SINut
Società Italiana di Nutraceutica

Il ruolo nutraceutico dei pasti sostitutivi nella terapia dell'obesità tramite VLCKD

PROF. GIOVANNI SPERA

N Engl J Med. 2015 Jul 2;373(1):82-3.

EDITORIAL

Another Agent for Obesity — Will This Time Be Different?

Elias S. Siraj, M.D., and Kevin Jon Williams, M.D.

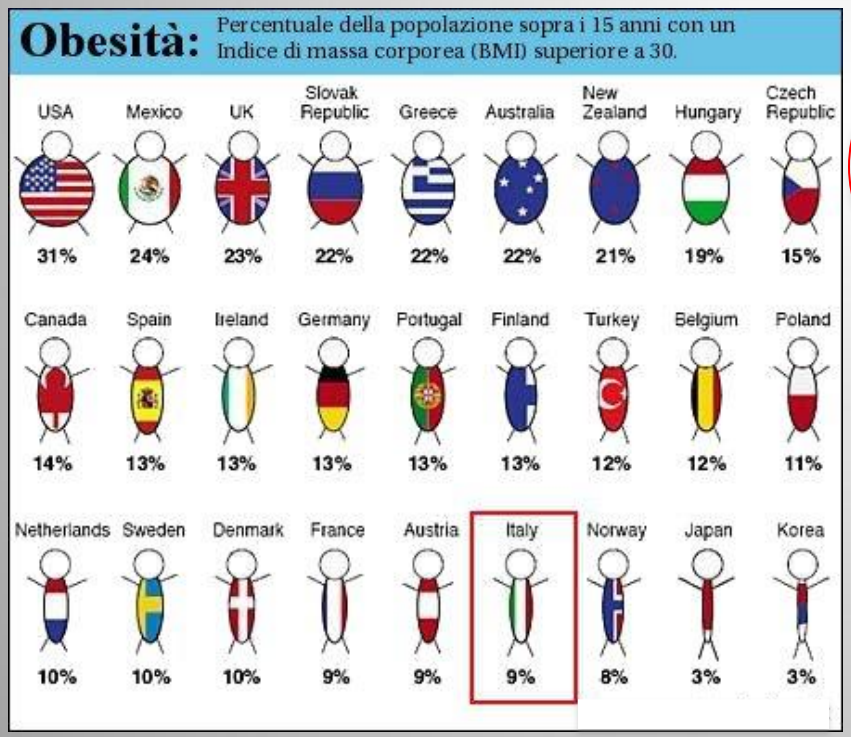
Although numerous randomized trials of lifestyle modification, medications, and bariatric surgery have shown that weight loss reduces morbidity, most patients cannot sustain sufficient weight loss. Despite decades of drug development, the **benefits of medications to treat obesity remain limited because of side effects and inadequate efficacy, especially in the long term.** Bariatric surgery results in the most weight loss and the highest rates of remission of type 2 diabetes, but the potential side effects are of concern. **Furthermore, performing bariatric surgery in approximately 400 million obese persons worldwide is not feasible.**

overconsumption of unhealthy foods.

N ENGL J MED 374;2 NEJM.ORG JANUARY 14, 2016

The Obesity Epidemic — Understanding the Disease and the Treatment

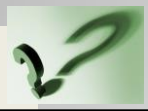
Caroline M. Apovian, M.D.



The prevention of severe obesity in adolescents is paramount, and bariatric surgery will not stop the progression of the disease. Continued efforts to work with government and the food industry to ensure that healthier food and increased physical activity are available for all children through communities, schools, and other avenues are important if the increase in severe obesity is to be halted. Because lifestyle interventions early in childhood may be effective, these should be instituted. But for adolescents with severe obesity for whom conservative medical treatment has failed, the present study indicates that surgery can result in substantial weight loss and resolution of coexisting conditions. Thus, it may be beneficial to consider such adolescents for bariatric surgery, before they reach adulthood, when some conditions become less reversible.

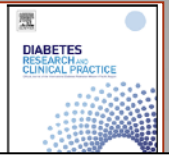
New approach to a better nutrition

Why VLCDs



Contents lists available at ScienceDirect

Diabetes Research and Clinical Practice



ELSEVIER

SURGERY FOR OBESITY AND RELATED DISEASES

Surgery for Obesity and Related Diseases 11 (2015) 230–237

OBES SURG (2015) 25:64–71
DOI 10.1007/s11695-014-1348-1

ORIGINAL CONTRIBUTIONS

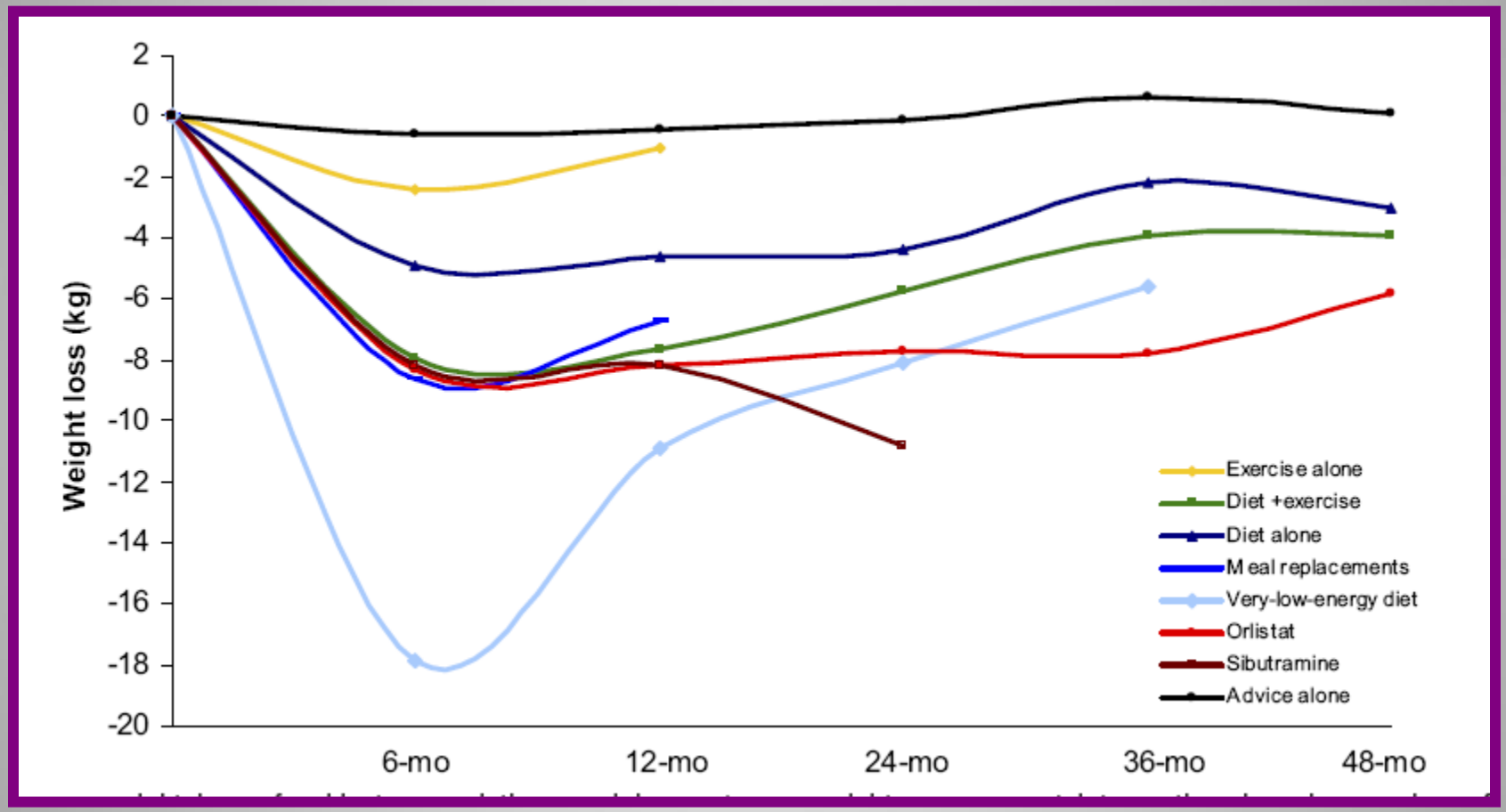
Very Low-Carbohydrate Ketogenic Diet Before Bariatric Surgery: Prospective Evaluation of a Sequential Diet

Frida Leonetti · Fabio Cesare Campanile · Federica Coccia · Danila Capoccia · Laura Alessandroni · Alessandro Puzziello · Ilenia Coluzzi · Gianfranco Silecchia

Diabetes Care May 2016 39:5 808-815;
published ahead of print March 21, 2016

Euro… 2013.116; published online 26 June 2013
Key… …; cardiovascular diseases

Weight-Loss Outcomes: A Systematic Review and Meta-Analysis of Weight-Loss Clinical Trials with a Minimum 1-Year Follow-Up



Average weight loss of subjects completing a minimum 1-year weight-management intervention; based on review of 80 studies (N26,455; 18,199 completers [69%]).

FRANZ M. J. *J Am Diet Assoc. 2007*

Endocrine
DOI 10.1007/s12020-014-0355-2

ORIGINAL ARTICLE

Safety and efficacy of a multiphase dietetic protocol with meal replacements including a step with very low calorie diet

Sabrina Basciani · Daniela Costantini · Savina Contini · Agnese Persichetti · Mikiko Watanabe · Stefania Mariani · Carla Lubrano · Giovanni Spera · Andrea Lenzi · Lucio Gnessi

Received: 6 May 2014 / Accepted: 29 June 2014

We evaluated **safety**, **adherence**, **acceptability** and **efficacy**, on weight loss and cardio-metabolic risk factors of a **commercially available multiphase, four-stage sequence, dietary intervention** based on **meal replacements**, which also includes an **initial period of VLCD** followed by **phases of dietary education** also based on a programmed reintroduction of carbohydrates in a group of obese patients.

Dipartimento di Medicina Sperimentale, Sez. di Fisiopatologia Medica, Endocrinologia, Scienza dell'Alimentazione, Università “Sapienza”, Policlinico Umberto I, Roma

Age
between 18 and 50 years
BMI
between 30 and 40kg/m^2

27 obese adults assessed for eligibility

3 excluded,
declined to participate

24 patients (7 males and 14 females) underwent inclusion

Phase I (30 days)
VLCD 670 Kcal/day
(4-5 meal replacements)

Phase II (30 days)
LCD 820 Kcal/day
(3-4 meal replacements and 1 protein dish)

Phase III (60 days)
LCD 1100 Kcal/day
(2 meal replacements, 2 protein dishes and reintroduction of small amounts of carbohydrate)

Phase IV (60 days)
HBD 1250 Kcal/day
(1 meal replacement, 2 protein dishes and complete reintroduction of carbohydrate)

Variazioni misure antropometriche e pressione arteriosa

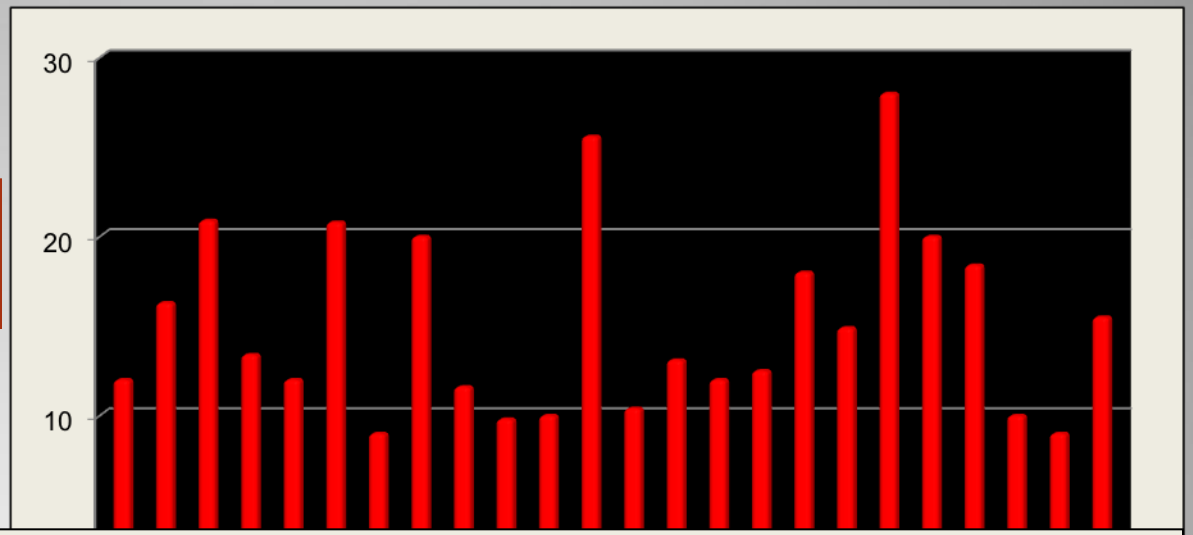
Table 1. Anthropometric measures, weight and blood pressure at the beginning of the program and during the four phases of the study.

	Baseline	Phase I	Phase II	Phase III		Phase IV		
	T0	T30	T60	T90	T120	T150	T180	T0-T180
Weight								
Mean (kg)	95.5±13	89.4±11.7	86.4±11.7	84.4±11	82.8±10.9	81.7±10.3	80.8±10.4	
Δ (kg)		Δ^{T0-T30} -6.1±2.0*	$\Delta^{T30-T60}$ -3.1±1.7*	$\Delta^{T60-T90}$ -2.0±1.6*	$\Delta^{T90-T120}$ -1.5±1.3*	$\Delta^{T120-T150}$ -1.1±0.7*	$\Delta^{T150-T180}$ -0.9±0.7*	-14.7±6.4*
Δ (%)		Δ^{T0-T30} -6.4±1.7*	$\Delta^{T30-T60}$ -3.3±2.0*	$\Delta^{T60-T90}$ -2.2±1.7*	$\Delta^{T90-T120}$ -1.8±1.6*	$\Delta^{T120-T150}$ -1.3±1.5*	$\Delta^{T150-T180}$ -1.1±0.9*	-15.4±5.4*
BMI								
Mean (kg/m^2)	33.8±3.2	31.7±2.9	30.6±3.0	29.9±3.0	29.3±3.1	29.0±3.1	28.6±3.1	
Δ (kg/m^2)		Δ^{T0-T30} -2.1±0.6*	$\Delta^{T30-T60}$ -1.1±0.6*	$\Delta^{T60-T90}$ -0.7±0.5*	$\Delta^{T90-T120}$ -0.6±0.4*	$\Delta^{T120-T150}$ -0.3±0.4*	$\Delta^{T150-T180}$ -0.4±0.3*	-5.2±1.9*
Δ (%)		Δ^{T0-T30} -6.2±1.7*	$\Delta^{T30-T60}$ -3.4±2.0*	$\Delta^{T60-T90}$ -2.2±1.6*	$\Delta^{T90-T120}$ -1.8±1.6*	$\Delta^{T120-T150}$ - 1.0±1.5*	$\Delta^{T150-T180}$ -1.1±0.9*	-15.4±5.4*
WC								
Mean (cm)	101.1±10.3	96.8±9.5	93.8±8.9	91.9±8.5	90.6±8.1	89.9±8.2	88.9±7.9	
Δ (cm)		Δ^{T0-T30} -4.3±1.9*	$\Delta^{T30-T60}$ -3.0±1.9*	$\Delta^{T60-T90}$ -1.9±1.5*	$\Delta^{T90-T120}$ -1.3±1.1*	$\Delta^{T120-T50}$ -0.7±1.2*	$\Delta^{T150-T180}$ -1.0±1.3*	-12.2±5.2*
Δ (%)		Δ^{T0-T30} -4.2±1.7*	$\Delta^{T30-T60}$ -3.1±1.8*	$\Delta^{T60-T90}$ -2.0±1.6*	$\Delta^{T90-T120}$ -1.3±1.1*	$\Delta^{T120-T150}$ -0.8±1.3*	$\Delta^{T150-T180}$ -1.1±1.3*	-12.0±4.5*
SBP								
Mean (mmHg)	125.8±9.3	126.3±9.0	123.1±7.2	121.0±6.7	121.0±6.7	121.5±6.0	119.8±6.2	
Δ (mmHg)		Δ^{T0-T30} +0.5±5.7*	$\Delta^{T30-T60}$ -3.2±6.4*	$\Delta^{T60-T90}$ -2.1±4.1*	$\Delta^{T90-T120}$ -0.0±2.5*	$\Delta^{T120-T150}$ +0.5±5.3*	$\Delta^{T150-T180}$ -1.7±5.0*	-6.0±6.2*
Δ (%)		Δ^{T0-T30} +0.4±4.6*	$\Delta^{T30-T60}$ -2.2±5.4*	$\Delta^{T60-T90}$ -1.6±3.3*	$\Delta^{T90-T120}$ -0.0±2.1*	$\Delta^{T120-T150}$ +0.5±4.4*	$\Delta^{T150-T180}$ -1.3±4.2*	-4.8±4.7*
DBP								
Mean (mmHg)	82.9±6.9	82.7±7.5	81.2±5.2	79.0±4.9	78.1±5.5	77.3±4.4	76.7±5.2	
Δ (mmHg)		Δ_{T0-T30} -0.2±5.4*	$\Delta_{T30-T60}$ -1.5±6.3*	$\Delta_{T60-T90}$ -2.2±5.9*	$\Delta_{T90-T120}$ -0.9±4.6*	$\Delta_{T120-T150}$ -0.8±5.2*	$\Delta_{T150-T180}$ -0.6±4.2*	-6.2±5.4*
Δ (%)		Δ_{T0-T30} -0.2±7.0*	$\Delta_{T30-T60}$ -1.8±8.3*	$\Delta_{T60-T90}$ -2.6±7.2*	$\Delta_{T90-T120}$ -1.1±1.1*	$\Delta_{T120-T150}$ -1.0±1.3*	$\Delta_{T150-T180}$ -0.7±1.3*	-7.5±4.5*

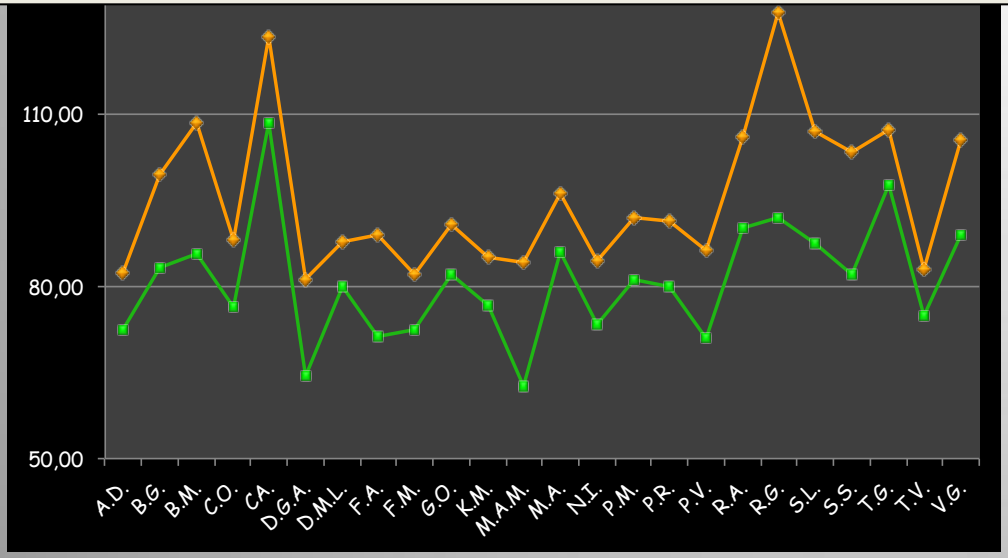
Data are expressed as mean values ±SD. *p<0.0001

Basciani S, Endocrine, 2014

Perdita di peso (%)
T0-T180 singoli pazienti



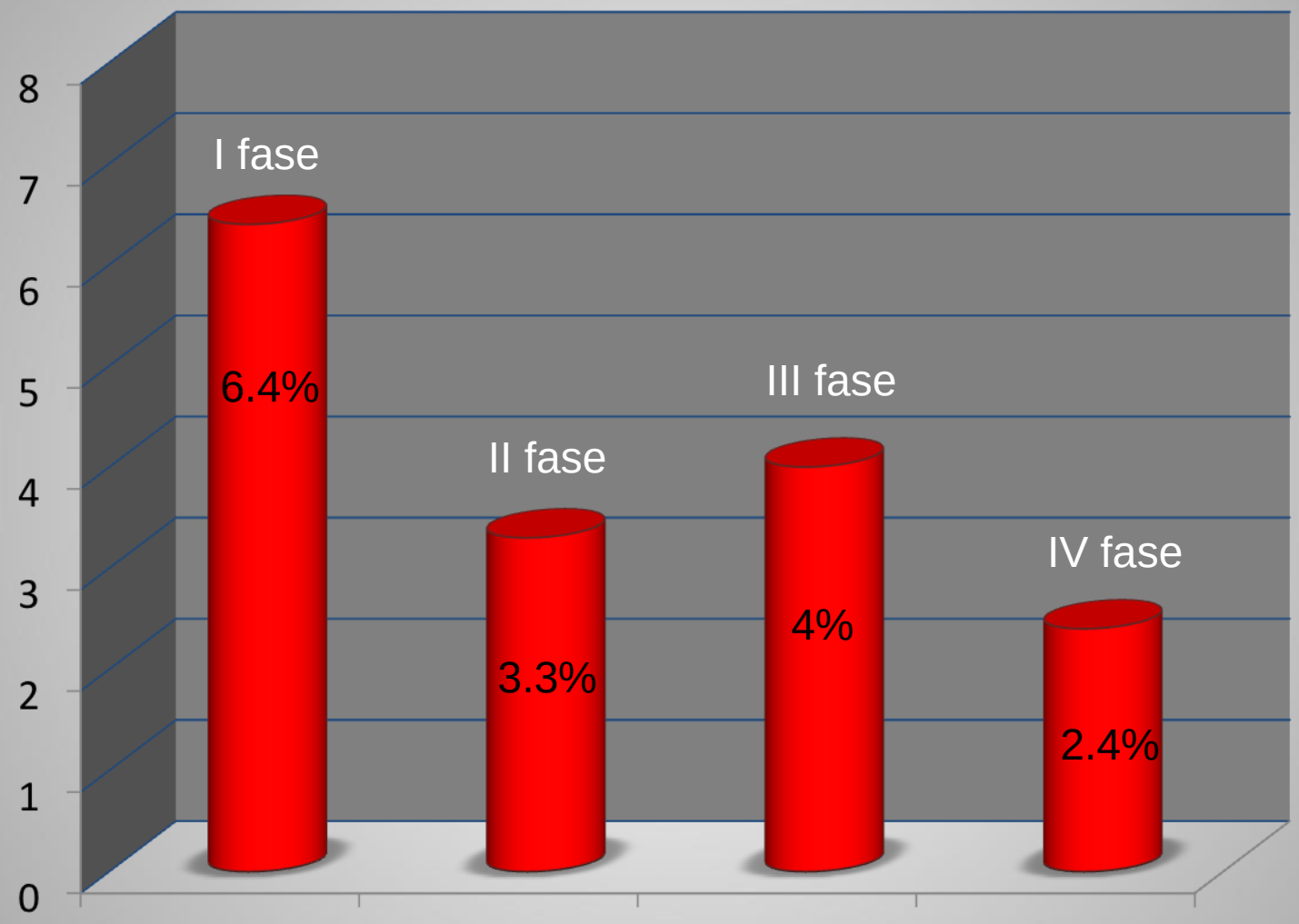
Grande variabilità soggettiva sia in termini di perdita di peso che di velocità di discesa. Accanto a pazienti che hanno perso in totale soltanto 8kg, con una velocità di discesa media globale pari a 1.3kg/mese, erano presenti pazienti che sono arrivati a perdere fino a 35.7kg in totale, con una velocità di discesa media globale di circa 6kg/mese.



Peso (kg) singoli pazienti a T0 ( ) e a T180 ( )

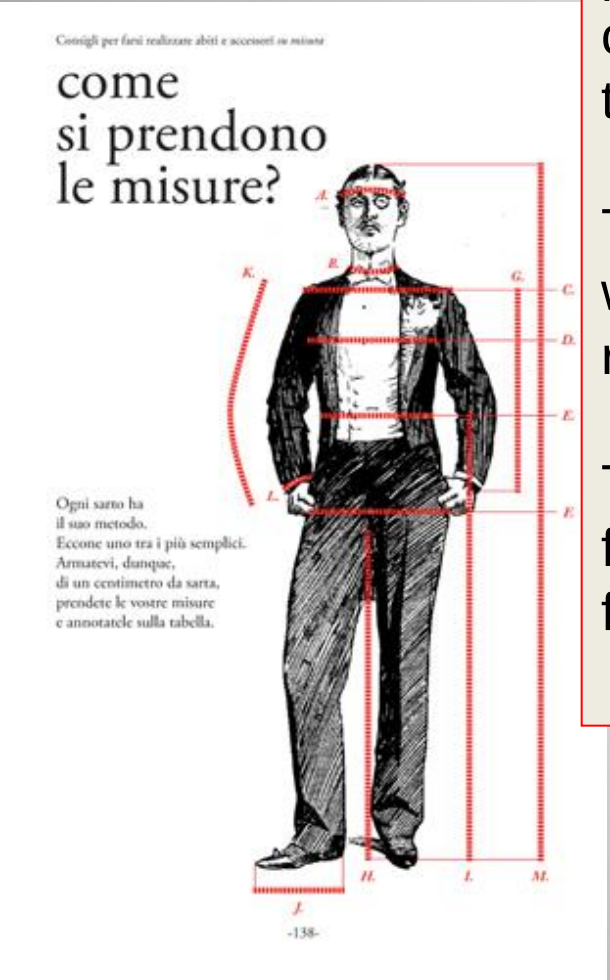
Basciani S, Endocrine, 2014

Variazioni di peso (%) singole fasi



T0-T30	T30-T60	T60-T120	T120-T180
-6.1Kg/mese	-3.1Kg/mese	-1.7Kg/mese	-1.0Kg/mese

Basciani S, Endocrine, 2014



A 12 % change in waist circumference was observed.

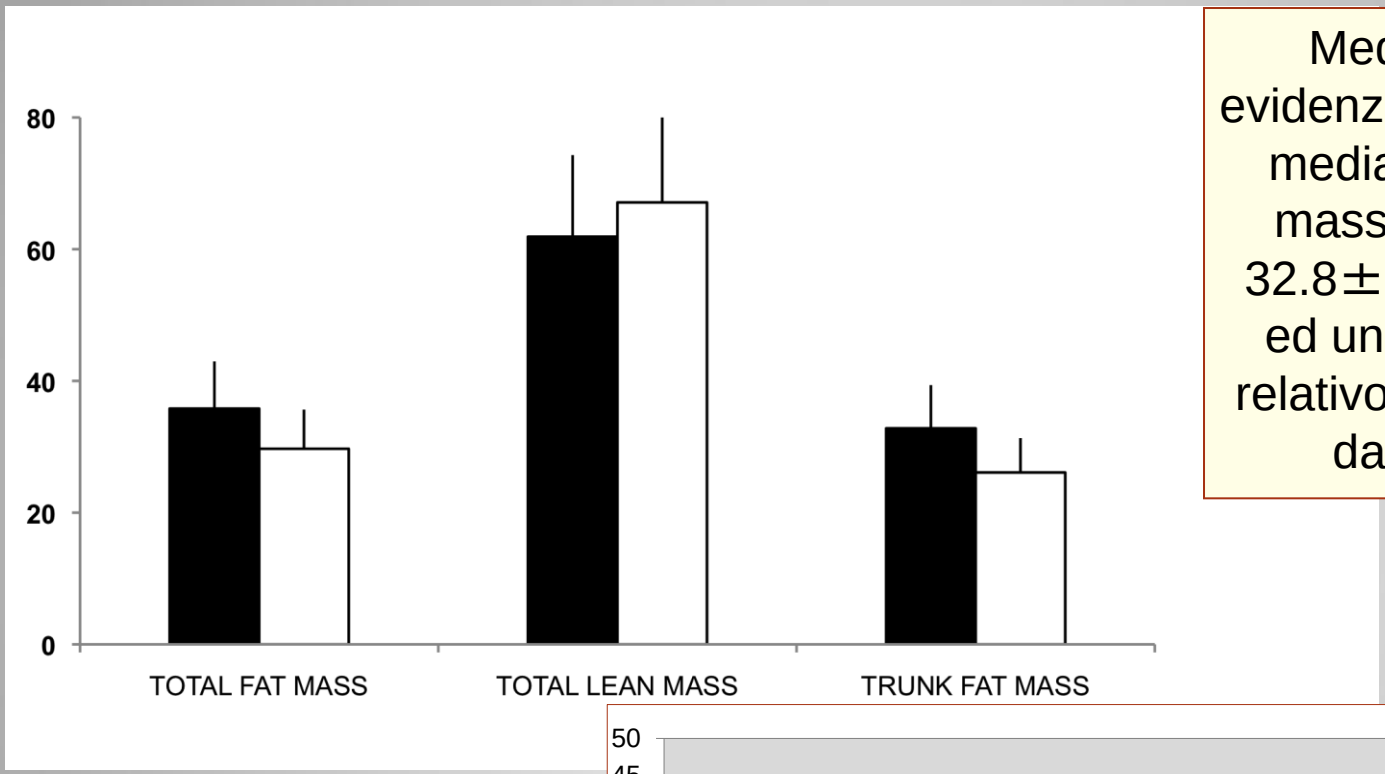
57 % of the male patients and 65 % of the female patients experienced a reduction of waist circumference from >102 to <94 and from >88cm to <80cm, respectively.

These results are important, being the decrease in waist circumference closely related to the reduction of cardiovascular risk.

The evaluation of a surrogate markers of visceral fat such as trunk fat % confirmed the reduction of fat mass in these areas.

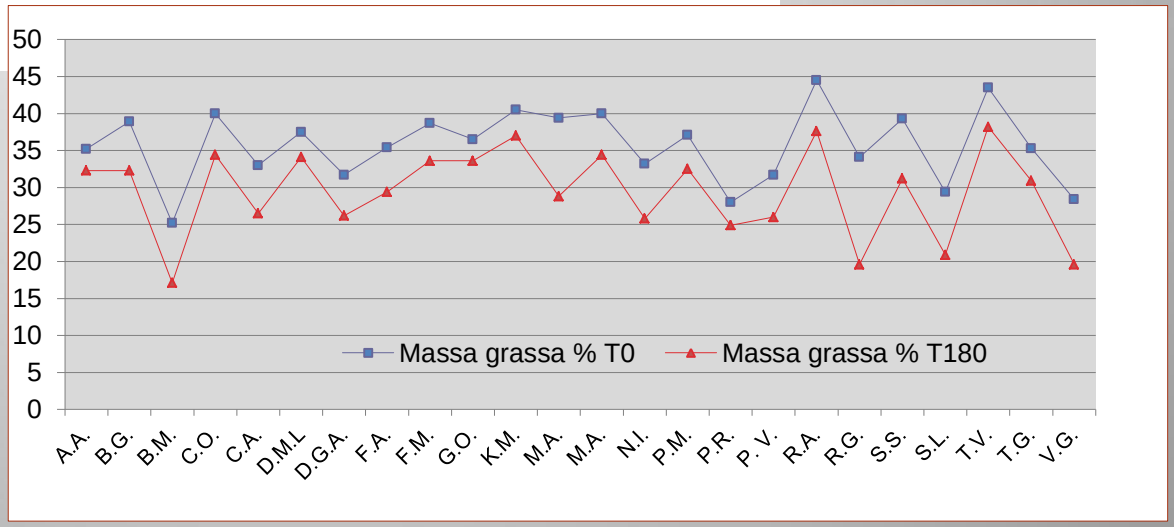
Basciani S, Endocrine, 2014

Variazione (%) di massa grassa e massa magra T0-T180



Mediante DEXA si è evidenziata una diminuzione media significativa della massa grassa totale da 32.8±4.7% a 26.1±6.3% ed un incremento medio relativo della massa magra dal 61.9±4.8% al 67.1±5.9%

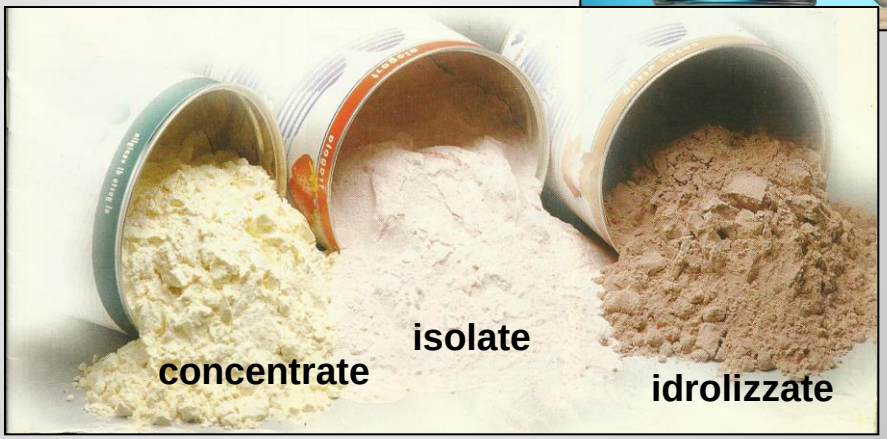
Massa grassa (%) a T0 e a T180 nei singoli pazienti



Basciani S, Endocrine, 2014

I pasti sostitutivi sono principalmente a base di proteine del siero del latte.

Le proteine del siero di latte sono ricche in AA essenziali e AA ramificati (BCAA).



I pasti sostitutivi contengono un mix di proteine del siero, isolate e idrolizzate (con prevalenza delle isolate), ottenute per microfiltrazione a flusso incrociato.

Le cosiddette proteine “isolate” sono la forma più pura di proteine del siero di latte, contengono quantità inferiori di umidità, di grassi e di lattosio, rispetto alle proteine concentrate.

Il processo utilizzato per ottenerle è la microfiltrazione a flusso incrociato. Questo metodo non sfrutta composti chimici o alte temperature e permette di ottenere formule con un contenuto tra l'85 e il 90% di proteine.

Vengono assimilate in tempi brevi e liberano nel sangue gli amminoacidi che possono così essere utilizzati per la sintesi proteica.

Le proteine del siero “idrolizzate” sono derivate dall'idrolisi, principalmente delle proteine isolate, mediante un processo artificiale di digestione enzimatica che prevede la scissione delle proteine in oligopeptidi.

Le proteine idrolizzate, in termini nutrizionali, sono equivalenti alle proteine da cui derivano, in quanto a composizione amminoacidica, ma la digestione enzimatica le rende più rapidamente digeribili e assorbibili.

Proc. Natl. Acad. Sci. USA
Vol. 94, pp. 14930–14935, December 1997
Physiology

Slow and fast dietary proteins differently modulate postprandial protein accretion

(amino acid turnover/postprandial protein anabolism/milk protein/stable isotopes)

YVES BOIRIE*, MARTIAL DANGIN*†, PIERRE GACHON*, MARIE-PAULE VASSON‡, JEAN-LOUIS MAUBOIS§, AND BERNARD BEAUFRÈRE*¶

*Laboratoire de Nutrition Humaine, Université Clermont Auvergne, Centre de Recherche en Nutrition Humaine, BP 321, 63009 Clermont-Ferrand Cedex 1, France; †Nestec, Ltd., Nestlé Research Center, P.O. Box 44, CH 1000 Lausanne 26, Switzerland; ‡Laboratoire de Biochimie, Biologie Moléculaire et Nutrition, Université Clermont Auvergne, BP 38, 63001 Clermont-Ferrand Cedex 1, France; and §Laboratoire de Technologie Laitière, Institut National de la Recherche Agronomique, 35042 Rennes Cedex, France

News and Views

Nature **391**, 843-845 (26 February 1998) | doi:10.1038/35993

Protein metabolism: Slow and fast dietary proteins

Gema Frühbeck[1]

Dietary amino acid absorption is faster with whey protein than with casein.

The rate limiting steps might be gastric emptying and/or luminal hydrolysis and/or amino acid mucosal absorption.

Slow and fast proteins differentially modulate whole-body protein deposition after a meal.

Whey, as a fast protein, is associated with a pronunced stimulation of protein synthesis and absence of protein breakdown inhibition.

The distinct amino acid composition of casein and whey protein may, therefore, trigger a discriminatory stimulus on protein synthesis and breakdown, due to differences in the secretion of insulin and glucagon.

Anabolic effect of whey protein

Variazione dei parametri metabolici T0-T180

Table 2. Clinical chemistry and blood count values during the phases of the study

	Baseline (a)	Phase I (b)	Phase II (c)	Phase III (d)	Phase IV (e)
	T0	T30	T60	T120	T180
Erythrocyte ($x10^6/\mu l$)	4.5±0.8	4.3±0.4	4.4±0.6	4.8±0.6	4.6±0.4
Leukocyte ($x10^3/\mu l$)	6.8±2.2	6.7±1.9	6.8±1.0	6.9±1.1	6.8±2.1
Hematocrit (%)	41.6±2.5	42.0±2.5	41.8±2.0	41.0±2.2	41.9±2.4
Hemaglobin (g/dL)	13.5±1.3	13.8±0.8	12.9±1.1	13.1±0.6	13.8±1.0
Glucose fasting (mg/dL)	96.0±8.1	89.5±8.3[ab]	85.7±10.6[ac,bc]	89.0±7.8[ad,cd]	81.5±6.3[ae,be,ce,de]
Insulin (μU/mL)	14.0±9.1	7.9±3.5[ab]	7.6±3.3[ac]	7.2±3.4[ad]	5.6±1.8[ae,be,ce,de]
Homa index	3.2±2.2	1.8±0.9[ab]	1.6±0.8[ac]	1.6±0.8[ad]	1.1±0.4[ae,be,ce,de]
Triglycerides (mg/dL)	116.3±68.5	90.0±45.9[ab]	81±42.0[ac]	89.0±54.1[ad]	72.1±37.0[ae,be]
Total Cholesterol (mg/dL)	193.8±34.4	167.4±22.5	179.3±27.2	184.2±21.1	178.6±21.2
HDL (mg/dL)	53.2±12.1	51.0±11.6	53.0±12.6	54.4±9.5	53.1±10.0
ALT (U/l)	29.5±24.4	29.6±19.0	27.3±15.1	20.5±9.1[ad,bd,cd]	19.1±8.4[ae,be,ce]
AST (U/l)	28.6±6.8	21.0±5.0	20.2±4.8	18.5±4.4[ad,bd]	18.3±4.1[ae,be]
BUN (mg/dL)	36.5±7.0	37.9±7.1	39.4±7.6	39.7±10.5	36.2±9.1

I pazienti che hanno perso la maggior quantità di peso (> 20kg) sono risultati essere quelli in cui l'indice Homa era maggiore.
In ogni caso, la riduzione dell'indice Homa è stata riscontrata anche in quelli con riduzioni di peso più contenute (< 10kg).

Basciani S, Endocrine, 2014

Available online at www.sciencedirect.com

SciVerse ScienceDirect

Journal of Nutritional Biochemistry 24 (2013) 1–5

Journal of Nutritional Biochemistry

REVIEWS: CURRENT TOPICS

Biochemical and metabolic mechanisms by which dietary whey protein may combat obesity and Type 2 diabetes☆

Daniela Jakubowicz[a,*], Oren Froy[b,*]

[a]Diabetes Unit E. Wolfson Medical Center, Tel Aviv University, Holon 58100, Israel
[b]Institute of Biochemistry, Food Science and Nutrition, Robert H. Smith Faculty of Agriculture, Food and Environment, The Hebrew University of Jerusalem, Rehovot 76100, Israel

Insulinotropic and glucose-lowering properties of whey protein in healthy and Type 2 diabetes subjects.

Whey protein leads to **increased** secretion of insulin.

Whey protein seems to induce these effects via **bioactive peptides** and **amino acids** generated during its gastrointestinal digestion.

The high content of **essential amino acids** (leucine, isoleucine, valine, lysine and threonine) released after whey protein digestion could be the mediator of its **insulinotropic response**.

In particular leucine **stimulates insulin secretion** from pancreatic β cells, either by its deaminated metabolite, alpha-ketoisocaproic acid (KIC) or by enhancing the oxidation of glutamate by allosterically activating glutamate dehydrogenase.

Another possible mechanism of whey protein effect, is the production of bioactive peptides that serve as endogenous **inhibitors of DPP-4** in the proximal gut, **preventing the degradation** of the insulinotropic **incretins** GLP-1 and GIP.

NON variazione dei parametri metabolici T0-T180

Table 2. Clinical chemistry and blood count values during the phases of the study

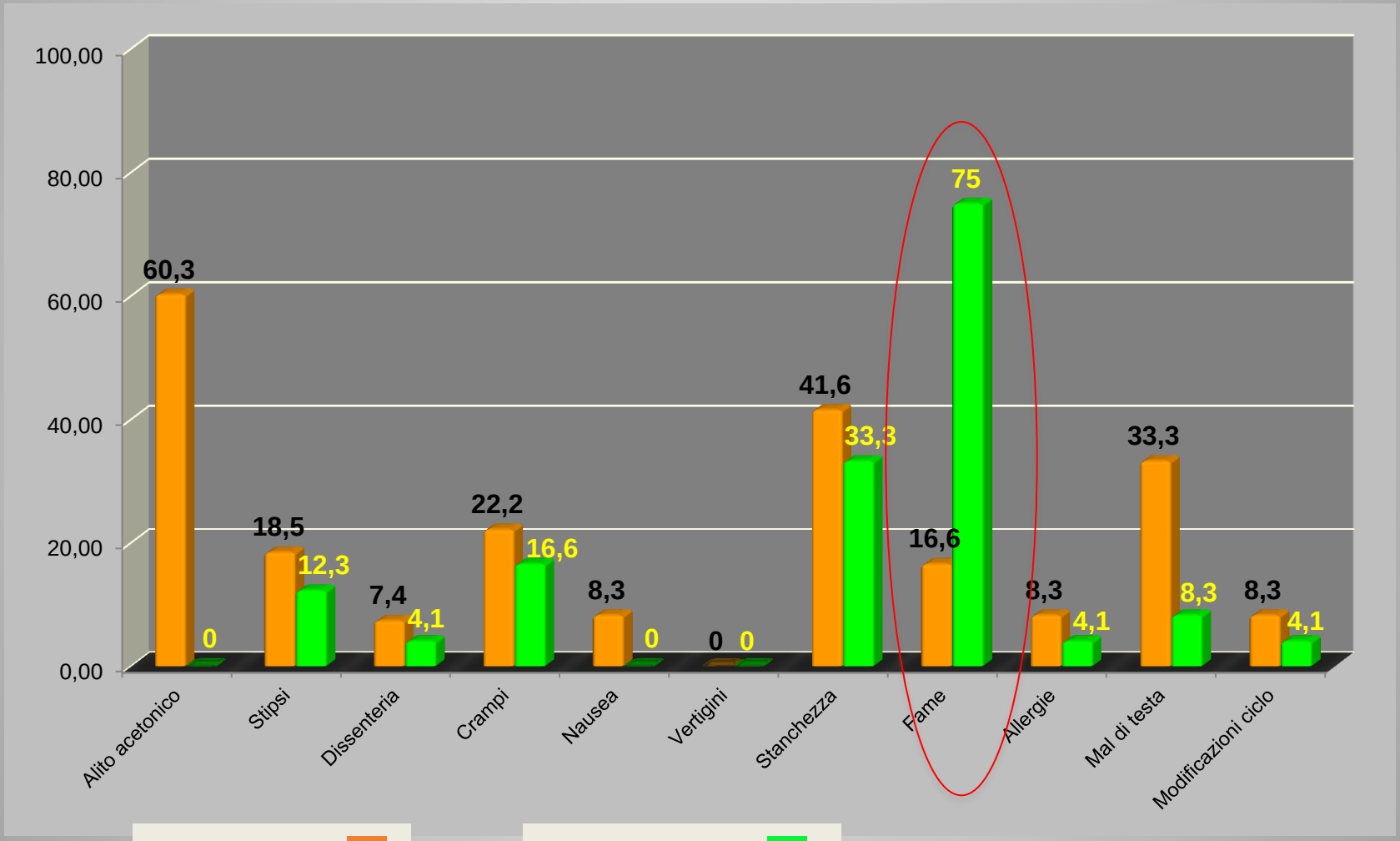
	Baseline (a)	Phase I (b)	Phase II (c)	Phase III (d)	Phase IV (e)
	T0	T30	T60	T120	T180
Erythrocyte ($x10^6/\mu l$)	4.5±0.8	4.3±0.4	4.4±0.6	4.8±0.6	4.6±0.4
Leukocyte ($x10^3/\mu l$)	6.8±2.2	6.7±1.9	6.8±1.0	6.9±1.1	6.8±2.1
Hematocrit (%)	41.6±2.5	42.0±2.5	41.8±2.0	41.0±2.2	41.9±2.4
Hemaglobin (g/dL)	13.5±1.3	13.8±0.8	12.9±1.1	13.1±0.6	13.8±1.0
Glucose fasting (mg/dL)	96.0±8.1	89.5±8.3[ab]	85.7±10.6[ac,bc]	89.0±7.8[ad,cd]	81.5±6.3[ae,be,ce,de]
Insulin (μU/mL)	14.0±9.1	7.9±3.5[ab]	7.6±3.3[ac]	7.2±3.4[ad]	5.6±1.8[ae,be,ce,de]
Homa index	3.2±2.2	1.8±0.9[ab]	1.6±0.8[ac]	1.6±0.8[ad]	1.1±0.4[ae,be,ce,de]
Triglycerides (mg/dL)	116.3±68.5	90.0±45.9[ab]	81±42.0[ac]	89.0±54.1[ad]	72.1±37.0[ae,be]
Total Cholesterol (mg/dL)	193.8±34.4	167.4±22.5	179.3±27.2	184.2±21.1	178.6±21.2
HDL (mg/dL)	53.2±12.1	51.0±11.6	53.0±12.6	54.4±9.5	53.1±10.0
ALT (U/l)	29.5±24.4	29.6±19.0	27.3±15.1	20.5±9.1[ad,bd,cd]	19.1±8.4[ae,be,ce]
AST (U/l)	28.6±6.8	21.0±5.0	20.2±4.8	18.5±4.4[ad,bd]	18.3±4.1[ae,be]
BUN (mg/dL)	36.5±7.0	37.9±7.1	39.4±7.6	39.7±10.5	36.2±9.1
Creatinine (mg/dL)	0.7±1.2	0.8±0.11	0.7±0.1	0.8±0.1	0.7±0.1
Uric acid (mg/dL)	4.5±1.5	4.5±1.4	4.6±1.5	4.2±1.1	4.3±1.1

Data are expressed as mean values ±SD

*the pairs of letters in the columns indicate statistical significance ($P < 0.05$) between the corresponding values.

Basciani S, Endocrine, 2014

Frequenza degli eventi avversi (%)



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ORIGINAL ARTICLE

Ketosis and appetite-mediating nutrients and hormones after weight loss

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BACKGROUND/OBJECTIVES: Diet-induced weight loss is accompanied by compensatory changes, which increase appetite and encourage weight regain. There is some evidence that ketogenic diets suppress appetite. The objective is to examine the effect of ketosis on a number of circulating factors involved in appetite regulation, following diet-induced weight loss.

SUBJECTS/METHODS: Of 50 non-diabetic overweight or obese subjects who began the study, 39 completed an 8-week ketogenic very-low-energy diet (VLED), followed by 2 weeks of reintroduction of foods. Following weight loss, circulating concentrations of glucose, insulin, non-esterified fatty acids (NEFA), β-hydroxybutyrate (BHB), leptin, gastrointestinal hormones and subjective ratings of appetite were compared when subjects were ketotic, and after refeeding.

RESULTS: During the ketogenic VLED, subjects lost 13% of initial weight and fasting BHB increased from (mean ± s.e.m.) 0.07 ± 0.00 to 0.48 ± 0.07 mmol/l ($P < 0.001$). BHB fell to 0.19 ± 0.03 mmol/l after 2 weeks of refeeding ($P < 0.001$ compared with week 8). When participants were ketotic, the weight loss induced increase in ghrelin was suppressed. Glucose and NEFA were higher, and amylin, leptin and subjective ratings of appetite were lower at week 8 than after refeeding.

CONCLUSIONS: The circulating concentrations of several hormones and nutrients which influence appetite were altered after weight loss induced by a ketogenic diet, compared with after refeeding. The increase in circulating ghrelin and subjective appetite which accompany dietary weight reduction were mitigated when weight-reduced participants were ketotic.

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Keywords: appetite; ketosis; very-low-energy diet; weight loss

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Journal of Nutritional Biochemistry

REVIEWS: CURRENT TOPICS

Biochemical and metabolic mechanisms by which dietary whey protein may combat obesity and Type 2 diabetes☆

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Whey protein decreases energy intake through mechanisms that influence appetite control.

Whey protein contains a high concentration of BCAAs, especially L-leucine.

Leucine enters the brain more rapidly than any other amino acid. It has recently been shown that intracerebroventricular injection of leucine is important for food intake suppression for 24 h, suggesting that whey protein may exert a central effect on appetite.

Elevation of dietary or brain leucine has been shown to suppress food intake via a mechanism involving mTOR, AMPK, and/or BCAA metabolism. Leucine reduces food intake via promoting mTOR signaling pathway in hypothalamus, especially in the region containing orexigenic neurons expressing both neuropeptide Y and agouti-related protein.

Other hormones are also involved in the regulation of food intake, either directly in the hypothalamus, such as ghrelin, or indirectly via the vagal nerve, such as cholecystokinin (CCK) and peptide YY (PYY).

Whey protein isolate influences energy balance and microbiota

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Protein Quality and the Protein to Carbohydrate Ratio within a
Influences Energy Balance and the Gut Microbiota In C57BL/6

Liam McAllan,[1,2] Peter Skuse,[1,3] Paul D. Cotter,[1,4] Paula O' Connor,[1] John F. Cryan,[4,5] R. Fitzgerald,[3] Helen M. Roche,[6] and Kanishka N. Nilaweera[1,*]

Darcy Johannsen, Editor

Diet is an important factor in determining the **composition** of the **gut microbiota** and specific gut microbiota signatures are associated with obesity phenotypes in animals and humans.

In particular **the class Clostridiales** are associated with the gut microbiota of animals fed a hight fat diet (HFD), while fasting reduces the levels of Clostridium. Notably, Clostridiaceae can produce short chain fatty acids as a product of their metabolism, which can play an important role in the regulation of immune cells and has been associated with inflammation and obesity. **Dietary whey protein isolate (WPI)** specifically normalises energy intake, increases lean mass, causes a trend towards a reduction in fat mass associated with prolonged high fat feeding and significantly **decreases Clostridiaceae/Clostridium.**

	HFD	20% WPI	30% WPI	40% WPI
Phylum				
Proteobacteria	0.36^{a}	0.63^{b}	0.34^{ab}	0.32^{a}
Actinobacteria	0.63^{a}	1.82^{b}	3.79^{b}	0.36^{c}
Deferribacteres	0.57^{a}	1.61^{b}	1.56^{ab}	2.03^{b}
Family				
Desulfovibrionaceae	0.12^{a}	0.31^{b}	0.21^{ab}	0.23^{ab}
Rikenellaceae	6.71^{ab}	7.54^{b}	3.9^{a}	6.4^{ab}
Bacteroidaceae	0.44^{a}	0.42^{a}	0.16^{b}	0.21^{b}
Lactobacillaceae	0.21^{a}	3.03^{b}	4.6^{b}	2.14^{b}
Bifidobacteriaceae	0.43^{a}	1.71^{b}	3.66^{b}	0.22^{c}
Deferribacteraceae	0.57^{a}	1.59^{b}	1.32^{ab}	2.03^{ab}
Peptostreptococcaceae	0.62^{a}	1.79^{a}	1.54^{a}	8.01^{b}
Succinivibrionaceae	0.13^{a}	0.15^{a}	0^{b}	0^{b}
Clostridiaceae	1.31^{a}	0^{b}	0^{b}	0^{b}
Veillonellaceae	0.02^{a}	0.12^{b}	0^{b}	0^{a}
Genus				
Anaerobiospirillum	0.13^{a}	0.15^{a}	0^{b}	0^{b}
Desulfovibrio	0.07^{a}	0.22^{b}	0.17^{ab}	0.15^{ab}
Alistipes	4.33^{ab}	4.41^{a}	2.24^{b}	3.76^{ab}
Rikenella	1.04^{ab}	0.49^{b}	0.68^{b}	1.08^{b}
Bacteroides	0.44^{a}	0.4^{a}	0.16^{b}	0.21^{ab}
Oscillibacter	0.24^{a}	0.67^{ab}	0.42^{ab}	0.52^{b}
Lactobacillus	0.2^{a}	3.03^{b}	4.6^{b}	2.39^{b}
Bifidobacterium	0.43^{a}	1.71^{b}	3.66^{b}	0.22^{c}
Mucispirillum	0.57^{a}	1.61^{b}	1.56^{ab}	1.92^{ab}
Coprococcus	0.11^{ab}	0.23^{b}	0.06^{a}	0.06^{ab}
Turicibacter	0.56^{a}	0.35^{a}	0.15^{ab}	0^{b}
Clostridium	1.3^{a}	0^{b}	0^{b}	0^{b}
Peptostreptococcus	0.1^{a}	0.14^{a}	0.12^{a}	0.78^{b}

[1]Data are means ± SEM (n = 10). Statistically significant differences generated using the Kruskal-Wallis algorithm. In each row values without a common letter significantly differ, $P \leq 0.05$.

Lavori in corso……

Studio aperto di confronto fra diete a basso contenuto di carboidrati in pazienti obesi.

Monocentrico, della durata di 3 mesi con un totale di 64 pazienti obesi, sarcopenici, affetti da Diabete di tipo II o iperinsulinismo suddivisi in 4 gruppi (16 pz/gruppo).

Gli obiettivi dello studio prevedono:

1. la valutazione della sicurezza e dell'efficacia di 4 programmi nutrizionali ipoglucidici, isoglucidici, con uno stesso quantitativo di grassi aggiunti, stessa quantità e qualità di fibre aggiunte, stessa quantità ma diversa qualità proteica. Si valuterà efficacia e sicurezza in termini di decremento ponderale, miglioramento dei parametri metabolici e in particolare miglioramento dell'assetto glicemico ed insulinemico, nonché gli effetti sulla composizione corporea;
2. la valutazione del grado di *compliance* dei pazienti nei confronti di tali programmi dietetici;
3. la valutazione degli effetti sul microbiota intestinale dei diversi piani alimentari.


CONCLUSIONI

L’approccio nutrizionale all’obesità si arricchisce oggi di un ulteriore valido strumento, costituito dall’uso di **nutraceutici sofisticati**, in grado di

In tal siddetti **pasti** , come dimost

In part **roteine del si** gli altri compo ficativo miglior lungo termin

Infine i essere considerato utile, non solo per il trattamento **dell’eccesso ponderale** con o senza **sindrome metabolica**, ma anche per la **riabilitazione** del paziente obeso verso un più **appropriato stile di vita**.