

# Glycogen storage: illusions of easy weight loss, excessive weight regain, and distortions in estimates of body composition<sup>1-3</sup>

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**ABSTRACT** Glycogen is stored in the liver, muscles, and fat cells in hydrated form (three to four parts water) associated with potassium (0.45 mmol K/g glycogen). Total body potassium (TBK) changes early in very-low-calorie diets (VLCDs) primarily reflect glycogen storage. Potassium released from glycogen can distort estimates of body composition during dieting. TBK changes due to glycogen mobilization were measured in 11 subjects after 4 d dieting with a VLCD. The influence of water-laden glycogen on weight fluctuations during the dieting process, the exaggerated regain if carbohydrate loading occurs, and the implications for weight control programs and overestimation of nitrogen losses with dieting are discussed. *Am J Clin Nutr* 1992;56:292S-3S.

**KEY WORDS** Very-low-calorie diet, VLCD, glycogen, total body potassium, fat-free mass, recidivism, dieting

## Introduction

The utilization of glycogen stores during weight reduction can have a significant effect on the apparent weight lost and the degree of recidivism after a period of dieting. The energy deficit required to reduce weight with glycogen as fuel (3400 kJ/kg) (1800 kcal/lb) is far less than fat (32 217 kJ/kg) (3500 kcal/lb) due to both the difference in energy contributions of fat versus carbohydrate and, even more, the large amounts of water associated with storage glycogen. Small energy deficits, therefore, early in a weight-reduction diet, can produce the illusion of significant fat lost. The ease with which this weight will be regained can be disheartening to a dieter. The apparent ease of early weight losses can also lead to unrealistic expectations of the ability of a modest energy restriction or even exercise to achieve significant weight losses. Quantitative estimation of glycogen storage would provide insight into early weight loss and rapid recidivism upon eating after a diet.

Glycogen is associated with potassium in the ratio of 0.45 mmol K to each gram of glycogen and glycogen is necessarily depleted in the earliest phases of any diet. The depletion of potassium during the first days of a ketogenic very-low-calorie diet (VLCD) should primarily reflect the labile glycogen.

## Methods

The study protocol was accepted by the ethical committees of Cambridge University, Swansea University, and Howard Foundation Research. All subjects gave informed consent.

Eleven female subjects of varying body mass indexes were placed on an identical, standardized 6694-kJ (1600-kcal/d) food program for 11 days before an extensive weight-reduction trial. The maintenance diet contained 201.7 g carbohydrate, 59.3 g fat, and 67.8 g protein. Four days before starting the 1700-kJ (405-kcal) VLCD (Cambridge Diet, Cambridge Nutrition Ltd, Norwich, UK), total body potassium (TBK) was measured in each subject using the whole-body counter at Addenbrookes Hospital, Cambridge, UK. TBK was again assessed at the same facility on the fifth day of the Cambridge Diet and at the end of the 10-week VLCD program.

Glycogen amount was calculated by taking the difference in TBK between prediet and day 5 of VLCD, expressed as mmol K. Because 0.45 mmol K represents 1 g glycogen, total glycogen loss is represented by mmol TBK/0.45.

## Results

Results are shown in **Tables 1 and 2**. Potassium changes during the first 4 d of VLCD of 180 mmol compare with only 104 mmol lost in the subsequent 10 weeks of dieting. It is assumed that the vast majority of the potassium lost in these initial days of dieting reflects primarily that bound up with glycogen. The estimate of  $\approx 400$  g stored glycogen matches other estimates of glycogen storage in healthy people.

## Discussion

Because considerable potassium is bound up with the hydrated glycogen (0.45 mmol K/g glycogen) (1), the loss of 400 g glycogen

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will result in the release of > 200 mmol K. Studies of body composition, using K40, assume that each kilogram of fat-free mass (FFM) is associated with 60 mmol potassium. The utilization of glycogen stores could therefore be misinterpreted as a loss of  $\approx$  3.5 kg nitrogen-rich FFM.


The mean glycogen losses by this subject group of 400 g are reasonable. This should not obscure the considerable intersubject variability, which in one subject reached 1 kg glycogen. Glycogen losses or gains are reported (2) to be associated with an additional three to four parts water, so that as much as 5 kg weight change might not be associated with any fat loss. As glycogen stores are readily replenished after conclusion of any weight-loss program, it is necessary to account for these losses before comparing effectiveness of weight-loss methods, before assessing recidivism, and certainly before criticizing dieters for lack of postdiet control. Bergstrom et al (3) demonstrated that muscle glycogen can be depleted to about one-third normal with low-carbohydrate diet for 3 d. After a high-carbohydrate diet the glycogen stored rose sixfold higher, to more than twice normal. This excessive glycogen repletion, with associated water, could follow inappropriate

TABLE 2  
Individual differences in estimated glycogen storage and initial weight change after 4 d of the VLCD

Subject	Weight on day of first TBK	Weight after 4 d VLCD	Weight loss after 4 d VLCD	Estimated glycogen storage
	kg	kg	kg	g
1	87.6	85.6	2.0	333
2	90.4	86.0	4.4	426
3	89.8	82.4	7.4	520
4	75.2	71.8	3.4	333
5	108.2	105.2	3.0	520
6	65.6	62.5	3.1	373
7	84.4	80.2	4.2	1066
8	107.6	100.9	6.7	80
9	78.8	75.4	3.4	146
10	83.0	80.7	2.3	333
11	104.0	98.0	6.0	240
12	95.4	89.7	5.7	613
$\bar{x} \pm$ SD	89.2 $\pm$ 13.1	84.8 $\pm$ 12.3	4.3 $\pm$ 1.8	415 $\pm$ 256

TABLE 1  
Average total body potassium (TBK) and estimated glycogen storage values of dieting subjects before the very-low-calorie diet (VLCD), after 4 d of the VLCD, and after 10 wk of the VLCD

TBK	
Before diet	3300 mmol
On day 5 of diet	3120 mmol
Lost after 4 d diet	180 mmol
Lost between days 5 and 70	104 mmol
Estimated glycogen storage	400 g

refeeding after dieting, leading to rapid and excessive nonfat weight regain. 

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