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Metabolic responses to food deprivation and refeeding in juveniles of *Rutilus rutilus* (Teleostei: Cyprinidae)

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Synopsis

Effect of food deprivation and refeeding on metabolic parameters were studied in juvenile *Rutilus rutilus*, weighing 280–460 mg. Tissue hydration increased with the length of the starvation period, reaching a new steady state after 4–5 weeks. Total protein concentration remained constant at about 60% of dry body mass. The concentration of glycogen decreased during food deprivation, a new steady state being reached at about 30% of control values after 4 weeks. Refeeding caused a dramatic increase of glycogen concentration which exceeded the value in fed controls by 6- to 9-fold. This is seen as a tactic for rapid storage of food energy, to be used later for the synthesis of body materials. With respect to their responses to food deprivation the 12 enzymes investigated formed four groups: (1) activity unaffected by food deprivation or refeeding (COX, THIOL, CK, GOT); (2) activity drops to about 60% of control value during the initial phase of food deprivation but remains constant thereafter (PK, LDH, Pase); (3) slow but continuous decrease in activity during the whole period of starvation, i.e. up to 7 weeks (PFK, OGDH, CS, FBPase); (4) activity increases during food deprivation, decreases again upon refeeding (GPT). A model is discussed which distinguishes between four phases in the general response of young fish to food deprivation and refeeding: *stress*, *transition*, *adaptation*, and *recovery*.

Introduction

Due to the temporal and spatial patchiness of food availability in the aquatic environment, periods of food deprivation are common events in the life of a fish. The metabolic consequences of such events being most pronounced in larvae and juveniles (Gadomski & Petersen 1988, Richard et al. 1991). In a previous paper (Wieser 1991) it has been pointed out that due to the high energy requirements and low metabolic scope for activity of small fish, selection might have favored the following sequential strategy in response to periods of food shortage: (1) when food becomes scarce, increase locomotor activity at first so as to raise the chance of finding a new food patch; (2) if the absence of food continues beyond a critical time limit, decrease locomotor and metabolic activities as much as possible; (3) once food becomes available again convert it into body substance as rapidly as possible.

Clear evidence for the operation of these strategies has been found in young cyprinids. Juvenile *Rutilus rutilus* deprived of food for about 24 h showed higher locomotor activity and higher rates of oxygen consumption in a respirometer than well-fed fish (Wieser et al. 1988a). In contrast, after two days of food deprivation, the routine rates of metabolism of the juveniles of three species of cypri-

nids decreased by 30–40% compared to the rates of recently fed specimens (Wieser et al. 1992). There was only a slight further decrease, of about 0.8% day^{-1}, when food deprivation was prolonged for another four weeks. When food was made available again, the young fish rapidly increased their rates of oxygen consumption and growth, the peaks of both variables increasing in proportion to the length of the starvation period.

These observations on the responses of young cyprinids to food deprivation were based on the measurement of oxygen consumption and growth rates following periods of starvation and refeeding. Here, we report on investigations designed to answer the question of whether the whole animal responses are accompanied by changes in the metabolic machinery of the fish. Since most of the whole animal responses are interpreted to reflect changes in either locomotor activity or muscle growth, the present investigation concentrated on the metabolic events following periods of food deprivation and refeeding occurring in the swimming musculature of juvenile cyprinids.

Material and methods

Fish and experimental protocol

Juvenile roach, *Rutilus rutilus* (L.), with initial body masses of 280 to 460 mg were used. The fish had been raised in the laboratory from parent fish originating from Seefelder See near Innsbruck (see Wieser et al. 1988b). Maintenance of the fish followed the protocol given in Wieser et al. (1992), except that in addition to *Artemia* nauplii the fish were also given pelleted trout feed and, occasionally, *Tubifex* sp. Maintenance and experimental temperatures were 20° C.

A large group of roach was transferred from the holding tanks into an aquarium where it was left without food. On days 7, 14, 22, 29, 36, and 49, samples of fish were removed and treated for the determination of body constituents and enzyme activities. On the same days, except the last one, other samples of fish were transferred from the starvation treatment to a tank where they were fed pelleted trout feed and *Tubifex* sp. ad libitum. After 7 and 14 days of refeeding, aliquots of fish were removed again for the determination of body constituents and enzyme activities. We have no evidence for the occurrence of cannibalism even during the longest period of food deprivation.

Determination of body constituents and enzyme activities

The fish sampled at specified intervals were weighed, head and tail were cut off, the body opened ventrally and the intestine with the internal organs removed. The remaining carcass was weighed (wet body mass: wbm) and frozen at − 70° C until further treatment. Aliquot samples of the fish carcasses were dried for 24 h at 80° C and reweighed (dry body mass: dbm). In agreement with the data on carp (Itazawa & Oikawa 1983) the carcass of *R. rutilus* represented about 60% of total body weight. Approximately 30% were contributed by the head and the tail, the remaining 10% by the internal organs.

Enzymes studied

Glycogen phosphorylase (Pase; EC 2.4.1.1.); phosphofructokinase (PFK; EC 2.7.2.11.); fructose-1,6-bisphosphatase (FBPase; EC 3.1.3.11.); pyruvate kinase (PK; EC 2.7.1.40.); lactate dehydrogenase (LDH; EC 1.1.1.27.); creatine kinase (CK; EC 2.7.1.40.); citrate synthase (CS; EC 4.1.3.7.); oxoglutarate dehydrogenase complex (OGDH); cytochrome oxidase (COX; EC 1.9.3.1.); acetoacetyl-CoA thiolase (THIOL; EC 2.3.1.9.); glutamate-oxaloacetate transaminase (GOT; EC 2.6.1.1.); glutamate-pyruvate transaminase (GPT; EC 2.6.1.2.).

Preparation of homogenates

Tissue samples were homogenized at 0° C with an Ultraturrax homogenizer for three periods of 10 s in 10 volumes of one of the following extraction media: Medium A (for PFK, PK, LDH, GOT, GPT, FBPase): 50 mM Tris-HCl buffer pH 7.5, 1 mM EDTA, 5 mM $MgCl_2$, 1 mM dithiothreitol, 0.1% Triton X-100. Medium B (for CS): 50 mM

Tris-HCl buffer pH 8.5, 1 mM EDTA, 0.1% Triton X-100. Medium C (for COX): 0.1 M phosphate buffer pH 7.0, 0.1% Triton X-100. Medium D (for OGDH): 50 mM triethanol-amine-HCl buffer pH 7.4, 1 mM EDTA, 1 mM dithiothreitol, 2 mM $MgCl_2$, 0.1 mM oxoglutarate, 2 M glycerol. Medium E (for THIOL): 75 mM Tris-HCl buffer pH 7.5, 3 mM $MgCl_2$, 5 mM EDTA, 1 mM dithiothreitol, 0.1% Triton X-100. Medium F (for CK): 50 mM Pipes/NaOH buffer pH 7.0, 1 mM EDTA, 4 mM dithiothreitol, 3 mM Mg acetate, 30 mM KCl, 0.1% Triton X-100. Medium G (for Pase): 50 mM Tris-HCl buffer pH 7.5, 1 mM EDTA, 20 mM NaF, 0.5 mg/ml bovine serum albumine.

Homogenates used for determination of PFK, LDH, PK, CS, GOT, GPT, FBPase, CK and Pase were centrifuged for 20 min at 15 000 × g, that for THIOL 10 min at 1 000 × g. The crude homogenate was used for determination of the OGDH and COX activities.

Assays

Measurements of enzyme activity were performed at 20° C, the final assay volume being 1 ml. Spectrophotometric assays followed standard procedures, as given by Hinterleitner et al. (1987, 1989) and Moon (1983). In the paper by Hinterleitner et al. (1987) the assay conditions given for GOT are actually those for GPT. We used the correct assay, of course. Protein was determined using the Lowry technique. Glycogen concentration was quantified by a spectrophotometric method. Samples were homogenized with an Ultraturrax homogenizer in 5 volumes of ice-cold 10% meta-phosphoric acid. A 50 µl sample of this homogenate was added to 0.5 ml 2 M acetate buffer pH 6.2. After mixing, a 100 µl sample was taken for the determination of free glucose. 20 µl of amyloglucosidase were added and the solution was incubated at room temperature for three hours. After the incubation, a 100 µl sample of the solution was centrifuged, and total glucose was determined in a system containing 20 µl ATP (5 mg ml^{-1}), 20 µl NADP (2 mg ml^{-1}), 800 µl buffer (0.3 M triethanolamine-HCl, pH 7.5, 4 mM $MgSO_4$) and 50 µl supernatant. 10 µl of glucose-6-P dehydrogenase were added and the increase in absorbance was followed until constancy was reached. Then 10 µl of hexokinase were added and the increase in absorbance again followed until constancy was reached. The two coupling enzymes were added in excess so as to reach the end point of the reaction within 10 minutes.

Biochemicals were obtained from Sigma Chemical CO., St Louis, U.S.A., inorganic chemicals from Merck-Schuchardt, Darmstadt, BRD.

Statistics

In order to decide whether enzyme activity changed during food deprivation, simple regressions of activity against length of starvation period (days) were calculated. If the slope of the regression was not significantly different from zero, average rates of enzyme activity during the whole period of food deprivation were calculated and compared with the rates of fed ('control') fish (t-test). The same procedure was used with enzyme activities after refeeding.

Results

Body constituents

Tissue hydration

Water content of the carcasses of juvenile roach increased with the length of the starvation period. However, not until 36 days was the water content significantly different from that of the controls; it had returned to the control level after one week of refeeding (Table 1).

Total protein

Despite significant changes in the activity of some enzymes during food deprivation, no change of total protein concentration could be detected in the dry mass of the carcasses used for the determination of enzyme activities. Protein concentrations in roach fed, starved, refed after 1 week, and refed after 2 weeks, amounted to 60.5 ± 1.2 (n = 8), 60.6 ± 3.8 (14), 59.6 ± 1.5 (7), and 60.5 ± 1.7 (4) per cent of dbm, respectively.

Glycogen

The concentration of this component changed dra-

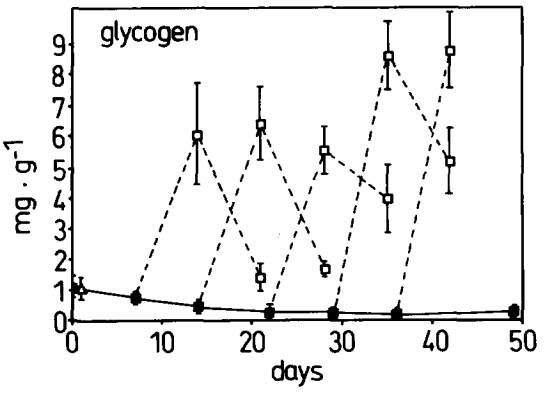

Fig. 1. Concentration of glycogen (mg gwbm^{-1}) in carcasses of juvenile *R. rutilus*, deprived of food (solid line, full symbols), and refed for one or two weeks (dashed lines, open symbols). Means and SD shown. Variability (means ± SD) of glycogen concentration of fed controls during the whole experimental period indicated by open triangle and vertical lines.

matically during starvation and refeeding, decreasing from 0.92 ± 0.4 (n = 15) to 0.28 ± 0.16 (4) mg gwbm^{-1} after about four weeks of food deprivation, and increasing when feeding was resumed. After one week of refeeding, the concentration of glycogen exceeded the value of the fed controls about six-fold in fish starved for less than 22 days, and about nine-fold in fish starved for longer periods (Fig. 1).

Enzyme activities

With respect to their responses to food deprivation the 12 enzymes investigated formed four groups.

Group 1 (Table 2a)

Enzyme activity was totally unaffected by food deprivation or refeeding. This group comprised two enzymes of aerobic catabolism, COX and THIOL, one muscle enzyme, CK, and one of the two transaminases, GOT.

Group 2 (Table 2b)

Enzyme activity dropped strikingly during the first week of food deprivation, but remained constant thereafter. The three enzymes belonging here (PK, LDH, and Pase) play important roles in glycolysis and glycogenolysis. The decrease in activity during the first 7 days of food deprivation amounted to 47–39% of control levels, and recovery was fairly slow. After one week of refeeding none of the three enzymes had returned to control level, and even after two weeks the activity of PK was significantly lower than that of the fed controls.

Group 3 (Table 3, Fig. 2)

Enzyme activity decreased continuously from day 7 to the end of the starvation period, the slope of the regression line being significantly different from zero ($p < 0.001$). The rate of change during this period ranged from 0.8 to 1.5% per day^{-1}. This group includes the key enzyme of glycolysis, PFK, as well as two key enzymes of the citrate cycle, CS and OGDH, and the gluconeogenic enzyme, FBPase. After one week of refeeding, three of the four enzymes had returned to control levels. PFK differed from all other enzymes studied in that its activity increased strongly with body size in the

Table 1. Tissue water content (% of wbm) in carcasses of juvenile *Rutilus rutilus* after varying periods of starvation and refeeding. Results are expressed as means ± SD of n individuals (in parentheses). Significantly different from the fed controls at a – $p < 0.05$, b – $p < 0.01$, c – $p < 0.001$.

Days of starvation	7	14	22	29	36	49
Starved	74.0 ± 1.3 (4)	75.0 ± 0.6 (3)	75.9 ± 2.1 (6)	76.6 ± 1.1[a] (6)	78.2 ± 2.1[b] (5)	78.6 ± 1.1[c] (5)
+ 1 week refeeding	73.5 ± 1.6 (3)	73.7 ± 1.3 (3)	75.8 ± 0.7 (5)	76.4 ± 0.4 (6)	76.0 ± 0.8 (6)	
+ 2 weeks refeeding	76.5 ± 1.4 (3)	74.1 ± 0.4 (3)	75.7 ± 0.2 (4)	75.1 ± 0.6 (4)		
Controls	75.3 ± 1.4 (18)					

Table 2. Activity (μmol min^{-1} gwbm^{-1}) at 20° C of enzymes extracted from carcasses of juvenile *R. rutilus*. Results (means, SD, and number of individuals) are presented for the fed controls, for fish starved 1–5 weeks, and for fish refed 1 week and 2 weeks. a – Enzymes unaffected by food deprivation and refeeding (group 1; see text). In none of the groups was activity significantly different from that of the fed controls. b – Enzymes with reduction of activity during the first week of food deprivation (group 2). Significantly different from controls at b = $p < 0.01$, c = $p < 0.001$.

Enzyme	Control	Starvation	Refeeding	
			1 week	2 weeks
a				
CK	1383 ± 350 (12)	966 ± 444 (18)	1013 ± 174 (12)	1057 ± 143 (12)
COX	8.5 ± 2.0 (13)	9.1 ± 1.2 (15)	9.3 ± 2.3 (12)	9.7 ± 2.2 (14)
THIOL	1.2 ± 0.3 (7)	1.3 ± 0.3 (16)	1.2 ± 0.2 (4)	1.2
GOT	16.9 ± 2.7 (17)	14.7 ± 1.8 (18)	16.4 ± 1.4 (11)	15.2 ± 2.6 (12)
b				
PK	65 ± 11.2 (12)	34 ± 9.4^{c} (17)	42 ± 6.3^{c} (12)	44 ± 7.6^{c} (13)
LDH	193 ± 51 (16)	118 ± 36^{c} (18)	137 ± 27^{b} (12)	166 ± 47 (13)
Pase	5.3 ± 1.3 (12)	2.8 ± 0.9^{c} (18)	3.9 ± 0.7^{b} (12)	4.8 ± 0.9 (13)

controls, at least up to an average weight of 1 g. During this period, activity of PFK in the control group is represented by the function y(μmol min^{-1} g^{-1}) = 2.74 + 0.106 x (d).

Group 4 (Fig. 3)

This group is represented by a single enzyme, GPT, the activity of which increased with food deprivation. The increase was gradual (from 2.7 ± 0.5 to 3.36 ± 1.28) during the first three weeks, but rather steep between days 22 and 29, when activity increased from 3.36 ± 1.28 to 5.68 ± 1.53 μmol min^{-1} g^{-1} (n = 5; $p < 0.05$; t-test). Refeeding caused enzyme activity to return to the lower val-

Table 3. As in Table 2, enzymes with continuously decreasing activity (group 3). Change of activity indicated by the regression of activity against days of starvation, i.e. y = a-bx. Also given is the mean activity during the whole period of food deprivation (7 weeks). The slopes of all four regression were significantly different from zero at $p < 0.001$.

Enzyme	Control	Regression	Starvation (×)	Refeeding	
				1 week	2 weeks
PFK	2.9 ± 0.4 (4)	2.3 − 0.037 x (38)	1.5	3.0 ± 0.5 (20)	3.1 ± 1.0 (20)
CS	6.5 ± 0.9 (18)	5.6 − 0.047 x (23)	4.6	5.8 ± 0.7 (12)	5.9 ± 0.7 (13)
OGDH	0.63 ± 0.04 (5)	0.55 − 0.007 x (23)	0.4	0.55 ± 0.07 (12)	0.56 ± 0.1 (13)
FBPase	0.22 ± 0.04 (17)	0.17 − 0.002 x (23)	0.13	0.21 ± 0.04 (12)	0.20 ± 0.04 (13)

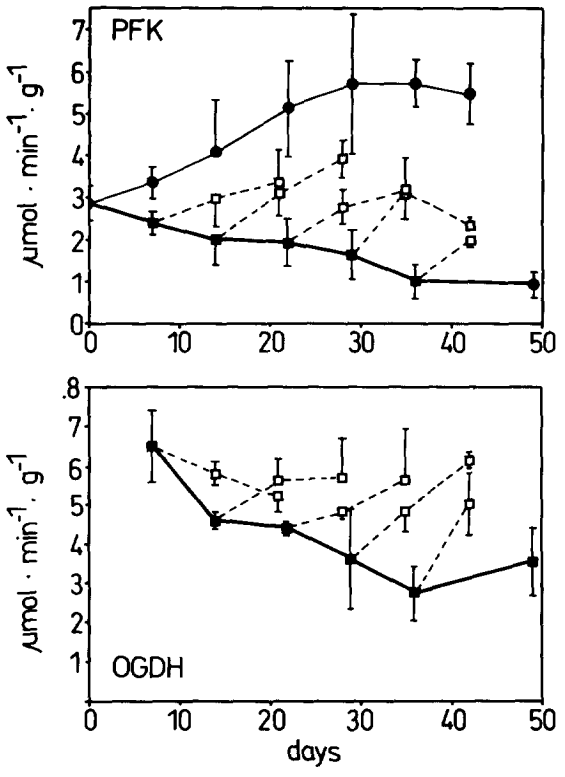

Fig. 2. Activity of PFK and OGDH in carcasses of juvenile *R. rutilus*, deprived of food (thick lines, full squares), refed for one or two weeks (dashed lines, open squares), and – for PFK only – continuously fed (thin lines, full circles). Means and s.d. are shown. Acclimation and experimental temperature 20° C. Enzyme activity referred to wet body mass.

ues of the fed controls, but only in the fish starved for less than 22 days was this process completed within two weeks. When food had been withheld for 29 days and longer, two weeks of refeeding were not sufficient to bring enzyme activity back to the level of the fed controls.

Discussion

Since lack of food is a frequent condition in the lives of animals, it is obvious that starvation is not just an experience passively endured, but involves a tightly controlled reorganization of metabolism and behavior (Newsholme & Start 1973, Lowery & Somero 1990). Despite much work on the morphological and metabolic consequences of starvation in fish (Ivlev 1961, Love 1970, Moon & Johnston

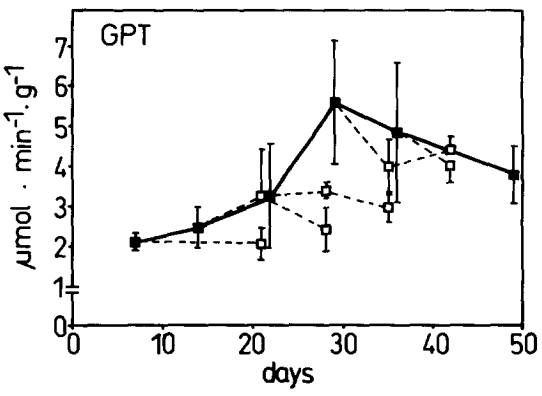

Fig. 3. Activity of GPT in carcasses of *R. rutilus*, deprived of food and refed. Symbols and conditions as in Table 2.

1980, Moon 1983, Lowery et al. 1987, Blaxter 1988, Moon et al. 1989, Lowery & Somero 1990) few generalizations have emerged which would allow a comparison with the much better understood situation in mammals. The main reason for this shortcoming is that interspecific variability of the responses of fish to food deprivation is huge. In addition, age and size, as well as temperature and other ecological factors, are of profound importance in setting the stage on which the reorganization of metabolism can take place.

On the basis of information so far available and by elaborating the ideas mentioned in the Introduction, we suggest that four phases can be distinguished in the sequence of responses of fish to food deprivation and refeeding. In order to provide a short system of reference, these phases will be called *stress, transition, adaptation,* and *recovery.*

(1) Stress

If food is withheld for a short time (the length of which depending mainly on species, age, and temperature) fish may enter a state of hyperactivity, reflecting a lowered appetitive threshold for food searching behavior. Due to their low energy reserves such a response will be strongest in larvae and young juveniles. Phases of hyperactivity in food-deprived fish have been mentioned by Huse & Skiftesvik (1985), and Blaxter (1988), and a quantitative assessment has been attempted by Wieser et al. (1988a). The larvae of *R. rutilus* kept on low rations and entirely deprived of food for

24 h before the experiment, consumed about 50% more oxygen during the first 10 hours in a respirometer than well-fed larvae of the same age. It is very likely that this response, expressed in levels of activity and patterns of behavior, is under hormonal and/or neural control and thus may be considered a true stress response. A more precise definition would require the determination of hormone levels in the plasma of food-deprived fish.

(2) Transition

After the phase of hyperactivity, young fish respond to continued food deprivation by reduction of the routine rate of oxygen consumption. In juvenile representatives of three species of cyprinids (*Leuciscus cephalus* (L.), *Chalcalburnus chalcoides mento* (Agassiz), and *Scardinius erythrophthalmus* (L.)), this phase of transition was completed after two days (Wieser et al. 1992). By following the rate of oxygen consumption during a 24 h cycle it was shown that the reduction concerned mainly the activity, not the maintenance, component of the energy budget. In this way about 40% of the metabolic energy dissipated by well-fed fish was saved by the starving fish. This finding on the whole animal level is matched by the results of the present investigation, showing that after 7 d of food deprivation the activity of some of the major glycolytic and glycogenolytic enzymes in the swimming muscles of juvenile roach, i.e. PK, LDH, and Pase, had decreased to 50–60% of control levels. At the same time the activity of COX and THIOL, two key enzymes of aerobic catabolism, remained unaffected by food deprivation. This suggests that during the phase of transition, energy metabolism in the swimming muscles of young roach shifted from a carbohydrate dominated to a – probably – lipid dominated form, as is the case in most mammals (Newsholme & Start 1973). This shift of energy metabolism parallels the reduction of carbohydrate-fuelled locomotor activity. In agreement with this finding, Lowery et al. (1987) and Lowery & Somero (1990) have shown in the barred sand bass, *Paralabrax nebulifer,* that starvation caused a reduction in the activities of glycolytic enzymes in white muscle, whereas protein synthesis in red muscle remained unaffected.

(3) Adaptation

This phase is characterized by the stability of whole body metabolic rate around the reduced level reached at the end of phase 2, indicating that a new steady state of energy turnover has been established. In the three cyprinid species studied previously, prolonged starvation led to only a slight further decrease of the rate of oxygen consumption of about 0.8% day^{-1}. This is in agreement with the stability of most of the enzymes of aerobic catabolism as documented in the present investigation. No change in activity was observed in COX and THIOL, whereas CS and OGDH, but also PFK and FBPase, exhibited a slow, albeit significant, decrease in activity of about 1% day^{-1} between 7 and 49 days of starvation. It is of interest that in the juvenile cyprinids studied, the rate of change of whole body metabolic rate is matched by the rate of change of the activity of the major regulatory enzymes of energy metabolism, particularly PFK and OGDH. However, the stability of whole body energy dissipation should not be taken as evidence that no metabolic reorganization is taking place during the phase of adaptation. In fact, after about four weeks of food deprivation a further shift of the metabolic state of juvenile roach seems to have occurred. By this time the water content of the muscles had stabilized at slightly higher (Table 1), and glycogen content at very much lower (Fig. 1) levels than those characteristic of the controls. In accordance with the data and conclusions of Cowey & Sargent (1979), Weatherley & Gill (1981), Black & Love (1986), Lim & Ip (1989), Miglavs & Jobling (1989), and others, it has to be assumed that up to this time lipids serve as the major metabolic fuel, which is gradually being replaced by proteins. The time course of this sequence is illustrated by the change in activity of GPT, the major link of the glucose/alanine cycle, a well-known feature of metabolism in starving mammals (Newsholme & Leech 1983). As Figure 3 demonstrates, the activity of GPT in juvenile roach increased slowly for about three weeks of food deprivation, but rose by about 70%, from 3.4 to 5.7 $\mu mol\ min^{-1}\ g^{-1}$, between days 22 and 29. An equally striking increase in GPT activity with starvation has been described in the red muscle of immature eels by Moon (1983). Han-

sen & Abraham (1989) have shown that alanine and serine are the preferred amino acids for gluconeogenesis in the fasted eel, whereas no such preference was apparent in fed eel. The increase in transaminase activity is coupled to an increase of proteolytic activity in the muscles of starving fish, as described, for example, by Beardall & Johnston (1985b) for the saithe, *Pollachius virens*.

(4) Recovery

Termination of the starvation period by ad libitum feeding leads to a rapid increase in the rates of oxygen consumption and growth of juvenile cyprinids. Two characteristic features were a day-by-day increment of the metabolic response, and a compensatory growth spurt correlating positively with the length of the starvation period (Wieser et al. 1992). The present investigation showed that, of the enzymes studied, only PK, LDH, and Pase, which had experienced the greatest reductions in activity during the initial period of food deprivation, failed to recover completely within one week of refeeding (Tables 2, 3). PFK, on the other hand, had recovered completely after 1 week of refeeding. This is in agreement with the fast recovery response of the same enzyme in *P. nebulifer*, which, according to Lowery et al. (1987), is due to its rapid shift in compartmentalization within the cytoplasm. There was no sign of an overshoot in the activity of any enzyme. In contrast, muscle glycogen displayed a striking overshoot, its concentration in the swimming muscles exceeding control levels by 6- to 9-fold after one week of refeeding.

A similar overshoot in glycogen storage following starvation and refeeding has been reported in pike (Ince & Thorpe 1976), saithe (Beardall & Johnston 1985a), and cod (Black & Love 1986), and may represent a tactic for the fastest and most efficient way of storing food energy, later to be mobilized for the synthesis of body materials (Kamra 1966, Black & Love 1986, Hansen & Abraham 1989). This interpretation is supported by our discovery that the overshoot in glycogen storage in the muscles of *R. rutilus* correlates with the compensatory growth response of the three species of cyprinids studied previously. After four weeks of starvation, refeeding led to initial growth rates of up to 30% day^{-1} in *C. chalcoides* and *S. erythrophthalmus*, whereas following shorter periods of starvation, the initial growth rates after refeeding ranged only from 5 to 20% day^{-1} (Wieser et al. 1992). Along the same line, glycogen storage in *R. rutilus* after one week of refeeding was considerably higher in fish starved for more than four weeks than in those exposed to shorter periods of food deprivation (see Fig. 1). Since the overshoot of the rate of glycogen synthesis after refeeding is not accompanied by similar changes in the activity of the single gluconeogenetic enzyme assayed, GPT, it may be speculated that enzyme activity is not a bottleneck of the process of glyconeogenesis in the muscle tissues.

The sequence of events described above may be considered a general model representing the metabolic responses of fishes (and other animals) to food deprivation and refeeding. The temporal structure of this model is bound to be strongly influenced by age, life style, and features of the environment of a given species. For example, the reduction in enzyme activity during starvation may be quite drastic, as in plaice (Moon & Johnston 1980), or nearly absent, as in the eel (Moon 1983). However, with time it should be possible to distinguish between general features and specific adaptations in the strategies employed by fish in dealing with one of the most ubiquitous properties of the environment: the temporal and spatial patchiness of food.

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