Decrease in Fat Oxidation Following a Meal in Weight-Reduced Individuals: A Possible Mechanism for Weight Recidivism

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This study examined the effect that dietary-induced weight loss has on body composition, energy metabolism, and substrate oxidation at rest and during the 5-hour period following a meal. Twenty older (age: mean ± SE, 61 ± 1 years; range, 56 to 70 y) obese (body mass index > 32 kg/m^2) subjects (12 women, eight men) completed an 11-week dietary restriction program in which they lost 9 ± 1 kg. Fat and fat-free mass were reduced ($P < .05$) by 15% and 5%, respectively. Resting metabolic rate decreased by 15% ($P < .05$). Overall, weight loss did not alter the percentage of energy derived from fat sources (≈47% of energy) under resting conditions. In contrast, the percentage of calories derived from fat during the 5-hour postmeal period decreased from baseline to post–weight loss from 38% ± 3% to 26% ± 4% ($P < .05$) of total calories expended. The reduction in fat oxidation subsequent to a meal may facilitate fat storage, and may be one mechanism by which one regains weight following weight loss.

OBESITY is prevalent in modern society. Since obesity is associated with a number of maladies, including increased incidence of high blood pressure, coronary heart disease, and diabetes,[1] relatively effective strategies for inducing weight loss have been developed.[2] However, although it is relatively easy to induce weight loss through dietary restriction, long-term maintenance of reduced body weight is problematic. A recent National Institutes of Health Technology Assessment panel examined weight recidivism following diet-induced weight loss and concluded that "... there is a strong tendency to regain weight, with as much as two-thirds of the weight lost regained within one year of completing the program and almost all by five years."[2] Although the reasons for weight recidivism following weight loss are likely multifactorial, much attention has recently focused on physiological adaptations that often occur following diet-induced weight loss.

Two areas that have been extensively researched are resting metabolic rate[3] and the effect of a meal on postprandial metabolism.[4] A reduction in either resting metabolic rate or postprandial metabolism would facilitate weight regain by decreasing daily energy requirements. The findings for both of these potential weight recidivism mechanisms have been equivocal, with many reports of dietary restriction either having no effect or reducing the energy expenditure associated with these processes.[3,4]

Surprisingly little research has been published with regard to the relationship between weight recidivism and the pattern of substrate utilization, especially fat. Unlike carbohydrate-induced increases in carbohydrate oxidation, excessive dietary fat intake does not seem to promote fat oxidation.[5] Because of this, Flatt[6] has hypothesized that body weight may be driven by fat intake. In other words, body weight will increase or decrease until the daily average rate of fat oxidation is equal to that of fat intake. Recent studies support this concept in that a relationship seems to exist between rates of fat oxidation and levels of obesity, with higher rates of fat oxidation being associated with higher levels of obesity.[7-9] Likewise, Calles-Escandón and Driscoll[10] and Calles-Escandón et al[11] report that rates of lipolysis are directly related to body weight and fat-free mass, respectively. Schutz et al[5] further report that for each kilogram reduction in body weight, daily fat oxidation rates (as determined via resting metabolism) are reduced by approximately 2 g.

Thus, according to Flatt's hypothesis, to maintain a body weight at a level of 10 kg less than current weight, one must reduce dietary fat intake by 20 g/d or weight regain is inevitable. Furthermore, dietary restriction and subsequent weight loss is associated with a facilitated ability to store fat, in that lipoprotein lipase activity has been shown to be enhanced following weight loss and to remain elevated for extended periods.[12] Consistent with this, Raben et al[13] recently reported that fat oxidation during a 4.5-hour period following ingestion of a high-fat meal was lower in postobese subjects as compared with never-obese subjects. This suggests that fat storage following feeding for postobese subjects may be facilitated as compared with that for individuals who have never been obese.

Therefore, weight regain may be partially linked to fat metabolism, resting energy expenditure, and meal-induced thermogenesis. The purpose of this study is to examine the effects that diet-induced weight loss has on factors potentially related to weight recidivism. Specifically, we looked at how dietary restriction and subsequent weight loss affects overall energy metabolism and rates of substrate oxidation during rest and following a meal.

SUBJECTS AND METHODS

Overview

This study assessed changes that occur in physical characteristics and metabolism (at rest and following feeding) as a result of diet-induced weight loss. Following provision of informed consent (as approved by the University of Vermont Human Research Committee), 22 older (aged 61.0 ± 0.9 years) obese (body mass index > 32 kg/m^2) subjects (14 women, eight men) volunteered to participate in an 11-week weight loss program. Following baseline measurements, each subject attempted to restrict dietary intake to

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900 to 1,100 kcal/d for the next 11 weeks. For the next week, they followed a low-fat, weight-appropriate (ie, designed for weight maintenance) diet, at which time they were again retested. All testing took place while the subjects were residing at the University of Vermont Clinical Research Center. Twenty of 22 subjects (two women dropped out) completed the weight loss program and subsequent retesting. Listed below are the methods used in this study. Dependent measures were made when the subjects were presumably weight-stable (ie, baseline and 1 week following cessation of dietary restriction).

Subjects

To be eligible for the study, subjects had to be 55 to 70 years old, have a body mass index greater than 32 kg/m^2, have no history, symptoms, or signs of heart disease, be nondiabetic, have a resting blood pressure less than 160/90 mm Hg, be a nonsmoker, and have an absence of any prescription or over-the-counter drugs that could alter metabolism. Subjects were recruited through advertisements in a local newspaper. To the best of our knowledge, all subjects were of northern European descent.

Weight Loss Program

Subjects were requested to restrict their dietary intake to 900 to 1,100 kcal/d and to balance their intake of fats, carbohydrates, and proteins such that they represented approximately 25%, 60%, and 15% of total calories, respectively. During the weight loss phase of the project, the subjects met weekly in small groups with dietary counselors. The weekly sessions included monitoring of body mass and review of food diaries. Subjects received instruction in behavior-modification principles (such as behavioral therapy in which the subjects learned strategies for self-monitoring, stimulus control, problem-solving, social assertion, goal-setting, and relapse prevention) and nutrition education. The weight loss program was supervised by a registered dietitian (J.R.H.).

Inpatient Testing/Feeding Protocols

Subjects were admitted to the Clinical Research Unit on the afternoon before testing started. The following day (day 1), resting metabolic rate was measured shortly after the subjects had awoken and while they were still postabsorptive. During day 1, the subjects' body composition was assessed via underwater weighing, and selected skinfold thicknesses and circumferences were measured. On day 2, the subjects' resting metabolic rate was again measured just after awakening while in a postabsorptive state. Following this, subjects consumed a high-fat meal, and their metabolic responses were monitored for the next 5 hours. Subjects were then dismissed from the Clinical Research Center. The same protocol was followed during all testing periods. Subjects were sedentary during the time in which they resided in the Clinical Research Center.

On the night before day 1 and day 2, the subjects were fed a standard 700-kcal meal that contained 50%, 30%, and 20% of its energy as carbohydrate, fat, and protein, respectively. For the other meals on day 1, subjects were allowed to select from a menu the foods they wished to eat. The dietary composition of the meals offered was generally similar to that described above in the standard meal. However, we cannot state with certainty the composition of the diet for an individual subject, nor whether their food choices changed between phases of testing. Furthermore, subjects were not instructed as to what foods to eat for the days before admission for inpatient testing.

Resting Metabolic Rate

Oxygen uptake and carbon dioxide production were measured while the subjects reclined in a supine position underneath a ventilated hood. The measurements were made approximately 12 hours following a standard meal. Resting metabolic rate determinations were made on 2 successive days and are reported as the average of these two determinations. Energy expenditure was estimated via the Weir equation.[14] With this procedure, urinary nitrogen excretion is used to estimate oxygen uptake and carbon dioxide production associated with protein metabolism, as well as the energy expenditure associated with these processes. These values are then subtracted from the overall oxygen and carbon dioxide values. The nonprotein respiratory exchange ratio (rate of CO_2 production divided by rate of O_2 consumption) is then calculated. Using the respiratory exchange ratio, one can determine both the percentage of energy derived from fat and carbohydrate sources and the energy value associated with a given quantity of oxygen. The intraclass correlation and the coefficient of variation for determination of energy expenditure in our laboratory have been shown to be .90 and 4.3%, respectively.[15]

Postprandial Metabolism

The effect of a high-fat meal (45% carbohydrate, 40% fat, and 15% protein) on resting metabolism was studied in each subject. Upon awakening, the resting metabolic rate of the subject was measured as described earlier. The subject then quickly ingested a liquid meal (15 kcal/kg baseline fat-free mass). The metabolism of the subject was then monitored for the next 5 hours. The subject spent the first 60 minutes under the ventilated hood and then alternated 30-minute periods of resting supine outside of and underneath the ventilated hood. The first 10 minutes of each measurement session were excluded (to allow for reequilibration), and the average was then taken of the remaining samples. Thus, the first hour represents the mean of 50 1-minute samples, and each of the following 4 hours is the mean of 20 1-minute samples. Energy expenditure was determined as described for resting metabolic rate. The thermic effect of the meal was the amount by which energy expenditure was increased above rest during the 5-hour postprandial period. The size of the high-fat meal was consistent across all measurement periods.

Anthropometric Measures

Body density was determined using underwater weighing with simultaneous correction for residual volume (helium dilution). Percent fat was than estimated using the Siri equation.[16] Tricep, suprailiac, abdomen, thigh, and subscapular skinfold thicknesses were measured in triplicate as described by Lohman et al.[17] Waist, hip, umbilicus, proximal thigh, and midthigh circumferences were measured in triplicate as also described by Lohman et al.[17] The waist to hip ratio represents the waist girth divided by the hip girth.

Statistical Analysis

Men and women were initially compared using a two-way (gender × time) ANOVA with repeated measures. When no statistically significant gender effects were detected, a paired t test was used to compare baseline and post–weight loss scores.[18] Means were reported as statistically different from each other when P values were .05 or less. All scores are reported as the mean ± SE.

RESULTS

Table 1 lists physical characteristics of the subjects at baseline and following the dietary intervention. The mean weight loss among subjects was 9 ± 1 kg ($P < .05$), with 29% ($P < .05$) and 71% ($P < .05$) of the weight loss representing fat-free and fat mass, respectively. There were also statistically significant ($P < .05$) reductions in most

Table 1. Physical Characteristics of the Subjects (mean ± SE)

Variable	Baseline (n = 20)	Post–Weight Loss (n = 20)
Weight (kg)	95.2 ± 3.2	86.2 ± 3.0*
Percent fat (%)	44.7 ± 1.5	42.0 ± 1.9*
Fat-free mass (kg)	52.6 ± 2.7	50.0 ± 2.5*
Fat mass (kg)	42.6 ± 1.8	36.2 ± 1.8*
SF (mm)		
Subscapular	34.0 ± 1.8	29.4 ± 1.6*
Tricep	36.2 ± 2.1	28.6 ± 2.3*
Suprailiac	43.9 ± 2.2	35.0 ± 7.1*
Thigh	44.2 ± 2.9	37.1 ± 3.6*
Abdomen	51.5 ± 2.1	47.8 ± 1.9
Sum of 5	209.8 ± 6.9	177.9 ± 8.9*
Waist to hip ratio	0.88 ± 0.02	0.87 ± 0.02
Waist girth (cm)	103.5 ± 2.7	95.4 ± 2.4*
Hip girth (cm)	117.6 ± 2.1	109.7 ± 2.1*
Umbilicus girth (cm)	112.4 ± 2.7	105.5 ± 2.7*
Proximal thigh girth (cm)	69.6 ± 2.4	63.9 ± 1.6*
Midthigh girth (cm)	59.1 ± 1.7	54.8 ± 0.8*

Abbreviation: SF, skinfold thickness.
*$P \leq .05$.

fat-related variables (ie, selected skinfold thicknesses and girths). For example, the sum of the five measured skinfold thicknesses decreased by approximately 32 mm (15%). Notably, the waist to hip ratio did not change from baseline to post–weight loss, suggesting that waist and hip girths were both reduced by similar percentages.

Table 2 reports how dietary restriction affects variables related to resting metabolism. The weight loss of 9 ± 1 kg was accompanied by a 15% decrease ($P < .05$) in overall resting energy expenditure. When expressed on a daily (ie, 24-hour) basis, this represents a 260-kcal decrease in daily metabolism. The decrease in resting metabolism is greater than one would predict from the weight loss alone, since energy expenditures per kilogram body mass (−5%) and per kilogram fat-free mass (−8%) were also significantly ($P < .05$) reduced. When one examines the substrate mix used to provide energy at rest, statistically significant ($P < .05$) reductions in the rates of fat oxidation were found (−18%), with carbohydrate- and protein-usage rates being slightly lower but not statistically different from

Table 2. Resting Metabolic Rate Determinations (mean ± SE)

Variable	Baseline	Post–Weight Loss
Total		
kcal/24 h	1,789.8 ± 80.2	1,528 ± 55.3*
kcal/24 h/kg BM	18.7 ± 0.04	17.7 ± 0.04*
kcal/24 h/kg FFM	33.8 ± 0.7	31.0 ± 0.9*
kcal/min	1.25 ± 0.06	1.06 ± 0.04*
Fat (kcal/min)	0.60 ± 0.05	0.49 ± 0.04*
Carbohydrate (kcal/min)	0.47 ± 0.04	0.41 ± 0.03
Protein (kcal/min)	0.18 ± 0.01	0.16 ± 0.01
Nonprotein RER	0.83 ± 0.01	0.84 ± 0.01
Percent kcal as fat (%)	47.2 ± 2.9	46.1 ± 2.9

Abbreviations: RER, respiratory exchange ratio calculated after the contributions of protein metabolism to oxygen uptake and carbon dioxide production had been removed; BM, body mass; FFM, fat-free mass.
*$P \leq .05$.

baseline values. Overall, the percentage of energy expenditure derived from fat sources at baseline and post–weight loss remained stable at about 47% of total energy expenditure (ie, fat, protein, and carbohydrate oxidation decreased similarly).

The individual relationships between weight loss and change in resting metabolism are shown in Fig 1. A statistically significant correlation ($P < .05$) was found between reductions in body weight and resting metabolism.

Metabolism during the 5-hour postprandial period obtained at baseline and post–weight loss is shown in Table 3. Weight loss did not alter the thermic effect (ie, increase in metabolism above resting) of a meal, with the thermic effect representing 6% to 7% of ingested calories. Total energy expenditure was reduced ($P < .05$) by 11%. Fat oxidation rates were reduced ($P < .05$) by 40%, and this represents a shift in substrate utilization, since the percentage of kilocalories from fat sources decreased from 38% ± 3% to 26% ± 4% of total kilocalories expended ($P < .05$). Thus, during the 5-hour period following the meal, post–weight loss fatty acid oxidation rates were substantially less than those found during baseline.

Figure 2 illustrates the effect of a high-fat meal on resting rates of fat metabolism at baseline and following weight loss. At baseline, postprandial rates of fat oxidation are not significantly reduced ($P > .05$) compared with those found at rest ($P < .05$). In contrast, although resting rates of fat oxidation are lower post–weight loss as compared with baseline, postprandial fat oxidation is depressed substantially ($P < .05$) compared with that present at rest. In addition, post–weight loss postprandial depression of fat metabolism (ie, rest *v* postmeal period) is significantly different ($P < .05$) from that found at baseline.

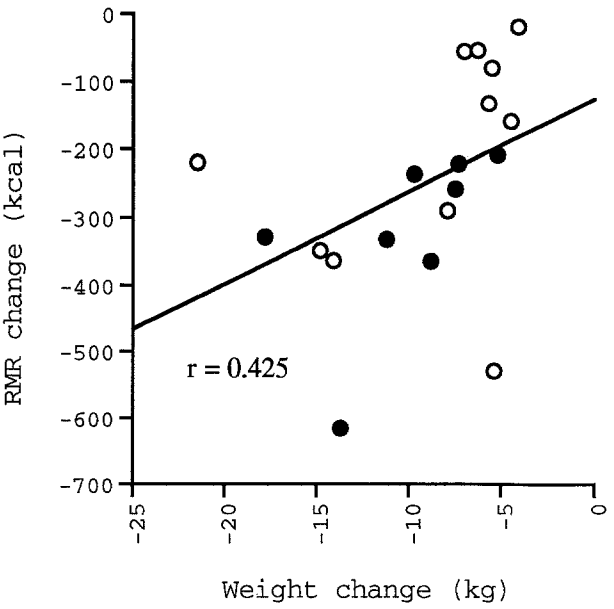

Fig 1. Relationship between change in body weight and resting metabolic rate (RMR). The decrease in body weight is associated with a concomitant decrease in RMR. (●) Men; (○) women. Regression line is calculated using the combined male and female data set.

Table 3. Postprandial Metabolism During the 5-Hour Period Following Ingestion of a High-Fat Meal (mean ± SE)

Variable	Baseline	Post–Weight Loss
Thermic effect (kcal/300 min)	49.6 ± 7.8	54.0 ± 5.6
Total metabolism (kcal/min)	1.40 ± 0.06	1.24 ± 0.05*
Fat (kcal/min)	0.54 ± 0.05	0.32 ± 0.05*
Carbohydrate (kcal/min)	0.69 ± 0.04	0.78 ± 0.06
Protein (kcal/min)	0.17 ± 0.02	0.15 ± 0.01
Percent kcal as fat (%)	37.5 ± 2.7	25.6 ± 3.7*
Nonprotein RER	0.87 ± 0.01	0.92 ± 0.01*
Meal size (kcal)	798.8 ± 38.1	798.8 ± 38.1

Abbreviation: RER, respiratory exchange ratio after subtracting oxygen and carbon dioxide contributions of protein metabolism. Per-minute energy and substrate oxidation values represent means of the 5-hour measurement period. Thermic effect is the difference between postprandial and resting energy expenditure, or the total amount that metabolism was increased above rest during the 5-hour period.

*$P \leq .05$.

DISCUSSION

Weight maintenance requires that one be in energy balance (ie, intake equals expenditure). Recent research also suggests that weight maintenance requires maintaining fat balance (ie, grams of fat intake equal expenditure) as well, or body weight will ultimately increase or decrease until fat balance (and energy balance) occurs.[6] Support for the fat-balance hypothesis of weight maintenance exists in a number of areas. For example, excess intake of fat does not stimulate higher rates of fatty acid oxidation,[9] but excess intake of carbohydrate does,[19] suggesting that stores of carbohydrate are actively regulated but stores of fat are not. Schutz et al[5] have also reported that rates of postabsorptive resting fatty acid oxidation are directly related to fat mass ($r = .56$), and that for every kilogram that body weight is reduced, daily fat oxidation decreases by 2 g (which is the same as that found here).

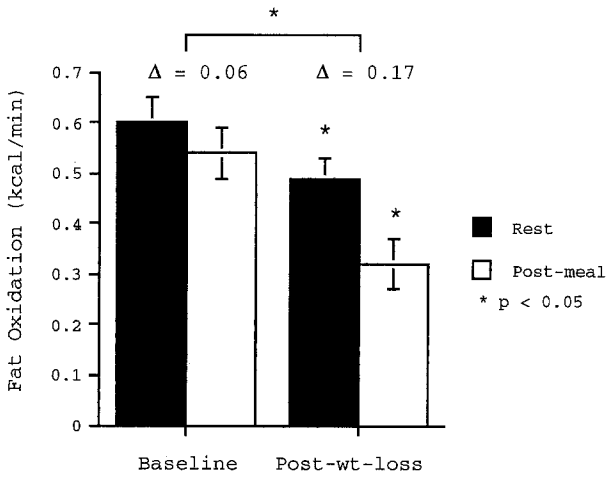

Fig 2. Fat energy expenditure immediately before (ie, from resting measurements) and during the 5-hour postprandial period following ingestion of a standard high-fat meal. Resting and postprandial fat energy expenditures represent a mean of 30 and 300 1-minute samples, respectively. A high-fat meal results in a depression of the rate at which fat is oxidized (compared with rest). Weight loss exaggerates this response. Δ, the difference between mean resting and postprandial fat energy expenditures found at either baseline or post–weight loss. Results are the mean ± SE.

Weight recidivism can then be examined in the context of the energy-balance and fat-balance hypotheses. Certainly, if one only ingests calories equal to those expended, weight maintenance is obligatory. Thus, body weight can always be controlled by regulating intake. However, there are physiological adjustments on the expenditure side of the energy- and fat-balance equations that can make weight maintenance more problematic. For example, resting metabolic rate is consistently reduced with diet-induced weight loss, and this is associated with the reduction in metabolic mass.[3] Less well-understood is the extent to which dietary restriction reduces resting metabolic rate by more than can be accounted for by body weight loss alone, as was found in this study. Keesey[20] has suggested that the type of obesity (ie, hyperplastic or hypertropic) may affect the response of resting metabolism to weight loss. Regardless, for this study, resting energy requirements of the subjects decreased by approximately 260 kcal/d, suggesting that intake must be reduced by this amount to maintain reduced body weight. Resting fat oxidation also decreased, and represents a decrease in fat oxidation of approximately 18 g when expressed on a 24-hour basis. However, this may be misleading, since most of one's day is not spent under conditions similar to those imposed when resting metabolism was measured.

The effects of a meal on total energy expenditure and rates of fatty acid oxidation are illustrative of this. The weight loss–induced decreases in the mean rates of energy expenditure following a meal (−0.16 kcal/min) and at rest (−0.19 kcal/min) are similar. This is not surprising, since resting metabolic rate represents a major portion of the postmeal energy expenditure and the thermic effect of a meal did not change. However, the effect of a meal on substrate oxidation is different post–weight loss. Before weight loss, the meal results in a decrease of the resting rate of fatty acid oxidation ($P > .05$) from 0.60 ± 0.05 kcal/min (Table 2) to 0.54 ± 0.05 kcal/min (Table 3). After weight loss, ingestion of a meal reduces resting rates of fatty acid oxidation significantly ($P < .05$) from 0.49 ± 0.04 kcal/min to 0.32 ± 0.05 kcal/min. In addition, the magnitude of the meal-induced reduction in fat oxidation was larger post–weight loss compared with baseline ($P < .05$). If one assumes that there were 15 hours under meal-fed conditions and 9 hours under resting conditions, fat oxidation rates for subjects in this study decreased from a baseline value of 90 g/d to 61 g/d post–weight loss.

Raben et al[13] recently completed a study similar to this one in which they compared resting and postprandial energy and substrate expenditure between postobese weight-stable and never-obese younger (38-year-old) women. The two groups' current body weights were similar. Resting energy expenditure and percentage of energy derived from fat, protein, and carbohydrate sources did not differ between groups. As in this study, ingestion of a high-fat (45%) meal resulted in a reduction of fat oxidation as compared with rest. The depression in fat oxidation in the postobese group over the 4.5-hour measurement period was more

than twice as great as that which occurred in the never-obese group. Consistent with this, rates of carbohydrate oxidation were significantly higher for the obese group, and the two groups did not differ with respect to either total energy expended during the 4.5-hour postmeal period or meal-induced elevation of energy expenditure. The weight loss–induced reduction (Δpost–weight loss minus Δbaseline) in postprandial fat oxidation found in this study (−0.11 kcal/min) was similar to that found by Raben et al[13] between postobese and never-obese individuals of the same weight (−0.95 kcal/min).

The postprandial decrease in fatty acid oxidation that occurred following weight loss could potentially affect maintenance of body weight. Whenever one overeats fat, the likelihood that it will be stored rather than oxidized is enhanced. As long as one remains in energy balance, weight recidivism will not occur, but the scales seem tipped in favor of weight regain. Schutz[19] presents data in a recent report that demonstrate the effect that overeating can have on substrate utilization. He measured day-long (24-hour) rates of substrate oxidation on 3 consecutive days in which obese subjects ingested 500, 4,380 to 4,860, and 500 kcal/d, respectively. During the overfeeding period, the respiratory exchange ratio increased from 0.79 to 0.90 as compared with the previous (500-kcal) day, reducing rates of fat oxidation and increasing carbohydrate utilization. Day-long energy expenditure also increased by 19%. On the subsequent day when the subjects were again underfed (500 kcal), rates of energy expenditure returned to the rates found during the initial underfeeding period. However, the day-long respiratory exchange ratio remained elevated as compared with day 1, indicating reduced rates of fatty acid oxidation.

Segal et al[21] examined the effect of meal-feeding on substrate oxidation in men with three levels of fatness. They noted that feeding increased respiratory exchange ratios from approximately 0.82 (60% from fat) during fasting conditions to a 3-hour average of 0.88 (40% from fat), with no differences in the responses of the three different groups to a meal. The change in respiratory exchange ratio with feeding is similar to that found in this study. Collectively (ie, this study, Raben et al,[13] and Segal et al[21]), these data suggest that when body weight (or fat) is reduced to levels less than those an organism finds desirable, fat storage is facilitated.

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