

## BIOCHEMICAL CONSEQUENCES OF THE PRITIKIN DIET IN CAUCASIAN WOMEN

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There is on-going interest in the ability of diets very low in fat and high in dietary fibre to minimise, if not reverse, the process of atherosclerosis, especially in so far as coronary heart disease is concerned. The Pritikin diet is the most renowned of such dietary approaches (Pritikin 1982). However there are some questions as to whether the high fibre content of the diet interfere with mineral nutrition and whether the low fat content of the diet interferes with fat soluble vitamin nutrition or plasma lipids. We have addressed these issues in the present study.

The study involved 10 women, who ate a Pritikin diet, and 20 women, who ate their normal diet, as the control group. For each subject, fasting blood was collected for blood lipid, minerals, and vitamin A and E analyse. All subjects completed a 14-day dietary record, and a food frequency questionnaire the day after the blood test. Anthropometric studies included height, weight, body mass index (BMI), waist circumference, hip circumference, waist to hip ratio (WHR), skin fold thickness of triceps, biceps, subscapular and supra-iliac, and body fat by impedance. Differences between groups have been assessed by students t-test

We have made comparisons of metabolic variables and fat soluble vitamin status in women adhering to the Pritikin diet with a control group of Anglo-Celtic Melbourne women. The women studied were middle aged (Pritikin  $54 \pm 3.2$  years; Control  $47 \pm 1.3$  years), were not obese (BMI: Pritikin  $24.4 \pm 0.9$ ; Control  $24.9 \pm 0.9$ ), and WHR was within acceptable limits (WHR: Pritikin  $0.83 \pm 0.01$ ; Control  $0.79 \pm 0.01$ ). The aim of a Pritikin diet is usually for the fat intake to be below 10% of energy intake. A mean of 16.6% was achieved by this group of women. The dietary fibre intake was 3-fold that of the control group, and the sodium intake was about 75% of control.

As far as the consequences of this dietary pattern for lipid status were concerned, plasma total cholesterol concentrations and LDL cholesterol concentrations, along with apo B concentrations, were less than those in the control. The HDL cholesterol concentrations and apo A1 concentrations were lower, and the LDL to HDL and apo B to Apo A1 were not different (Table). Whilst plasma concentrations of calcium, phosphorus, potassium, and sodium are not necessarily indicative of dietary intakes, it is interesting to observe that the plasma potassium was actually lower in the Pritikin adherents than in their non-Pritikin counterparts. Of particular interest were the low serum retinol concentrations in the Pritikin adherents, whilst alpha-tocopherol concentrations were not different (Table). However homeostasis of serum retinol is usually achieved in the face of a varied vitamin A intake, unless this is at very low intakes, so that here as in other studies, and may simply reflect the low fat intakes (Rabuco et al. 1991).

Since the Pritikin adherents had a higher overall carotenoid intake than their controls, provided that the bioavailability of these carotenoids is not compromised by the low fat diet it might be expected that, where a low LDL concentration is achieved, the carotenoid protection of this lipoprotein in the Pritikin adherents may exceed that in their non-Pritikin counterparts. More work is required to resolve this question, but the failure to achieve improvements in the relationships between apo B and apo A1, may not be as disappointing for Pritikin adherents as appears at first sight. Whether or not lower serum retinol concentrations disadvantage those on a Pritikin diet remains an open question.

Subject	n	TC mmol/L	HDL-C mmol/L	LDL-C mmol/L	L:HDL-C	TRIG mmol/L	APO A1 g/L	APO B g/L
Pritikin	10	5.0±0.4*	1.37±0.17	3.2±0.4*	2.66±0.46	0.99±0.14	1.18±0.11***	0.77±0.10**
Control	20	6.2±0.2	1.52±0.07	4.2±0.2	2.89±0.25	1.17±0.13	1.76±0.05	1.09±0.06

  

Subject	n	APO B:A1 mmol/L	Ca mmol/L	P mmol/L	K mmol/l	Na mmol/L	Retinol µmol/L	a-toc µmol/L
Pritikin	10	0.66±0.07	2.42±0.04	1.05±0.05	4.1±0.1***	141.3±1.0	1.59±0.15**	28.4±2.9
Control	20	0.63±0.05	2.39±0.09	1.17±0.04	5.1±0.1	140.0±0.4	2.57±0.18	26.4±0.8

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$

PRITIKIN, N. (1982). *Preventive Medicine* 11: 733.

RABUCO, L.B., RUTISHAUSER, I.H.E. and WAHLQVIST, M.L. (1991). *Ecol. Food and Nutrition* 26: 97.