Intermittent Fasting

Megan J. Ramos

The Fasting Method | CEO & Co-founder

Disclosures

- Co-founded The Fasting Method by Intensive Dietary Management with Dr. Jason Fung

- Intermittent fasting, low carb/ketogenic diets are responsible for reversing my:
  - NAFLD (age 12)
  - PCOS (age 14)
  - 86 lbs weight loss (age 27)
  - DMII (age 27)

the Fasting Method

Background

- Launched in June 2012 in Toronto, Canada
- Worked with 14,000 people worldwide
- Started with obesity and DMII management
- Now:
  - DMI
  - Latent onset DMI
  - PCOS
  - NAFLD
  - Cancer
  - Neurological diseases, i.e. Parkinson's, MS, etc.

Outline

- The Why: Physiology of Fasting

- How: Fasting Protocols

- The Results

The Why: Physiology of Fasting

Isn't fasting just starvation?

No. Fasting differs from starvation in one crucial way: **control**. Starvation is the ***involuntary*** absence of food for a long time.

Fasting is the ***voluntary*** avoidance of food for spiritual, health, or other reasons. It's done by someone who is not underweight and has enough stored body fat to live off.

What is intermittent fasting?

Intermittent fasting involves cycling between periods of fasting and eating — and it's recently become very popular

Intermittent fasting can provide significant health benefits if it is done right, including weight loss, type 2 diabetes reversal, PCOS, fatty liver disease, etc.

At its very core, intermittent fasting simply allows the body to use its stored energy, by burning off excess body fat

Constantly Fueling

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https://share.getcloudapp.com/kpuKrmgA

- Eating several times a day

- Constant snacking

- Eating beyond satiation

- Stress, fun, boredom, mindless

Body Fat = Unused Glucose

Treatment of Diabetes Mellitus 1916

*"That temporary periods of undernutrition are helpful in the treatment of diabetes will probably be **acknowledged by all** after these two years of experience with fasting"* (page 676)

*"The practice, observed by many clinicians of the old school, who advantageously fasted their diabetics one day a week have given the cue to **intermittent fasting**"* (page 679)
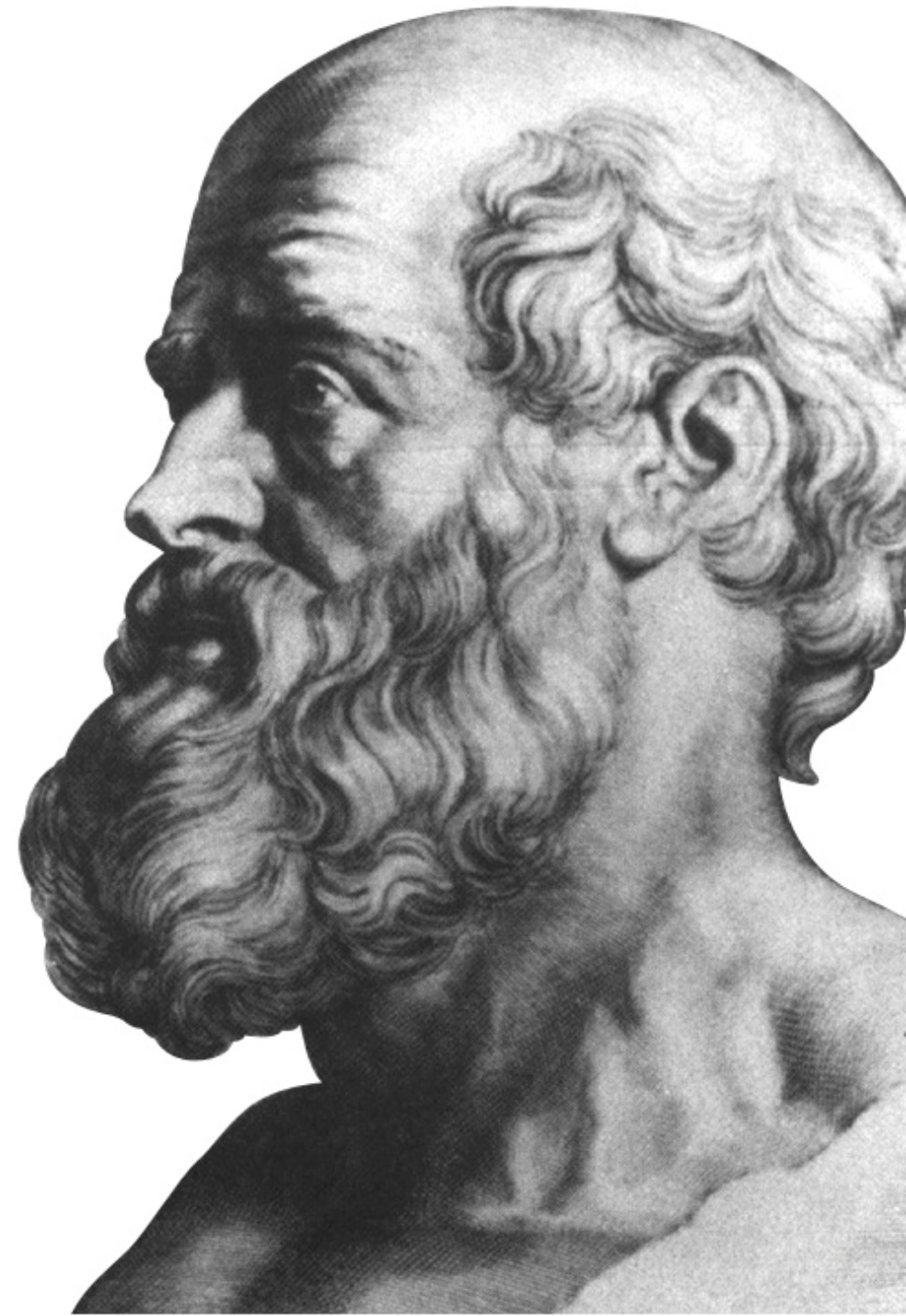
Can Med Assoc J. Aug 1916; 6(8): 673-684

Dr. Elliot P. Joslin

Hippocrates (460-370 BC)

Our food should be our medicine. Our medicine should be our food. But to eat when you are sick is to feed your sickness."
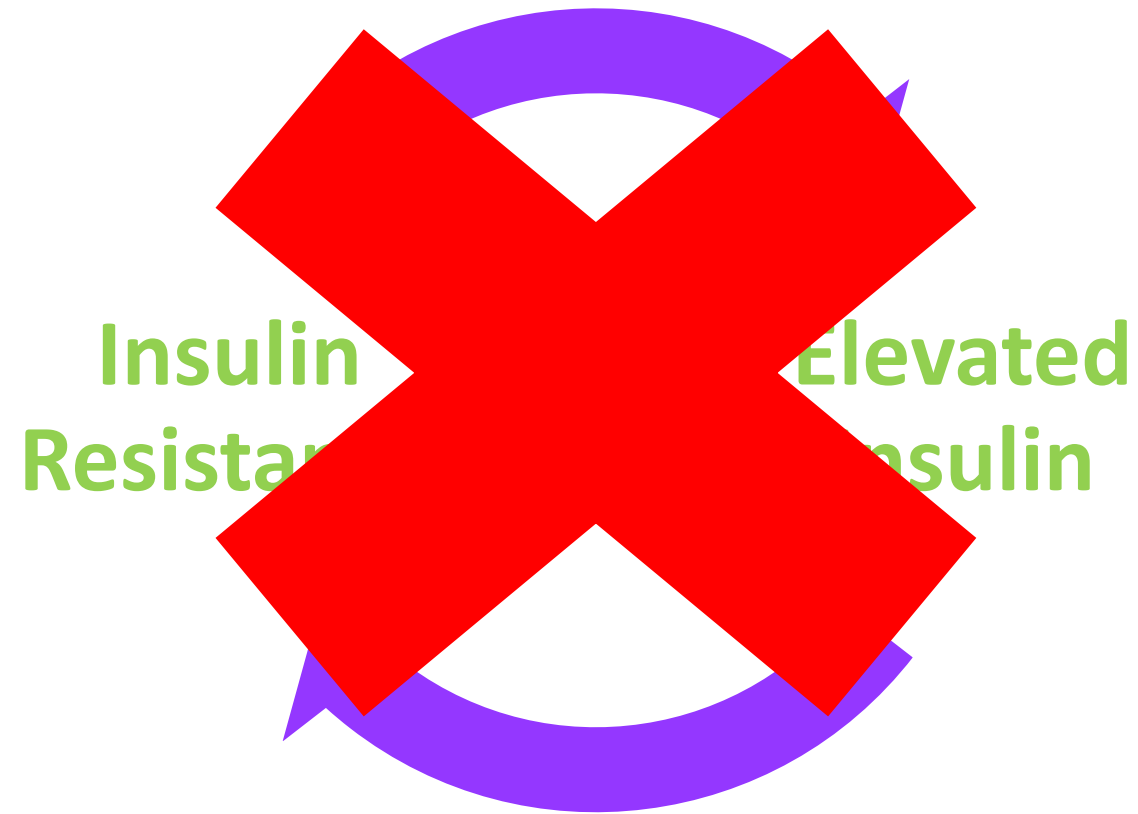
The fasting instinct

Why Fast for Type 2 Diabetes (DMII)?

- DMII is caused by a condition called **insulin resistance – toxic levels of insulin within the body**

- **What you eat:** being mindful of what you eat (LCHF/keto) will stop adding insulin to your system

Cycle of Insulin Resistance

When We Eat Matters

Obesity

A Randomized Pilot Study Comparing Zero-Calorie Alternate-Day Fasting to Daily Caloric Restriction in Adults with Obesity

Victoria A. Catenacci[1,2], Zhaoxing Pan[3], Danielle Ostendorf[2,4], Sarah Brannon[5], Wendolyn S. Gozansky[6], Mark P. Mattson[7,8], Bronwen Martin[9], Paul S. MacLean[1,2], Edward L. Melanson[1,10], and William Troy Donahoo[1,6]

Objective: To evaluate the safety and tolerability of alternate-day fasting (ADF) and to compare changes in weight, body composition, lipids, and insulin sensitivity index (Si) with those produced by a standard weight loss diet, moderate daily caloric restriction (CR).

Methods: Adults with obesity (BMI $\geq$30 kg/m^2, age 18-55) were randomized to either zero-calorie ADF ($n = 14$) or CR (-400 kcal/day, $n = 12$) for 8 weeks. Outcomes were measured at the end of the 8-week intervention and after 24 weeks of unsupervised follow-up.

Results: No adverse effects were attributed to ADF, and 93% completed the 8-week ADF protocol. At 8 weeks, ADF achieved a 376 kcal/day greater energy deficit; however, there were no significant between-group differences in change in weight (mean $\pm$ SE; ADF -8.2 ± 0.9 kg, CR -7.1 ± 1.0 kg), body composition, lipids, or Si. After 24 weeks of unsupervised follow-up, there were no significant differences in weight regain; however, changes from baseline in % fat mass and lean mass were more favorable in ADF.

Conclusions: ADF is a safe and tolerable approach to weight loss. ADF produced similar changes in weight, body composition, lipids, and Si at 8 weeks and did not appear to increase risk for weight regain 24 weeks after completing the intervention.

TABLE 4 Changes in anthropometric measures at the end of the 8-week intervention and after 24 weeks of unsupervised follow-up[a]

Outcome variable and group	Assessment period			Week 8–baseline	P	ES	Week 32–baseline	P	ES	Week 32–week 8	P	ES	P value for overall interaction
	Baseline	Week 8	Week 32										
Weight (kg)													
CR	114.0 (4.6)	106.9 (4.5)	109.0 (4.7)	−7.1 (1.0)	<0.001		−5.0 (1.6)	0.005		2.1 (1.0)	0.047		
ADF	94.8 (4.4)	86.5 (4.4)	89.1 (4.5)	−8.2 (0.9)	<0.001		−5.7 (1.5)	0.001		2.6 (1.0)	0.013		
CR–ADF	19.3 (6.3)	20.4 (6.3)	19.9 (6.5)	1.1 (1.3)	0.409	−0.35	0.7 (2.2)	0.774	−0.12	−0.5 (1.4)	0.739	0.14	0.559
Weight (%)													
CR				−6.2 (0.9)	<0.001		−4.4 (1.6)	0.011		−1.8 (1.0)	0.082		
ADF				−8.8 (0.9)	<0.001		−5.9 (1.5)	<0.001		−2.9 (1.0)	0.006		
CR–ADF				2.6 (1.3)	0.056	−0.84	1.5 (2.2)	0.496	−0.29	1.1 (1.4)	0.456	−0.32	0.456
BMI (kg/m²)													
CR	39.5 (1.4)	37.1 (1.5)	37.8 (1.6)	−2.4 (0.3)	<0.001		−1.7 (0.6)	0.007		0.8 (0.4)	0.045		
ADF	35.8 (1.4)	32.6 (1.4)	33.6 (1.5)	−3.2 (0.3)	<0.001		−2.2 (0.5)	<0.001		1.0 (0.4)	0.008		
CR–ADF	3.7 (2.0)	4.5 (2.1)	4.2 (2.2)	0.7 (0.5)	0.136	−0.64	0.5 (0.8)	0.548	−0.25	−0.2 (0.5)	0.65	0.19	0.207
Total fat mass (kg)													
CR	48.8 (2.7)	45.1 (2.6)	46.3 (2.9)	−3.7 (0.5)	<0.001		−2.5 (1.1)	0.028		1.2 (0.8)	0.162		
ADF	37.7 (2.6)	33.9 (2.5)	33.5 (2.8)	−3.7 (0.5)	<0.001		−4.2 (1.0)	<0.001		−0.4 (0.8)	0.605		
CR–ADF	11.1 (3.7)	11.1 (3.6)	12.8 (4.0)	0.0 (0.8)	0.995	0	1.6 (1.5)	0.291	−0.45	1.6 (1.2)	0.173	−0.59	0.371
Total fat mass (%)													
CR	43.4 (1.7)	42.4 (1.7)	42.7 (1.7)	−1.0 (0.3)	0.007		−0.7 (0.5)	0.222		0.3 (0.5)	0.53		
ADF	40.3 (1.6)	39.2 (1.6)	38.0 (1.7)	−1.1 (0.3)	0.002		−2.4 (0.5)	<0.001		−1.3 (0.5)	0.015		
CR–ADF	3.1 (2.3)	3.2 (2.4)	4.7 (2.4)	0.1 (0.5)	0.826	−0.09	1.7 (0.8)	0.035	−0.93	1.6 (0.7)	0.032	−0.95	0.078
Trunk fat mass (kg)													
CR	26.0 (1.8)	23.9 (1.7)	24.7 (1.8)	−2.1 (0.4)	<0.001		−1.3 (0.7)	0.054		0.8 (0.4)	0.093		
ADF	20.9 (1.7)	18.5 (1.7)	18.2 (1.7)	−2.4 (0.4)	<0.001		−2.7 (0.6)	<0.001		−0.3 (0.4)	0.436		
CR–ADF	5.1 (2.5)	5.4 (2.4)	6.5 (2.5)	0.3 (0.5)	0.637	−0.2	1.4 (0.9)	0.144	−0.63	1.1 (0.6)	0.083	−0.76	0.215
Trunk fat mass (%)													
CR	23.1 (1.1)	22.4 (1.2)	22.7 (1.1)	−0.7 (0.3)	0.017		−0.3 (0.4)	0.34		0.3 (0.3)	0.274		
ADF	22.1 (1.1)	21.2 (1.1)	20.3 (1.1)	−0.9 (0.3)	0.001		−1.8 (0.3)	<0.001		−0.8 (0.3)	0.005		
CR–ADF	1.0 (1.6)	1.3 (1.7)	2.4 (1.5)	0.3 (0.4)	0.484	−0.3	1.4 (0.5)	0.009	−1.19	1.2 (0.4)	0.007	−1.24	0.016
Lean mass (kg)													
CR	60.9 (3.0)	58.2 (2.8)	59.3 (2.8)	−2.6 (0.6)	<0.001		−1.6 (0.6)	0.022		1.1 (0.5)	0.051		
ADF	53.2 (2.8)	50.0 (2.7)	52.1 (2.7)	−3.2 (0.6)	<0.001		−1.2 (0.6)	0.072		2.0 (0.5)	<0.001		
CR–ADF	7.7 (4.1)	8.2 (3.8)	7.2 (3.9)	0.5 (0.9)	0.539	−0.26	−0.4 (0.9)	0.64	0.2	−1.0 (0.7)	0.197	0.55	0.424
Lean mass (%)													
CR	54.2 (1.6)	55.1 (1.6)	54.8 (1.7)	0.9 (0.3)	0.016		0.5 (0.5)	0.309		−0.3 (0.5)	0.509		
ADF	57.1 (1.5)	58.0 (1.6)	59.3 (1.6)	0.9 (0.3)	0.009		2.2 (0.5)	<0.001		1.3 (0.5)	0.012		
CR–ADF	−2.8 (2.2)	−2.9 (2.3)	−4.5 (2.3)	−0.1 (0.5)	0.921	0.04	−1.7 (0.7)	0.026	0.99	−1.6 (0.7)	0.026	0.99	0.061

TABLE 5 Changes in resting metabolic rate at the end of the 8-week intervention and after 24 weeks of unsupervised follow-up[a]

Outcome variable and group	Assessment period			Week 8–baseline	P	ES	Week 32–baseline	P	ES	Week 32–week 8	P	ES	P value for overall interaction
	Baseline	Week 8	Week 32										
Unadjusted RMR (kcal/d)[b]													
CR	1,892.5 (67.7)	1,719.3 (69.3)	1,807.3 (72.2)	−173.2 (35.2)	<0.001		−85.2 (39.0)	0.039		88.0 (22.2)	<0.001		
ADF	1,640.1 (65.1)	1,539.7 (66.8)	1,567.2 (69.2)	−100.4 (34.1)	0.007		−72.9 (37.3)	0.063		27.5 (22.0)	0.223		
CR–ADF	252.4 (93.9)	179.6 (96.2)	240.1 (100.0)	−72.8 (49.0)	0.151	0.62	−12.3 (54.0)	0.822	0.09	60.5 (31.3)	0.065	−0.81	0.096
Adjusted RMR (kcal/d)[b,c]													
CR	1,757.6 (37.0)	1,646.0 (32.8)	1,681.53 (18.6)	−111.6 (36.9)	0.006		−76.1 (35.9)	0.045		35.6 (22.4)	0.126		
ADF	1,689.0 (34.2)	1,672.8 (33.5)	1,659.8 (20.1)	−16.2 (36.6)	0.662		−29.2 (35.2)	0.416		−13.0 (22.5)	0.569		
CR–ADF	68.6 (51.1)	−26.8 (48.1)	21.7 (29.8)	−95.4 (51.4)	0.076	0.77	−46.9 (49.7)	0.356	0.39	48.5 (31.8)	0.14	−0.64	0.14

[a]Linear mixed-effects model analysis with unstructured covariance was used to assess the efficacy of intervention on each outcome variable. Test of time by group interaction was used to test the efficacy of intervention (see P value for overall interaction). Results are mean (SE). Significant P values (P <0.05) are indicated in bold. Effect size (ES) is calculated as (2 × t value)/√DF, where degrees of freedom (DF). Hand calculations for between- and within-group differences may not be equal to data shown because all data were rounded to 0.1 decimal place. For CR: $n = 12$ for baseline and week 8; $n = 10$ for week 32; for ADF: $n = 13$ for baseline and week 8; $n = 11$ for week 32; non-missing observations: $n = 71$.
[b]RMR results exclude one observation at week 32 for one subject in ADF because the value was physiologically implausible.
[c]RMR results adjusted for fat-free mass (FFM) and fat mass (FM).
RMR, resting metabolic rate.

Counter-Regulatory Hormones

- Sympathetic Nervous System
- Adrenalin and Noradrenalin
- Growth Hormone
- Cortisol

Human Growth Hormone

- Increase the availability and utility of fats for fuel
- Preserves muscle mass and bone density
- Decreases with age
- One of the most potent stimuli is fasting

Lean Mass Loss – Clinical Example

Female, 53-year-old

Fasting Regimen (2 years):

4 days (96 hours) once a month

16-24 hour fasts daily

Exercise:

Weight-training, 2x/week

Diet: Ketogenic

Adjusts protein accordingly to support elevated level of lean mass

# Loss of Lean Mass

Summary-TotalBodyCompositionAnalysis

ThetotalbodycompositiontablesummarizesthemetricsofyourentirebodyanddisplaysyourTotalBodyFat%,TotalMass(lbs),FatTissue
(lbs),LeanTissue(lbs),BoneMineralContent(BMC),andVisceralFat(lbs).

MeasuredDate	TotalBodyFat%	TotalMass	FatTissue	LeanTissue	BMC	VisceralFat
07/05/2017	45.3%	250.5lbs	113.5lbs	130.4lbs	6.6lbs	3.61lbs
03/15/2017	46.2%	247.3lbs	114.4lbs	126.3lbs	6.6lbs	3.47 lbs

Gained 4.1 lbs of lean mass

Growth Hormone

Integrated GH concentration

pulsatile
non-pulsatile

p < 0.002
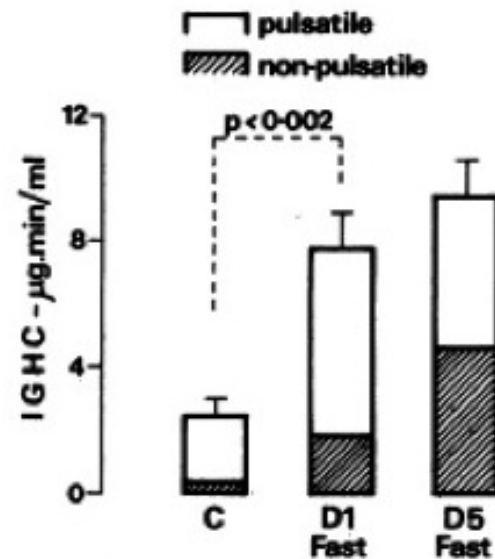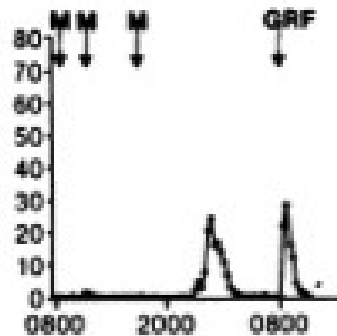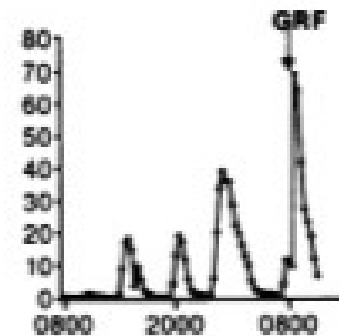
J Clin Invest. 1988 April; 81(4): 968–975 Ho KY

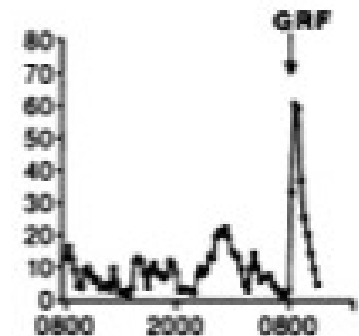Fasting increases Growth Hormone

CONTROL   DAY 1   DAY 5

J Clin Invest. 1988 April; 81(4): 968–975 Ho KY

How: Fasting Protocols

Intermittent Fasting Protocols

Duration

- 16:8 or 18:6
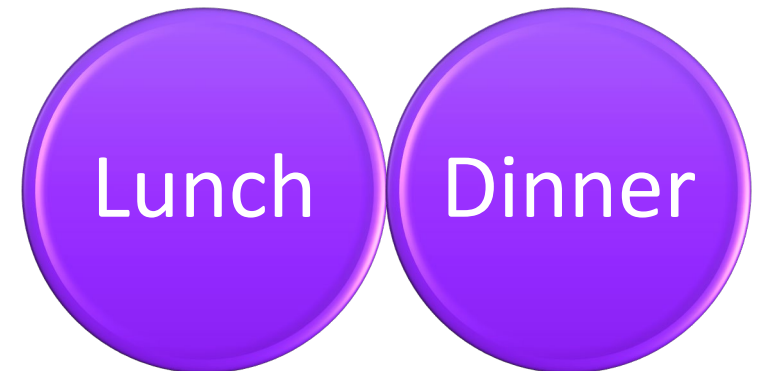- 24 hours
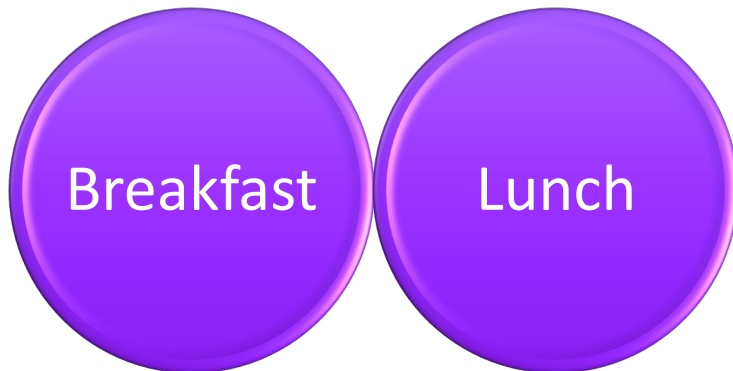- 36 hours
- 42 hours

Frequency

- 16 or 18: daily
- 24, 36, 42: 3 times per week

The 16:8 or 18:6 Fast

Who should do it?

Healthy individuals looking to maintain good health or drop a couple of pounds

Breakfast   Lunch

Lunch   Dinner

The 24-hour Fast

Who should do it?

- Healthy people or very sick people
- No history of dieting
- <20 lbs of weight-loss

How does it work?

- Skip 2 consecutive meals

Day	Breakfast	Lunch	Dinner
Day 1	Fast	Fast	Eat
Day 2	Eat or Fast	Eat	Eat
Day 3	Fast	Fast	Eat
Day 4	Eat or Fast	Eat	Eat
Day 5	Fast	Fast	Eat

The 36-hour Fast

Who should do it?

- Healthier people with metabolic syndrome
- >20 lbs of weight-loss

How does it work?

- Missing three consecutive meals in one day

Day	Breakfast	Lunch	Dinner
Day 1	Fast	Fast	Fast
Day 2	Eat	Eat	Eat
Day 3	Fast	Fast	Fast
Day 4	Eat	Eat	Eat
Day 5	Fast	Fast	Fast

The 42-hour Fast

Who should do it?

- Healthier people with metabolic syndrome
- >20 lbs of weight-loss
- Not hungry in the mornings

How does it work?

- Never eat breakfast
- Miss lunch and dinner three times per week
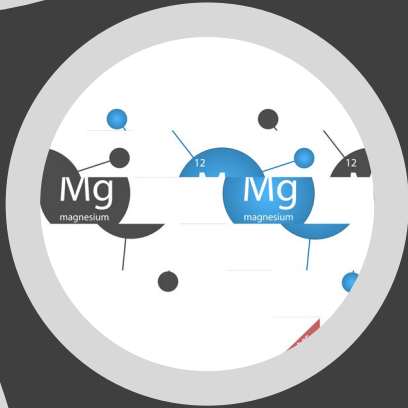
Day	Breakfast	Lunch	Dinner
Day 1	Fast	Fast	Fast
Day 2	Fast	Lunch	Dinner
Day 3	Fast	Fast	Fast
Day 4	Fast	Lunch	Dinner
Day 5	Fast	Fast	Fast

Extended Fasting (EF) Protocols

Duration and Frequency

- 48 hrs, 2 times per week
- 66 hrs + 48 hours, once a week
- 72 hrs, once a week
- 5 days, once a month
- 7+ days, 1-4 times per year
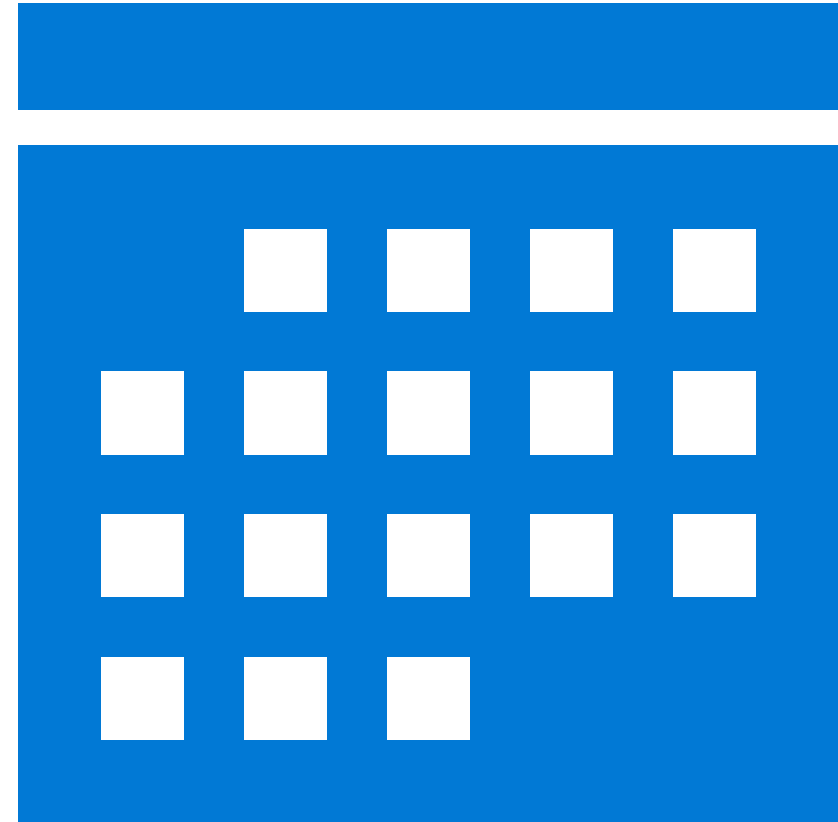
Fasting for Insulin Related Conditions

Fasting for Disease
Prevention/Cancer/Neurological

The Results

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Myth exploded

CASE REPORT

Therapeutic use of intermittent fasting for people with type 2 diabetes as an alternative to insulin

🔓 OPEN ACCESS 📄

Suleiman Furmli[1], **Rami Elmasry**[2,3], **Megan Ramos**[4], **Jason Fung**[4,5]

Table 1	Patient characteristics				
	Age	Sex	Years with type 2 diabetes	Comorbidities	Fasting frequency/duration
Patient 1	40	Male	20	Hypertension. Hypercholesterolaemia.	3×/week for 7 months
Patient 2	52	Male	25	Chronic kidney disease. Renal cell carcinoma (nephrectomy 2004). Hypertension. Hypercholesterolaemia.	3×/week for 11 months
Patient 3	67	Male	10	Hypertension. Hypercholesterolaemia.	Alternating days for 11 months

Table 2 Changes in glycaemic and other health parameters from baseline to end of follow-up

	Initiation HbA1c (%), (mmol/mol)	Final HbA1c (%), (mmol/mol)	Initial diabetic medications	Final diabetic medications	Initial weight (kg)	Final weight (kg)	Initial waist circumference (cm)	Final waist circumference (cm)	Number of days to come off insulin
Patient 1	11, 96.7	7, 53	insulin glargine 58. insulin aspart 22. canagliflozin 300 mg. metformin 1 g.	canagliflozin 300 mg	83.8	73.8	100	87	5
Patient 2	7.2, 55.2	6, 42.1	insulin lispro mix 25–38/32 IU 25.	None	61	50.4	89	70	18
Patient 3	6.8, 50.8	6.2, 44.3	metformin 1000 mg. insulin lispro mix 25–30/20 IU.	None	97.1	88.1	123	110	13

HbA1C, haemoglobin A1C.

Nutrient dense feasting is just as important as fasting

- Try to eat a variety of foods to get a range of nutrients, i.e. can do so on carnivore and omnivore

- Try to use the savings you have from fasting to buy better quality foods, i.e. TruLocal – extra convenient door-to-door delivery

- Eat until you feel satiated – don't be afraid to feel full!

Thank You

www.thefastingmethod.com