BRAIN AND MALNUTRITION
Chronic Diseases Association ONLUS

VI Congresso Nazionale B&M
Nutrizione e Neurodegenerazione

SESSIONE IV: RELATORI

- ***Dieta chetogenica nelle cefalee***

Dott. Cherubino Di Lorenzo
Medico Specialista presso il Polo Pontino dell'Università degli studi di Roma "La Sapienza"

MILANO | 11-12 MAGGIO | 2017

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DIETA CHETOGENICA NELLE CEFALEE

SAPIENZA
UNIVERSITÀ DI ROMA

25 OTTOBRE 2009 DON CARLO GNOCCHI BEATO
Fondazione Don Carlo Gnocchi Onlus

Cherubino Di Lorenzo

Dipartimento di Scienze medico-chirurgiche e Biotecnologie, Università La Sapienza Roma - Fondazione Don Gnocchi Onlus

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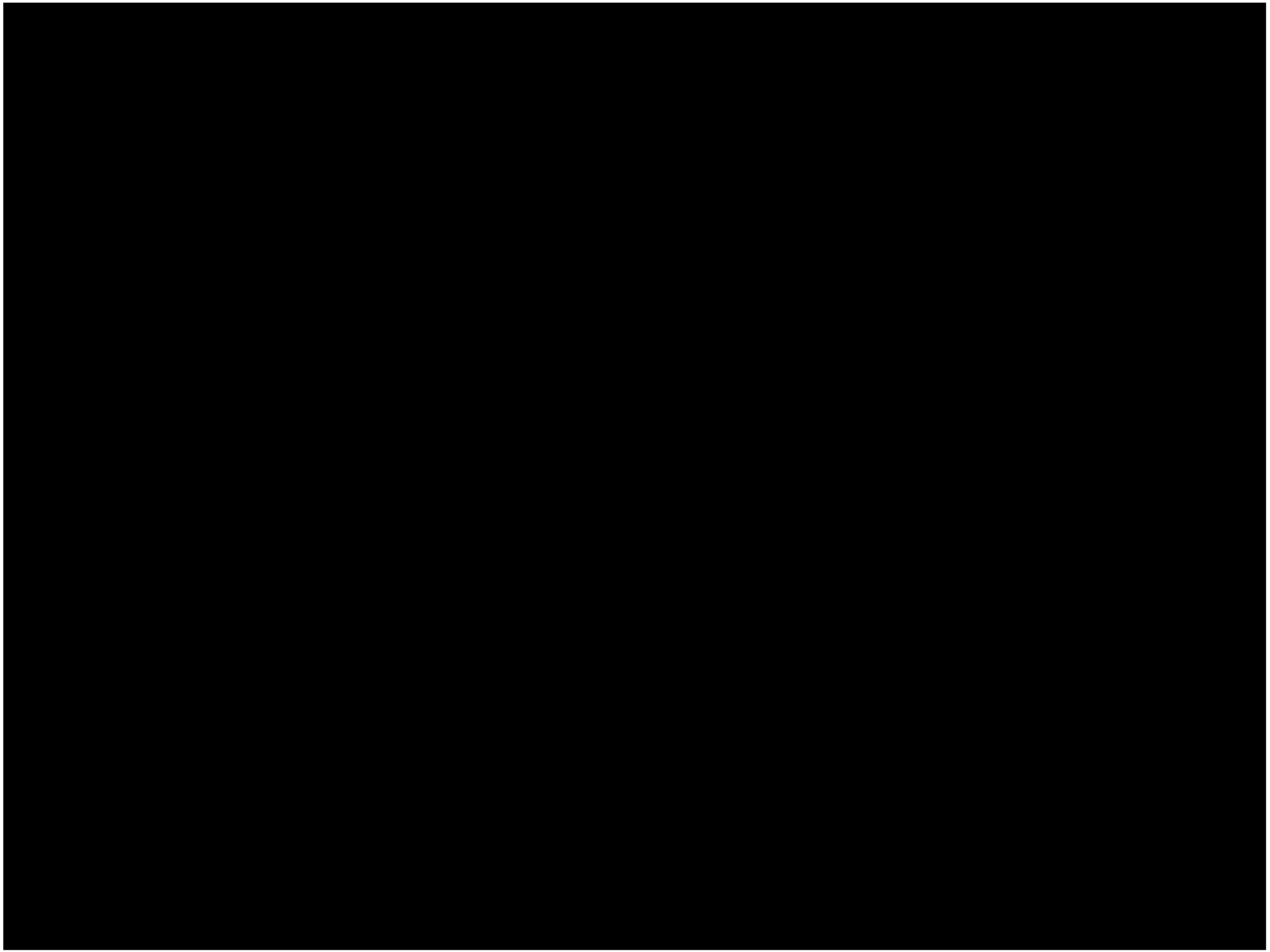

The long way of ketogenesis

Bible: Epilepsy can be cured with starvation and prayers

1921: diets c… epilep…

1928: "… ketogenic dietary in migraine

1997: "First Do No Harm " The Charlie Foundation.

2008: "The ketogenic diet: uses in epilepsy and other neurologic diseases" (suggestions for Alzheimer disease, Parkinson disease, amyotrophic lateral sclerosis, brain tumours and autism)

Mc. 9,28 "…hoc genus in nullo potest exire nisi in oratione et ieiunio"

(Fischer B, Gryson R, Weber R. Biblia sacra: iuxta vulgatam versionem (ISBN 3- 438-05303-9). Stuttgart: Deutsche Bibelgesellschaft, 1994)

Blood levels	*Normal diet*	*Ketogenic diet*	*Diabetic ketoacidosis*
Glucose (mg/dl)	80-120	65-80	>300
Insulin (µU/l)	6-23	6.6-9.4	≅ 0
Glucagon	Low	High	High
KB produc. (gr/day)	Low	115-180	400
KB conc. (mmol/dl)	0.1	4-10	>20
pH	7.4	7.4	<7.3

Therapeutic uses of ketogenic diets
A Paoli *et al*

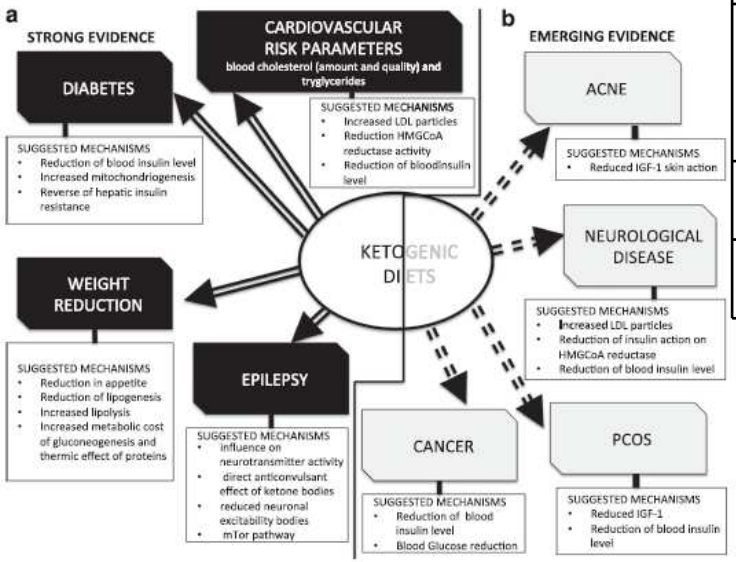

Figure 1. Suggested mechanisms for the therapeutic action of ketogenic diets in pathologies for which there exists strong (**a**) and emerging (**b**) evidence.

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Ketogenesis and Epilepsy

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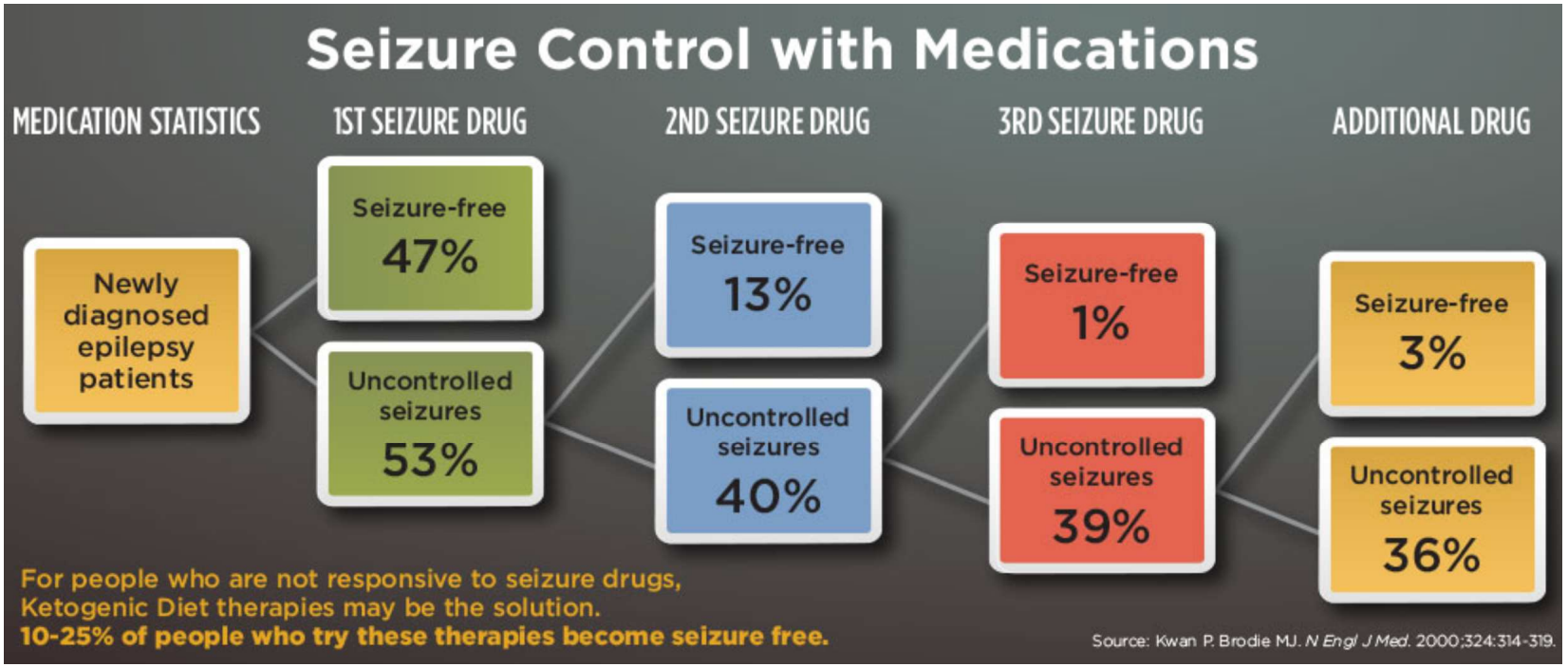
Ketogenic diet and Epilepsy

Why?



KD in GLUT1 and Pyruvate dehydrogenase deficit

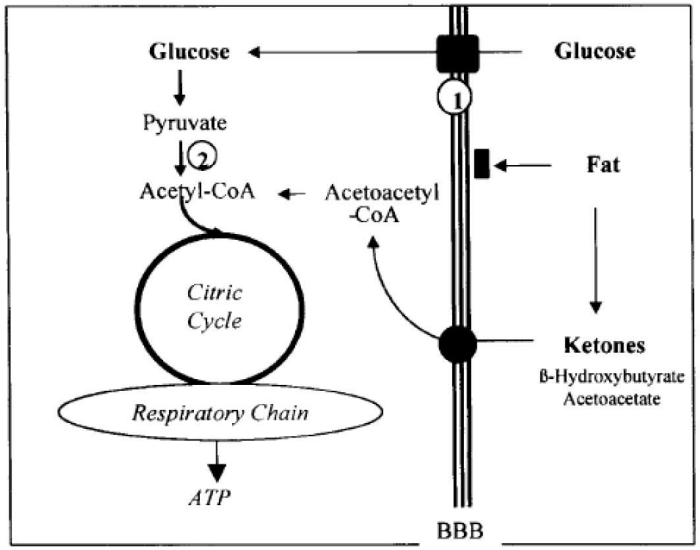
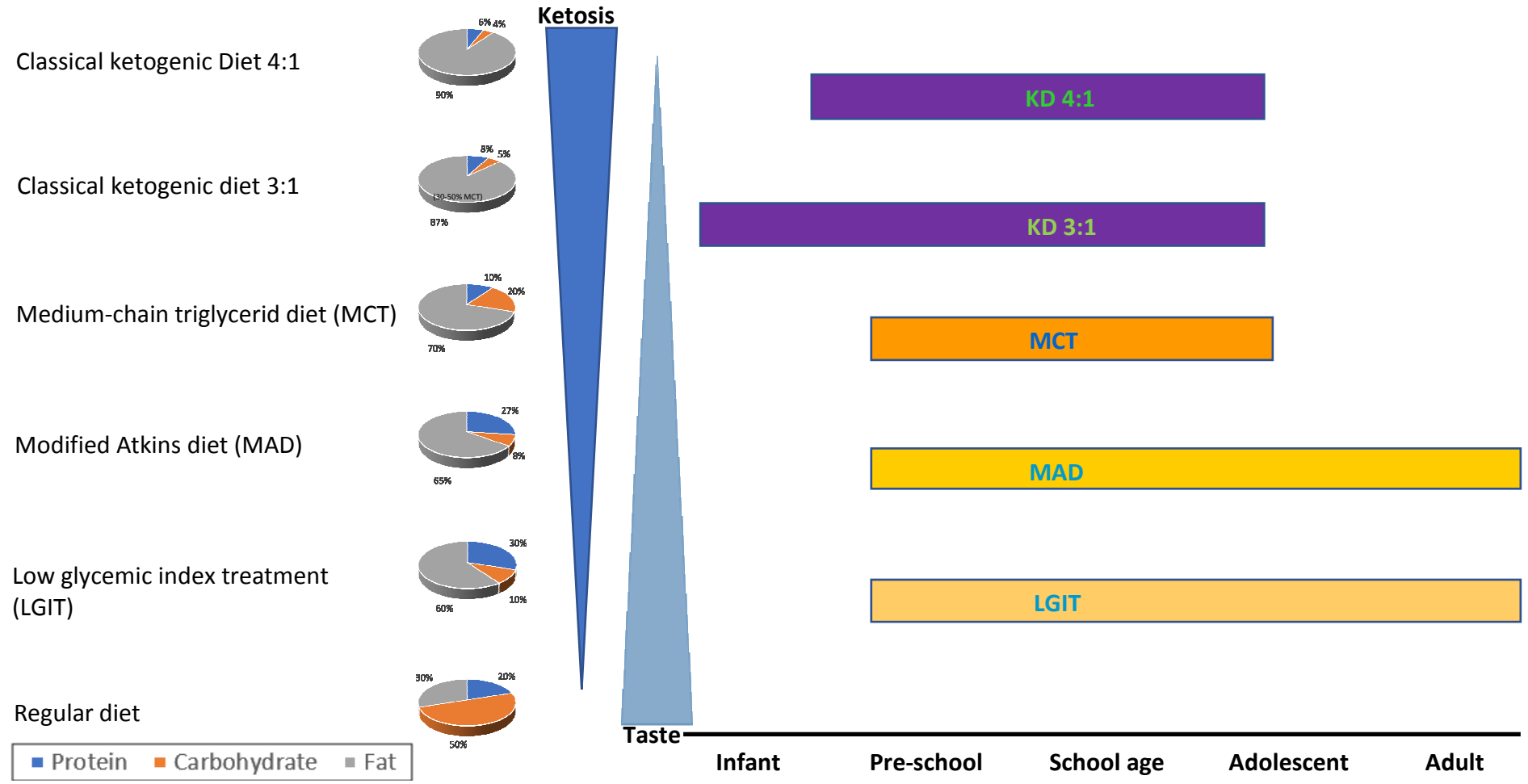


Figure 1 Ketosis and brain energy metabolism. Glucose enters the brain via the facilitated glucose transporter GLUT1 (■); ketones penetrate the blood–brain barrier (BBB) via the MCT1-transporter (●). Both substrates enter the citric acid cycle as acetyl-CoA for energy production. ① GLUT1 DS is caused by a defect in GLUT1-mediated glucose transport into brain. ② Pyruvate dehydrogenase deficiency impairs acetyl-CoA production. In both conditions, ketones bypass the transport/enzyme defect as acetoacetyl-CoA and provide acetyl-CoA.


Classical ketogenic Diet 4:1
6%
4%
90%
Classical ketogenic diet 3:1
8%
5%
(30-50% MCT)
87%
Medium-chain triglycerid diet (MCT)
10%
20%
70%
Modified Atkins diet (MAD)
27%
8%
65%
Low glycemic index treatment (LGIT)
30%
10%
60%
Regular diet
30%
20%
50%
Protein
Carbohydrate
Fat
Ketosis
Taste
KD 4:1
KD 3:1
MCT
MAD
LGIT
Infant
Pre-school
School age
Adolescent
Adult

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VI Congresso Nazionale B&M
Nutrizione e Neurodegenerazione

Ketogenesis and Migranie

Rediscovering an ancient treatment

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1928

An Experience with a Ketogenic Dietary in Migraine*

By TRUMAN G. SCHNABEL, M.D., *Philadelphia, Pa.*

THE practice of dietary restriction for the control of hemicranial attacks is well known and doubtless dates back to a time when men or perhaps more often women first experienced such crises. Some migraine victims, long before consulting a physician, have learned to associate their sickness with the ingestion of certain foods and have accordingly omitted them from their dietary often with satisfactory results. The practice has been further extended by physicians who interdict for their patients as nearly as feasibility permits some one of the three great food groups. Even this course has undoubtedly been suggested by patients themselves when they have offered the observation that an increased intake of either carbohydrates, proteins, or fats has been followed by sick headaches and that restriction in one of these three types of food has seemingly been of some benefit. Here and there, either on their own initiative or under direction, individuals have practiced either prolonged or periodic fasting for bilious headaches not without some good effect as it would seem at least to those who have gone through this experience. Up to the present time various explanations have been offered for the apparent effectivity of either complete or partial dietary restriction in the control of migraine, but many of these explanations are largely theoretical and are open to justifiable adverse criticism.

With a knowledge that diet would seem to have a relationship to the migraine attacks of some individuals it must have occurred to many that the starvation treatment as advocated in epilepsy by Guelpa and Marie (1) abroad and by Geylin in this country must have some reasonable logic in its application. When Wilder (2) in 1921 suggested a high fat diet for the treatment of epilepsy on the hypothesis that the ketone bodies are responsible for the favorable effect of starvation in epilepsy, the conviction of the logic of a ketogenic diet must also have been born home to those who had been observing migraine in relationship to diet. It was only when Peterman (3) in 1925 reported results in the treatment of epilepsy by ketogenic diet that the applicability of this type of diet in migraine suggested itself to me.

There seemed, however, at the time

*Read before the American College of Physicians, March 8, 1928, New Orleans, La.

VOLUME 95
NUMBER 24

1930

MIGRAINE

RESULTS OF TREATMENT BY KETOGENIC DIET IN FIFTY CASES *

CLIFFORD J. BARBORKA, M.D.
ROCHESTER, MINN.

Early in my experience with the ketogenic diet,[1] it was tried in cases of migraine. Since then Lennox and Cobb,[2] in their monograph on epilepsy, have stated that it would be of interest to know whether the induction of acidosis is of benefit in cases of migraine. Since the original report from the Mayo Clinic,[3] Schnabel [4] has reported his experience with a ketogenic diet in cases of migraine.

Some of the recent etiologic theories and therapeutic suggestions are of interest in considering the justification of a ketogenic regimen in cases of migraine. R. and S. Weissmann-Netter [5] found apparent changes in the acid-base balance: the hydrogen ion concentration and alkali reserve are normal in the periods of freedom from attention to the use of large doses of calcium lactate in an effort to lessen the irritability of the nerves.

The suggestion that migraine is sometimes a phenomenon of protein sensitization is not new. Pagniez [6] and his associates assumed that migraine is an anaphylactic manifestation. Miller and Raulston [9] continued the work in this country. Vaughan,[10] Rowe,[11] and many others have considered migraine from the standpoint of an allergic manifestation. Curtis-Brown [12] proposed the theory of inherited impaired metabolism with intolerance of nitrogenous foods, a protein-poison theory. They advocated various forms of treatment from the use of peptone to the restriction of certain proteins.

Chiray,[13] Duval,[14] Diamond,[15] Hetinyi,[16] McClure and Huntsinger,[17] and others have approached the problem from the standpoint of dysfunction of the liver and duodenum. They have called attention to biliary stasis as an etiologic factor. The French literature has emphasized duodenal migraine; in it evidence is presented of disturbed hepatic function as estimated

TABLE 1.—*Observations on Patients Whose Conditions Had Been Controlled*

Case	Age	Sex*	Attacks Before Treatment	Disease, Years	On Diet, Months	Ketosis	Comment
1	28	♀	About once a week, lasting two to three days	13	23	Always present	Attacks disappeared after two months
2	26	♀	Twice a week, lasting about two days.....	19	7	Always present	Headaches frequent first six weeks on diet; none since
3	29	♀	Two to three time a week, lasting twelve to eighteen hours	19	29	Periodic	Controlled after first two months
4	37	♀	Once a week, lasting twelve to thirty-six hours	27	9	Always present	First month on diet less severe headache every week; then entirely free since
5	33	♀	Two to three a month, lasting two to three days	8	23	Periodic	After first two months free from attacks
6	44	♀	At least two a month.....	32	16	Periodic	Attacks disappeared after two months
7	21	♀	One to two a week, lasting several hours...	4	14	Always present	After two months some relief; after three months arrested
8	58	♀	One to two a week, lasting one to two days, menopause at thirty-eight; no alteration in attacks	25	16	Always present	After two months no headaches
9	23	♀	Cyclic vomiting beginning at age of two years; migraine at 14; attacks about every five to six weeks	21	15	Always present	Controlled since ketosis developed
10	31	,,	Once and sometimes twice a week.....	21	18	Almost always present	No headaches after first three weeks
11	30	♀	Three to six weeks.....	17	13	Always present	Attacks controlled since being on diet
12	27	♀	One to three a week lasting about twelve hours	15	9	Periodic	Attacks controlled since fourth week on diet
13	54	♂	Cephalic migraine thirty-eight years; abdominal migraine last two years	38	36	Present one year, periodic since	Both abdominal and cephalic migraine disappeared after six weeks
14	25	♀	Every two to four weeks.....	8	0	Almost always present	One attack in first three months; none since

* In the tables, ♂ indicates male; ♀, female.

Serendipity!

2006

182 *January 2006*

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Syracuse, NY

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1. *BusinessWeek.* September 5, 2005; 78.
2. Silberstein SD, Neto W, Schmitt J, Jacobs D. Topiramate in migraine prevention. *Arch Neurol.* 2004;61:490-495.

induced and monitored on a weekly basis. A caloric restriction of 600 to 800 calories per day is maintained. Blood pressure, blood chemistries, and electrocardiograms are monitored regularly. Most patients stay in ketosis for 4 months or longer, depending on how much weight needs to be lost.

After going into ketosis, my wife went from having almost daily headaches to being completely free of migraines. Her last migraine was in late April, 2004. She maintained ketosis and the modified fast for almost 7 months and then went

Which kind of diet?

tory is significant for severe migraines on the paternal side.

The migraines were described as a "throbbing, burning, hot knife" sensation in one temple. During her adulthood, the headaches progressed and were occurring many times a week. She tried multiple lifestyle changes without any change in the frequency of the headaches. Exercise, dieting, and two pregnancies did not alter the frequency.

Numerous medications were prescribed by neurologists over the years. Agents that helped the most included Imitrex, Amerge, and Fioricet. Pharmacy profiles show that in 2004, at the age of 43, the patient was filling prescriptions about every 6 weeks for: Imitrex 50 mg #18, Amerge 2.5 mg #9, and Fioricet #30.

In an effort to lose the weight gained during pregnancy, the patient enrolled in a diet program under medical supervision. Patients undergo a modified fast, taking 3 to 4 high-protein, low-carbohydrate shakes a day. Each shake is 200 calories, and the shakes are the sole calorie source. Ketosis is

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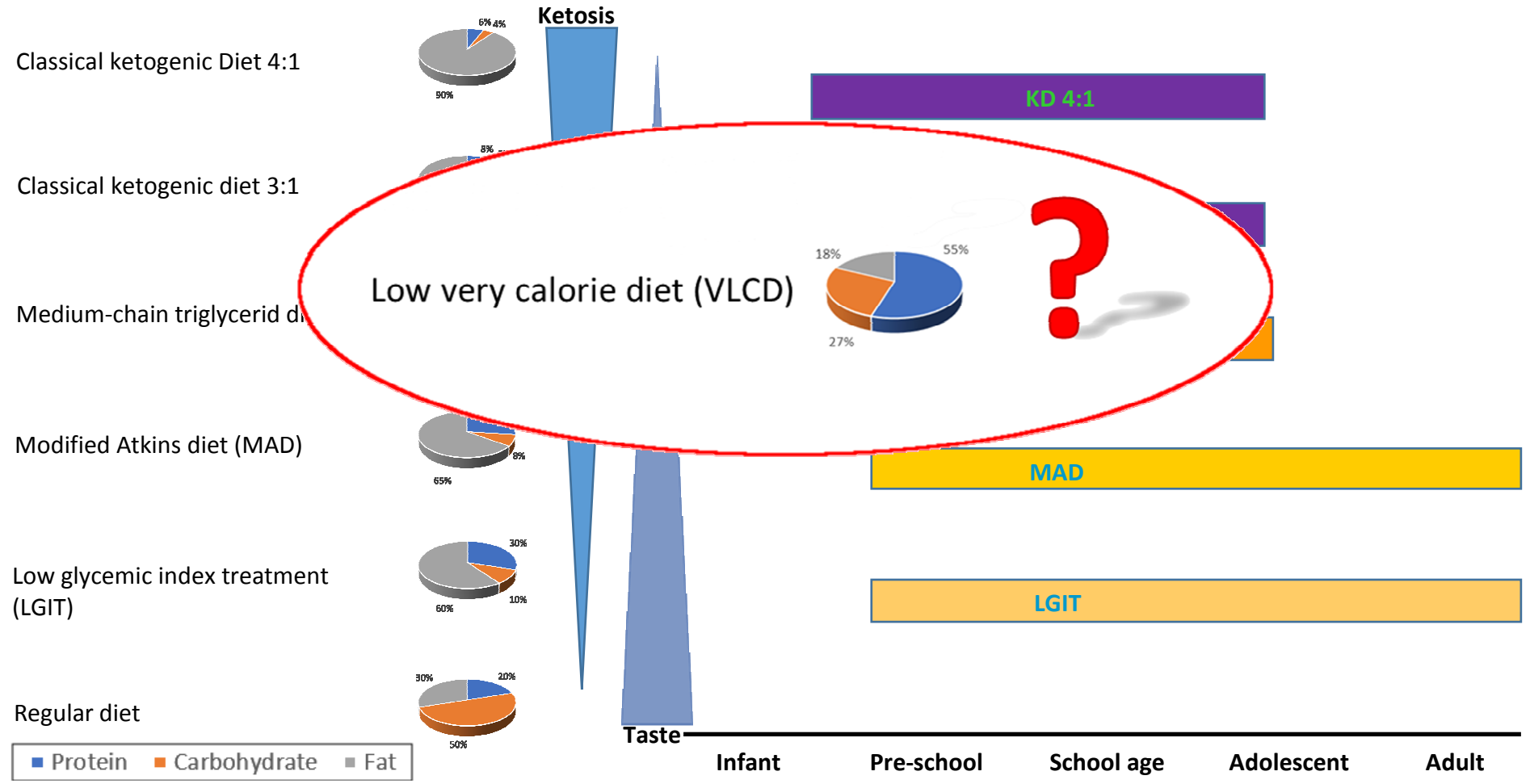
Ketosis
Classical ketogenic Diet 4:1
Classical ketogenic diet 3:1
Medium-chain triglycerid di
Modified Atkins diet (MAD)
Low glycemic index treatment (LGIT)
Regular diet
Low very calorie diet (VLCD)
KD 4:1
MAD
LGIT
Taste
Infant
Pre-school
School age
Adolescent
Adult
Protein
Carbohydrate
Fat

Very low calorie diet (VLCD)

- 400 – 850 Kcal/day
- Low carbohydrate: Ketogenic (VLCKD)
- Hihg carbohydrate: non-Ketogenic (?) (VLCD)

Is it a difficult diet?
No!

Water (2 lit./day) 

Protein supplement (10-15 gr X4) 

Up to 200 gr X2 (well dressed) 

100-200 gr/day 

(+ KCl & Mg)

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Nutrizione e Neurodegenerazione

Diet transiently improves migraine in two twin sisters: possible role of ketogenesis?

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Giulio Sirianni, MD[c]
Gianluca Coppola,PhD[d]
Martina Bracaglia, MD[e]
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Summary

The ketogenic diet is a high-fat, low-carbohydrate diet long used to treat refractory epilepsy; ketogenesis (ketone body formation) is a physiological phenomenon also observed in patients following low-carbohydrate, low-calorie diets prescribed for rapid weight loss.
We report the case of a pair of twin sisters, whose high-frequency migraine improved during a ketogenic diet they followed in order to lose weight.
The observed time-lock between ketogenesis and migraine improvement provides some insight into how ketones act to improve migraine.

KEY WORDS: ketogenic diet, migraine, prophylaxis, weight loss.

Patient Zero

Pt1	Months											
Days	January	February	March	April	May	June	July	August	September	October	November	December
1	3		Diet on	2		Diet on			Diet on			
2	3			2	2	2						
3	3				3							
4					2						2	
5		2									2	
6		3			3		2			2		
7	2				3		3			3		
8	3	3			2					2		
9	3	3							1'			
10		3							1'			
11	3							2				
12	3				3			2				
13		2			2			2				
14		3		2								2
15		3		3							2	1
16	3										2	
17	3									2	2	
18	2	3								2		
19												
20												
21												
22	3	2										2
23		3					2					2
24		3					2					3
25		3										
26	2	2										
27	3							2				2
28	3		Diet off	2		Diet off		2	Diet off		2	3
29				3							2	
30				1				2				
31								2				
	6/15/1.3	5/14/1.4	0/0/0	3/7/0.5	3/8/0.7	1/1/0.1	2/4/0.3	3/7/0.5	1/2/0.1	2/5/0.3	3/7/0.5	3/7/0.5

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Nutrizione e Neurodegenerazione

ORIGINAL ARTICLE

Migraine improvement during short lasting ketogenesis: a proof-of-concept study

C. Di Lorenzo[a], G. Coppola[b], G. Sirianni[c], G. Di Lorenzo[d], M. Bracaglia[e], D. Di Lenola[e], A. Siracusano[d], P. Rossi[f,g] and F. Pierelli[h]

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European Journal of Neurology 2015, **22:** 170–177

doi:10.1111/ene.12550

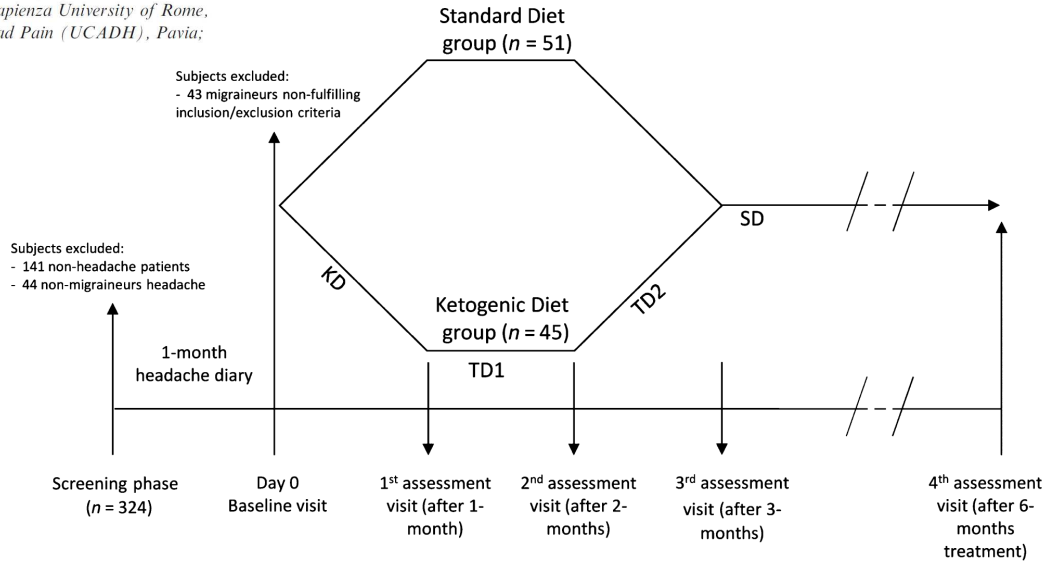

Figure 1 Outline of the study design. SD, standard low-calorie diet; KD, ketogenic very-low-calorie diet, supplemented by nutraceutical integrators; TD1, transitional diet supplemented by nutraceutical integrators; TD2, transitional diet without nutraceutical integrators.

ORIGINAL ARTICLE

Migraine improvement during short lasting ketogenesis: a proof-of-concept study

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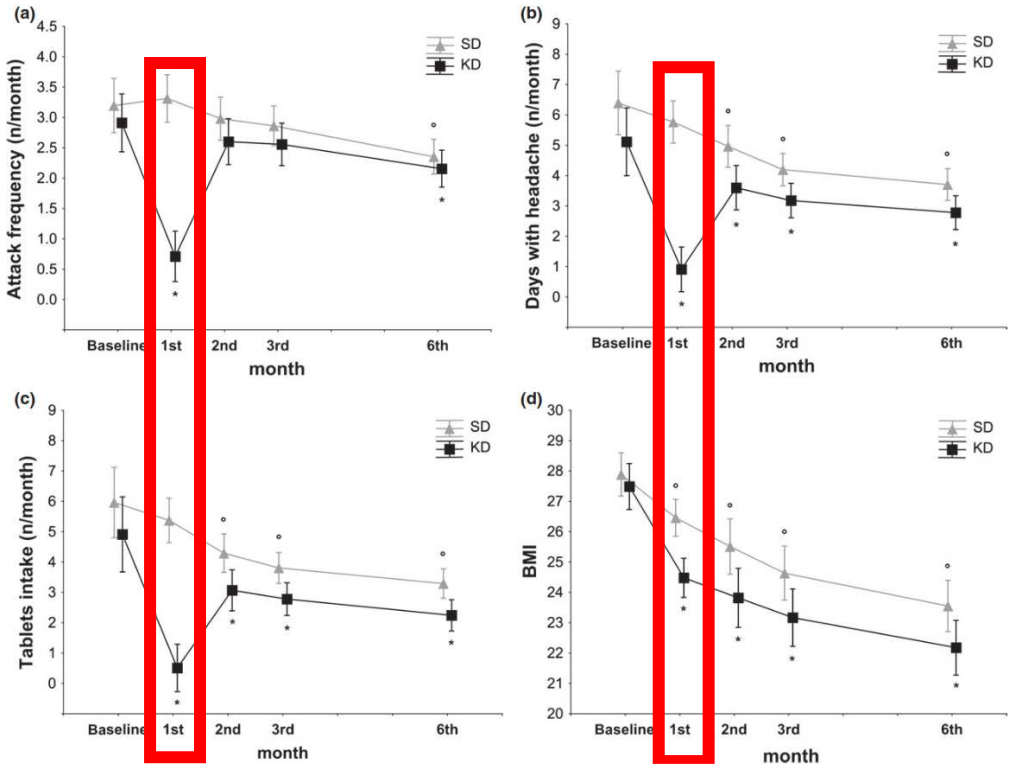

Warning!!!

It is noteworthy that weight increase is a common side effect of the most of preventive treatments for migraine. In particular, flunarizine and amitriptyline induces a weight increase related to higher levels of insulin, leptin and C-peptide [Berilgen et al., 2005], together with changes in the levels of hypothalamic orexinergic peptides [Caproni et al., 2010].

The increase of weight, insulin and leptin (leptin resistance?), although induced by migraine preventive drugs, could counteract the treatment and explain why in some cases these prophylactic therapies lose their efficacy, and potentially, on a long lasting, could worsen the preexisting migraine if the weight does not remain under control (the “prophylactic paradox”).

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Ketogenic Diet, Migraine and neurophysiology

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Schematic overview of hypotheses for an effect of the ketogenic diet in migraine prevention.

FFA : Free Fatty Acids

PUFA : Polyunsaturated Fatty Acids

PPAR : Peroxisome Proliferator-Activated Receptor

TCA : Tricarboxylic Acid Cycle

GABA: γ–Aminobutyric Acid

SNS : Sympathetic Nervous System

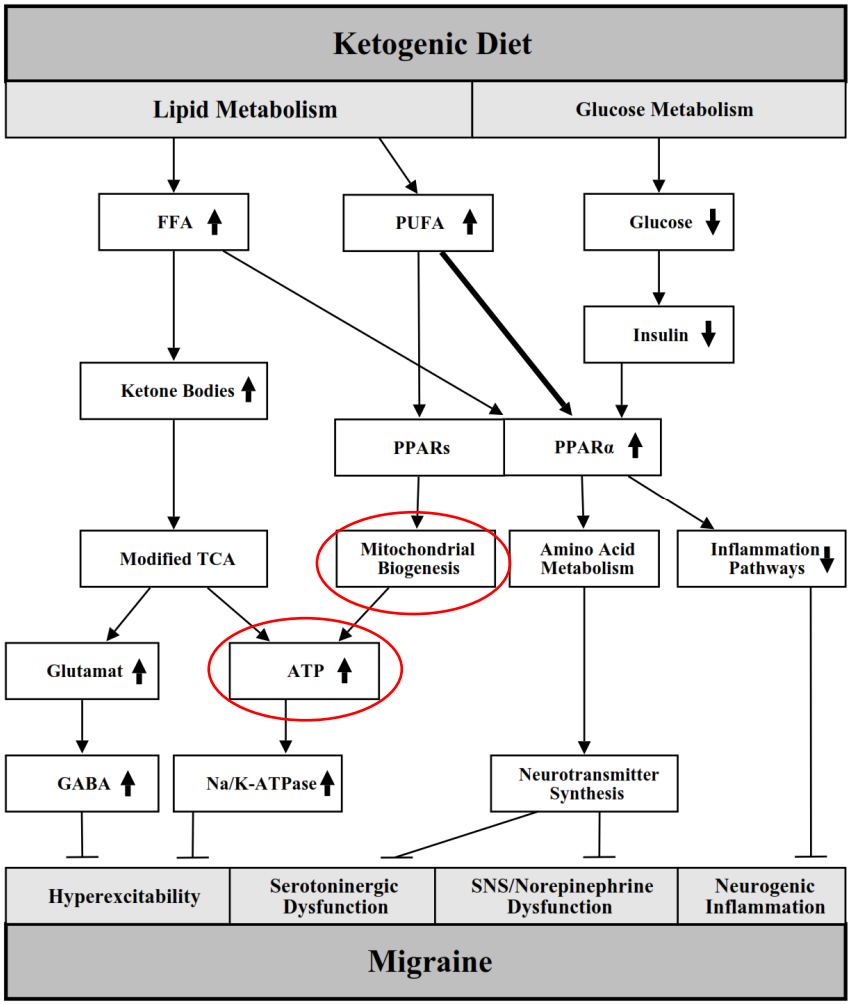
: Down-Regulation

: Up-Regulation

A → B : A affects B

A ⟹ B : A affects B strongly

A ⊣ B : A inhibits B



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Nutrizione e Neurodegenerazione

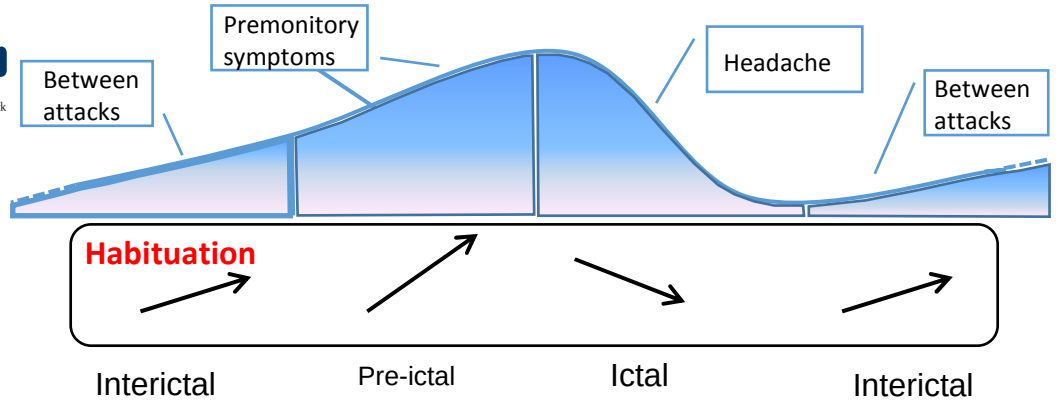
Di Lorenzo et al. The Journal of Headache and Pain (2016) 17:58
DOI 10.1186/s10194-016-0650-9

The Journal of Headache and Pain

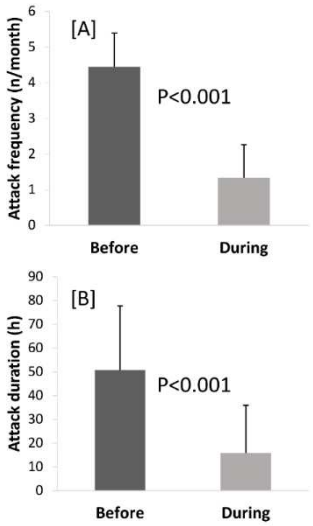
RESEARCH ARTICLE Open Access

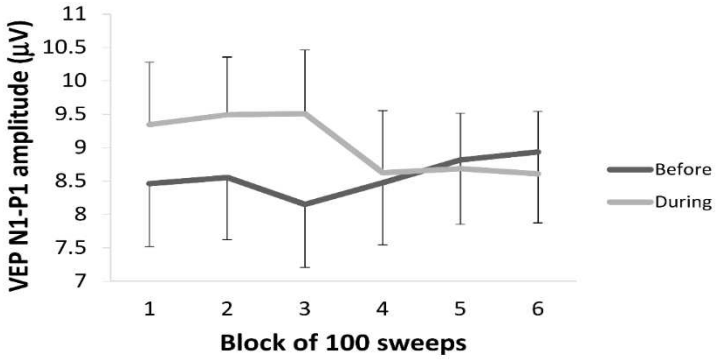
Cortical functional correlates of responsiveness to short-lasting preventive intervention with ketogenic diet in migraine: a multimodal evoked potentials study

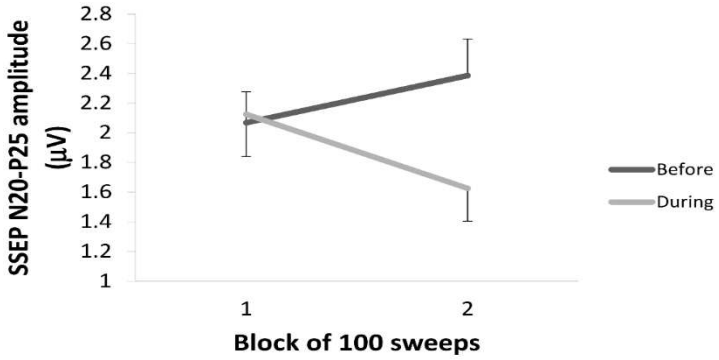
Cherubino Di Lorenzo[1*], Gianluca Coppola[2], Martina Bracaglia[3], Davide Di Lenola[3], Maurizio Evangelista[4], Giulio Sirianni[5], Paolo Rossi[6], Giorgio Di Lorenzo[7], Mariano Serrao[3], Vincenzo Parisi[2] and Francesco Pierelli[3,8]



Coppola et al., 2007; 201







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Conclusions

Far from considering our data suggestive for the introduction of KDs as an alternative prophylactic treatment for migraine, this observation may provide a useful strategy for migraineurs who need to treat headache, being overweight and/or weight increase resulting as side effect of cyclic prophylactic treatments.

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Ketogenesis and Cluster Headache

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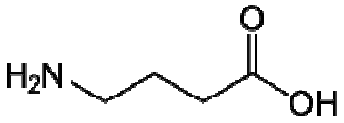
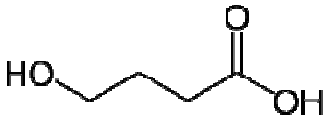
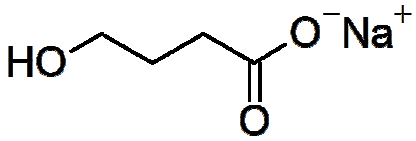
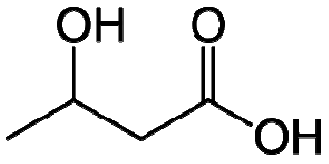
Ketogenic Diet and Cluster headache: why?

Ketogenic Diet and Cluster headache: why?

- Gamma-aminobutyric acid (GABA) 
- Gamma-hydroxybutyric acid (GHB) 
- Sodium Oxybate 
- Beta-hydroxybutyric acid (BHB) 

☐ Efficiency of **sodium oxybate** in episodic **cluster headache**.

1. Hidalgo H, Uhl V, Gantenbein AR, Sándor PS, Kallweit U.
Headache. 2013 Oct;53(9):1490-1. doi: 10.1111/head.12068. Epub 2013 Mar 6.
PMID: 23463909
Similar articles

☐ Targeting sleep disruption using **sodium oxybate** in chronic **cluster headache** prophylaxis.

2. Silberstein SD, Robbins MS.
Neurology. 2011 Jul 5;77(1):16-7. doi: 10.1212/WNL.0b013e3182231445. Epub 2011 May 25. No abstract available.
PMID: 21613603
Similar articles

☐ Long-term efficacy of **sodium oxybate** in 4 patients with chronic **cluster headache**.

3. Khatami R, Tartarotti S, Siccoli MM, Bassetti CL, Sándor PS.
Neurology. 2011 Jul 5;77(1):67-70. doi: 10.1212/WNL.0b013e31822313c6. Epub 2011 May 25.
PMID: 21613599
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Looking for information out of conventional channels…

Herbert Markley · 24.63 · 83.41 · University of Massachusetts Medical School

I have followed 4 patients with chronic cluster headache who became headache free as long as they could maintain a strict Atkins diet. They monitored their urinary ketosis with test strips. They all found that the headaches returned when they stopped spilling ketones, improved when they got ketotic again. All three gave up on this management because it was too taxing to follow the diet protocol. All three have done much better now with Botox.

Methods

We enrolled consecutive drug resistant (or not compliant) patients with cluster headache and propose them to follow a MAD for at least 12 weeks.

- Food low in carbohydrates were allowed
- Protein intake was about 0.7-1 gr/Kg/day
- Carbohydrate intake was about 10-50 gr/day
- KD ratio (fat:non-fat) about 1:1
- Lipid supplementation with specific products

Results

29 CH patients were recruited and underwent to a M.A.D.

- 11 ECH: 11/11 responders within 4 weeks.
- 18 CCH +2 (did not followed the diet for more than 3 consecutive days).
  - 15/18 responders (7 in 4 wks, 3 in 8 wks, 5 in 12 wks)
    - 11/15 crisis fully disappeared
    - 4/15 reduction of attacks >50%
    - 12/15 decided to continue the diet over the 12 weeks
    - 3/15 discontinued the diet (in one case, recurrence of headache in 7 weeks; in one case, recurrence in 10 weeks; the other, recurrence in 6 months)
  - 2/16 ineffectiveness during 12 weeks of diet
  - 1/16 early response, diet discontinuation before 12 weeks, recurrence after further 2 weeks, absence of response after diet restart (difficulty to stay in ketogenesis?).

Discussion

MAD ameliorated CH in the most of patients, both episodic and chronic, in absence of any relevant side effect (blood test were performed before, at 4th, and 12th week od diet).

Patients were strongly motivated to perform the diet, maybe this is the reason of their very high compliance.

Our preliminary results seem to be promising, further studies are auspicated.

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Thank you for your kind attention

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