10.3 ± 6.2 (6)	8.2 ± 4.7 (13)	13.3 ± 4.6 (8)	9.1 ± 6.4 (18)	F = 1.43 P = 0.22

inflammation or possibly even some resolution (wound healing). More acute infections will show infections with bacteria and microsporeans (the primary agents identified in this population of cod) with less inflammation.

Resistance to stress and diseases might also have been influenced by maturation. The neuroendocrine and immune systems of fish interact and several hormones associated to reproduction have been shown to either enhance or depress immunocompetence (Harris and Bird, 2000). Compared with immature fish, a larger proportion of maturing fish survived in the present study. This does not necessarily mean that the immune system of maturing fish was enhanced. Most deaths were recorded during the first half of the feeding period; survivors had more time to develop larger gonads than cod which died early in the feeding period.

The range of target condition factors in our experiment encompassed the extreme condition factors observed in the spring period in the early to mid-1990s in the Gulf of St Lawrence (Lambert and Dutil, 1997a; Schwalme and Chouinard, 1999). Poor condition in winter and around spawning time has been shown to negatively affect several aspects of the cod life history. Cod spawn in the spring. Low condition factors associated with spawning increase mortality (Dutil and Lambert, 2000) and reduce fecundity (Lambert and Dutil, 2000). Furthermore, controlled feeding experiments have shown that muscle metabolic capacities decrease progressively as fish condition deteriorates (Lemieux et al., 2004). Depressed metabolic capacities in the red and white muscle of fish in poor condition were found to be correlated with a much lower capacity for sustained swimming and a slower recovery following burst swimming in fish in poor condition (Martínez et al., 2003, 2004). These results suggest that poor condition may lead to increased mortality and slower growth through a reduced capacity of cod to catch mobile prey and to escape predation or mobile fishing gears. The present study adds to this list by suggesting an increased vulnerability to disease by poor condition fish. Poor condition is observed regularly in less productive stocks living in more extreme environments (Dutil et al., 1999; Dutil and Brander, 2003) and may thus increase natural mortality through energy exhaustion as well as through increased vulnerability to pathogenic diseases.

Growth rates achieved in our laboratory experiment may have been slowed by two factors: disease and maturation. Although we have examined growth in survivors exclusively, some of the survivors may have experienced disease. This is less likely to have occurred in the group with the highest condition factors, which did not experience any mortality during the experiment. Hence, faster growth rates in other groups over 12 weeks would suggest that this effect was not great. Our experiment was conducted in the autumn-winter period, i.e. during maturation. Maturation was more advanced in groups with higher condition factors at the start of the feeding experiment. This may have contributed to their slower growth later into the experiment. Assuming a gonads-somatic index value of 2% at the start of the experiment, roughly 12% of the change in total mass in the group with the highest condition factors would be ascribed to an increase in the mass of the gonads.

The main goal of the present study was to determine whether prolonged starvation resulting in sublethal condition factor values would have a detrimental effect on short-term growth capacity upon refeeding. Prolonged starvation alters the structure and composition of many organs (Beardall and Johnston, 1985; Black and Love, 1986) and these changes could lower the capacity of fish to feed and process food. Growth compensation proceeded similarly irrespective of growth history in two groups of fish with similar initial condition factors at the onset of refeeding (Zhu et al., 2003). No other study has examined catch-up growth as a function of condition, but several studies have investigated growth following a prolonged period of starvation. Larval fish starved during 2 weeks and experiencing high mortality rates due to energy exhaustion were able to catch up to continuously fed larvae within 2 weeks, suggesting that survivors were able to maintain or scale up their metabolic capacities upon refeeding (Molony, 1993). Compensatory growth was also observed in juvenile turbot, with faster growth taking place in fish having experienced the most severe food restriction (Saether and Jobling, 1999). Similarly, in rainbow trout starved for several weeks and having experienced a 32% weight loss, growth exceeded that of controls upon refeeding, but the timing of that recovery was not investigated (Weatherley and Gill, 1981). In cod, fish in poor condition grew more rapidly than fish in good condition, suggesting catch-up growth took place even in fish with the lowest condition factors. The fish were not food-deprived and their feeding history was unknown, and fish with higher condition factors were used (Pedersen and Jobling, 1989) than those fish in the present study. The fish had been submitted to food deprivation for several weeks in the present study, but catch-up growth was hypothesized to be suppressed in fish reaching to extremely low condition factors. Our results indicate that extremely low condition factors had a detrimental effect on growth capacity. Growth was slow during the first 3 weeks upon refeeding. Slow growth in poor condition fish was short-lived however and higher specific growth rates were observed in poor condition fish than in control fish over the next few weeks. Compensatory growth is usually associated with hyperphagia and increased efficiency of food utilization (Ali et al., 2003) suggesting that digestive capacities are increased upon refeeding. Blier et al. (1997) suggested fish growth is limited by the digestive processes. The increased activity of citrate synthase in the pyloric caeca during growth compensation may indicate an enhanced aerobic production of ATP to fuel higher rates for the synthesis of digestive enzymes, for instance trypsin (Lemieux et al., 1999). Early in the post-starvation period, fish increase the relative mass of pyloric caeca and intestine which suggests that regeneration of the digestive functions and increased capacity to produce digestive enzymes and to absorb nutrients across the gut wall are necessary steps in the process of catch-up growth (Bélanger et al., 2002). The present study suggests that poor condition may temporarily limit the capacity of cod to bank on increased food availability.

The duration of the feeding experiment in the present study (12 weeks) was representative of the duration of the feeding period in the northern Gulf of St Lawrence in 1994, when condition factors were minimal in June and peaked in late-August (Lambert and Dutil, 1997a). Poor condition fish grew in length and in mass as much during this period as fish with higher condition factors. Nevertheless, their somatic condition factor was on average lower at the end of the experiment (0.85 ± 0.12, 0.92 ± 0.12, 0.99 ± 0.08, 0.99 ± 0.09, for groups 2, 3, 4 and 5, respectively). These fish would thus be at a greater risk of mortality over winter (Dutil et al., 2003). Results obtained on subadult sole led to similar conclusions (Paul et al., 1995). Furthermore, fish in the laboratory experiment were fed to satiation three times a week. In situations where food is limited or distributed unevenly in time and space, fish in poor condition may not recover to the point where catch-up growth takes place. Our data also indicate that

mass recovery after refeeding starts earlier at higher condition factors. In the wild, condition factor as well as physiological condition of the digestive system at the beginning of the growth season might be key determinants of growth performance over summer. The first few weeks after spawning in the spring and early summer period would thus appear to be critical to the survival of adult cod in the Gulf of St Lawrence.

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Author's address: Jean-Denis Dutil, Ministère des Pêches et des Océans, Institut Maurice-Lamontagne, C. P. 1000, Mont-Joli, Québec G5H 3Z4, Canada.
E-mail: dutiljd@dfo-mpo.gc.ca