Int J Biochem Cell Biol. 2015 Jun;63:55-9. doi: 10.1016/j.biocel.2015.01.022. Epub 2015 Feb 7.

Mitochondria: The ketogenic diet-A metabolism-based therapy.

Vidali S[1], Aminzadeh S[1], Lambert B[2], Rutherford T[2], Sperl W[3], Kofler B[4], Feichtinger RG[1].

Author information

Abstract

Mitochondria are the energy-producing organelles of the cell, generating ATP via oxidative phosphorylation mainly by using pyruvate derived from glycolytic processing of glucose. Ketone bodies generated by fatty acid oxidation can serve as alternative metabolites for aerobic energy production. The ketogenic diet, which is high in fat and low in carbohydrates, mimics the metabolic state of starvation, forcing the body to utilize fat as its primary source of energy. The ket
transporter type 1 and py
is a hallmark of cancer c
the antitumor effect of ch
Therapies.

Cancer Metab. 2015 Mar 25;3:3. doi: 10.1186/s40170-015-0129-1. eCollection 2015.

Treatment of glioma patients with ketogenic diets: report of two cases treated with an IRB-approved energy-restricted ketogenic diet protocol and review of the literature.

Schwartz K[1], Chang HT[2], Nikolai M[3], Pernicone J[4], Rhee S[4], Olson K[5], Kurniali PC[1], Hord NG[6], Noel M[7].

Author information

Abstract

BACKGROUND: Based on the hypothesis that cancer cells may not be able to metabolize ketones as efficiently as normal brain cells, the ketogenic diet (KD) has been proposed as a complementary or alternative therapy for treatment of malignant gliomas.

CASE PRESENTATION: We report here our experience in treating two glioma patients with an IRB-approved energy-restricted ketogenic diet (ERKD) protocol as monotherapy and review the literature on KD therapy for human glioma patients. An ERKD protocol was used in this pilot clinical study. In addition to the two patients who enrolled in this study, we also reviewed findings from 30 other patients, including 5 patients from case reports, 19 patients from a clinical trial reported by Rieger and 6 patients described by Champ. A total of 32 glioma patients have been treated using several different KD protocols as adjunctive/complementary therapy. The two patients who enrolled in our ERKD pilot study were monitored with twice daily measurements of blood glucose and ketones and daily weights. However, both patients showed tumor progression while on the ERKD therapy. Immunohistochemistry reactions showed that their tumors had tissue expression of at least one of the two critical mitochondrial ketolytic enzymes (succinyl CoA: 3-oxoacid CoA transferase, beta-3-hydroxybutyrate dehydrogenase 1). The other 30 glioma patients in the literature were treated with several different KD protocols with varying responses. Prolonged remissions ranging from more than 5 years to 4 months were reported in the case reports. Only one of these patients was treated using KD as monotherapy. The best responses reported in the more recent patient series were stable disease for approximately 6 weeks. No major side effects due to KD have been reported in any of these patients.

CONCLUSIONS: We conclude that 1. KD is safe and without major side effects; 2. ketosis can be induced using customary foods; 3. treatment with KD may be effective in controlling the progression of some gliomas; and 4. further studies are needed to determine factors that influence the effectiveness of KD, whether as a monotherapy, or as adjunctive or supplemental therapy in treating glioma patients.

Surg Neurol Int. 2015 Apr 16;6:61. doi: 10.4103/2152-7806.155259. eCollection 2015.

The role of metabolic therapy in treating glioblastoma multiforme.

Maroon JC[1], Seyfried TN[2], Donohue JP[1], Bost J[1].

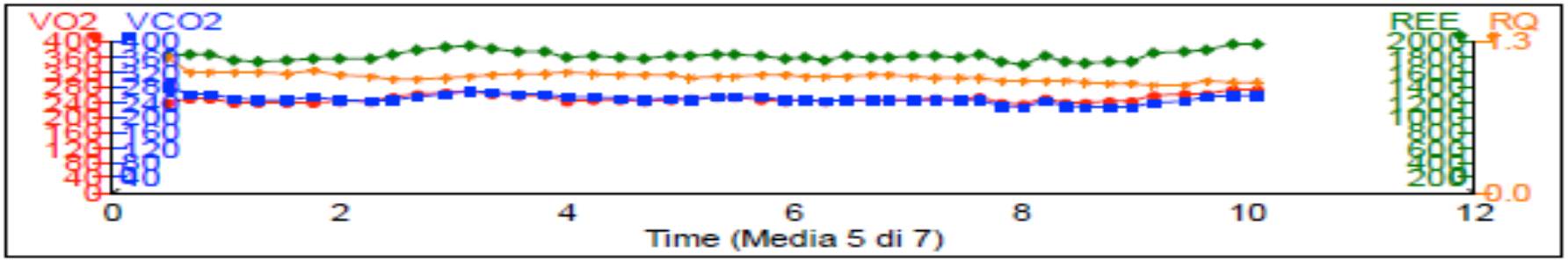
Author information

Abstract

Glioblastoma multiforme (GBM) is an aggressive and nearly uniformly fatal malignancy of the central nervous system. Despite extensive research and clinical trials over the past 50 years, very little progress has been made to significantly alter its lethal prognosis. The current standard of care (SOC) includes maximal surgical resection, radiation therapy and chemotherapy and temozolomide (TMZ), including the selective use of glucocorticoids for symptom control. These same treatments, however, have the potential to create an environment that may actually facilitate tumor growth and survival. Research investigating the unique metabolic needs of tumor cells has led to the proposal of a new metabolic treatment for various cancers including GBMs that may enhance the effectiveness of the SOC. The goal of metabolic cancer therapy is to restrict GBM cells of glucose, their main energy substrate. By recognizing the underlying energy production requirements of cancer cells, newly proposed metabolic therapy is being used as an adjunct to standard GBM therapies. This review will discuss the calorie restricted ketogenic diet (CR-KD) as a promising potential adjunctive metabolic therapy for patients with GBMs. The effectiveness of the CR-KD is based on the "Warburg Effect" of cancer metabolism and the microenvironment of GBM tumors. We will review recent case reports, clinical studies, review articles, and animal model research using the CR-KD and explain the principles of the Warburg Effect as it relates to CR-KD and GBMs.

Nome	[redacted]iovanni	ID:	010447gd	BSA:	1.93	Data:	31/03/2009
Tech:		Altezza:	169.00	Età:	61	Prov.:	
Dott.:	Armando D'Orta	Peso:	82.00	Sesso:	M	Etnia:	Caucasica

Diagnosis:
Dyspnea: Cough: Wheeze:
Tbco Prod: Yrs Smk: Pks/Day: Yrs Quit:
Medications:
Pre Test Comments:
Post Test Comments:



	Valore Medio	PRED
Time (min)	10:05	
FIO2 (%)		

REE (Kcal/day)	1813	1628
RQ	1,00	
REE/Pred (%)	111	

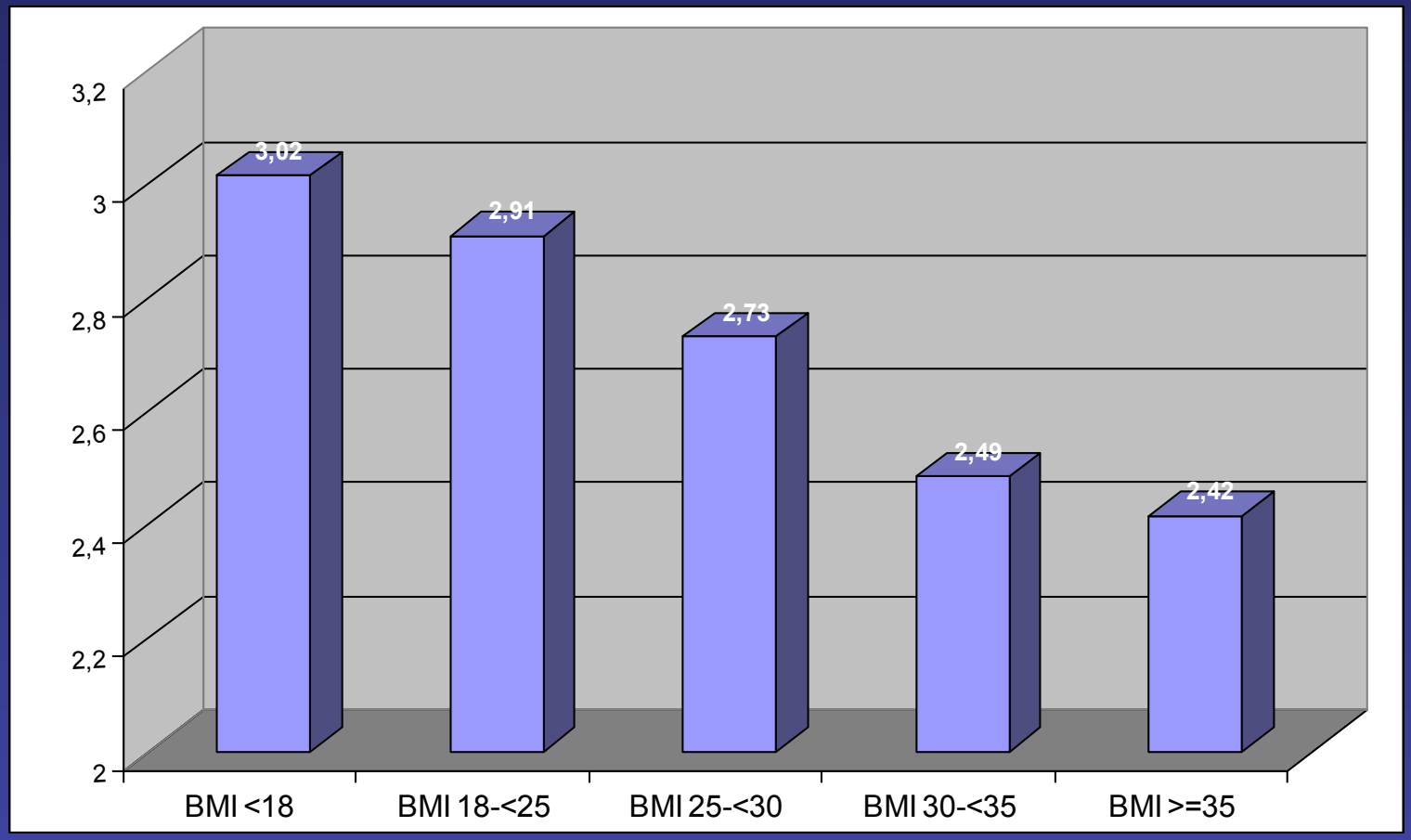
RR (br/min)	14,04	
Vt BTPS (mL)	736	
VE BTPS (L/min)	10,2	

VCO2 (mL/min)	249	
VO2 (mL/min)	249	

Urinary N2 (g/day)		
CHO/REE (%)	99	
Fat/REE (%)	1	
Prot/REE (%)		

REE_Covar (%)	3	

Il volume di Ossigeno/kg/min in rapporto al BMI (Centro Regionale di Fisiopatologia della Nutrizione ASL Teramo)



Enpab

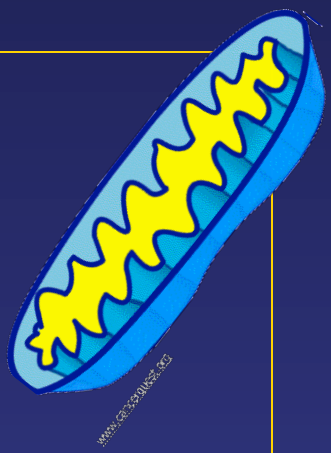
1 g di glucosio= ↓ 0,8 l di O_2 e ↑ 0,8 l di CO_2.
RQ (CO_2/O_2) del glucosio è = 1.

1 g di proteine = ↓ 0,97 l di O_2 e ↑ 0,78 l di CO_2.
RQ delle proteine è = 0,8.

1 g di lipidi = ↓ 2 l di O_2 e ↑ 1,4 l di CO_2.
RQ dei grassi è = 0,7.

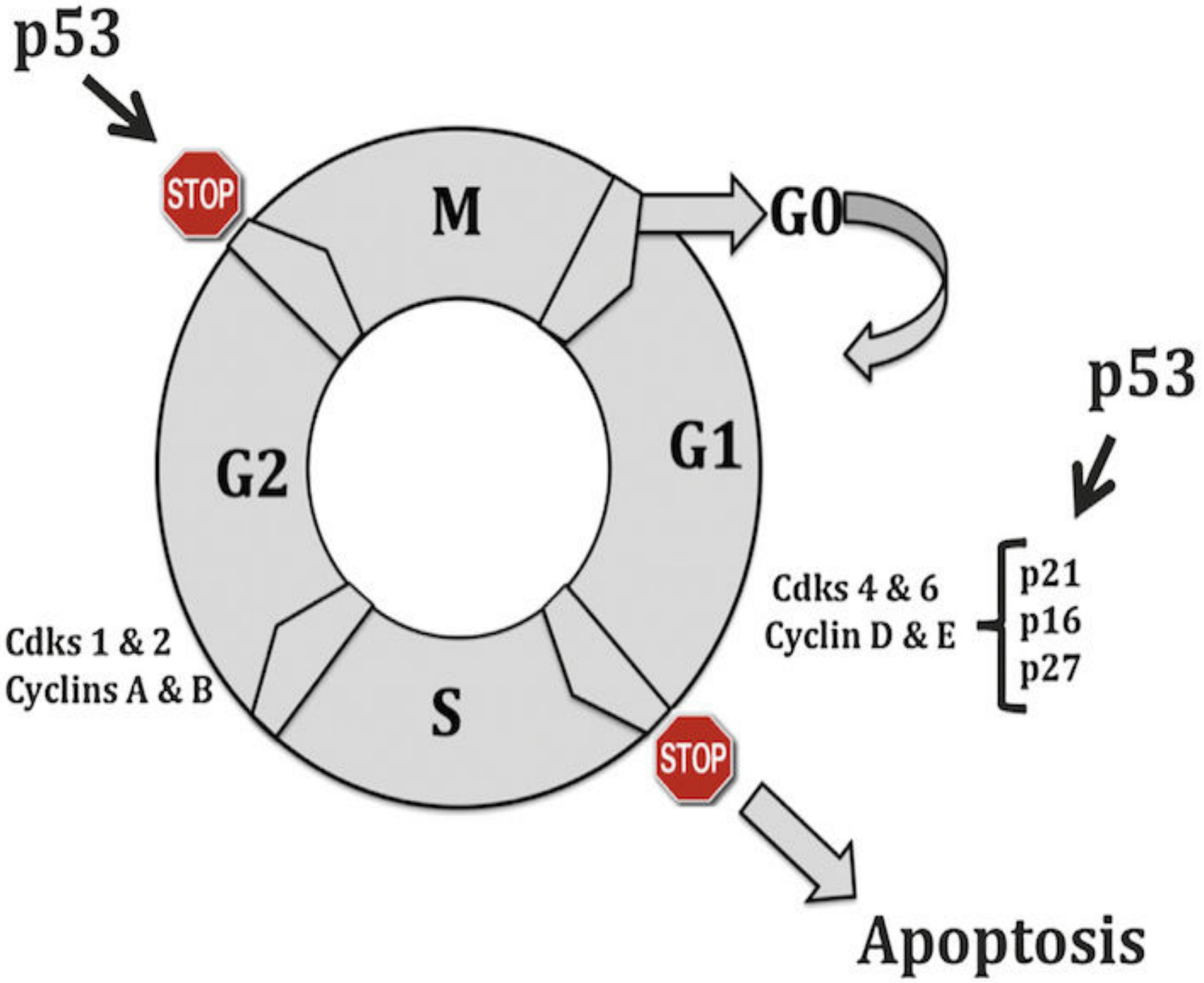
Enpab

- Il Quoziente Respiratorio
- Atti Respiratori / minuto
- Variazione del consumo di O2
- Variazione del Metabolismo



… sono espressione del numero e grandezza dei Mitocondri (Potenza Metabolica)

Enpab


p53
STOP
M
G0
G1
G2
S
STOP
p53
Cdks 4 & 6
Cyclin D & E
p21
p16
p27
Cdks 1 & 2
Cyclins A & B
Apoptosis

1 . SWITCH metabolico – 2. mutazioni acquisite

Trasformazione

3. Insulina: Insulin Like Grow Factos (IGF-1, somatomedine etc)

Proliferazione

Inflammation and insulin resistance

Steven E. Shoelson, Jongsoon Lee, and Allison B. Goldfine

Author information ▶ Copyright and License information ▶

This article has been corrected. See J Clin Invest. 2006 August 1; 116(8): 2308.
This article has been corrected. See J Clin Invest. 2006 August 1; 116(8): 2308.

Abstract

Go to:

Over a hundred years ago, high doses of salicylates were shown to lower glucose levels in diabetic patients. This should have been an important clue to link inflammation to the pathogenesis of type 2 diabetes (T2D), but the antihyperglycemic and antiinflammatory effects of salicylates were not connected to the pathogenesis of insulin resistance until recently. Together with the discovery of an important role for tissue macrophages, these new findings are helping to reshape thinking about how obesity increases the risk for developing T2D and the metabolic syndrome. The evolving concept of insulin resistance and T2D as having immunological components and an improving picture of how inflammation modulates metabolism provide new opportunities for using antiinflammatory strategies to correct the metabolic consequences of excess adiposity.

4. ESPLOSIONE di citochine infiammatorie chemiotattiche, in particolar modo Tumor Necrosis Factor – alpha (TNF-α)

Invasività locale e metastatica

Effetti del Digiuno Terapeutico

- Niente digestione ! (Effetto laminare)
- Desaturazione dei recettori insulinici (effetto anti down-regulation)
- Riduzione della insulino-resistenza

Enpab

Effetti del Digiuno Terapeutico

- **Riduzione della glicolisi con riattivazione del mitocondrio**
- **Ripristino dell'anabolismo (Autofagia)**
- **Ripristino della fluidità di membrana grazie all'uso degli Oli**

Enpab

Dieta Chetogena… Vegetale

Dieta Chetogena Vegetale

- Estrattore di succhi verdi
- Passati di Verdure
- Olio
- Acqua

L'INCREDIBILE ILLUSIONE: SANI IN UN MONDO MALATO