

Dietoterapia e approcci innovativi

Elisa Mazza

RD, PhD

Dipartimento di Scienze Mediche e Chirurgiche

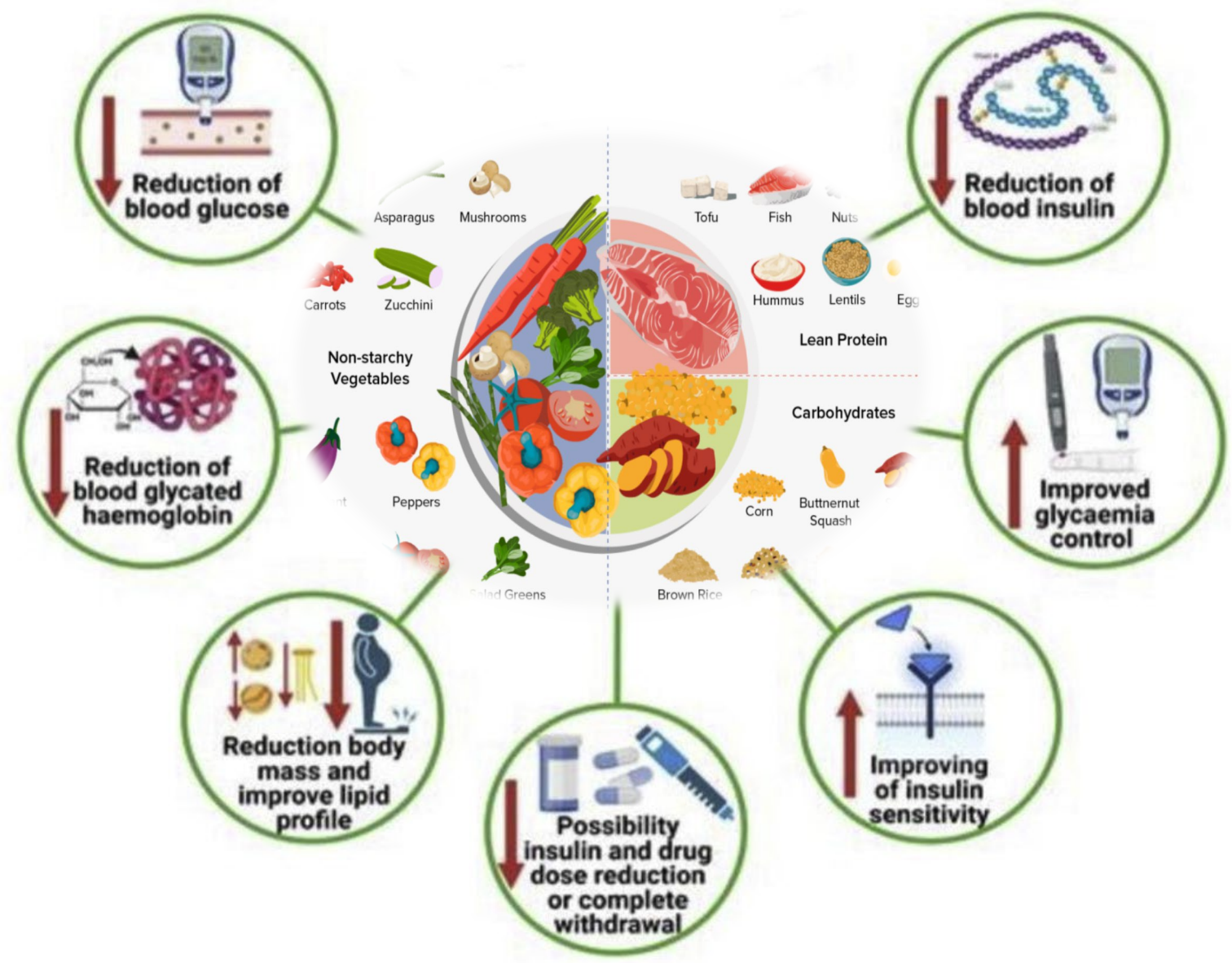
Università Magna Græcia di Catanzaro

La sottoscritta Elisa Mazza dichiara di NON aver ricevuto negli ultimi due anni compensi o finanziamenti da Aziende Farmaceutiche e/o Diagnostiche

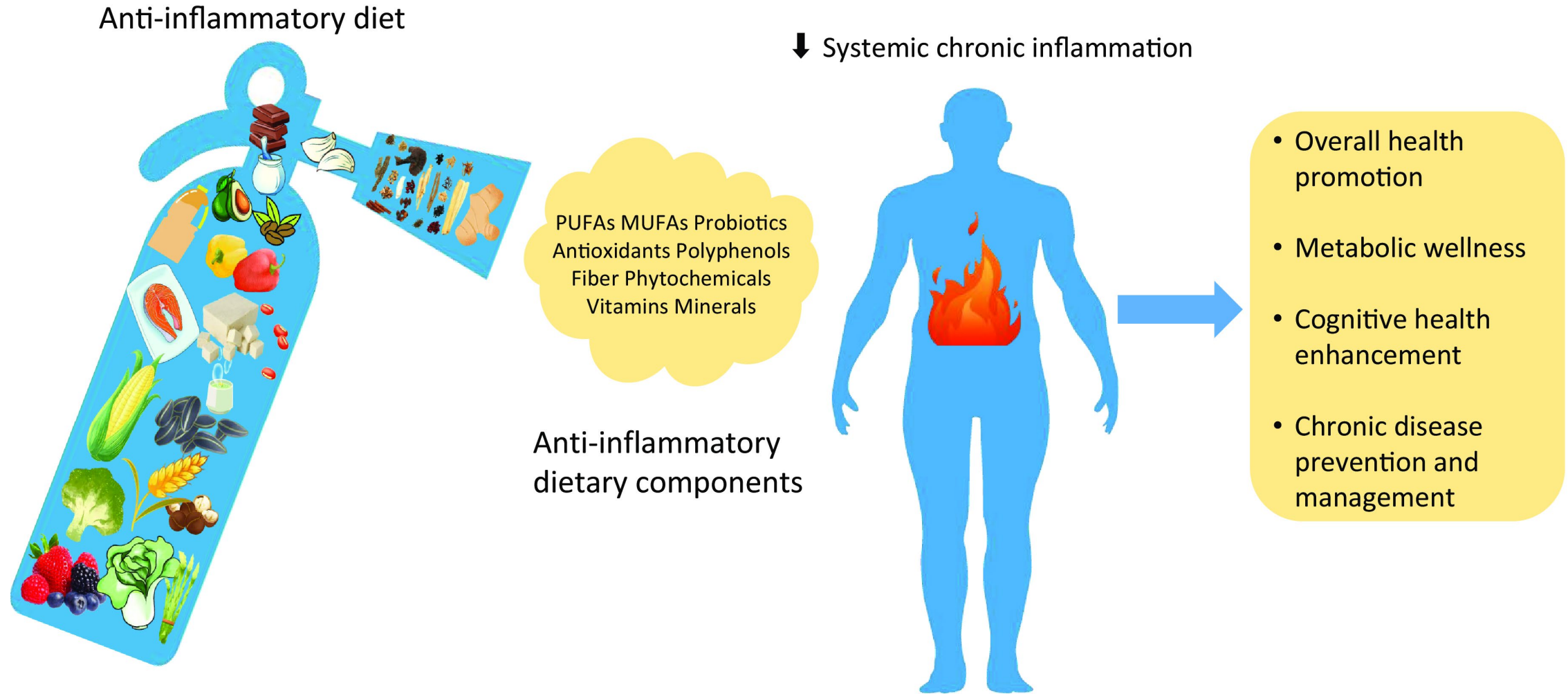
Dichiara altresì il proprio impegno ad astenersi, nell'ambito dell'evento, dal nominare, in qualsivoglia modo o forma, aziende farmaceutiche e/o denominazione commerciale e di non fare pubblicità di qualsiasi tipo relativamente aspecifici prodotti di interesse sanitario (farmaci, strumenti, dispositivi medico-chirurgici, ecc.).

A handwritten signature in black ink, appearing to read 'Elisa Mazza', with a stylized, cursive script.

The Role of Nutrition in the Treatment of Diabetes



ANTI-INFLAMMATORY DIET AND CHRONIC INFLAMMATION IN DM



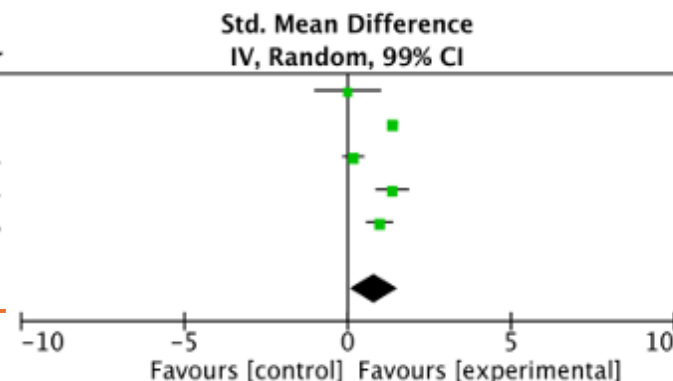
The Effect of Dietary Patterns on Inflammatory Biomarkers in Adults with Type 2 Diabetes Mellitus: A Systematic Review and Meta-Analysis of Randomized Controlled Trials

Abril I. Sánchez-Rosales ¹, Ana L. Guadarrama-López ², Laura S. Gaona-Valle ³, Beatriz E. Martínez-Carrillo ¹ and Roxana Valdés-Ramos ^{1,*} 

Study or Subgroup	Experimental			Control			Std. Mean Difference			Year
	Mean	SD	Total	Mean	SD	Total	Weight	IV, Random, 99% CI	Std. Mean Difference IV, Random, 99% CI	
Golan Rachel	7.3	2.8	15	7.3	2.6	12	15.9%	0.00 [-1.00, 1.00]		2012
L. Maria Belalcazar	7.9	3.1	922	3.1	3.9	836	21.9%	1.37 [1.23, 1.51]		2012
Dylan Thomson	5.79	1.7	248	5.5	1.7	99	21.3%	0.17 [-0.14, 0.48]		2014
Lasa, A.	7.9	3.1	74	3.1	3.9	67	20.2%	1.36 [0.88, 1.85]		2014
Maiorino Maria Ida	8.87	1.9	102	7.1	1.7	97	20.8%	0.98 [0.59, 1.36]		2016
Total (99% CI)			1361			1111	100.0%	0.81 [0.06, 1.56]		

Heterogeneity: $\tau^2 = 0.38$; $\chi^2 = 96.28$, $df = 4$ ($P < 0.00001$); $I^2 = 96\%$

Test for overall effect: $Z = 2.80$ ($P = 0.005$)



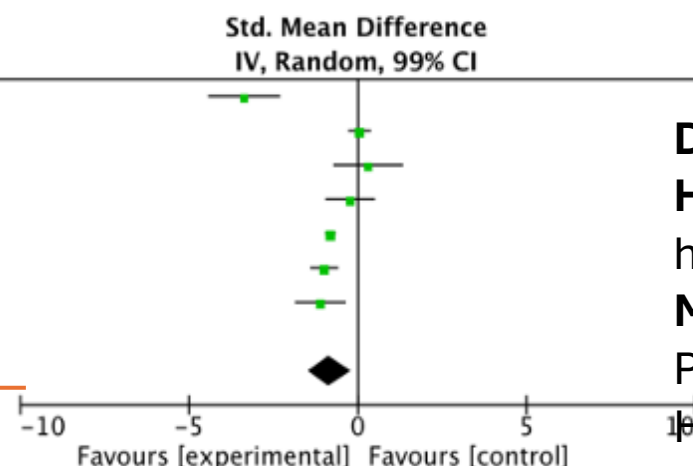
SOLO PER:
Mediterranean Diet,
Diabetes Prevention
Program, American Heart
Association's Therapeutic
Lifestyle Changes diet

(A) Adiponectin

Study or Subgroup	Experimental			Control			Std. Mean Difference			Year
	Mean	SD	Total	Mean	SD	Total	Weight	IV, Random, 99% CI	Std. Mean Difference IV, Random, 99% CI	
Azadbakht Leila	2.04	0.2	31	2.91	0.3	31	11.8%	-3.37 [-4.41, -2.33]		
Dylan Thomson	1.68	3	248	1.53	3.4	99	16.1%	0.05 [-0.26, 0.35]		
Golan Rachel	4.6	3.4	15	3.6	2.9	12	12.0%	0.30 [-0.70, 1.31]		
Itsiopoulos C.	2.38	0.46	27	2.49	0.5	27	14.0%	-0.23 [-0.93, 0.48]		
L. Maria Belalcazar	2.8	1.35	922	4.02	1.64	836	16.6%	-0.82 [-0.94, -0.69]		
Maiorino Maria Ida	1.94	0.78	102	2.84	1.01	97	15.7%	-1.00 [-1.38, -0.61]		
Sauder, Katherine	1.98	0.16	30	2.16	0.16	30	13.9%	-1.11 [-1.83, -0.39]		
Total (99% CI)			1375			1132	100.0%	-0.83 [-1.49, -0.17]		

Heterogeneity: $\tau^2 = 0.39$; $\chi^2 = 104.17$, $df = 6$ ($P < 0.00001$); $I^2 = 94\%$

Test for overall effect: $Z = 3.26$ ($P = 0.001$)

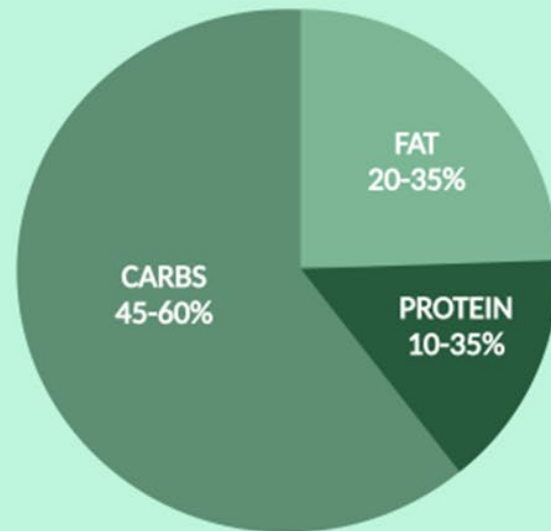


**Dietary Approaches to Stop
Hypertension, Diabetes UK
healthy eating guidelines,
Mediterranean Diet, Diabetes
Prevention Program, American
Heart Association's Therapeutic
Lifestyle Changes diet**

(B) CRP

APPROCCI INNOVATIVI - KETOGENIC DIET

Regular Diet



Glucose



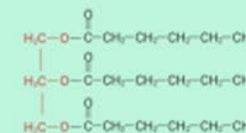
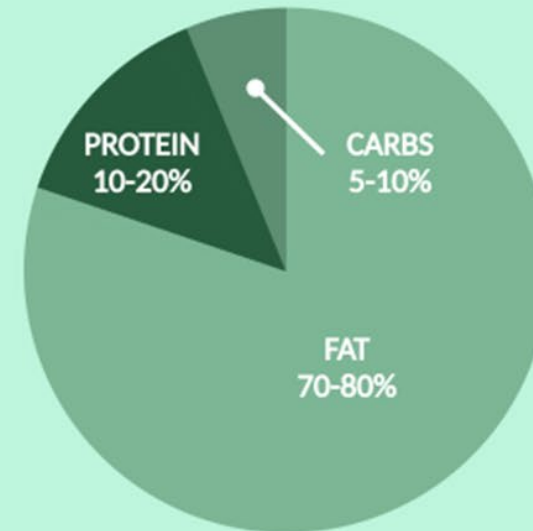
glucose levels rise

pancreas secretes insulin

insulin shuttles glucose into cell

energy

True Ketogenic Diet



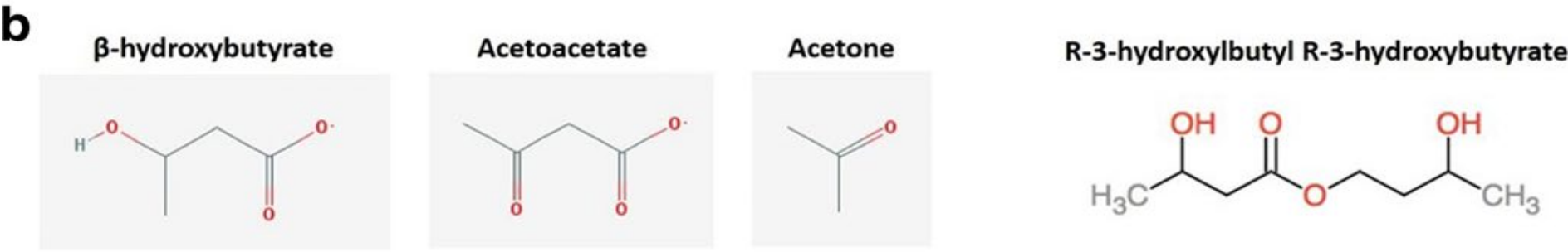
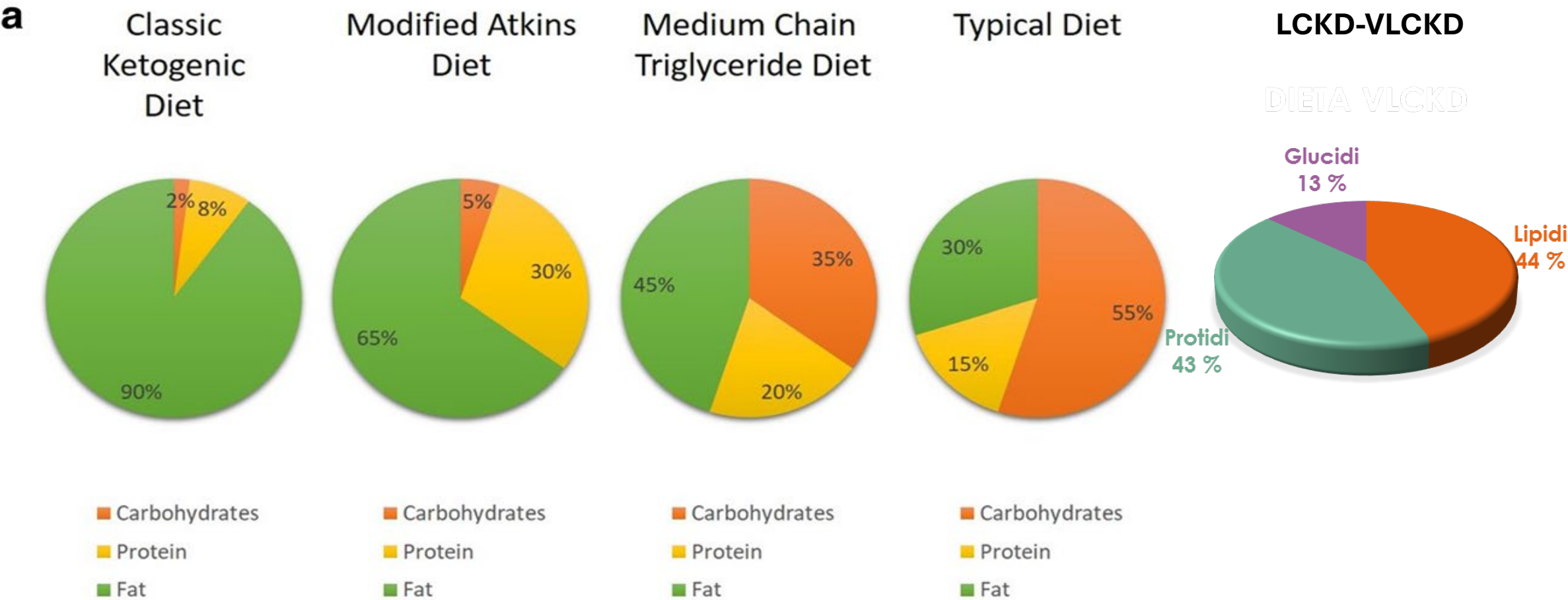
glucose levels fall

lipase releases stored triglycerides

fatty acids travel to liver

liver produces ketones

PROTOCOLLI DI DIETA CHETOGENICA



VARIANTI DELLA DIETA CHETOGENICA LOW-CALORIE

Table 1. Characteristics of classical ketogenic diet (KD), low-calorie ketogenic diet (LCKD) and very low-calorie ketogenic diet (VLCKD).

	KD	LCKD	VLCKD
Caloric intake	Normocaloric	800–1200 Kcal/day	<800 Kcal/day
Carbohydrate (%)	5–10	13	13
Protein (%)	15–20	29	44
Fat (%)	70–80	58	43
Foods	Vegetable oils, fish, eggs, meat, cheese, olives, avocado, coconut	Natural high biological value proteins (1–2 servings) including meat, fish, eggs, processed meat	Replacement meals with high biological value proteins composed by 18 g of proteins, 4 g of carbohydrates and 3 g of fats
Recommendations for use [1]	• Epilepsy and resistance to antiepileptic therapy • Gliomas and glioblastomas • Neurodegenerative diseases (Alzheimer or Parkinson's disease) • Neurocognitive disorders • Brain trauma	• Obesity BMI 25–35 kg/m² • Obesity associated with arterial hypertension or type 2 diabetes mellitus or hypertriglyceridaemia or heart failure or polycystic ovary syndrome • Paediatric obesity with epilepsy and/or insulin resistance	• Severe obesity • Obesity complicated by type 2 diabetes or arterial hypertension or hypertriglyceridaemia or metabolic syndrome or OSAS or arthropathies • Obesity with indication of bariatric surgery • Adolescents with severe obesity

FASI DEL PROTOCOLLO VLCKD-LCKD

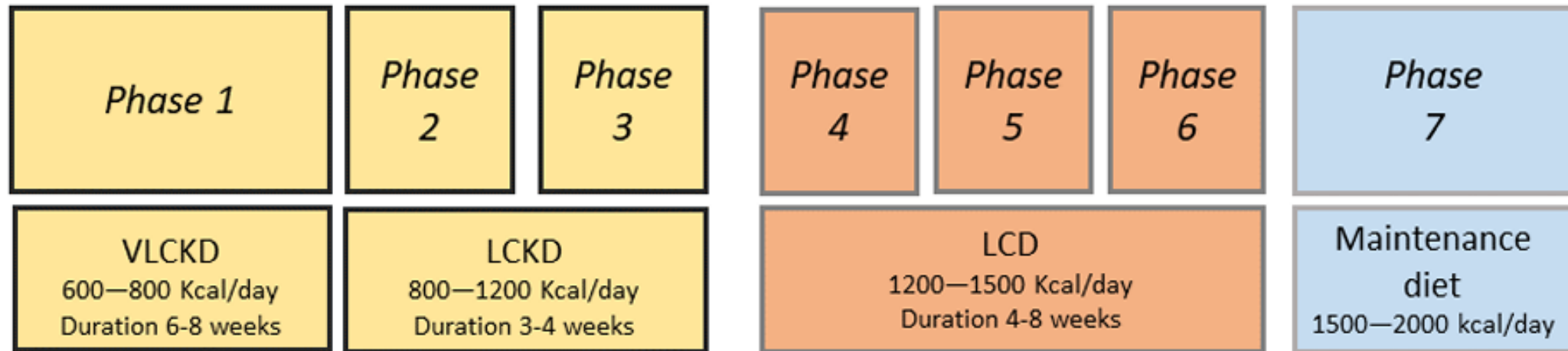
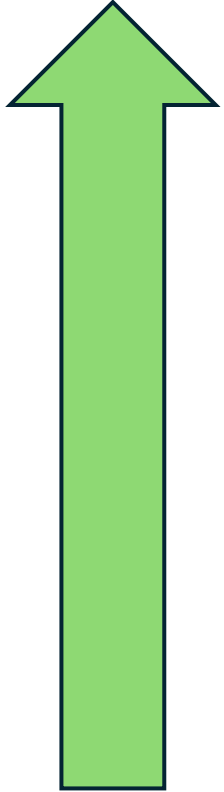


Figure 1. Phases of very low-calorie ketogenic diet (VLCKD) protocol. LCKD: low-calorie ketogenic diet. LCD: low carbohydrate diet.

Effetti in campo metabolico



Soppressione dell'appetito

Riduzione della lipogenesi

Aumento della lipolisi

Riduzione del grasso viscerale

Aumento dei costi metabolici della gluconeogenesi e dell'effetto termico delle proteine

Riduzione dei fattori infiammatori

DM1 e L-CarbDs

RESEARCH ARTICLE

Low-carbohydrate diets for type 1 diabetes mellitus: A systematic review

Jessica L. Turton¹, Ron Raab², Kieron B. Rooney^{3*}

FLCD: false low-carbohydrate diet= **130g/d**;
TLCD: true low-carbohydrate diet= **50–130g/d**;
VLCKD: Very low-carbohydrate ketogenic diet = **<50g/d**

Table 2. Effect of intervention and comparator diets on type 1 diabetes management outcomes (primary and secondary).

Study ID	I/C ^a	CHO ^b	Class ^c	Follow-up ^d	Pre (units) ^e	Post (units) ^e	Significance ^f	
							Within Group	Between Groups
Haemoglobin A1c (%HbA1c (mmol/mol))								
Anderson 1991	I	221 ± 35 (~39%)	FLCD	4 weeks (<i>n</i> 8)	9.0 ± 0.5 (75 ± 5.5)	8.1 ± 0.4 (65 ± 4.4)	NS	NS
	C	363 ± 76 (~68%)	-	4 weeks (<i>n</i> 8)	9.0 ± 0.5 (75 ± 5.5)	8.0 ± 0.3 (64 ± 3.3)	NS	
Chantelau 1982	I	156 ± 46 (34 ± 5%)	FLCD	4–6 months (<i>n</i> 10)	9.7 ± 1.9 (83 ± 20.8)	7.3 ± 0.5 (56 ± 5.5)	P < 0.0025	-
Ireland 1992	I	~87 (22 ± 6%)	TLCD	2 weeks (<i>n</i> 8)	11.1 ± 1.7 (98 ± 18.6)	11.6 ± 2.3 (103 ± 25.1)	NS	-
Knight 2016	I	162 (143–204) (42 ± 7%)	FLCD	12 months (<i>n</i> 46)	7.9 ± 1.2 (63 ± 13.1)	7.9 ± 1.6 (63 ± 17.5)	NS	-
Krebs 2016	I	103 ± 22 (~30%)	TLCD	12 weeks (<i>n</i> 5)	7.9 ± 0.9 (63 ± 9.8)	7.2 ± 0.4 (55 ± 4.4)	NS	NS
	C	203 ± 92 (~44%)	-	12 weeks (<i>n</i> 5)	7.4 ± 0.9 (57 ± 9.8)	7.4 ± 0.9 (57 ± 9.8)	NS	
Nielsen 2012	I _A	≤75 (15–20%)	TLCD	4 years (<i>n</i> 48)	7.6 ± 1.0 (60 ± 10.9)	6.9 ± 1.0 (52 ± 10.9)	P < 0.001	-
	I _B	-	-	4 years (<i>n</i> 23)	7.7 ± 1.0 (61 ± 10.9)	6.4 ± 0.8 (46 ± 8.7)	P < 0.001	-
O'Neill 2003	I	30	VLCKD	18 months (<i>n</i> 8)	6.8 ± 1.1 (51 ± 12.0)	5.5 ± 0.8 (37 ± 8.7)	P = 0.003	-
Vernon 2003	I	20–50	VLCKD	3 months (<i>n</i> 1)	16.8 (160)	5.3 (34)	NA	-
Severe Hypoglycemia (episodes/year)								
Bernstein 1980	I	15%	TLCD	5 years (<i>n</i> 1)	730.0 ^g	12.0	NA	-
Knight 2016	I	162 (143–204) (42 ± 7%)	FLCD	12 months (<i>n</i> 46)	3.7 ± 15.7	0.2 ± 1.1	P = 0.014	-
Total Daily Insulin (units/day)								
Bernstein 1980	I	15%	TLCD	5 years (<i>n</i> 1)	80.0	25.0	NA	-
Ireland 1992	I	~87 (22 ± 2%)	TLCD	2 weeks (<i>n</i> 8)	41.1 ± 3.5	35.3 ± 4.1	P < 0.05	-
Krebs 2016	I	103 ± 22 (~30%)	TLCD	12 weeks (<i>n</i> 5)	66.4 ± 25.3	44.2 ± 16.5	P < 0.05	P < 0.05
	C	203 ± 92 (~44%)	-	12 weeks (<i>n</i> 5)	40.6 ± 7.8	44.8 ± 12.4	NS	
Nielsen 2012	I	≤75 (15–20%)	TLCD	1 year (<i>n</i> 36)	42.6 ± 10.3	31.6 ± 8.5	NA	-
O'Neill 2003	I	30	VLCKD	8–61 months (<i>n</i> 10)	47.0	30.0	NA	-
Body Mass Index (kg/m²)								
Krebs 2016	I	103 ± 22 (~30%)	TLCD	12 weeks (<i>n</i> 5)	27.5 ± 2.2	25.8 ± 1.0	NS	NS
	C	203 ± 92 (~44%)	-	12 weeks (<i>n</i> 5)	27.7 ± 6.2	27.6 ± 6.1	NS	
Nielsen 2012	I	≤75 (15–20%)	TLCD	4 years (<i>n</i> 48)	25.9 ± 3.5	25.7 ± 3.8	NS	-
Vernon 2003	I	20–50	VLCKD	3 months (<i>n</i> 1)	20.5	23.3	NA	-
Mean Blood Glucose (mmol/L)								
Krebs 2016	I	103 ± 22 (~30%)	TLCD	12 weeks (<i>n</i> 5)	10.2 ± 2.3	8.9 ± 0.8	NS	NS
	C	203 ± 92 (~44%)	-	12 weeks (<i>n</i> 5)	9.3 ± 1.9	10.1 ± 2.9	NS	

Turton, J. L., Raab, R., & Rooney, K. B. (2018). PloS one, 13(3), e0194987.

Table 1. Effect of ketogenic diets as prescribed for children with type 1 diabetes.

Gender and Age	Disease	Dietary Intervention	Benefits	Adverse Effects	References
Boy, 4 years	Type 1 diabetes and myoclonic-astatic epilepsy	Ketogenic diet	1. Acceptable control of epileptic seizures 2. Improvement of cognitive functions 3. Maintenance of the target glycemia values	No severe episodes of hypoglycemia or ketoacidosis were observed	[56]
Girl, 14 years	Type 1 diabetes	Ketogenic diet	1. Significant improvement of subjective sensations 2. Significant improvement in glycemia	Not observed	[57]
Girl, 3.5 years	Type 1 diabetes, right hemiparesis, epilepsy	Ketogenic diet	1. Absence of epileptic seizures 2. Improvement of development, motor functions, and activity 3. Proper glycemic control and improvement in the HbA1c value	1 episode of ketoacidosis, apart from which no adverse effects were observed	[58]
A 9-year-old child (no information on gender)	Type 1 diabetes	Paleolithic ketogenic diet	1. Improvement of insulin and glucose levels 2. Discontinuation of insulin treatment	Not observed	[59]
Girl, 2 years	Type 1 diabetes and epilepsy	Ketogenic diet	1. No episodes of epileptic seizures 2. Improvement (reduction) of glycated hemoglobin level 3. No new episodes of diabetic ketoacidosis	Mild hypoglycemia episodes	[60]
Girl, 4 years	Pyruvate dehydrogenase deficiency, diabetic ketoacidosis, static encephalopathy, convulsive disorders	Ketogenic diet	1. Proper glycemic control 2. Improvement of activity level 3. Significant developmental achievement 4. Compensation of linear growth from < 5th to the 50th percentile	No major adverse effects were observed	[9]

KD BAMBINI DM1
EFFETTI

Table 2. Effect of a ketogenic diet in adults with type 1 diabetes.

Gender and Age	Disease	Dietary Intervention	Benefits	Adverse Effects	References
Group of 17 women and 7 men. Mean age 51 ± 10 years	Type 1 diabetes	Low-carbohydrate diet (70–90 g carbohydrates)	1. Significant reduction in hypoglycemia episodes and glycated hemoglobin concentration (from 7.5% to 6.4%), 2. Significant reduction in the postprandial requirement for insulin (from 21.1 IU daily to 12.4 IU daily) 3. Reduction of triglyceride concentration by 16% on average	Diabetic gastroparesis was observed in 6 individuals	[62]
Group of 7 men and 4 women. Mean age 36.1 ± 6.8 years	Type 1 diabetes	Ketogenic diet	1. Normal HbA1c concentration (The mean HbA1c levels were 35 ± 4 mmol/mol (5.3 ± 0.4%)) 2. Slight changes in glycemia values (little daily glycemic variability (1.5 ± 0.7 mmol/L)	Hypoglycemia episodes and dyslipidemia were observed	[63]
Group of 4 men and 1 woman. Mean age 44.5 ± 10.4 years (in the normal carbohydrate amount group, there were an additional 3 men and 2 women, mean age 44.8 ± 8.3 years)	Type 1 diabetes	Target: ketogenic diet, 50–75 g carbohydrates as a result: low-carbohydrate diet, up to 100 g of carbohydrates	1. Improved glycemic control (significant reductions in HbA1c (63 to 55 mmol/mol)) 2. Reduction of insulin doses (significant reductions in daily insulin use (64.4 to 44.2 units/day)) 3. Body mass reduction (83.2 to 78.0 kg)	One participant reported a higher irritability and another one a greater number of minor diseases	[64]
Group of 316 women and men. Mean age 27 ± 19	Type 1 diabetes	Carbohydrates of 36 g on average, i.e., a ketogenic diet	1. Improved glycemic control (mean HbA1c was 5.67% ± 0.66%) 2. Lower requirement for insulin	A group of 7 individuals in a year, who reported a hospitalization associated with diabetes (including ketoacidosis and hypoglycemia); in the remaining majority of cases, there were no adverse effects	[65]



Low carb diets and type 2 diabetes

Efficacy and safety of low and very low carbohydrate diets

Summary



On the basis of moderate to low certainty evidence, patients adhering to a low carbohydrate diet for six months might experience diabetes remission without adverse consequences

Study design



Systematic review and meta-analysis

Published and unpublished randomized trial data

Patients with type 2 diabetes

Data sources



23 studies total
14 included participants using insulin



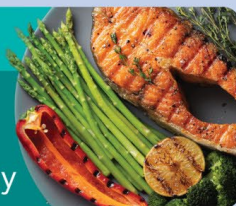
1357 participants
Primarily overweight and obese
Age range was 47 to 67 years

Interventions

Low and very low carbohydrate diets

Low 23/23 studies <26% of daily calories or <130 g/day

Very low 12/23 studies <10% of daily calories or <50 g/day



Comparators

18/23 trials used low fat diets as control comparators

Outcomes

Favors low carb diet
Favors control diet

Remission (HbA_{1c} <6.5%)

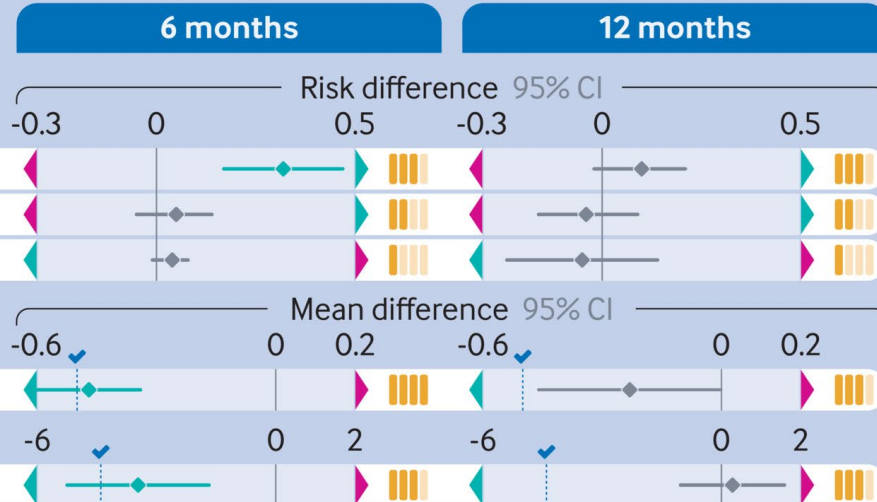
+ no diabetes medication

Adverse events

Minimal clinically important difference

HbA_{1c} (%)

Weight change (kg)



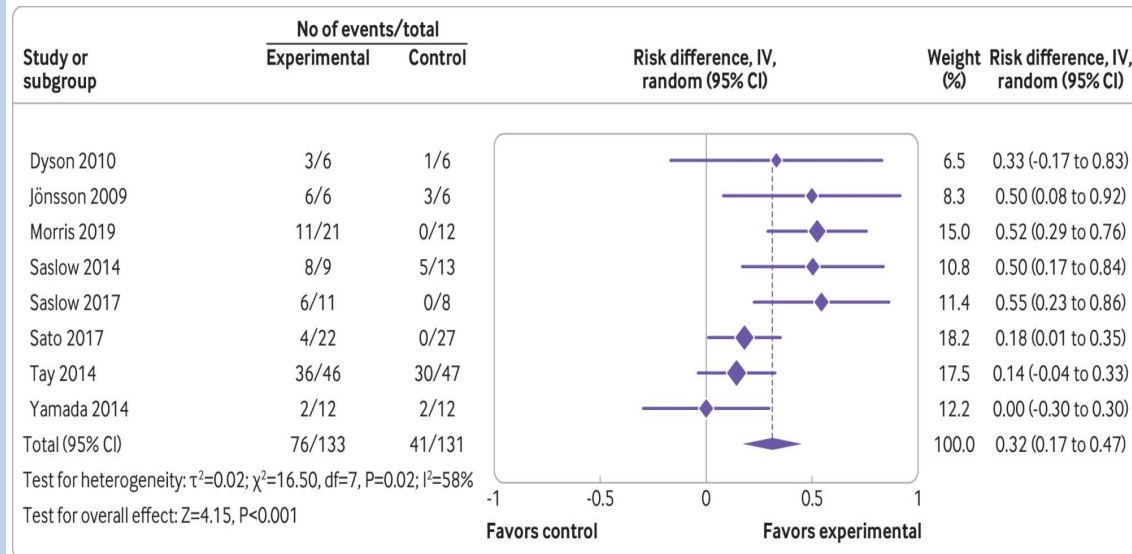
GRADE certainty of evidence rating

Very low Low Moderate High

© 2020 BMJ Publishing group Ltd.

DM2 e L-CarbDs a lungo termine

Remission (HbA_{1c} <6.5%) at six months

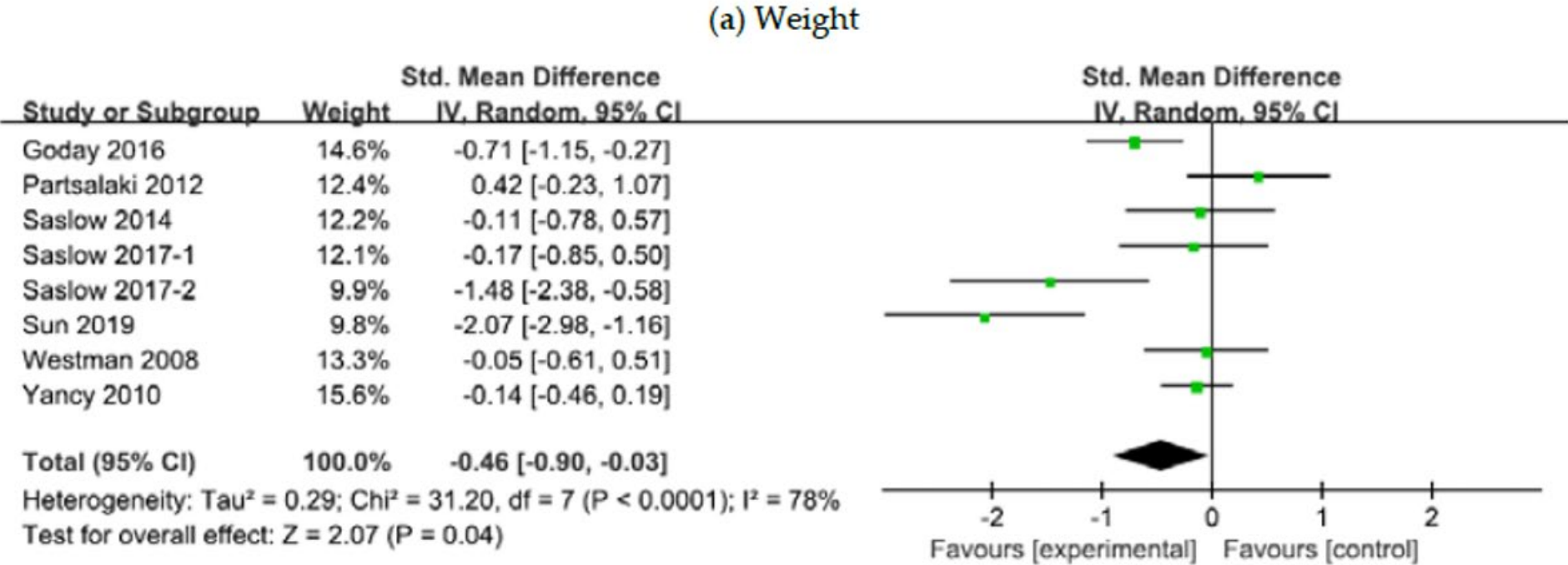


LCD hanno aumentato le remissioni di n. 32 ogni 100 pazienti seguiti e in terapia farmacologica, **ma si perdono già a 12 mesi**

Impact of a Ketogenic Diet on Metabolic Parameters in Patients with Obesity or Overweight and with or without Type 2 Diabetes: A Meta-Analysis of Randomized Controlled Trials

Yeo Jin Choi ^{1,†}, Sang-Min Jeon ^{2,3,†} and Sooyoung Shin ^{2,3,*}

Soggetti sovrappeso o obesi con o senza DM2
tutta la popolazione
Cambiamento del peso corporeo



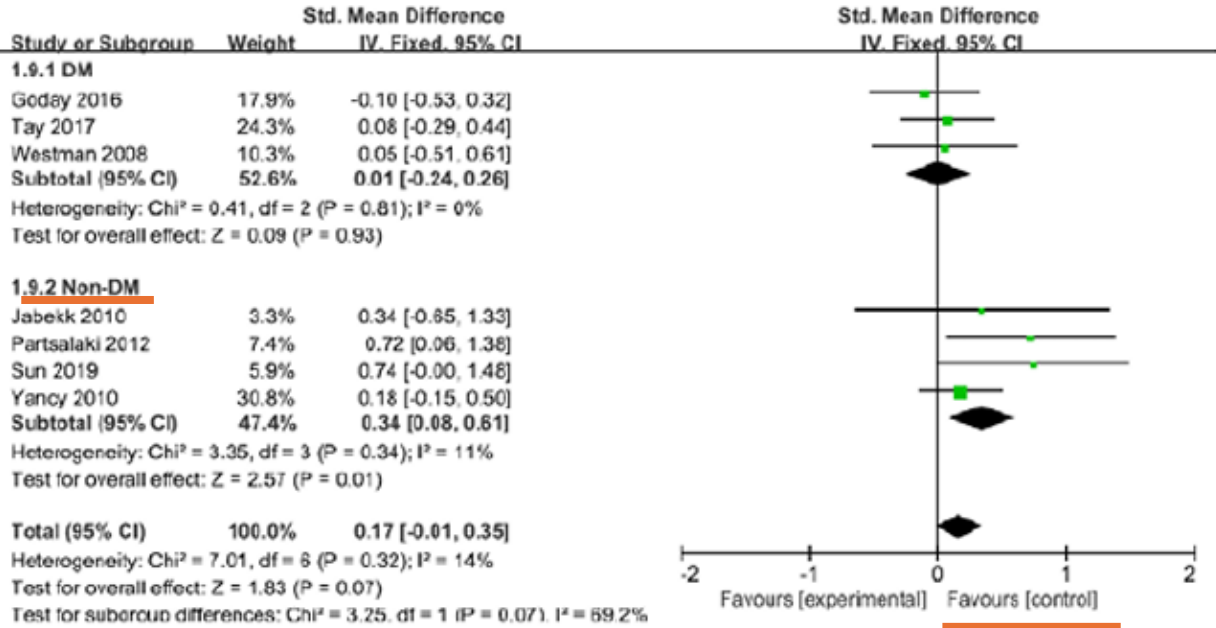
Impact of a Ketogenic Diet on Metabolic Parameters in Patients with Obesity or Overweight and with or without Type 2 Diabetes: A Meta-Analysis of Randomized Controlled Trials

Yeo Jin Choi ^{1,†}, Sang-Min Jeon ^{2,3,†} and Sooyoung Shin ^{2,3,*}

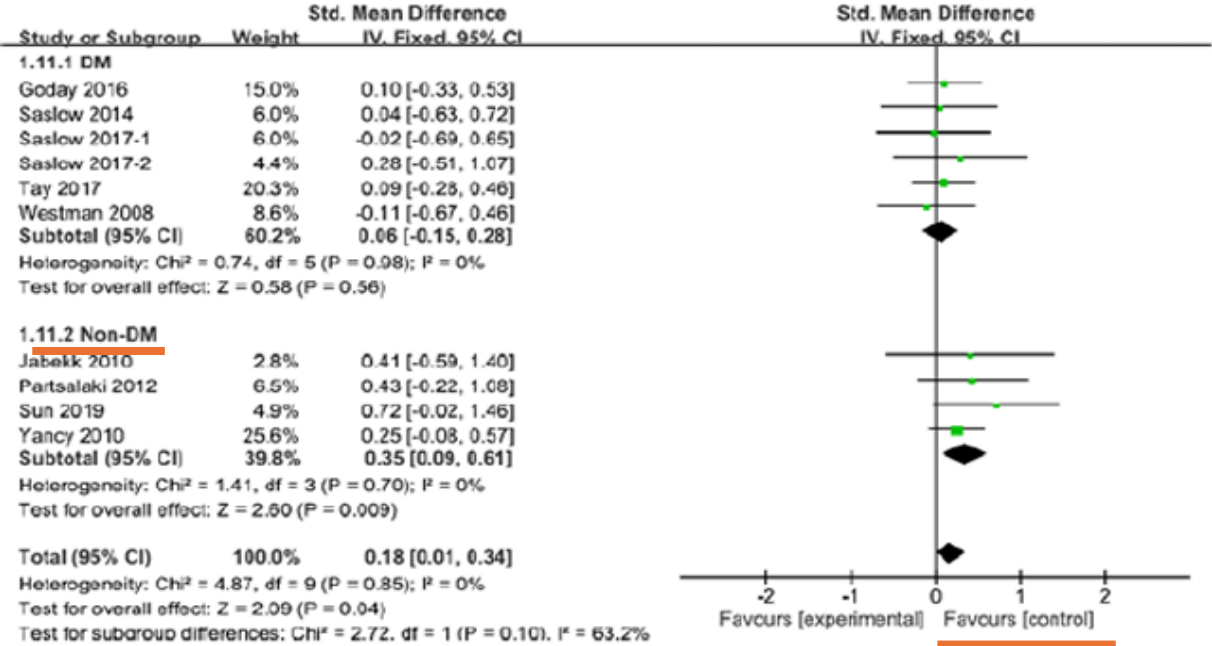
Soggetti sovrappeso o obesi con o senza DM2

Effetti sfavorevoli negli Ob Non-DM della KD su T-Col e LDL-Col

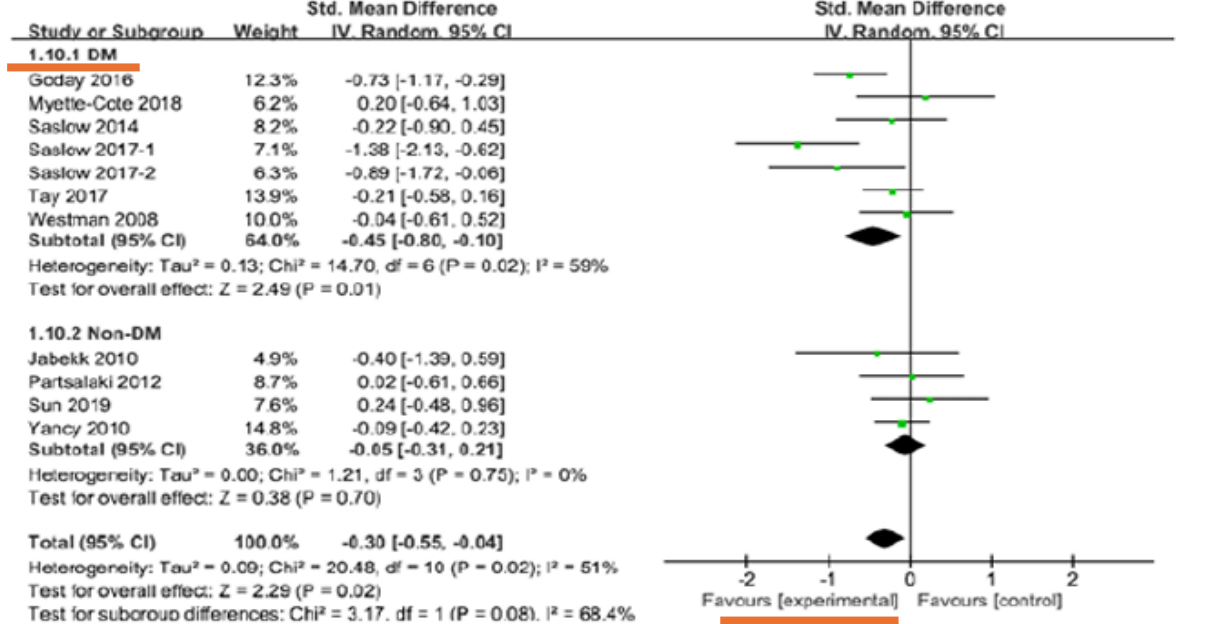
(a) Total cholesterol



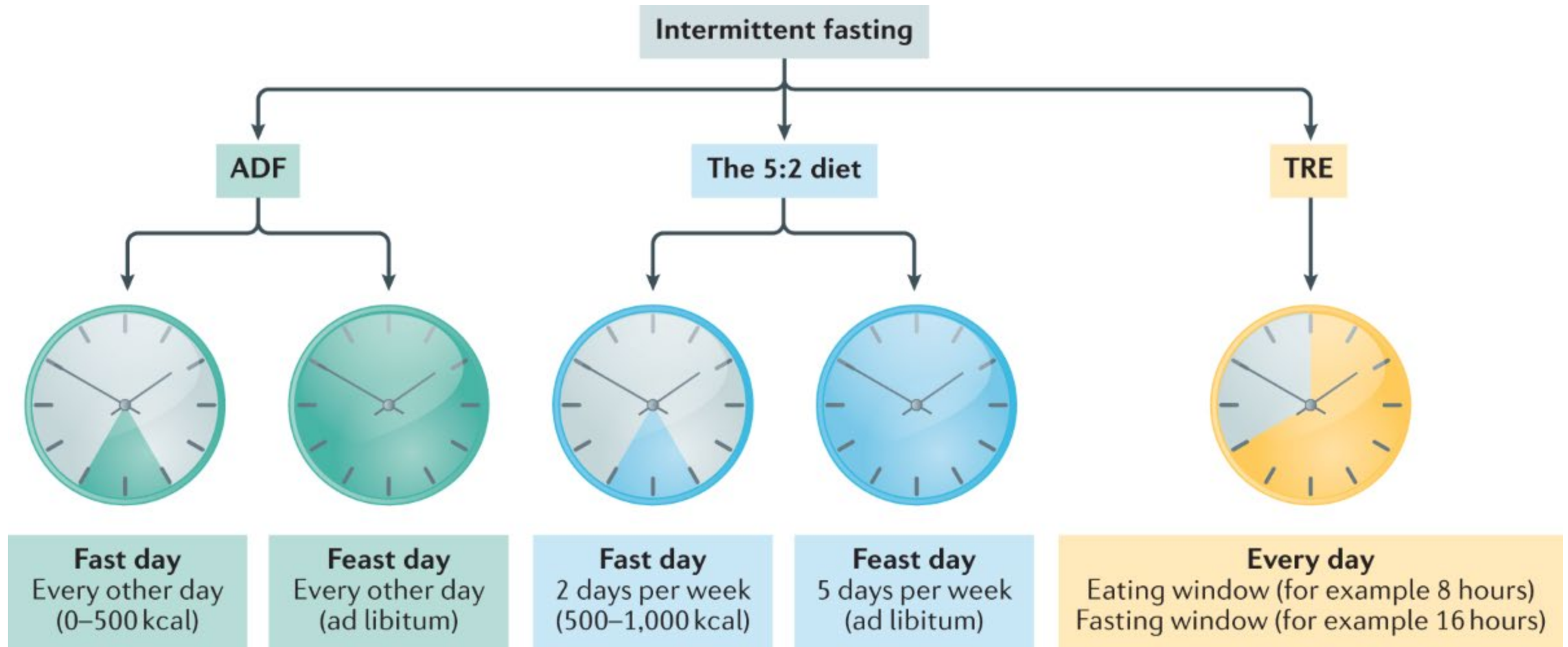
(c) LDL



(b) Triglyceride



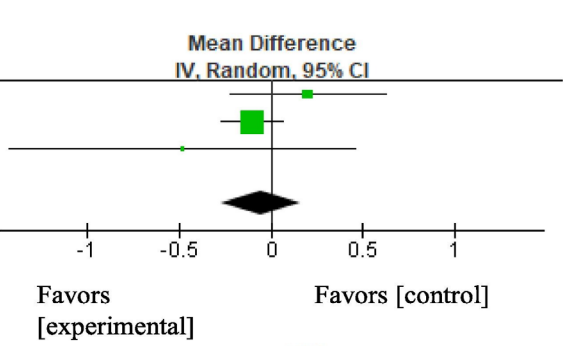
INTERMITTENT FASTING



1. HbA1c

Study or Subgroup	Experimental			Control			Weight	Mean Difference	
	Mean	SD	Total	Mean	SD	Total		IV, Random, 95% CI	
Carter 2018	-0.3	0.8	70	-0.5	1.6	67	21.3%	0.20 [-0.23, 0.63]	
Sundfær 2018	-0.3	0.5	54	-0.2	0.4	58	73.7%	-0.10 [-0.27, 0.07]	
Williams 1998	-0.71	1.59	16	-0.23	1.04	15	5.0%	-0.48 [-1.42, 0.46]	
Total (95% CI)			140			140	100.0%	-0.06 [-0.27, 0.16]	

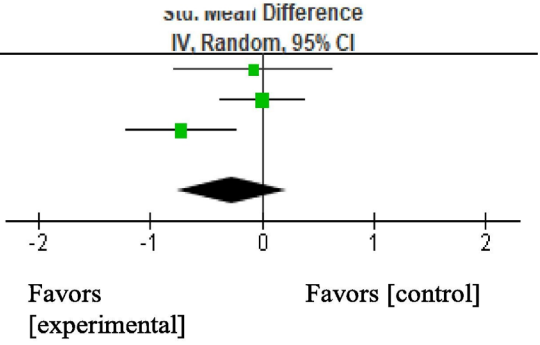
Heterogeneity: Tau² = 0.01; Chi² = 2.39, df = 2 (P = 0.30); I² = 16%
Test for overall effect: Z = 0.50 (P = 0.62)



2. Fasting glucose

Study or Subgroup	Experimental			Control			Weight	Std. Mean Difference		Year
	Mean	SD	Total	Mean	SD	Total		IV, Random, 95% CI		
Williams 1998	-1.9	1.5	16	-1.8	0.9	15	25.1%	-0.08 [-0.78, 0.63]		1998
Sundfær 2018	-0.2	0.9	54	-0.2	0.6	58	40.4%	0.00 [-0.37, 0.37]		2018
Parvaresh 2019	-5	6.82	35	0	6.85	34	34.5%	-0.72 [-1.21, -0.24]		2019
Total (95% CI)			105			107	100.0%	-0.27 [-0.76, 0.22]		

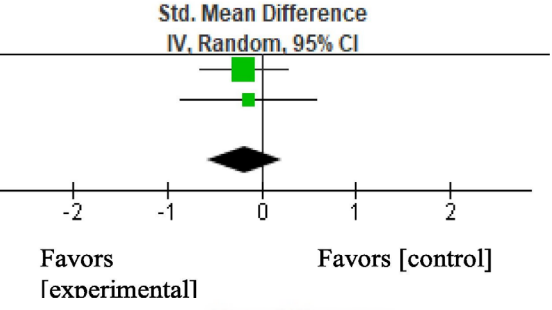
Heterogeneity: Tau² = 0.12; Chi² = 5.58, df = 2 (P = 0.06); I² = 64%
Test for overall effect: Z = 1.08 (P = 0.28)



3. Fasting insulin

Study or Subgroup	Experimental			Control			Weight	Std. Mean Difference	
	Mean	SD	Total	Mean	SD	Total		IV, Random, 95% CI	
Parvaresh 2019	-2.41	3.21	35	-1.56	5.41	34	69.7%	-0.19 [-0.66, 0.28]	
Williams 1998	-39	47	16	-33	36	14	30.3%	-0.14 [-0.86, 0.58]	
Total (95% CI)			51			48	100.0%	-0.17 [-0.57, 0.22]	

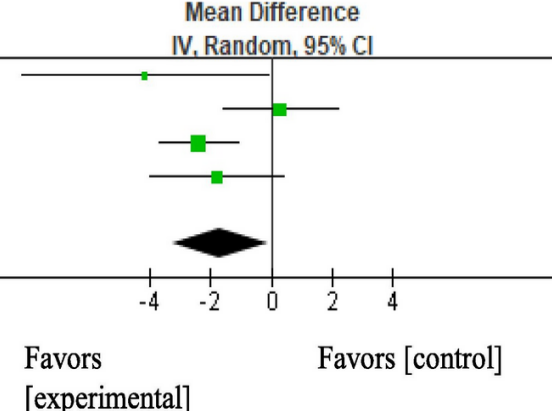
Heterogeneity: Tau² = 0.00; Chi² = 0.01, df = 1 (P = 0.91); I² = 0%
Test for overall effect: Z = 0.86 (P = 0.39)



1. Weight

Study or Subgroup	Experimental			Control			Weight	Mean Difference	
	Mean	SD	Total	Mean	SD	Total		IV, Random, 95% CI	
Williams 1998	-9.6	5.7	16	-5.4	5.9	15	11.4%	-4.20 [-8.29, -0.11]	
Sundfær 2018	-9.1	5	54	-9.4	5.3	58	28.1%	0.30 [-1.61, 2.21]	
Parvaresh 2019	-4.1	3.65	35	-1.7	1.49	34	35.8%	-2.40 [-3.71, -1.09]	
Carter 2018	-6.8	6.7	70	-5	6.5	67	24.7%	-1.80 [-4.01, 0.41]	
Total (95% CI)			175			174	100.0%	-1.70 [-3.28, -0.11]	

Heterogeneity: Tau² = 1.38; Chi² = 6.76, df = 3 (P = 0.08); I² = 56%
Test for overall effect: Z = 2.10 (P = 0.04)



Review

Intermittent fasting versus continuous energy-restricted diet for patients with type 2 diabetes mellitus and metabolic syndrome for glycemic control: A systematic review and meta-analysis of randomized controlled trials

Xue Wang^a, Qifei Li^a, Yan Liu^a, Hua Jiang^{b,c,d}, Wei Chen^{a,*}

DIGIUNO INTERMITTENTE vs Restrizione calorica continua

Trials clinici: 355 pazienti

← Gruppo IF -1,7 kg

Clinical Assessment of Intermittent Fasting With Ketogenic Diet in Glycemic Control and Weight Reduction in Patients With Type II Diabetes Mellitus: A Systematic Review and Meta-Analysis

Review began 10/25/2022
Review ended 10/27/2022
Published 10/30/2022

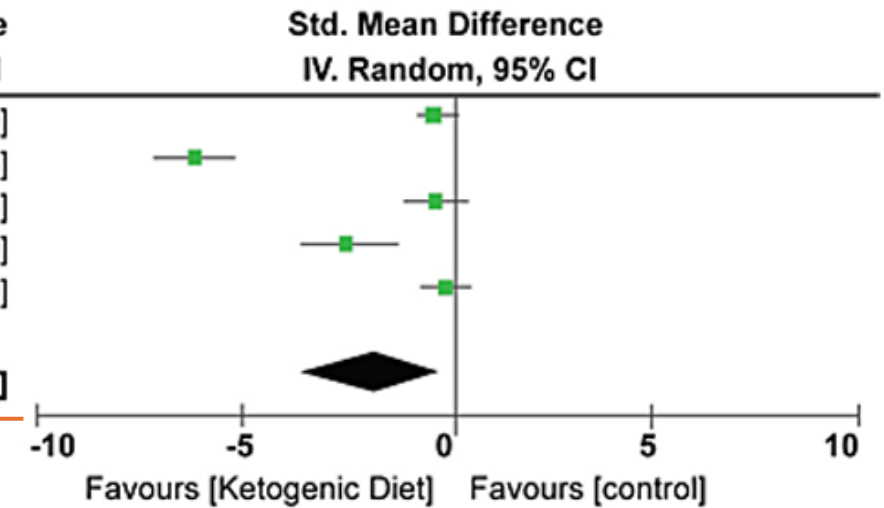
© Copyright 2022
Zaki et al. This is an open access article
distributed under the terms of the Creative

Hany A. Zaki¹, Haris Iftikhar¹, Abeer Abdalrub², Nood Dhafi R. Al-Marri¹, Mohammed Gafar Abdelrahim¹, Mohamed Fayed¹, Mohamed Abdelgadir M. Elgassim¹, Mohamed A. Elarref^{3,4}

KD e IF su HBA1c

Study or Subgroup	Ketogenic Diet			Control			Weight	Std. Mean Difference IV. Random, 95% CI
	Mean	SD	Total	Mean	SD	Total		
Godoy et al. 2016 [20]	6	0.7	45	6.4	0.8	40	20.6%	-0.53 [-0.96, -0.10]
Hussain et al. 2012 [19]	6.25	0.1	78	7.2	0.25	24	19.6%	-6.35 [-7.34, -5.35]
Moriconi et al. 2021 [21]	6.2	0.66	15	6.5	0.55	15	20.2%	-0.48 [-1.21, 0.25]
Saslow et al. 2017 [23]	6.3	0.28	12	6.9	0.15	13	19.3%	-2.61 [-3.73, -1.50]
Westman et al. 2008 [18]	7.3	1.5	21	7.8	2.1	29	20.4%	-0.26 [-0.83, 0.30]
Total (95% CI)			171			121	100.0%	-2.00 [-3.76, -0.25]

Heterogeneity: $\tau^2 = 3.85$; $\chi^2 = 130.89$, $df = 4$ ($P < 0.00001$); $I^2 = 97\%$
Test for overall effect $Z = 2.24$ ($P = 0.03$)



Study or Subgroup	Intermittent Fasting			Control			Weight	Std. Mean Difference IV. Random, 95% CI
	Mean	SD	Total	Mean	SD	Total		
Carter et al. 2016 [14]	-0.6	0.8	31	-0.8	1	32	25.8%	0.22 [-0.28, 0.71]
Carter et al. 2018 [16]	-0.3	0.1	70	-0.5	0.2	67	27.2%	1.27 [0.90, 1.63]
Corley et al. 2018 [17]	-0.7	0.1	19	-0.6	0.9	18	23.8%	-0.15 [-0.80, 0.49]
Li et al. 2017 [13]	-0.2	1.1	16	-0.2	0.8	16	23.2%	0.00 [-0.69, 0.69]
Total (95% CI)			136			133	100.0%	0.36 [-0.37, 1.10]

Heterogeneity: $\tau^2 = 0.48$; $\chi^2 = 23.00$, $df = 3$ ($P < 0.0001$); $I^2 = 87\%$
Test for overall effect $Z = 0.97$ ($P = 0.33$)

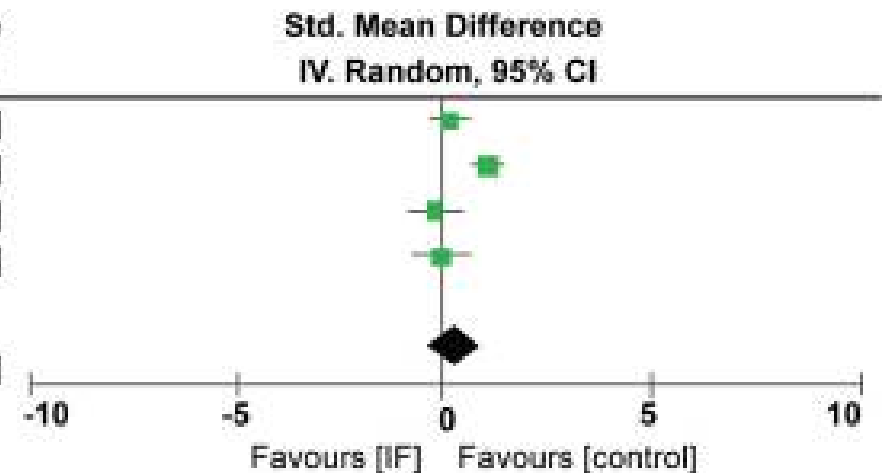


Table 2
Mean changes in weight, body composition, glycaemic measures and total energy intake in each group.

	SEM	VLCD	COMB
Weight (kg)	−6.0 ± 0.7	−13.1 ± 1.9 ^{##}	−13.5 ± 1.7 ^{##}
Weight (%)	−5.9 ± 0.6	−11.3 ± 1.8 [#]	−12.9 ± 1.4 ^{##}
BMI (kg.m ^{−2})	−2.0 ± 0.2	−4.3 ± 0.7 [#]	−4.7 ± 0.5 ^{##}
FM (kg)	−4.0 ± 0.5	−9.0 ± 1.3 ^{##}	−9.0 ± 1.2 ^{##}
LBM (kg)	−1.7 ± 0.6	−3.8 ± 0.8	−3.3 ± 1.0
HbA1c (mmol/mol)	−9.7 ± 3.1	−14.9 ± 3.6	−20.9 ± 3.9
Fasting glucose (mmol/l)	−2.3 ± 0.6	−1.6 ± 0.5	−3.2 ± 0.8
Total energy intake (kcal)	−480.0 ± 223.9	−1146.6 ± 131.7 [#]	−1136.5 ± 173.5 [#]

Data presented as mean ± SEM. Statistical significance assessed using one-way ANOVA. #p < 0.05 v SEM, ##p < 0.01 v SEM.

AGONISTI DEL GLP-1 E DIETA

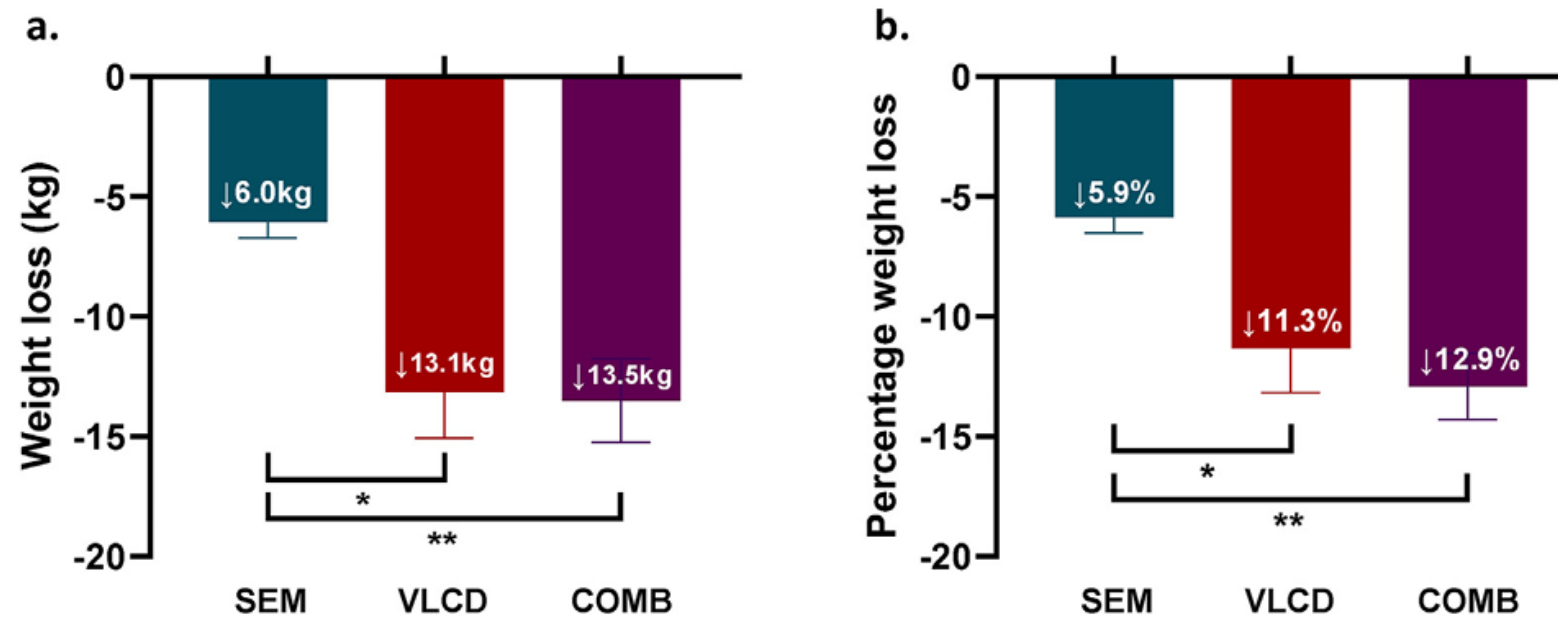


Fig. 1. Absolute weight loss (a) and percentage weight loss (b) in each group. *p < 0.05, **p < 0.01.

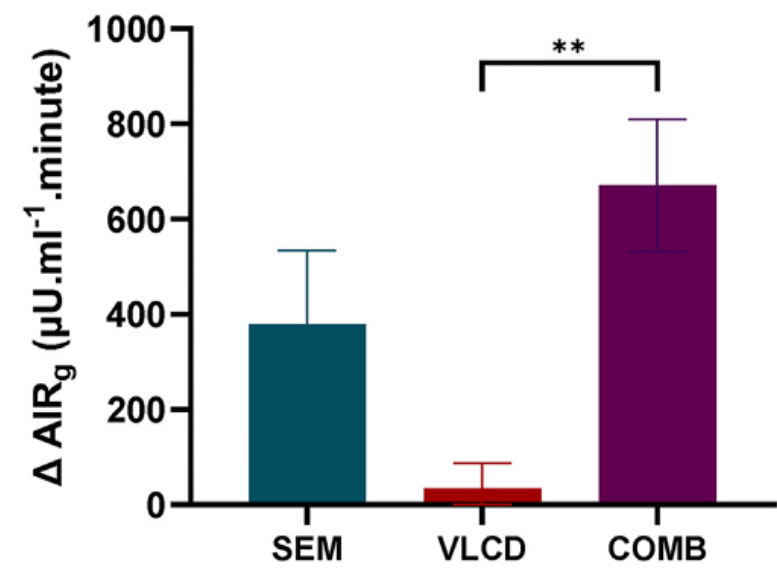


Fig. 2. Mean change in first-phase response in each group. **p < 0.01.

AIRg =Acute Insulin Response to Glucose

CRONONUTRIZIONE NEL DIABETE

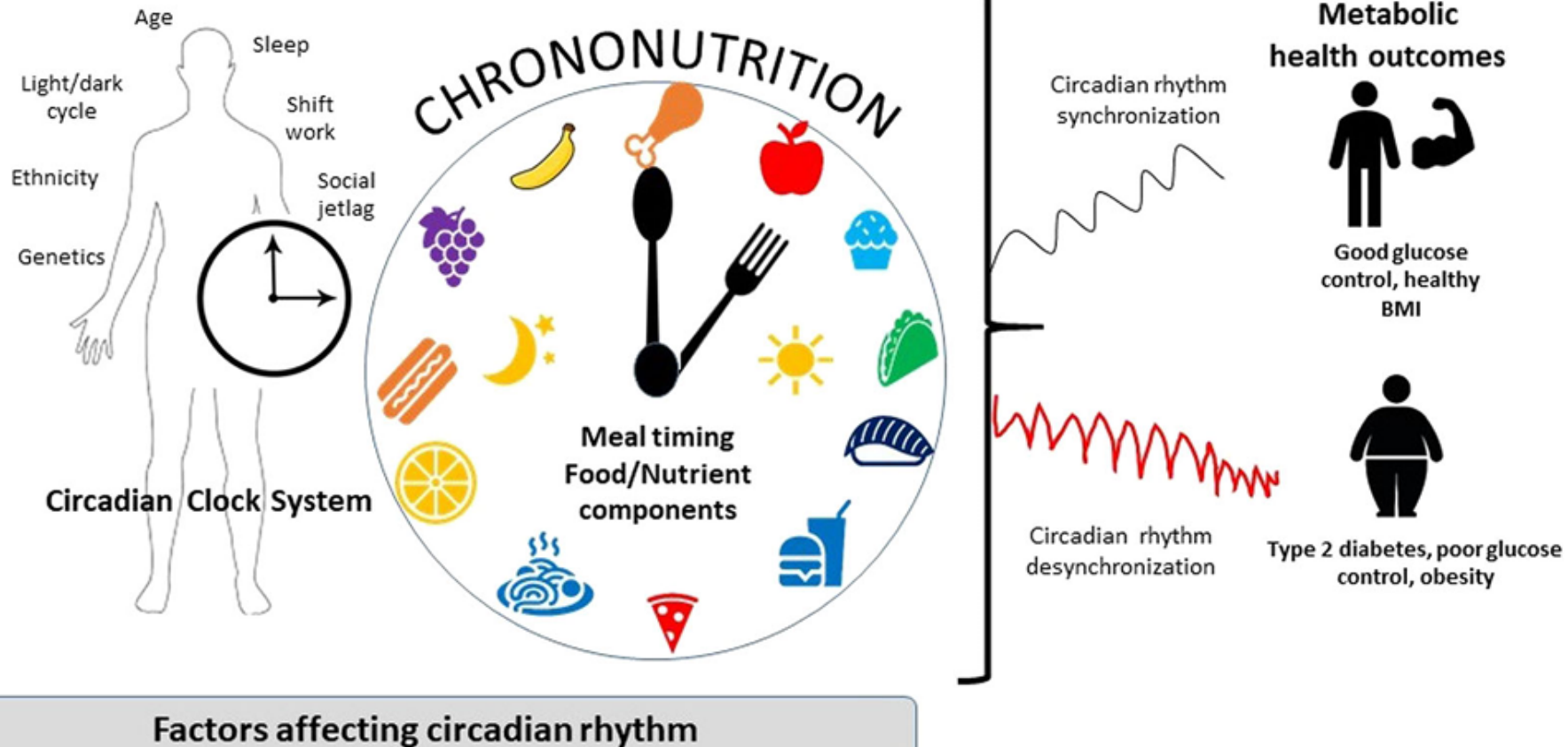


Fig. 1 A schematic representation outlining the factors affecting the circadian clock system. Meal timing and dietary components (chrononutrition) play an important role in regulating circadian clocks, to enhance metabolic health and reduce the risk of type 2 diabetes.

CRONONUTRIZIONE NEL DIABETE

Impatto del timing dei pasti sul DM2

**Effetti del consumo di
carboidrati di sera**

**Aumento della
glicemia postprandiale
rispetto a un pasto
identico consumato al
mattino**

**Modifica
composizione dei
macronutrienti**

**Aumentare le proteine
e i grassi nei pasti
serali, può aiutare a
migliorare la risposta
glicemica**

CRONONUTRIZIONE NEL DIABETE

Impatto del timing dei pasti sul DM2

**Incoraggiare i pasti
all'inizio della
giornata**

**Migliore controllo
glicemico**

**Effetto dei cibi a
basso IG di mattina**

**Risposta glicemica più
efficace rispetto al
consumo serale**

CRONONUTRIZIONE NEL DIABETE

Impatto del timing dei pasti sul DM2

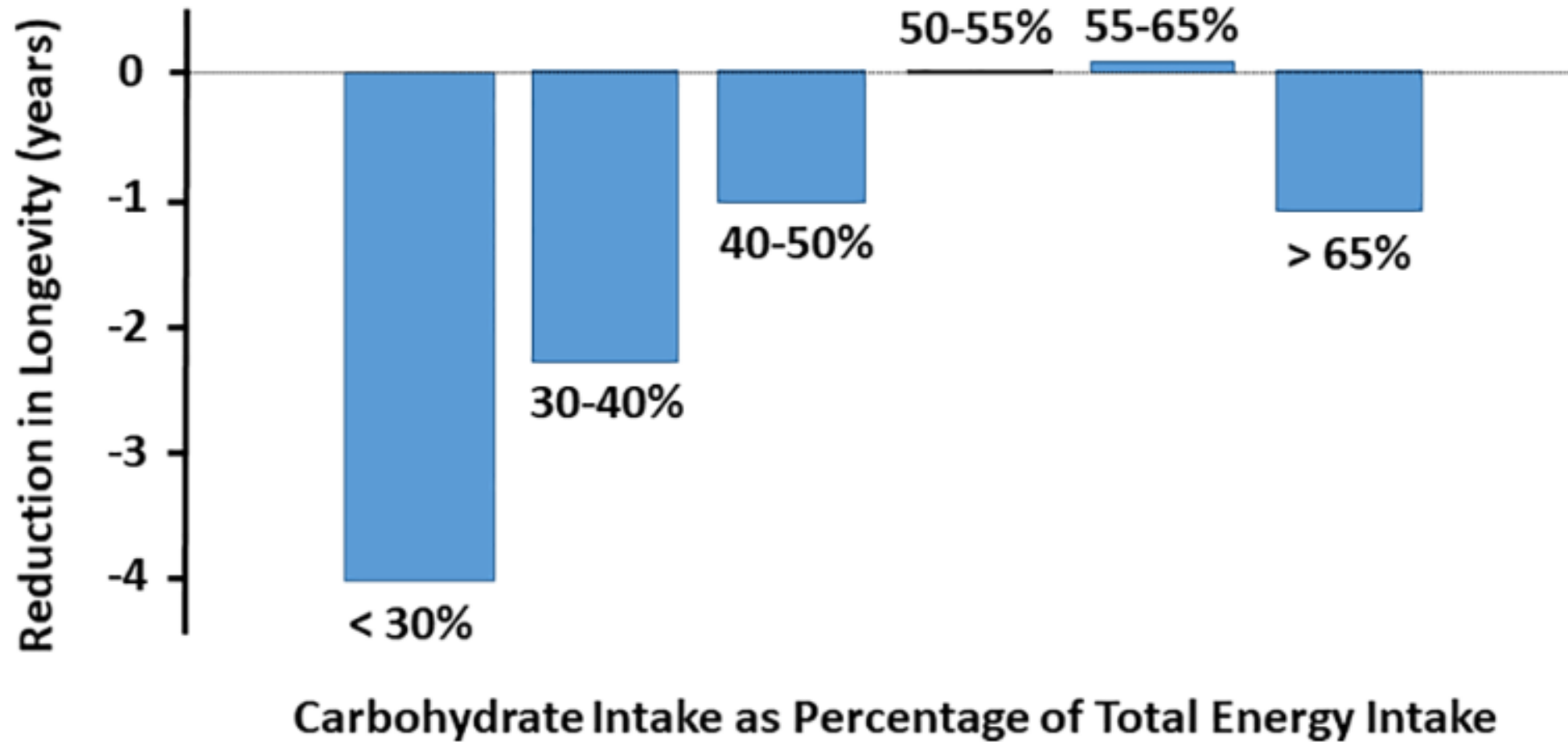
**Timing del consumo
di grassi e proteine
con carboidrati**

**grassi e proteine
(inclusi amminoacidi)
insieme ai carboidrati,
può ridurre la risposta
glicemica**

**Ordine di presentazione
degli alimenti sulla
glicemia**

**mangiare prima le
verdure, poi la carne e
infine il riso può ridurre
la glicemia
postprandiale**

INFINE.....CARBOIDRATI E LONGEVITA'





**Take
home message*

Diete Antinfiammatorie: Migliorano controllo glicemico e riducono rischio cardiovascolare.

Dieta Chetogenica:

DM 1: Supporta stabilità glicemica (monitoraggio necessario per chetosi) ma effetti negativi, non sufficientemente supportata dagli studi;

DM 2: Favorisce perdita di peso e sensibilità insulinica, ma effetti negativi su CVC.

Digiuno Intermittente: Promettente per sensibilità insulinica e riduzione di farmaci, riduce il peso (richiede ulteriori studi).



**Take
home message*

Conteggio dei Carboidrati: Strategia ottimale per il DM1 per gestione precisa del glucosio e insulina.

Carboidrati e Longevità: Consumo 50-65% En carboidrati di alta qualità migliora longevità e salute metabolica.



**Take
home message*

- **Personalizzazione:** Adattare le strategie nutrizionali alle caratteristiche individuali (tipo di diabete, profilo metabolico, preferenze, genere) per massimizzare l'efficacia
- **Educazione e Adesione:** Promuovere un'educazione nutrizionale continua per migliorare l'adesione e mantenere risultati duraturi.
- **Ricerca e Innovazione:** Ampliare la ricerca per validare l'efficacia a lungo termine degli interventi nutrizionali innovativi e sviluppare linee guida basate sull'evidenza.

Grazie per
l'attenzione!!