Restoration of Body Energy Reserves during Refeeding in Rats Is Dependent on Both the Intensity of Energy Restriction and the Metabolic Status at the Onset of Starvation

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Abstract

During starvation, after a short dynamic period of adaptation (phase I), a metabolic steady state is reached in which proteins are spared and lipids provide most of the energy expended [phase II (P2)]. However, protein breakdown increases dramatically once a lower threshold of body lipids is reached [phase III (P3)]. Body composition, energy intake, energy expenditure, and energy efficiency were determined in 8 groups of rats (fed, food-deprived up to P2 or P3 of starvation and refed for 3 d, 7 d, or until body mass restoration) to determine whether the kinetics of lipid and/or protein reserve recovery may be slowed down when refeeding occurs after the lipid threshold has been reached. Despite larger losses, P3 refed rats restored their body reserves as efficiently as those refed in P2. Whatever the nutritional status at the onset of refeeding, rehydration occurred first and hyperphagia played a more important role than hypometabolism in the restoration of the lost reserves. However, the pattern of body component gains was different during early refeeding. In P3 refed rats, body lipids were restored preferentially by significant contribution from endogenous lipid production. Thus, the extent of lipid depletion has important consequences for the restoration pattern of the body reserves. It depends not only on the intensity of the energy restriction (partial or total) as already demonstrated but also on the metabolic status at the onset of refeeding. These results may have significant implications on the way refeeding should be conducted after severe energy depletion. J. Nutr. 138: 861–866, 2008.

Introduction

Periods of negative energy balance resulting from restricted feeding or total starvation are common events (1,2). In humans, such situations can arise due to disease, eating or psychological disorders, or hunger strikes. In wild animals, total or partial food deprivation can be a consequence of environmental conditions or occur as part of the natural life cycle (3–6). Rehabilitation after a period of negative energy balance is possible (1,2) even when an extensive depletion in body reserves has occurred (7–9). The kinetics of body fuel restoration is related to the severity of the restriction (10). Body fat is recovered earlier than body protein in intake restricted, refed rats (10,11), independent of the composition of the diet refed (12). In contrast, in starved-refed rats, a body fat deficit persists when body mass and protein are restored, within 7–12 d (9,10). This asymmetrical pattern occurs in both males and females (9–11) even if the depletion of body lipids and proteins occurs to the same extent after a partial or total food deprivation (10). Thus, the extent of lipid restoration depends on whether the loss was a result of partial or total food deprivation. To better understand body restoration following starvation, it is necessary to relate the dynamics of the restoration of body reserves to the extent of their depletion (13). To date, the possible impact of the metabolic phases reached during prolonged starvation (4,14) has not been taken into account. As previously shown in different species and both sexes, use of body reserves is not linear throughout starvation (1,2,4,9). After a short period of adaptation (phase I), a steady state is reached [phase II (P2) of starvation] (4) in which lipids provide 80–95% of the energy expended and proteins are spared. However, once ~80–90% of the lipid reserves have been used, protein catabolism increases dramatically (P3 of starvation) (4,8,9,15). During P3, fat stores fall below a lower defended level. Animals in this metabolic situation increase their exploratory activity to search for food (4,8) and we hypothesize that the dynamics of body store recovery depends on whether refeeding is initiated before or after the lipid threshold is reached.
The aim of this study was to determine the effect of the metabolic status reached during starvation on the kinetics of recovery of body stores, particularly during early refeeding. Rats were refed in P2 or in P3 of starvation. Changes in energy intake and body composition were determined until the prestarvation body mass was recovered.

**Materials and Methods**

**Animals.** Male Wistar rats (IFAA-CREDO) were housed individually in a controlled room (23 ± 1°C, 50–60% humidity) with a 12-h-light–dark cycle. They were fed a standard powder diet (A03 from UAR; w/w: wt composition: proteins 23.5%, carbohydrates 53.8%, fat 5.0%, cellulose 4%, minerals 5.6%, and water 12%; metabolizable energy content, 13.4 kJ/g) supplied in cups covered with a grid to avoid spillage. The animals had free access to food (except when starved) and tap water and were adapted 1 wk before the beginning of the experiment. Body mass and food consumption were recorded daily (±0.1 g). The experiment complied with the “Principles of Animal Care” publication no. 86–23, revised 1985 of the NIH and with current legislation (L87–848) on animal experimentation in France.

**Experimental procedures.** Rats were randomly assigned to experimental groups. Body mass and age ranged from 410 ± 1 g to 412 ± 1 g and from 15.2 ± 0.1 wk to 15.5 ± 0.2 wk, respectively, and did not differ among the groups. Experimental groups consisted of a fed control group, 3 groups that were starved for 5 d up to the middle of P2 (P2 groups), and 4 groups that were starved until they had been in P3 for 2 d, i.e., starving for a total duration of 11–12 d (P3 groups). The criteria used to stop starvation of the P3 groups were: body mass loss (>30%), increased daytime locomotor activity, piloerection, and 2 consecutive days of an increased rate of body mass loss. From previous experiments, rats starved up to the early stages of P3 are capable of restoring their body mass and refed up to the initial body mass recovery; P3R0, rats starved up to phase III and not refed; P3R3, rats starved up to phase III and refed for 3 d; P3R7, rats starved up to phase III and refed for 7 d).

Blood was collected in heparinized tubes and centrifuged at 1000 g for 10 min. The liver, the retroperitoneal (RP) and epididymal (EPI) fat pads, the intestine, the stomach, and leg muscles (soleus and extensor digitorum longus (EDL)) were dissected and weighed (±0.001 g). The intestine and the stomach were cleared of their contents. The fat pads, intestines, and stomach were replaced with the remaining carcass.

The liver, muscles, carcass, and plasma were kept frozen (−20°C) until analysis.

**Analytical methods.** The carcass was ground roughly under liquid nitrogen. The muscles, the liver, and 100 g of the carcass were freeze-dried until constant mass was achieved. The dry aliquots of carcass and the liver were then finely ground to obtain a homogenous powder. Total lipids were determined on the carcass and on the liver (16). The Test-Combination 240 052 (Boehringer) was used to determine the triacylglycerol content of carcasses. Nitrogen content was measured in the muscles, carcass, and liver using the Kjeldahl method. Proteins were calculated as 6.25-nitrogen (9). Plasma nonesterified fatty acids (NEFA) and β-hydroxybutyrate concentrations were measured using the NEFA C test (Wako Chemicals) and an enzymatic Test-Combination (Boehringer-Mannheim), respectively.

**Calculations and statistics.** The specific daily change in body mass (dm/mdt) was the difference in body mass between 2 consecutive days divided by mean body mass for the 2 d. Net body mass was the body mass minus digestive contents. Total body water was net body mass minus the dry mass of the carcass, liver, and dissected muscles. Structural lipids were calculated as the body fat minus body triacylglycerols. The increase in body components during the acclimation period in control-fed rats was estimated assuming that the contribution of water, proteins, and lipids to body mass was constant. In control and refed rats, an endogenous lipid production corresponding to the synthesis of lipid from the animal body was calculated when the intake could not account for the storage.

Energy expenditure or gain calculations between groups followed the procedure of Hill et al. (17,18). The individual losses during starvation were calculated by subtracting the mass of body components in individual rats of the P2R0 and P3R0 groups from the mean masses in the prestarvation group. The body component gains during the refeeding period (R0 to RT, R0 to R3, R3 to R7 or RT, and R7 to RT) were obtained similarly. Energy intake was calculated from food intake and its metabolizable energy content. The amount of food in the digestive tract in P3 rats was subtracted from the energy intake during the first 3 d of refeding, because R0 rats had no food in the digestive tract. According to Hill et al. (17,18), the ingested metabolizable energy was calculated as the sum of: 1) energy stored in the animal; 2) cost of accumulation of energy reserves (0.36 kJ to store 1 kJ of fat and 1.25 kJ to store 1 kJ of protein); and 3) energy required for maintenance (e.g., locomotion, thermoregulation, and metabolic events). The energy stored during refeding was calculated as the gain in lipid and protein mass multiplied by their energetic equivalent (39.3 and 18.0 kJ g⁻¹, respectively) (9). The negligible energy stored as carbohydrates (11,19) was not determined. Daily energy expenditure was daily energy intake minus daily energy storage (ES). Energy efficiency (%) was: stored energy/energy intake × 100 (20).

Results are presented as means ± SEM. Data were analyzed by using Sigmastat 3.0 software. Arcsin transformations were performed on percentages before statistical analysis. Nonparametric tests were used when normality or homoscedasticity failed. A Student’s t test was used to compare the ratio duration of fasting/duration of refeding. In each group of rats, lipid intake and storage was compared by using paired t tests (except in P3R3 rats where a nonparametric Wilcoxon’s test on rank was used). One-way ANOVA followed by Student-Newman-Keuls tests for multiple group comparisons were used to compare age, body mass, body mass loss, energy intake, whole body composition, liver composition, body component gains, organ masses (except for RP fat masses), energy expenditure and efficiency, and time of food deprivation between the different groups. Within groups, dm/mdt values and energy intakes were compared by the use of ANOVA for repeated measures. The relationship between body composition, time of food deprivation, time of refeding, and age was analyzed through regression lines. A nonparametric Kruskal-Wallis test followed by Dunn’s test for multiple group comparisons was used to compare plasma metabolites, triacylglycerol:total lipid mass ratio, water:protein ratio of the body mass gain (W:Pratio) or for body reserves (W:Pratio) and RP fat masses. Differences were considered significant at P < 0.05.

**Results**

**Nutritional status of starved rats.** Within 2 d of starvation, dm/mdt values declined by one-half (P < 0.001) and reached...
3.0 ± 0.1 g·100 g body mass$^{-1}$·d$^{-1}$ in P2 and P3 rats during P2. The day prior to refeeding, dm/mdt values of P3 rats increased by 45–50% ($P < 0.001$). NEFA concentration was 0.15 ± 0.03 mmol·L$^{-1}$ and β-hydroxybutyrate plasma concentration was 0.05 ± 0.01 mmol·L$^{-1}$ in control rats. They increased ($P < 0.01$) in P2 rats to 0.66 ± 0.08 mmol·L$^{-1}$ and 2.86 ± 0.30 mmol·L$^{-1}$ and decreased in P3 rats to 0.23 ± 0.06 and 0.46 ± 0.05 mmol·L$^{-1}$, respectively ($P < 0.01$). Protein and lipid accounted for 16 and 84% of the energy expenditure in P2 rats and for 52 and 48% during the last 2 d of starvation in P3 rats, respectively. The above results are similar to those previously published for rats in the same metabolic phases (9,19).

**Changes in body mass.** The rats recovered the body mass lost during starvation within 6.7 ± 0.3 d and 11.5 ± 0.4 d of refeeding in P2 and P3 rats, respectively. The duration of refeeding:duratio of starvation ratio in P3 refed rats (1.07 ± 0.05) was lower than in P2 rats (1.34 ± 0.06; $P < 0.003$).

**Energy intake.** Considering total energy intake, P3 refed rats consumed less total energy compared with P2 rats during the first 48 h ($P < 0.05$), but the specific energy intake (i.e. per unit body mass) did not differ during this period (Supplemental Fig. 1). In P2 rats, the specific energy intake remained constant during refeeding and was higher than in control-fed rats ($P < 0.05$). In P3 rats, it increased after 48 h of refeeding ($P < 0.025$). Maximum energy intake in P3 refed rats was higher than during the fed period ($P < 0.001$) and than in P2 refed rats ($P < 0.002$). P3RT rats were still hyperphagic at body mass recovery ($P < 0.01$; Supplemental Fig. 1).

**Energy expenditure and energy efficiency.** The energy expenditure for maintenance (EEM), ES, and the energy cost of storage in control rats accounted for 85 ± 2%, 8 ± 1%, and 7 ± 1% of the ingested metabolizable energy, respectively. EEM values did not differ between P2 refed and control rats. ES values in P2RT rats were only elevated compared with those of control and P2R3 rats ($P < 0.01$). In P3 refed rats, EEM values were lower than in control rats and P2 refed rats ($P < 0.05$) (Supplemental Fig. 2). In P3R7 and P3RT refed rats, ES was higher ($P < 0.05$) than in control rats. In P3RT rats, values were greater ($P < 0.05$) than in P3R3 and P2RT rats. Energy cost of storage was higher ($P < 0.05$) than in control rats for refeeding durations longer than 3 d (Supplemental Fig. 2). Energy efficiency values in P3 refed (25.4 ± 4.4 to 37.7 ± 4.1%) and P2RT rats (24.5 ± 4.6%) were higher than in control (8.4 ± 0.9%) and P2R3 rats (12.4 ± 2.9%) ($P < 0.01$). In P3RT rats, energy efficiency was 51% higher than in P2RT and P3R3 rats ($P < 0.02$).

**Overall changes in body fat, protein, and water masses during starvation and refeeding.** Body lipid mass in P3R0 rats was lower compared with P2R0 rats ($P < 0.05$; Table 1). The ratio of triacylglycerol:total lipid mass decreased during starvation ($P < 0.02$), a consequence of a slower net catabolism of structural lipids. At body mass recovery, total body fat remained lower in refed than in control rats ($P < 0.05$) as a result of a deficit in body triacylglycerol mass, structural lipids having been fully restored (Table 1).

These body components were recovered within 3 d of refeeding in P2 rats and at body mass recovery in P3 rats (Table 1). The hydration of the lean mass was 73.0 ± 0.1% in control rats, decreased in starved rats to 72.4 ± 0.1% in P2 rats and 70.8 ± 0.1% in P3 rats, ($P < 0.05$), and was restored on d 3 of refeeding in P2 and P3 refed rats.

**Rate of recovery of body components.** The daily gains in body and water masses during recovery were greatest during early refeeding and were significantly higher in P3 than in P2 rats (Supplemental Fig. 3). Water accounted for three-quarters of the body mass gain in R3 rats, but its fraction of weight gain was lower in the other groups of refed rats ($P < 0.05$). The W:Pgains (21.6 ± 2.5) in P3R3 rats was higher than in P2R3 rats (6.1 ± 0.5; $P < 0.001$). W:P preserves in R3 refed rats were higher than the W:Preserves in control rats (3.2 ± 0.1; $P < 0.001$). Later during refeeding, W:P gains were similar to W:P preserves in control rats.

The daily protein gain was lower in P3R3 rats than in P2R3 rats ($P < 0.05$), opposite of the daily body lipid gain (Supplemental Fig. 3). Later in the refeeding period, daily gains for lipid and protein tended to increase in P2 and P3 rats (Supplemental Fig. 3). Protein gains became higher in P3 rats than in P2-RT rats ($P < 0.05$; Supplemental Fig. 3), whereas lipid gains no longer differed. In control and P2 rats, lipid storage was not significantly greater than intake (Table 2). However, P3 refed rats had endogenous lipid production ($P < 0.05$; Table 2). In P2R3 rats, three-quarters of the stored lipids were structural, whereas they

| Table 1 | Net body mass, whole body water, protein, total lipid, triacylglycerol, and structural lipid masses in control fed, starved, and refed rats$^1$ |
|---|---|---|---|---|---|---|---|
| Group$^2$ | n | Net body mass$^3$ | Body water | Body proteins | Total body lipids | Triacylglycerols | Structural lipids |
| CONT | 10 | 402.8 ± 1.5$^a$ | 263.6 ± 1.9$^a$ | 83.2 ± 0.8$^{ab}$ | 41.8 ± 2.3$^a$ | 34.6 ± 2.2$^a$ | 7.2 ± 0.5$^a$ |
| P2R3 | 10 | 333.7 ± 1.4$^d$ | 225.5 ± 0.9$^d$ | 75.2 ± 0.5$^d$ | 22.2 ± 1.2$^d$ | 16.8 ± 2.2$^d$ | 5.3 ± 0.5$^d$ |
| P2R3 | 12 | 376.6 ± 1.9$^b$ | 258.4 ± 1.8$^b$ | 81.0 ± 0.8$^b$ | 23.4 ± 1.0$^b$ | 17.2 ± 1.2$^b$ | 6.2 ± 0.3$^b$ |
| P3RT | 10 | 405.2 ± 1.5$^b$ | 274.6 ± 1.5$^b$ | 85.1 ± 0.6$^b$ | 29.9 ± 1.4$^b$ | 23.0 ± 1.0$^b$ | 7.0 ± 0.6$^b$ |
| P3RT | 10 | 259.6 ± 2.8$^d$ | 179.7 ± 1.8$^d$ | 65.3 ± 1.1$^d$ | 5.7 ± 0.6$^d$ | 1.4 ± 0.3$^d$ | 4.3 ± 0.3$^d$ |
| P3RT | 11 | 316.6 ± 4.2$^b$ | 223.5 ± 2.9$^b$ | 67.8 ± 1.0$^b$ | 11.3 ± 1.0$^b$ | 6.8 ± 0.9$^b$ | 4.5 ± 0.2$^b$ |
| P3RT | 9 | 356.6 ± 3.8$^c$ | 245.5 ± 2.8$^c$ | 74.5 ± 1.3$^c$ | 21.3 ± 1.1$^c$ | 15.2 ± 0.9$^c$ | 6.1 ± 0.3$^c$ |
| P3RT | 10 | 406.5 ± 1.3$^a$ | 273.5 ± 1.8$^a$ | 84.3 ± 0.7$^a$ | 33.3 ± 1.3$^a$ | 26.9 ± 1.0$^a$ | 6.4 ± 0.9$^a$ |

$^1$ Values are means ± SEM. Means in a column without a common letter differ, $P < 0.05$.

$^2$ CONT: control fed rats.

$^3$ Net body mass: body mass minus digestive tract contents.
TABLE 2 Lipid intake and storage and endogenous lipid production in control fed and refed rats

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Lipid intake</th>
<th>Lipid storage</th>
<th>Endogenous lipid production</th>
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<td>CONT</td>
<td>10</td>
<td>6.1 ± 0.2*</td>
<td>2.2 ± 0.3*</td>
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<tr>
<td>P2R3</td>
<td>12</td>
<td>4.5 ± 0.1*</td>
<td>1.2 ± 1.0*</td>
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<tr>
<td>P2RT</td>
<td>10</td>
<td>5.4 ± 0.4</td>
<td>6.6 ± 0.7</td>
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<tr>
<td>P3R3</td>
<td>11</td>
<td>3.9 ± 0.2*</td>
<td>5.7 ± 0.7</td>
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<tr>
<td>P3RT</td>
<td>9</td>
<td>5.8 ± 0.4*</td>
<td>10.5 ± 0.3*</td>
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<tr>
<td>P3RT</td>
<td>10</td>
<td>6.8 ± 0.6*</td>
<td>12.0 ± 0.5*</td>
<td>5.2 ± 0.9</td>
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</table>

1 Values are means ± SEM. Means in a row without a common letter differ, P < 0.05.
2 Lipid intake and storage are the mass accumulated over the last 5 d in the control group and during the first 3 d, from d 4 to 7 and up to the end of the refeeding period in refed groups of rats.
3 Endogenous lipid production was calculated when the lipid storage was significantly higher than the intake.

Discussion

On a mass-specific basis, P3 refed rats were normophagous compared with the acclimation period and to P2 refed rats (7; this study). This does not support the concept of hypophagia during early refeeding after prolonged starvation (21). In fact, our results are consistent with the lipostatic theory (22) and with data obtained in restricted-refed rats (12) that relate the intensity of hypophagia to the extent of fat and lean mass depletion. Hypophagia that is abolished when the lost reserves are repleted (12) persists in P2 and P3 rats at body mass recovery. This is likely due to their deficit in lipid stores and the concomitant overshoot in the lipoprotein-lipase activity and food efficiency that occurs until fat cell size recovers to that of prestarvation (13,23). This high food intake is linked to a rapid restoration of digestive capabilities (24), liver composition, and rehydration of the body (this study), irrespective of the starvation state reached. Moreover, despite a low contribution of the liver to the overall recovery of body stores, high rates of deposition during early refeeding were found for lipid, protein, and water, expanding results previously obtained for protein in P2 refed rats (25) (Supplemental Table 2). At the whole body level, the W:Pred ratio in early refeeding is 2- to 7-fold higher than in growing rats of the same body mass (26). Particularly in P3R3 rats, which store limited amounts of protein, it makes sense that a large proportion of the water gained is more associated with tissue rehydration rather than with protein accretion. The digestive tract may play a large part in this process, because there is no need to rehydrate the muscular compartment.

In early refeeding, the restoration of lipid and protein stores followed different patterns depending on the phase of starvation reached. In P3 rats, recovery is not consistent with the concept of a deficit in body lipid accretion after total food deprivation (9,10, 13,20). In P2 rats, the daily lipid accretion was low and similar to the rate in fed rats, whereas in P3 rats, this was the case for protein. It is unlikely that a limitation in lipid or protein intake occurs in P2 or P3 rats, respectively, because intake was higher than storage. In starved nonobese animals, survival is limited by triacylglycerol availability (19). Furthermore, at the start of P3 of starvation, a lower threshold in the lipid level is attained (4,8). In rats of this body mass (9), the threshold triacylglycerol mass (7–8 g) is intermediate between the level in P3 and P2 rats. The metabolism of P3 rats seems then to be directed toward triacylglycerol storage to rapidly increase its mass above the lower threshold. This is achieved at the expense of an energetically costly endogenous lipid production (27), emphasizing the necessity to rapidly increase triacylglycerol stores above the threshold level. This is supported by self-choice diet experiments in which lipid intake is higher in P3 than in P2 rats in early refeeding (28). In contrast, the driving force to restore proteins is less obvious (28). Several hypotheses may explain such a metabolic orientation in P3 refed rats. First, the high rate of fat deposition in restricted refed rats is partly under corticosteroid control (29) and yet P2 and P3 of starvation are characterized by low and high corticosterone plasma concentrations, respectively (9,19). This hormonal pattern would favor fat deposition in P3 refed rats, in contrast to P2 refed rats. Second, hypometabolism during restricted feeding persists during refeeding, the saved energy being preferentially directed toward lipid storage (12,30). Accordingly, EEM was significantly lower in P3 than in P2 rats in early refeeding.

During energy restriction, fat pad and muscle mass decreases show different responses (13,14,19,20). Their restoration in early refeeding corresponded to some extent to their sensitivity to food deprivation (Supplemental Table 1). For example, P3 refed rats first stored lipids at the whole body level, but only the fat pad most sensitive to starvation increased in mass. The same was true for protein and muscle mass in P2 refed rats. However,
the extent of the fuel storage at the whole body level modulates the extent of restoration in individual tissues. When protein is low (P3 rats), muscle masses do not increase regardless of their sensitivity to starvation. The same applies in P2 rats for lipid storage and fat pad masses.

After the first days of refeeding, EEM differences between groups persisted, but the kinetics of body fuel restoration became more similar. However, when body protein was restored, a deficit in body fat remained that, for individual fat deposits, was independent of its previous response to starvation. In starved-refed rats, lipid restoration is slower than for protein regardless of the timing of refeeding (this study). Thus, as indicated by P-ratios, prolonged starved- and restricted-refed rats have different overall patterns of body reserve restoration (Supplemental Fig. 4). This occurs despite persistent hypometabolism in P3 refed rats.

Body reserve restoration occurs through a combination of hyperphagia and hypometabolism, the contribution of the former being the highest in P2 refed rats (17,31; this study). In P3 refed rats, the same situation occurs; the effect of hypometabolism alone could account for 1.4–2.0 times the actual recovery. Similarly, hyperphagia alone can account for 2.0–4.0 times the actual gain and appears to play the major role (approximately two-thirds of the gain) in the recovery of body reserves. Nevertheless, hypometabolism during refeeding prolongs the energy-saving processes established during starvation. Furthermore, during restricted refeeding, EEM may be adjusted (reduced up to 40–50%) to maintain body reserves at a constant level (17,18). Remarkably, the same occurs in refed P3 rats, because EEM values were similar in hyperphagic (400 g body mass) (this study) and restricted refed rats (250 g body mass) (17,18). This suggests that energy expenditure in such extreme situations is matched to the minimum required to maximize body reserve recovery or conservation, as is exemplified by the high food efficiency we found.

To conclude, despite the fact that P3 rats exhibited a more severe degradation of the digestive mucosa than P2 rats (24), were more dehydrated, starved for a longer duration, and reached threshold amounts of body fuels (this study), they are not disadvantaged during the restoration of body reserves. This provides additional evidence against the previous theory that P3 is a premortal state leading to a final, irreversible degeneration of the cells (7,9 for review). Instead, these findings support the concept of P3 being a highly adaptive metabolic situation and provide a physiological basis for the concept of the signal for refeeding (2,4,8) operating at the P2/P3 transition. However, our data demonstrate that the depletion of lipid stores below a lower threshold influences the restoration pattern of body reserves. Further studies should be carried out to determine the influence of the composition of the diet on the kinetics of the recovery of protein and lipid in animals refed in P2 or P3 of starvation.

Acknowledgments

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Literature Cited


ERRATUM


There was an error in the title. The correct title is below.

Restoration of Body Energy Reserves during Refeeding in Rats Is Dependent on both the Intensity of Energy Restriction and the Metabolic Status at the Onset of Refeeding.