



Loss of total body potassium during rapid weight loss does not depend on the decrease of potassium concentration in muscles. Different methods to evaluate body composition during a low energy diet

M Krotkiewski^{1*}, K Landin², D Mellström³ and J Tölli⁴

¹Department of Rehabilitation Medicine, Sahlgrenska University Hospital, Göteborg; ²Department of Endocrinology, Sahlgrenska University Hospital, Göteborg; ³Department of Geriatrics, Sahlgrenska University Hospital, Göteborg; ⁴Department of Radiation Physics, Sahlgrenska University Hospital, Göteborg, Sweden

OBJECTIVE: The aim of the study was to elucidate whether combustion of skeletal muscle glycogen during a very low calorie diet (VLCD) was associated with decreased muscle potassium content. A comparison between different methods was also performed to evaluate body composition during a VLCD and a low calorie diet (LCD).

DESIGN: Dietary treatment of obese women by VLCD and LCD. Measurements after 1 and 2 weeks of VLCD and 6 months of LCD.

SUBJECTS: Fifteen perimenopausal obese women aged 46.5 ± 1.3 y and 15 of 48.0 ± 0.7 y of age.

MEASUREMENTS: Skeletal muscle biopsies under local anaesthesia. Body composition measurements by means of dual-energy X-ray absorptiometry (DEXA), and measurements of total body potassium (^{40}K) and total body nitrogen (TBN). Measurements of electrolytes and glycogen concentration in muscle samples.

RESULTS: In the first study (1 week of VLCD) skeletal muscle glycogen decreased ($P < 0.01$), but muscle potassium increased ($P < 0.01$). Muscle sodium decreased ($P < 0.01$), while muscle magnesium was unaltered. Body weight decreased by 2.9 ± 0.5 kg and ^{40}K decreased. Fat-free mass (FFM) calculated from ^{40}K and DEXA decreased by 2.7 vs 1.9 kg ($P < 0.001$). Body fat measured with DEXA decreased by 1.1 kg ($P < 0.01$), but not body fat calculated from ^{40}K . TBN decreased by 0.03 ± 0.01 kg ($P < 0.05$) and FFM calculated from TBN by 2.9 ± 0.5 kg ($P < 0.002$). In the second study, 6 months on the LCD resulted in 17.0 ± 2.0 kg weight reduction and this was mainly due to reduced body fat, 14.0 ± 2.0 kg measured with DEXA and from ^{40}K ($P < 0.001$). The decrease in FFM was slight.

CONCLUSION: One week of VLCD resulted in muscle glycogen depletion but increased muscle potassium content in spite of decreased total body potassium. FFM contributed to the main part of body weight loss during short periods of severe energy restriction, but remained unchanged during long-term dietary treatment. Body fat became mostly responsible for the body weight loss during long-term LCD. Calculations of changes of FFM from ^{40}K and TBN seem to overestimate the FFM decrease associated with short-term VLCD.

International Journal of Obesity (2000) 24, 101–107

Keywords: skeletal muscle potassium; skeletal muscle glycogen; body composition; weight reduction

Introduction

Thirty years ago Forbes and Lewis published their results concerning total body potassium (^{40}K) on four male cadavers.¹ Chemical analysis showed potassium concentration in human bodies to be 66.5, 66.6, 72.8 and 66.8—on average 68.0 mmol per kilogram fat-free mass (FFM). During the subsequent 30 y, numerous studies used the average content of 68 mEq K per kg as a base for extrapolation used for further calculation of total body potassium and FFM, and by subtraction from body weight for estimation of body fat.

This so-called two-compartment model has been widely used when referring to body composition. This model and the technique of whole-body counting of ^{40}K to calculate the total body potassium has also been used to follow changes in body composition during low (LCD) and very low calorie diets (VLCD).

Our own experience with these types of calculation showed disproportionately high and rapid decrease of FFM even after a short period of energy reduction.^{2,3} It was also remarkable that a decrease of FFM during VLCD was found to be unusually high in comparison with other methods used for estimation of body composition. As proposed by Kreitzman *et al*,⁴ the natural explanation for this phenomenon could be glycogen depletion already in the earliest phase of any low or very low calorie diet. The authors suggested that potassium released from glycogen distorts estimates of body composition during dieting.⁴

*Correspondence: M Krotkiewski, Department of Rehabilitation Medicine, Sahlgrenska University Hospital, Guldhedsgatan 19 III tr, 413 45 Göteborg, Sweden. E-mail: mpab@algonet.se.
Received 9 November 1998; revised 5 May 1999; accepted 27 July 1999

The aim of the present study was to elucidate whether combustion of muscle glycogen during VLCD is associated with a decrease of potassium concentration in muscle tissue. Another question addressed by us when designing the study was associated with finding a possible explanation for the observed discrepancies between the different methods of measuring body composition during VLCD and during long-term LCD treatment.

Methods

Subjects and study design

Fifteen middle-aged perimenopausal (age 45.1 ± 1.8 y), obese (body mass index, BMI 32.5 ± 1.1 kg/m²) women (Table 1) were subjected to a weight reduction programme for 1 week. Patients received a VLCD by means of formula diet preparation enriched with medium chain triglycerides (MCT; Swedish Formula Diet, 'Extra Diet', NovoVital, Vimmerby, Sweden). All patients daily consumed three meals of 30 g (108 kcal) dissolved in 2.5 dl skim milk (87.5 kcal) with a total daily intake of 586.5 kcal. The protein consumed per day was 28.5 g, fat 9.9 g and carbohydrates 30.3 g. The Swedish Formula Diet powder was fortified with vitamins, minerals and dietary fibre (guar gum, xanthan gum and oat bran fibres, 16 g per day). Medium chain triglycerides consisted of copyric (C₈) and capric (C₁₀) acids, 9.02 g per 100 g of formula diet. Body composition was determined and skeletal muscle biopsies were performed before and after the week.

A second group of 15 obese female patients, mean age 48.0 ± 0.7 y, were randomly selected to participate in a longer VLCD study. These patients consumed the same type of VLCD as in the 1 week study (Swedish Formula Diet, NovoVital, Vimmerby, Sweden) for 2 weeks. After the termination of VLCD the patients were allocated to dietary group treatment with group meetings every second week, and were followed up by one physician (MK) for 6 months. Dietary treatment consisted of a low-calorie, low-fat, 1200 kcal diet (LCD). All patients received phosphates (Fosfatin, MultiPharma Sweden AB, Gothenburg Sweden) in order to diminish the risk of osteopenia. Body composition was evaluated after termination of the 2 weeks and the 6 months, respectively.

The study was approved by the ethical committee at Sahlgrenska University Hospital and all patients gave their formal written consent.

Skeletal muscle glycogen and electrolytes

Skeletal muscle biopsies were taken under local anaesthesia with alligator forceps from the middle lateral portion of the right vastus muscle on two different occasions. The first biopsy was taken after 2 weeks of verified weight stability on habitual diet.

The second biopsy was taken after 1 week of VLCD treatment—as a rule in the same line, but somewhat higher, above the end of the cicatrix from the first incision to avoid inflamed tissue at the site of the first biopsy. No complications occurred.

Two muscle specimens were taken on each occasion. The specimens were frozen immediately in liquid nitrogen for later determination of muscle glycogen concentration and electrolytes.

The muscle sample of 10–20 mg wet weight was frozen rapidly in liquid nitrogen and kept at -80°C until determination of the skeletal muscle electrolytes. All analyses were performed on the same occasion. Sodium and potassium contents were determined with a digital flame photometer (IL 543, Scandia Metric, Gothenburg Sweden) and expressed in $\mu\text{mol/g}$ wet weight. The calculations were carried out on mean values of duplicate samples. The within-assay coefficient of variation was 4% for potassium and 8% for sodium. The reproducibility is in accordance with earlier studies.^{2,3,5} Muscle glycogen concentration was determined according to Kasrllsson and Saltin.⁶

Anthropometric and body composition measurements

Anthropometry. Height was recorded without shoes to the nearest cm and body weight in underwear to the nearest 0.1 kg. Body mass index (BMI) was calculated as body weight divided by height squared (kg/m²). Waist circumference was measured at the lowest girth between the chest cage and umbilicus and the hip circumference at the largest firth around the hips in a standing position. The ratio of these two girths (WHR) was then calculated.

Total body potassium. Total body potassium was measured by counting the gamma radiation from the naturally occurring radionuclide ⁴⁰K in a whole body counter, with consideration taken for a shielding factor associated with body size. Fat-free mass (FFM) was obtained according to Forbes' formula.¹ The method had a high precision, and corrected for body fat quenching.^{2,3} Body fat (BF) was estimated as a value obtained by subtraction of FFM from body weight.

Total body nitrogen (TBN). The TBN was measured by *in vivo* neutron activation analysis of nitrogen. In this work we used the spontaneous fission neutrons from a ²⁵²Cf source. The activity of the source was at the time 7.4 GBq. The method is based on capture of thermal neutrons by nitrogen nuclei in a ¹⁴N(n, γ)¹⁵N-reaction. The 10.8 MeV γ-rays emitted in the reaction are characteristic for nitrogen and were detected by two large size NaI(Tl)-detectors. The relation between TBN and total body protein (TBP) was expressed as $\text{TBP} = 6.25 (\text{TBN})$.

The accuracy in a single TBN measurement is about 4% and the equivalent radiation dose was less than 0.4 mSv if a quality factor of 20 was used for the neutrons. The FFM was also calculated from the relation between TBN and FFM as described by Tölli *et al.*⁷

$$\text{TBN/FFM} = 32.7 \pm 1.8 \text{ g/kg (females)}$$

$$\text{TBN/FFM} = 32.9 \pm 2.9 \text{ g/kg (males)}$$

Dual energy X-ray absorptiometry (DEXA). DEXA (Lunar DPX, Scanexport Medical, Helsingborg, Sweden) was used to measure the bone mineral content and body composition. The body fat, FFM, total bone mineral density (BMD) and total bone calcium content were assessed. The error (1 s.d.) for total body BMD was < 0.01 g/cm², for fat 1.4%, fat mass 1.0 kg and FFM 0.8 kg. These results correspond to a relative error of 0.8% for total body BMD and 1.5% for FFM. There is a limitation to the estimation of body composition by DEXA. A sensitivity to the anterior/posterior thickness of the body a pixel that contains a small amount of bone may fail to be counted as bone. This appears as 'very clean' tissue because the pixel average absorption coefficient is closer to that of lean tissue than that of bone. The FFM calculated from DEXA measurements is denoted FFM DEXA.⁸

Statistical methods

All variables presented in the tables and figures are expressed as means \pm s.e.m. and were calculated by means of the Stat-View McIntosh statistical programme. For comparison within groups the Wilcoxon test for paired comparisons was used. For comparisons between groups, the Mann-Whitney test was used. For comparisons between more than two groups of variables one-way ANOVA or ANOVA with repeated measures was used. Correlations were performed using simple regression analysis and $P < 0.05$ was chosen as the level of significance.

Results

Results of 1 week on the VLCD

One week on VLCD resulted in a decrease of 2.9 ± 0.5 kg ($P < 0.001$) body weight and BMI

Table 1 Anthropometric data before and after 1 week of a VLCD in 15 women; means \pm s.e.m.

	Before	After	P-level
Age (y)	46.5 \pm 1.8		
Height (cm)	168.5 \pm 1.71		
Weight (kg)	91.9 \pm 2.81	89.0 \pm 2.77	$P < 0.001$
Body mass index (kg/m ²)	32.5 \pm 1.07	31.4 \pm 1.05	$P < 0.001$
Waist (cm)	102.6 \pm 2.82	99.0 \pm 2.64	$P < 0.01$
Hip (cm)	116.9 \pm 2.44	114.5 \pm 2.20	$P < 0.01$
Waist/hip ratio	0.88 \pm 0.01	0.86 \pm 0.01	n.s.

($P < 0.001$) (Table 1). Both waist and hip circumferences decreased ($P < 0.01$), while the WHR remained unchanged (Table 1).

The concentration of potassium in the lateral portion of the vastus muscle increased ($P < 0.01$), while the concentration of sodium decreased ($P < 0.01$). Skeletal muscle magnesium was unaltered (Figure 1). The concentration of glycogen in the muscle tissue decreased by nearly 40% during 1 week of the VLCD ($P < 0.001$; Figure 1).

Total body potassium decreased by 187 mmol after 1 week of the VLCD (from 3434 ± 97 mmol to 3247 ± 89 mmol, $P < 0.001$; Table 2). Body fat, calculated from ⁴⁰K measurements and Forbes' formula, decreased by 0.03 ± 0.6 kg (ns) and FFM decreased by 2.7 ± 0.5 kg ($P < 0.001$; Table 2, Figure 2). Calculated from DEXA measurement, body fat decreased by 1.1 ± 0.2 kg ($P < 0.01$) and FFM 1.9 ± 0.3 kg ($P < 0.001$; Table 2, Figure 2).

The mean loss of TBN was 0.122 ± 0.015 kg ($P < 0.05$) corresponding to 0.763 ± 0.09 kg of the total protein loss, assuming that the ratio of protein to nitrogen is 6.25. The loss of protein ranged from 0.131 to 1.242 kg. FFM was also calculated from TBN, where it was assumed that nitrogen was present in the FFM at a concentration of 32.7 g/kg FFM. The FFM loss calculated from TBN was -2.89 ± 0.5 kg ($P < 0.001$). The mean loss of TBN per kg of body weight loss was 0.015 ± 0.001 kg, while body fat was unchanged (Table 2, Figure 2).

Total body protein, calculated from an assumption of 1 g nitrogen per 6.25 g of protein, was calculated as 10.7 ± 0.3 kg before and 10.5 ± 0.3 kg after with a difference of -0.20 ± 0.1 kg ($P < 0.05$). As the loss of TBN corresponded to 30 g, losses of protein corresponded to 187.5 g. Protein constitutes 25% of body cell mass, this would correspond to a loss of 1 kg of FFM. One gramme of nitrogen corresponds to 3 mmol of potassium, so 30 g nitrogen loss corresponds to a 90 mmol decrease of potassium. Out of the measured 187 mmol decrease of potassium, nearly 100 mmol was therefore not associated with a decrease of protein and could possibly account for losses of potassium bound to liver and muscle glycogen.

The mean TBN loss in relation to body weight reduction by each subject was 7.5 ± 0.9 g nitrogen per kg of body weight loss.

BMD decreased after 1 week of VLCD ($P < 0.05$), while total bone calcium content was unchanged (Table 2).

Results of 2 weeks of the VLCD and 6 months of the LCD

Six months of LCD treatment resulted in $17.0 \pm$ kg weight reduction ($P < 0.001$) and 5.6 ± 0.8 kg/m² decrease in body mass index ($P < 0.001$; Figure 3). In contrast to VLCD, most of the weight loss depended on reduction of body fat. In fact, the 6

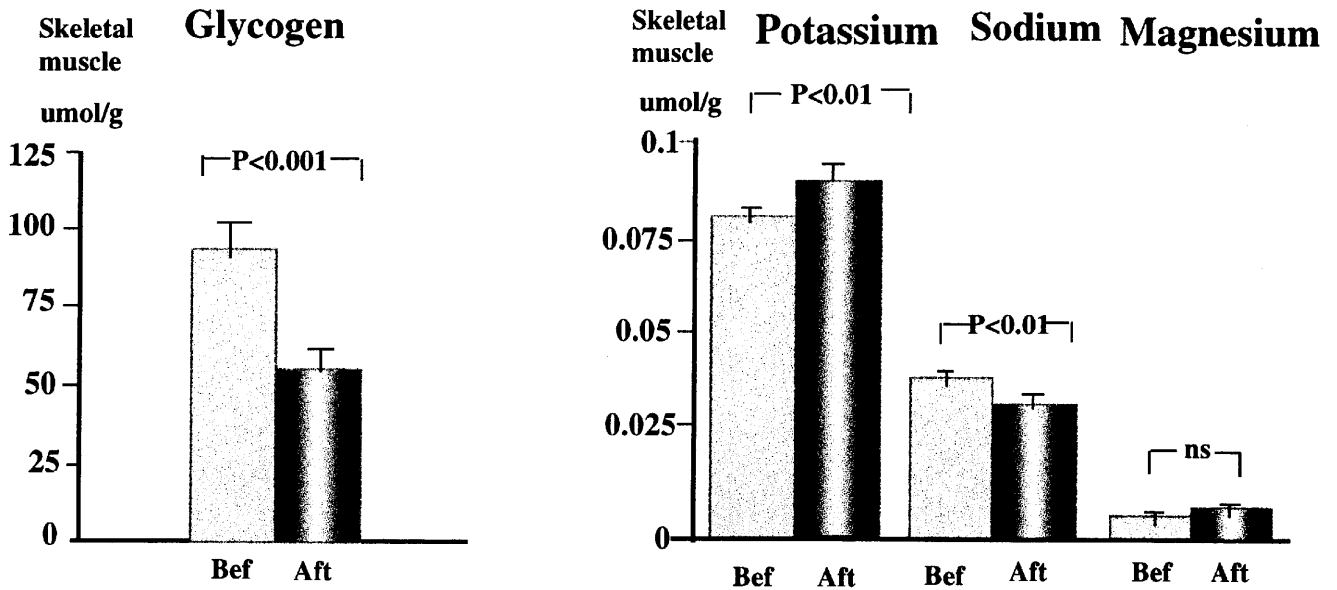


Figure 1 Effects of 1 week of a VLCD on skeletal muscle glycogen and electrolyte content in 15 women; means \pm s.e.m. Bef = before, Aft = after the treatment period.

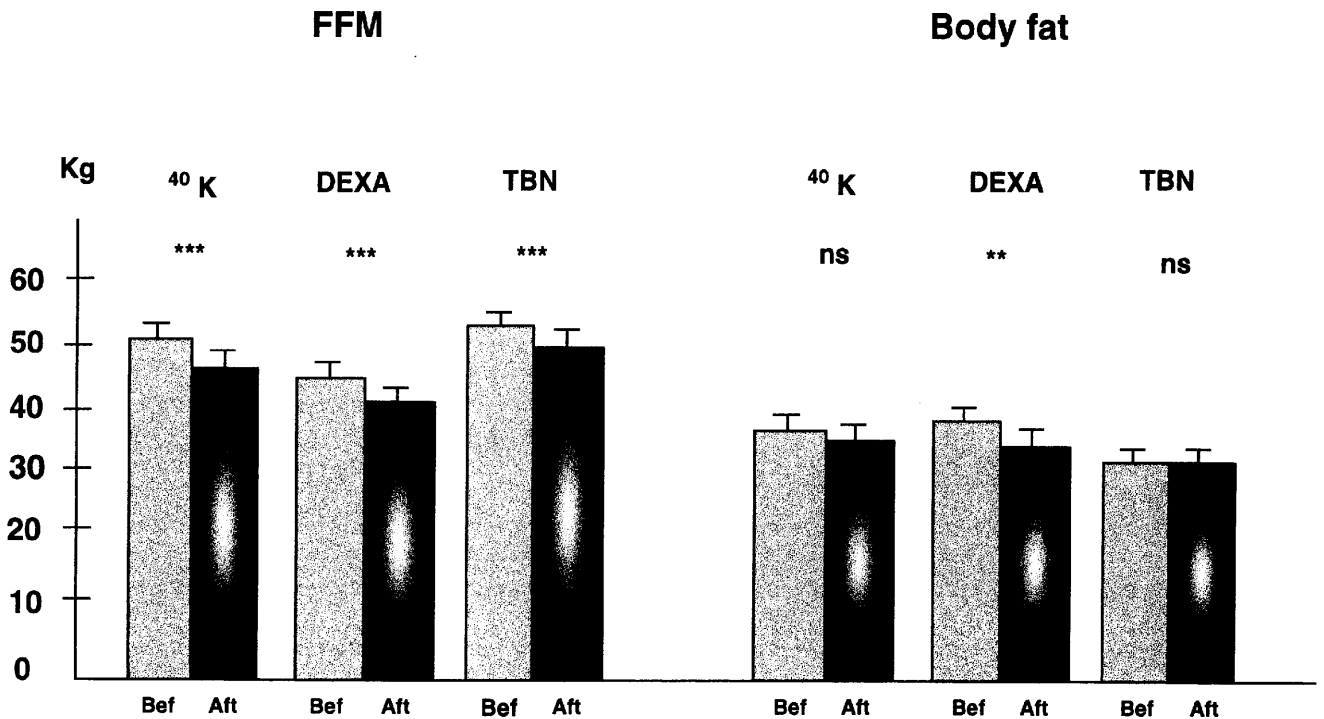


Figure 2 Effects of 1 week of a VLCD on fat-free mass (FFM) and body fat calculated from total body potassium (^{40}K), dual energy X-ray absorptiometry (DEXA) and total body nitrogen (TBN) in 15 women; means \pm s.e.m. Body fat is calculated by subtraction of FFM (measured with ^{40}K) from body weight. For details see Methods section. ** $P < 0.01$, *** $P < 0.001$. Bef = before, Aft = after the treatment period.

months of LCD did not contribute to further decrease of FFM (Figure 3).

The FFM loss calculated from ^{40}K was 3.6 kg after 2 weeks ($P < 0.001$) and only 2.7 kg after 6 months in comparison with baseline ($P < 0.01$). The same was true for FFM measured by DEXA where FFM decreased only 2.4 kg after 2 weeks of the VLCD ($P < 0.001$) and 1.9 kg after 6 months of the LCD compared with the initial FFM before the diet ($P < 0.01$; Figure 3).

The changes in body fat followed the pattern of changes for FFM. Thus, there was only a small change after 2 weeks of VLCD with a mean decrease of 1.7 ± 1.5 kg ($P < 0.001$) when calculated with Forbes' formula using ^{40}K measurements, and a mean decrease of 2.4 ± 1.2 kg ($P < 0.001$) when measured with DEXA (Figure 3). Body fat decreased markedly after six months of LCD, 14.2 ± 1.8 kg ($P < 0.001$) calculated from ^{40}K , and a similar amount when measured with DEXA, 14.0 ± 1.9 kg ($P < 0.001$; Figure 3).

Table 2 Body fat, fat-free mass (FFM), total body potassium, nitrogen, protein, bone mineral density and total bone calcium before and after 1 week of VLCD in 15 women; means \pm s.e.m.

	Before	After	P-level
Body fat, ⁴⁰ K (kg)	41.5 \pm 2.5	41.3 \pm 2.2	n.s.
Body fat, DEXA (kg)	42.3 \pm 2.1	41.2 \pm 2.1	<i>P</i> < 0.01
FFM, ⁴⁰ K (kg)	50.4 \pm 1.4	47.7 \pm 1.3	<i>P</i> < 0.001
FFM, DEXA (kg)	46.0 \pm 1.0	44.1 \pm 1.1	<i>P</i> < 0.001
FFM (TBN \times 33.1)	53.1 \pm 1.5	50.2 \pm 1.4	<i>P</i> < 0.001
Total body potassium (mmol)	3434 \pm 96.8	3247 \pm 89.0	<i>P</i> < 0.001
Total body potassium (g/kg)	1.47 \pm 0.05	1.43 \pm 0.04	<i>P</i> < 0.05
Total body nitrogen (kg)	1.71 \pm 0.05	1.68 \pm 0.05	<i>P</i> < 0.05
Total body protein (kg)	10.7 \pm 0.31	10.5 \pm 0.31	<i>P</i> < 0.05
Bone mineral density (g/cm ²)	1.23 \pm 0.01	1.21 \pm 0.01	<i>P</i> < 0.01
Total bone calcium (g)	1054 \pm 21.3	1037 \pm 23.4	n.s.

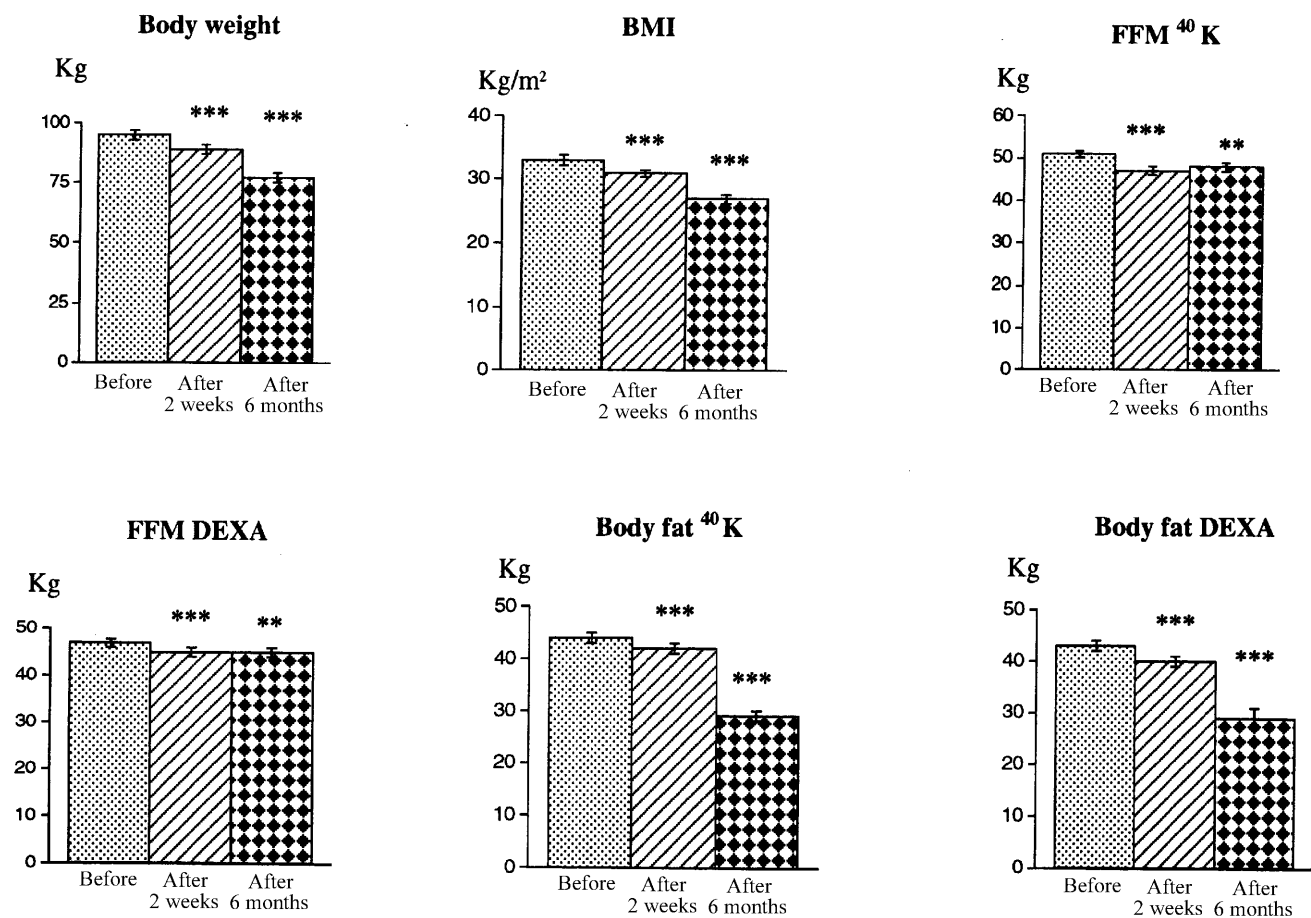


Figure 3 Effects of 2 weeks of a VLCD and 6 months of a LCD on body composition in 15 women, BMI = body mass index; FFM = fat-free mass; ⁴⁰K = total body potassium; means \pm s.e.m. ** *P* < 0.01, ****P* < 0.001.

BMD decreased after 2 weeks of VLCD (from 1.22 \pm 0.01 to 1.21 \pm 0.01 g/cm², *P* < 0.05), but total bone calcium was unaltered as compared with initial levels before the diet (not shown).

Discussion

The results of the present study show that the concentration of muscle potassium increased in spite of decreased concentrations of glycogen in the muscle

tissue. The increase of muscle potassium concentration was associated with a concomitant decrease of muscle sodium and magnesium. This is in accordance with earlier studies after weight reduction showing increased muscle potassium and decreased glycogen content in spite of decreased total body potassium.^{9,10}

No conclusive explanation for this phenomenon is readily to hand, but natriuresis associated with the ketogenic diet of the type applied in the study can theoretically induce an outflow of intracellular sodium and even bivalent ions like magnesium with the secondary inflow of potassium from the extracellular space.

It was assumed by Kreitzman *et al*⁴ that a VLCD was associated with depletion of glycogen and in the earliest phase was associated with a secondary depletion of potassium, which could distort estimates of body composition during dieting. Potassium in muscles is bound to glycogen in the ratio of 0.45 mmol of potassium to each gram of glycogen, and a decrease of glycogen following fasting during VLCD should be associated with a decrease of potassium concentration in muscles.

As reported previously by Bergström,¹¹ muscle glycogen is already seriously depleted after 1 week of a VLCD. The expected losses of potassium based on the observed depletion of glycogen could be calculated to account for a loss of ca. 98.5 mmol potassium. This corresponds to a loss of nearly 50% of the normal glycogen content.

Since considerable potassium is bound to hydrated glycogen (0.45 mmol/g glycogen), the loss of 187 mmol of potassium would correspond the loss of nearly 400 g glycogen, that is close to the total amount of glycogen stored in the body. Calculating the glycogen losses from the losses of TBK, Kreitzman *et al*⁴ found that some of the patients lost even more than 1 kg of glycogen. Obviously potassium remains in the muscles in an unbound form in spite of relative glycogen depletion. Furthermore, it is known and confirmed in our study that the ketogenic diet induces the increase of sodium urinary excretion without an increase of urinary excretion of potassium. The increase of muscle potassium concentration is even more unexpected in view of the concomitant decrease of total protein. It is possible to speculate about a decrease of intracellular water, but this explanation for increased concentration of potassium must simultaneously assume a changed activity of the Na⁺/K⁺ transport across the cellular membrane as the concentration of sodium increases.

Landin *et al* reported previously on decreased skeletal muscle potassium in obesity.¹² The skeletal muscle Na/K ratio tended to be higher in obese men but was not related to body weight in women. Skeletal muscle magnesium was higher in obese men than in lean women. The authors interpreted their findings as being compatible with a disturbance of the N/K pump activity across the muscle cell membrane. One possible explanation of the findings in the present study could be changed availability of intracellular ATP and/or insulin sensitivity, both having a known influence on Na/K pump activity.¹²

The present study showed a 2.9 kg decrease in body weight but only 1 kg FFM loss after 1 week when calculated from changes in TBN. Nearly 2 kg FFM were lost when calculated from DEXA and nearly 3 kg when using different calculations based on results of ⁴⁰K measurement by whole body counting.

Similar differences were observed after 2 weeks of VLCD. Again, the mean loss of FFM calculated from ⁴⁰K was close to 4 kg, while only 2.4 kg loss of FFM was found by DEXA measurement.

The results of the present study confirm the previous observations regarding the discrepancies between different methods of measuring body composition during body weight reduction.^{13–18} Some of the methods used in this study belong to the two-compartment methods (body fat and FFM). However, it was emphasized previously¹⁹ that the composition of the FFM (water, minerals, protein and glycogen) is not constant.

The rapid decrease of FFM was higher than the decrease of body fat after 1 week of VLCD. This contradicts the concept of a protein-sparing effect of ketone bodies (augmented by the supplementation by MCT) and/or free fatty acids, which reach the peak values during the first few days of fasting.^{20,21}

On the basis of the above observations it seems reasonable to assume that ⁴⁰K measurements after short periods of rapid weight reduction are grossly overestimating the losses of FFM.

The results of the present study seem to indicate that the estimations of body composition are likely to have some methodological errors influencing the estimates of body composition during the first few weeks of rapid weight reduction with a VLCD.

FFM loss after 1 week of VLCD constituted 94% of the total weight loss when calculated according to Forbes' formula (from ⁴⁰K), 66% when calculated from DEXA measurements and 99% when calculated from TBN. After 2 weeks of VLCD the corresponding numbers were 68% for ⁴⁰K and 46% for DEXA, and after 6 months of LCD 25% for ⁴⁰K and 12% for DEXA. Thus it seems plausible to assume that the ⁴⁰K and TBN measurement-based calculations tended to overestimate the FFM losses associated with short-term rapid weight losses.

Apart from methodological differences the present findings indicate that a positive nitrogen balance which increases successively during ongoing energy reduction is associated with consecutively increasing contribution of body fat to the total body weight loss.

This phenomenon may be associated with a well known metabolic adaptation dependent on successive change from glycolysis to lipolysis and increased oxidation of fat. After the first week nitrogen balance becomes less negative and the contribution of FFM to the total body weight loss decreases. Nevertheless short-term VLCD is inevitably associated with a proportionally higher loss of FFM than those observed during the LCD.

At the baseline, there was a significant correlation between DEXA and ⁴⁰K measurements. The choice between these two methods at base-line may not be so important as when trying to evaluate the changes of body composition related to reduction of body weight. DEXA seems to be the most adequate method and ⁴⁰K and TBN overestimate losses of FFM after short-term calorie deprivation. The same applies for the bioimpedance method with a tendency to overestimate FFM after weight loss.¹⁷ Results of the present study indicate anyway that DEXA is the best method to evaluate changes in body composition during short- and long-term dietary treatment.

Beside the decrease in soft tissue, BMD also decreased on a VLCD. Whether this is secondary to reduced body size (BMD is calculated in relation to body mass) or a true decrease in bone area is not known. Total bone calcium content, however, was not affected by the diet.

The formula diet used in the dietary treatment in both groups of patients contained MCT. MCT are metabolized in a different way from long chain triglycerides (LCT). Their oxidation rate is higher and calorie value lower than LCT.²² Furthermore, MCT are more ketogenic than LCT and are therefore responsible for a specific anorectic and protein-sparing effect.^{22,23}

In conclusion, 1 week of VLCD resulted in muscle glycogen depletion but increased muscle potassium content in spite of decreased total body potassium. FFM contributed to the main part of body weight loss during short periods of severe energy restriction, but remained unchanged during long-term dietary treatment. Body fat became mostly responsible for the body weight loss during a long-term LCD. Calculations of changes of FFM from ⁴⁰K and TBN seem to overestimate the FFM decrease associated with short-term VLCD.

Acknowledgement

The statistical and organizational help from Carl-Johan Tham is gratefully acknowledged.

References

- 1 Forbes GB, Gallup J, Hirsch JB. Estimation of total body fat from potassium-40 content. *Science* 1961; **133**: 101–103.
- 2 Krotkiewski M, Toss L, Björntorp P, Holm G. The effect of a low calorie diet with and without chronic exercise on thyroid and sex hormones, plasma proteins, oxygen uptake, insulin and C-peptide concentrations in obese women. *Int J Obes* 1981; **5**: 281–293.
- 3 Krotkiewski M, Grimby G, Holm G, Szczepanik J. Increased muscle dynamic endurance associated with weight reduction on a very low calorie diet. *Am J Clin Nutr* 1990; **51**: 321–330.
- 4 Kreitzman S, Coxon AY, Szaz KF. Glycogen storage, illusions of easy weight loss, excessive weight regain and distortions in estimates of body composition. *Am J Clin Nutr* 1992; **56**: 292–335.
- 5 Landin K, Petruson B, Jakobsson K-E, Bengtsson B-Å. Skeletal muscle sodium and potassium changes after successful surgery in acromegaly: Relation to body composition, blood glucose, plasma insulin and blood pressure. *Acta Endocrinol* 1993; **128**: 418–422.
- 6 Karlsson J, Saltin B. Diet, muscle glycogen and endurance performance. *J Appl Physiol* 1971; **31**: 203–206.
- 7 Tölli J, Bengtsson BA, Bosaeus I, Johannsson G. A comparison of different methods to measure body composition in patients. *Appl Radiat Isot* 1998; **49**(5–6): 469–472.
- 8 Lukaski HC. Methods for the assessment of human body composition, traditional and new. *Am J Clin Nutr* 1987; **46**: 537–556.
- 9 Allenberg K, Nilsson M, Landin K, Lindgärde F. Glycogen and lactate synthetic pathways in human skeletal muscle in relation to obesity, weight reduction and physical training. *Eur J Clin Invest* 1988; **18**: 250–255.
- 10 Landin K, Lindgärde F, Saltin B. Skeletal muscle potassium increases after diet and weight reduction in obese subjects with normal and impaired glucose tolerance. *Acta Endocrinol* 1989; **121**: 21–26.
- 11 Bergström J. Muscle electrolytes in man. *Scand J Clin Lab Invest* 1962; **14**(Suppl 68): 1–110.
- 12 Landin K, Lindgärde F, Saltin B, Wilhelmsen L. Decreased skeletal muscle potassium in obesity. *Acta Med Scand* 1988; **223**: 507–513.
- 13 Apfelbaum M, Ficker J, Apfelbaum LJ. Low and very low calorie diets. *Am J Clin Nutr* 1987; **45**: 1126–1134.
- 14 Wadden TA, Van Itale TB, Blackburn GL. Responsible and irresponsible use of very low calorie diets in the treatment of obesity. *JAMA* 1990; **263**: 83–85.
- 15 Barrous K, Snook JT. Effect of high protein, very low calorie diet on body composition and anthropometric parameters of obese middle-aged women. *Am J Clin Nutr* 1987; **45**: 381–388.
- 16 Deurenberg P, Westrate JA, Hautvast J. Changes in fat free mass during weight loss measured by bioelectrical impedance and by densitometry. *Am J Clin Nutr* 1989; **49**: 33–36.
- 17 Deurenberg P, Westrate JA, Van der Kooy K. Body composition changes assessed by bioelectrical impedance measurements. *Am J Clin Nutr* 1988; **49**: 401–403.
- 18 Ryde JS, Saunders NH, Birks JL et al. The effect of VLCD on body composition. In Kreitzman SN, Howard AN (eds). *Smith Gordon: London*, 1993; pp 31–54.
- 19 Kreitzman SN, Howard AN (eds). *The Swansea trial*. Smith Gordon: London, 1993, pp 15–24.
- 20 Pairan GLS, Semple SJG. Effect of 3-hydroxybutyrate in obese subjects on very low energy diets. *Proc Nutr Soc* 1980; **39**(2): 1–48A.
- 21 Sherwin RS, Hendler RG, Felig PL. Effect of ketone infusion on amino-acid and nitrogen metabolism in men. *J Clin Invest* 1975; **55**: 1382–1390.
- 22 Bach AC, Babayan VK. Medium chain triglycerides, an update. *Am J Clin Nutr* 1982; **63**: 950–962.