

A NEW DIETARY PARADIGM?

Low-carbohydrate and high-fat intake can manage obesity and associated conditions: Occasional survey

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This study analyses 127 communications from individuals self-reporting their weight change following adoption of a low-carbohydrate, high-fat (LCHF) eating plan. Total combined self-reported weight loss was 1 900 kg (range 5 kg gain to 84 kg loss). The mean \pm standard deviation weight loss of 15 (\pm 12) kg is among the largest yet described. Sixteen subjects reported the LCHF 'cured' (i.e. medications no longer required) one or more of their medical conditions, most commonly type 2 diabetes mellitus (T2DM) ($n=14$), hypertension ($n=8$) and hypercholesterolaemia ($n=7$). Another 9 subjects with either type 1 diabetes mellitus or T2DM reduced medications as did 7 patients with hypertension; 8 no longer suffered from irritable bowel syndrome. These data show that significant and rapid weight loss is possible on an unsupervised eating plan that severely restricts daily carbohydrate intake to approximately <75 g/day. Better weight loss on a carbohydrate-restricted LCHF eating plan than on an iso-caloric high-carbohydrate, low-fat (HCLF) diet is well described in the literature, probably due to a paradoxical reduction of hunger by carbohydrate restriction. A randomised controlled clinical trial is urgently required to disprove the hypothesis that the LCHF eating plan can reverse cases of T2DM, metabolic syndrome and hypertension without pharmacotherapy.

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Since William Banting self-published his *Letter on Corpulence*^[1] in 1862, describing how William Harvey^[2] had cured Banting's life-long obesity through the avoidance of starchy foods, there has been interest in the low-carbohydrate, high-fat (LCHF) diet for the treatment of obesity.

It was promoted by William Osler in the United States in 1892 as *the* treatment for obesity^[3] and by Vilhjalmur Stefansson who had lived on an exclusive fat/protein diet for more than a decade while living with the Inuit.^[4,5] Stefansson's experience inspired a long lineage of LCHF diets in the US. The popularly maligned Atkins diet (1972)^[6] is one of the more recent examples.

The 'hormonal' or 'insulin' theory of obesity predicts that the carbohydrate content of the diet must be an important regulator of body mass since 'a functioning pancreas is essential for the fattening process'.^[7] This theory, originally promoted by German-speaking scientists, also noted that fat accumulated in specific 'lipophilic' areas of the body, more readily in the obese than in the lean.^[8] Germany's defeat in World War II with the adoption of English as the universal scientific language, buried this theory until recently.^[9-11] Instead, the energy balance model^[12] which posits that obesity is due simply to the ingestion of too many calories regardless of macronutrient composition, became the favoured explanation.^[9-11]

Following a description of my successful adoption of the LCHF diet,^[13] I received correspondence from individuals self-reporting their experiences with this eating plan, popularly known in South Africa (SA) as the 'Noakes diet'.

These data have significant limitations; they are self-reported and few have been substantiated so they lack the scientific certainty of a prospective randomised controlled clinical trial (RCT). Nevertheless, they are of value. First, successful weight loss was achieved without expensive medical or other support. Second, subjects lost weight by following a diet that is the opposite of that currently promoted for

health, weight loss and the management of type 2 diabetes mellitus (T2DM) in SA.^[14] Third, these data suggest that the LCHF has the potential to 'cure' some individuals with morbid obesity, T2DM, hypertension or metabolic syndrome. These findings invite disproof from an appropriately designed RCT.

I received 173 unsolicited communications from individuals self-reporting their experience following adoption of the LCHF eating plan^[15] that reduces total daily carbohydrate intake to below ~ 75 g/day or lower if required. This analysis is restricted to the 127 communications reporting exact weight losses. Five cases are presented in this paper.

Of the 127 respondents, 41 (32%) were female. Fig. 1 shows the body weight changes reported by these subjects. Total combined weight loss was 1 900 kg. The mean \pm standard deviation weight loss for the total group was 15 (\pm 12) kg; 16 (\pm 12) kg in men and 13 (\pm 10) kg in women. One patient with type 1 diabetes mellitus (T1DM) intentionally increased her body weight by 5 kg, at the same time halving her daily insulin requirements; all other subjects lost weight. Symptoms of hunger were reduced or absent on the diet in 27 subjects. Six asserted that their results were either 'life-changing' or 'life-saving'.

Medications were no longer required to treat chronic medical conditions in 16 subjects including: T2DM ($n=14$), hypertension ($n=8$) and hypercholesterolaemia ($n=7$). Some individuals were 'cured' of more than one condition. Another 9 with either T1DM or T2DM reduced their medications, as did 7 patients with hypertension; 8 were 'cured' of irritable bowel syndrome, one after 40 years. Exercise capacity increased markedly in 32 subjects. In one individual, the change was astonishing (Case 5). Five subjects reporting apparently 'miraculous outcomes' are briefly described with their permission.

Case 1

A 44-year-old Cape Town man was unable to fit into an economy class airplane seat. During the flight, the adjacent passenger complained

incessantly about his large size. 'Depressed, embarrassed and ashamed' he drove directly from the airport to his general practitioner (GP) who found his blood pressure (160/100 mmHg) and blood glucose concentration to be raised. She advised immediate treatment for both conditions and to consult a dietician. He declined, searched the internet, discovered the 'Noakes diet' and decided to adopt it.

His weight loss began immediately and continued for 7 months, during which time he lost 84.6 kg. After 6 months, his GP failed to recognise him. His blood pressure and fasting blood glucose and cholesterol concentrations had reverted to the normal range (Table 1).

He concluded that the LCHF diet had 'saved his life' and that 'label reading has opened my eyes to the almost criminal levels of carbs in everyday processed foods and the propensity of these foods to cause weight gain' (personal communication, 19 March 2013).

Case 2

A 44-year-old male restaurant critic who had always been 'fat' as a child, never participating in sports or exercise, reached the weight of 153 kg in October 2011. He had associated complications of T2DM and was receiving treatment for hypertension. He required non-steroidal anti-inflammatory drugs for relief of intermittent bilateral lower limb and chronic low-back pain. He had tried several methods of weight loss without lasting success. He sought bariatric surgery, but was refused as he posed too great an anaesthetic risk. As a final resort he decided to try the 'Noakes diet'.

Over the next 18 months, he lost 73 kg, at the same time curing his T2DM, hypertension (Table 1), neuropathy and chronic low-back pain. He is now a regular exerciser with a resting heart rate of 45 bpm compared to 80 bpm a year previously. He takes no medications.

Despite a dramatic reduction in calorie intake, his food cravings have disappeared: 'I totally subscribe to the view that sugar and carbohydrates are drugs to the body. I was an addict' (personal communication, 22 April 2013).

Case 3

A 57 year-old rural GP played rugby and athletics at school and was lean while at university (78 kg; body mass index (BMI) 21 kg/m²). His wife's

Table 1. Biological measurements in 5 cases showing the greatest beneficial changes following adoption of the LCHF eating plan

Variable	Case 1		Case 2		Case 3		Case 4		Case 5	
	Before	After	Before	After	Before	After	Before	After	Before	After
Weight (kg)	July 2012	March 2013	October 2011	April 2013	January 2008 - May 2012	February 2013	January - September 2012	June 2013	May 2012	April 2013
Height (cm)	163.4	78.8	153	82	126 - 135	102	120	75	83	68
BMI (kg/m ²)	174	174	176	176	193	193	167	167	179	179
BP (mmHg)	54	26	49	26	34 - 36	27	43	27	26	21
Blood glucose (mmol/l)	160/100	140/80	140/85	120/80	140/90 - 160/90	130/80	-	-	-	-
Blood insulin (mIU/l)	8.1 - 10.2	3.1 - 4.2	5.1	4.8	10 - 18.5	5.0	6.1	-	-	-
Blood HbA1c (%)	-	-	12.0	-	-	-	-	-	-	-
Blood cholesterol (mmol/l)	7.4	3.9	9.6	4.8	-	4.8	5.6	-	-	-
Blood LDL-C (mmol/l)	-	-	5.5	4.3	5.7 - 6.1	5.4	8.6	-	-	-
Blood HDL-C (mmol/l)	-	-	3.5	2.7	-	-	-	-	-	-
Blood triglyceride (mmol/l)	-	-	0.99	1.08	0.9	1.0	-	-	-	-
Blood CRP (mg/l)	-	-	2.54	0.63	2.5	1.3	-	-	-	-
GFR (ml/min)	-	-	81.4	54.5	-	-	20.2	-	-	-
Medication for:	-	-	-	-	59	>90	-	-	-	-
hypertension	No	No	Yes	No	Yes	No	No	No	No	No
type 2 diabetes mellitus	No	No	Yes	No	Yes	No	Yes	No	No	No
hypercholesterolaemia	No	No	No	No	Yes	No	No	No	No	No

LCHF = low-carbohydrate, high-fat; BMI = body mass index; BP = blood pressure; HbA1c = glycosylated haemoglobin; LDL-C = low-density-lipoprotein cholesterol; HDL-C = high-density-lipoprotein cholesterol; CRP = C-reactive protein; GFR = glomerular filtration rate.

culinary skills, his love of 'healthy' fruits and forced inactivity due to a knee injury at age 34, resulted in progressive weight gain, reaching 135 kg in January 2008. He had developed T2DM, hypertension, atrial fibrillation, hypercholesterolaemia, sleep apnoea, fatigue and stucco keratosis. He was treated with perindopril, bisoprolol, amlodipine and metformin and advised to follow a conventional low-calorie, high-carbohydrate, low-fat (HCLF) (Weight Watcher's) diet. Although he lost 10 kg, he was perpetually hungry. He developed diabetic neuropathy and gout and his hypercholesterolaemia worsened, and he was placed on additional medications – gliclazide, rosuvastatin and warfarin. In early 2012, he informed his wife that he was unlikely to live beyond age 65.

In May 2012 he read about the 'Noakes diet', concluded that he was carbohydrate intolerant and would benefit from the LCHF eating plan. Over the next 8 months, he lost 25 kg, curing all his medical conditions (Table 1). He now offers the LCHF dietary option for all his patients suffering from any of the conditions, of which he is now rid.

Case 4

Despite regular physical activity, a 23-year-old mother had always struggled with her weight, reaching 73 kg in grade 9 of high school. At 13 years of age her gall bladder was removed and she was diagnosed with polycystic ovarian syndrome. Weight-loss attempts always failed as she would eventually 'cheat', reverting to her addictive food choices,^[16] quickly regaining any lost weight. Her health deteriorated and she ceased sport because she was frequently ill.

In the third trimester of her first pregnancy in March 2012, her weight reached 96 kg and she developed gestational diabetes and hypertension, requiring caesarean section due to uncontrolled hypertension. After the birth of her child, she began to eat addictively. Her weight increased to 120 kg, her surgical wound became infected and she suffered frequent and repeated skin infections culminating in a facial cellulitis that responded only to intravenous clindamycin. She was diagnosed with T2DM and treated with metformin.

She adopted the 'Noakes diet' and by June 2013 her weight had dropped to 75 kg, a loss of 45 kg. She no longer requires medication for T2DM and concluded that '... for the life of me I don't know why I struggled so much (to control my weight) since it really isn't that difficult. It was more of a lifestyle change for me than a diet' (personal communication, 3 June 2013).

Case 5

A 37-year-old science correspondent, who initially weighed 75 kg at university and was a competitive athlete, reported that his weight increased to 80 kg by the time he was age 25 years – despite competitive running. This weight gain caused skin chaffing and painful knees – in spite of which he managed 70 km/week in training. He participated regularly in marathons and ultra-marathons completing the Two Oceans 56 km ultra-marathon in between 5h:55min and 6h:45min. In October 2011, he decided to lose weight by completing 9 marathons in 9 provinces in 9 consecutive weeks. However, rather than losing weight, his weight increased to 83 kg and he ran poorly. In April 2012, he completed the Two Oceans ultra-marathon in his slowest-ever time (6h:57min:57sec) in 7 668th place.

He began the 'Noakes diet' on 11 June 2012. Fig. 2 shows his weight and distance training. He lost the most weight when he ran the least. His racing performances improved dramatically. On 16 March 2013, he completed the Two Oceans ultra-marathon in 3:59:42 in 208th position, nearly 3 hours and 7 460 positions better than his performance 12 months earlier. He concluded: 'This is the important point to all of this. The weight loss enabled my training, not the other way around. I feel like I have won my life back' (personal communication, 27 April 2013).

Discussion

The study has several potential limitations. First, all data are self-reported and were not verified but it is unlikely that all participants

would fabricate this information. Second, there is no record of exactly what each person ate. Third, all reports describe only short-term outcomes. To collect this information as part of an RCT involving 254 subjects would have been very costly.

Despite these substantive limitations, this information challenges current conventional wisdom (widely taught at medical schools); the higher fat content of the LCHF diet (*i*) must cause weight gain because of the higher energy density (per g) of fat than of either carbohydrate or protein; (*ii*) will either cause abnormal or worsen already abnormal lipid profiles, and (*iii*) will worsen blood pressure and blood glucose control. The data also suggest that the macronutrient composition of the diet is the key driver of weight gain and obesity.

Proposition 1: High-fat diets cause weight gain or are inferior to hypo-caloric HCLF diets in producing sustained weight loss

The average weight loss (15 kg) exceeds that reported in expensive clinical trials typically involving the HCLF diet with intensive medical supervision. By comparison, low-fat (HCLF) diets produce an average weight loss of just 1.6 kg.^[17] Another study found an average weight loss of 1.3 kg at 1 year and 0.2 kg at 2 years.^[18] Even an intensive combined diet and physical activity intervention in severe obesity produced an average weight loss of only ~10 kg at 6 and 12 months.^[19] There is extensive evidence that the LCHF is either 'at least as effective'^[20] or more effective^[21] than the hypo-caloric HCLF in producing weight loss.

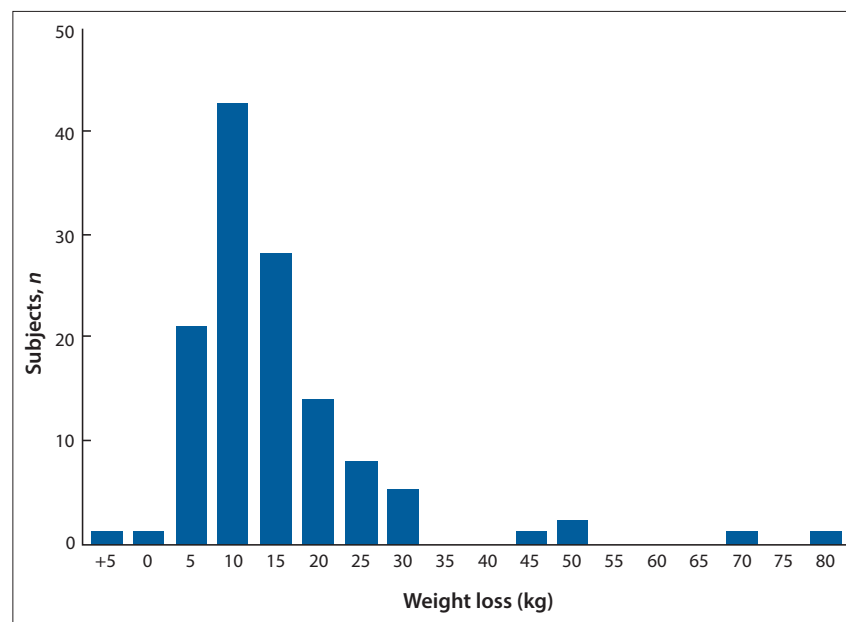


Fig. 1. Distribution of self-reported body weight losses in 127 subjects following adoption of a carbohydrate-restricted, low-carbohydrate, high-fat (LCHF) eating plan.

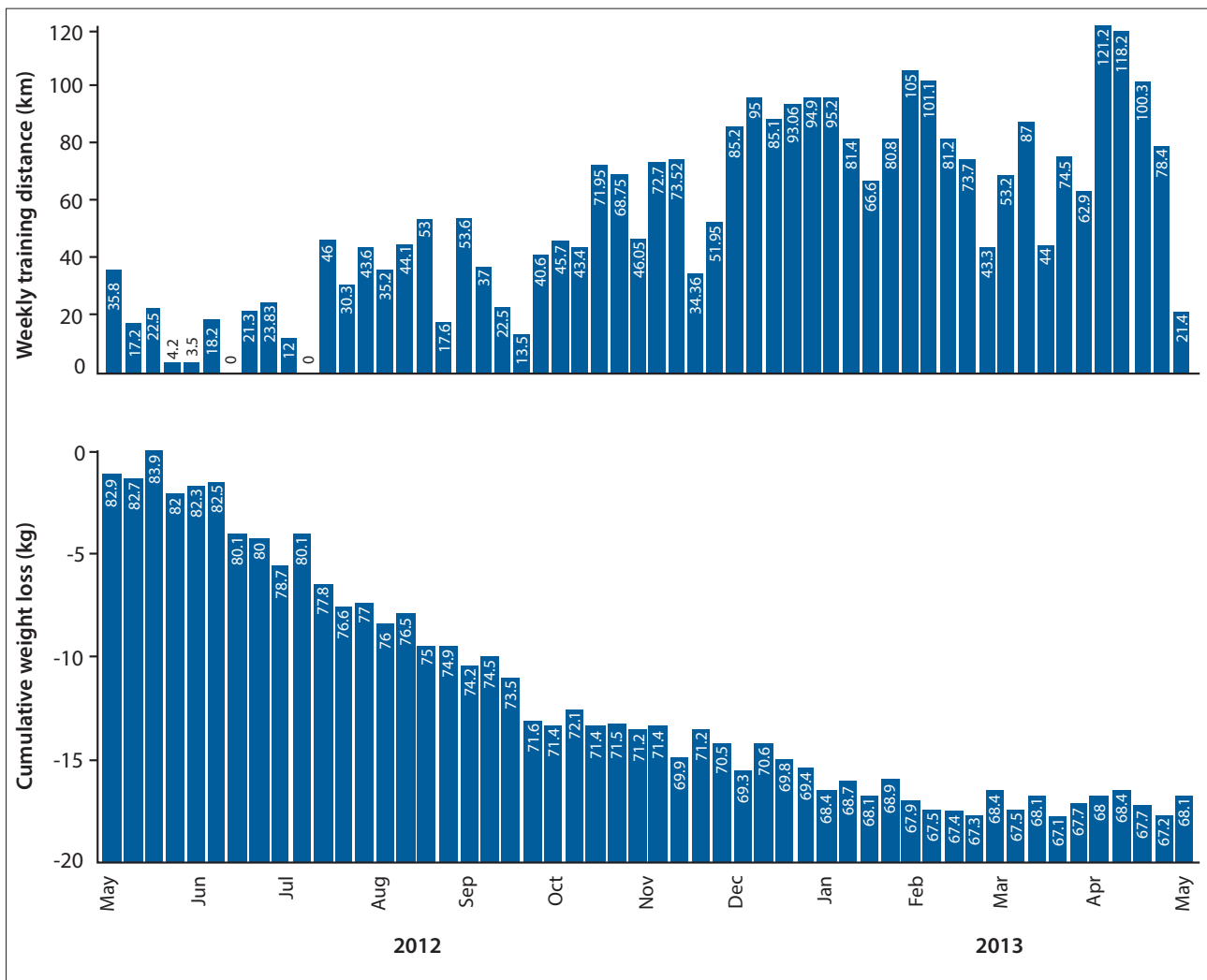


Fig. 2. Case 5. Weekly running training distances (top panel) and cumulative body weight changes (bottom panel). Note that subject lost the most weight when he exercised the least.

The key determinant of long-term successful weight loss is the extent to which any intervention reduces hunger and consequently caloric intake.^[6,22-24] Cases 1 and 2 each lost more than 70 kg *without hunger* despite eating a fraction of the calories previously needed to satisfy their food cravings. This paradoxical, and important phenomenon was previously reported by Yudkin and Carey.^[23] The LCHF diet is especially effective in producing satiation on a reduced energy intake.^[6,24] As Yudkin noted, 'the high-fat diet is in fact a low calorie diet.'^[24]

Proposition 2: A high-fat diet will either cause or worsen abnormal lipid profiles especially in those with hypercholesterolaemia

Five subjects normalised their elevated blood total cholesterol concentrations on the LCHF diet. Meta-analyses confirm that the LCHF is at least as effective^[25] as the HCLF in improving all common parameters predicting coronary heart disease (CHD) risk.^[20,21,25-29]

Besides body weight reduction and normalised blood pressure, this includes greater reductions in parameters of abnormal glucose metabolism, in plasma triglyceride and very-low-density-lipoprotein (VLDL)-cholesterol concentrations, and small dense low-density-lipoprotein (LDL)-cholesterol particle numbers, while significantly increasing plasma high-density-lipoprotein (HDL)-cholesterol

concentrations. This occurs without significant increases in LDL-cholesterol concentrations.^[26] In contrast, a HCLF diet produces changes^[30-33] that are considered to be atherogenic.

The evidence that the LCHF produces the greatest benefits in the most unhealthy (Cases 1-4) suggests that the LCHF is the safer option for those who are the most ill.^[28,29,31,34]

Proposition 3: A high-fat diet will elevate blood pressure in those with hypertension and worsen blood glucose control in those with T2DM

Eight subjects reported that the LCHF cured their hypertension; another 7 reduced their anti-hypertensive medications; 14 reported that they no longer require medications to control their blood glucose concentrations indicating that the LCHF 'cured' their T2DM. That the LCHF improves glucose control more effectively than does the HCLF diet is well established in the literature.^[27,31,34-36]

The role of sloth and gluttony in the development of obesity and related co-morbidities

The energy balance model of human obesity^[9-12,37] predicts that the overweight are consciously slothful and gluttonous. This information shows that many presumed slothful and gluttonous persons have a remarkable capacity to lose weight on the LCHF eating plan.

Cases 1 - 5 had repeatedly failed to lose weight on a 'healthy' HCLF diet as had many of the others. Only his poor state of health saved Case 2 from bariatric surgery, while Case 3 concluded that he would be dead by age 65 years. Case 4 had never been informed that many processed foods are addictive.^[16] Despite much training, the marathon runner (Case 5) gained weight progressively with age. On the LCHF eating plan his weight loss was greatest when he exercised the least (Fig. 2).

If sloth and gluttony alone cause obesity, then none of the respondents should have reversed their obesity as such character defects are presumably immutable.

Summary

This uncontrolled clinical study describes 127 subjects who collectively lost 1 900 kg while following a diet that is the opposite of that currently prescribed as ideal for weight control and the management of T2DM.^[14] This suggests that the dietary cause(s) of obesity require a fundamental re-appraisal.^[7-11] An RCT is urgently needed to disprove the hypothesis that the LCHF diet can reverse T2DM, metabolic syndrome and hypertension without pharmacotherapy.

This report, backed by extensive published evidence,^[6,9,20,21,23-29,31,34-36,38] suggests that we need to start radically re-thinking the advice that we give to patients. The Swedish National Board of Health and Welfare has concluded that 'low carb diets can today be seen as compatible with scientific evidence and best practice for weight reduction for patients with overweight or diabetes type 2, as a number of studies have shown effect in the short term and no evidence of harm has emerged ...'^[39]

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