The GALEN Foundation

WEBSITE:

WWW.ORLANDOCVI.COM

FACEBOOK:

WWW.FACEBOOK.COM/CVIORLANDO

TWITTER:

WWW. TWITTER.COM/ORLANDO_CVI

THE BITTERSWEET TRUTH

PRADIP JAMNADAS, MD, MBBS, FACC, FSCAI, FCCP, FACP

GALEN IS A FEDERALLY RECOGNIZED 501(C)(3) NON-PROFIT ORGANIZATION THROUGH WHICH DONATIONS ARE TAX DEDUCTIBLE.

PLEASE DONATE GENEROUSLY

WWW.GALEN.ORLANDOCVI.COM

CASH OR CHECK TO ONE OF OUR VOLUNTEERS JUST OUTSIDE OF THE AUDITORIUM

The Galen Foundation

Galen is a federally recognized 501(c)(3) non-profit organization through which donations are tax deductible.
Please donate generously

www.Galen.OrlandoCVI.com

Cash or Check to one of our volunteers just outside of the auditorium

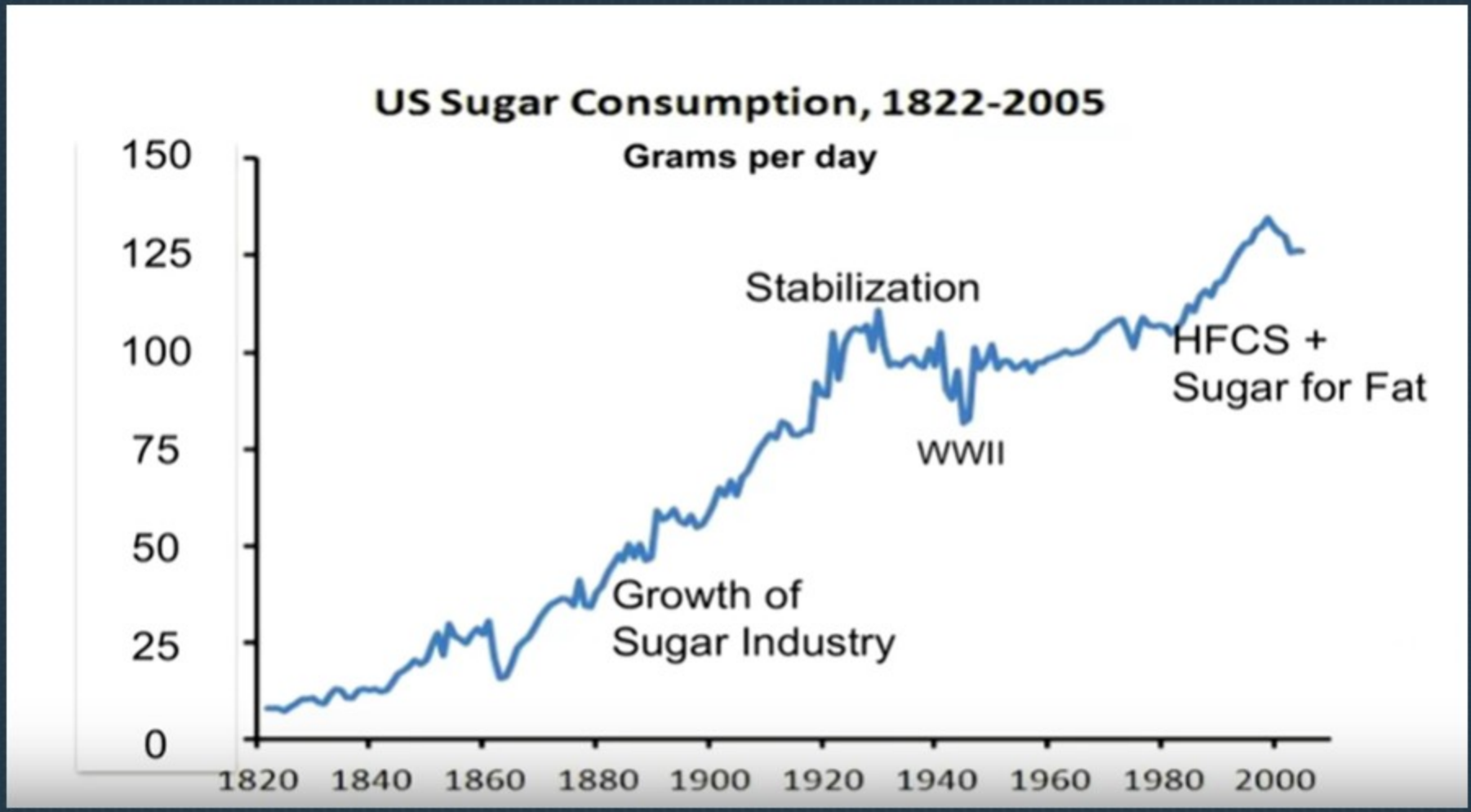
Please join us at the next talk on

Tuesday June 18th, 2019 At 7am & 7pm

FASTING FOR SURVIVAL

PRADIP JAMNADAS, MD
MBBS, FACC, FSCAI, FCCP, FACP

The founder and chairman of The Galen Foundation is a medical graduate of the University College of London, England with post-graduate training at Yale University in Cardiology. He has a successful specialty practice in Orlando, Florida since 1990 and performs interventional procedures and is a consultant cardiologist with a large diversified inpatient and outpatient practice. He has been recognized in Orlando Magazine as Top Doctor in Cardiology for multiple years over the past decade. He is also a clinical assistant professor of medicine at The Florida State University and University of Central Florida. He is a renowned lecturer and teacher, with a passion for high tech interventions when called for, yet places greater emphasis on prevention measures.



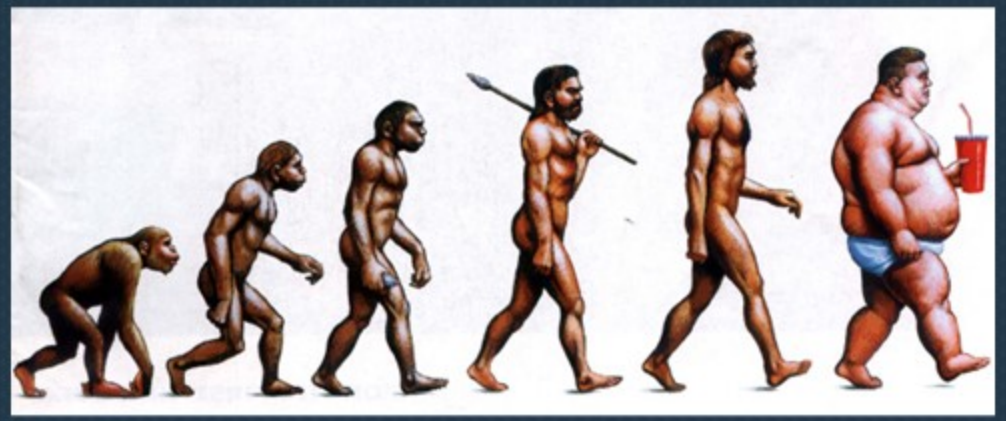
U.S. Commerce Service 1822:1910, combined with Economic Research Service, USDA 1910-2010

Lustig, Robert [JumpStartMD]. (2019, February 1). *What is Metabolic Syndrome Anyway?* Retrieved from https://www.youtube.com/watch?v=zx-QrilOoSM

MAN IS EVOLVING

We are NOT genetically modified humans

Instead, we ARE NOW hormonally-modified humans
- Mainly by INSULIN but many others as well.



BALANCE OF HORMONES

GOALS OF LECTURE

#1. Disprove the calorie-in-out theory

#2. Prove the hormonal imbalance theory

#3. Sugar and carbohydrate drive metabolic states of the body

#4. High carbohydrate diet is driving atherosclerosis, diabetes, hypertension, obesity, and likely dementia and cancer

HORMONAL MODIFICATION RESULTS IN METABOLIC SYNDROME (MS)

- Low HDL (<40mg/dL for males and <50 mg/dL for Females)
- High Triglycerides (>150 mg/dL)
- Large waist line (>40" for males and >35" for Females)
- High blood pressure (>130/>85)
- High blood glucose (>100mg/dL)

Best way to cause MS is to:

- Increase sugar intake
- Add refined wheat flour
- Add polyunsaturated fats

JAMA Intern Med. 2013 Aug 12;173(15):1439-44. doi: 10.1001/jamainternmed.2013.8198.

WHAT IS DRIVING CORONARY ARTERY DISEASE?

ANSWER: METABOLIC SYNDROME

2015 Euroaspire study

-1/3 of MI victims had DM

- 2/3 had undiagnosed DM

- 21% discovered new DM

- 26% had insulin resistance

- 24% were normal (pre-diabetic)

Eur Heart J. 2015 May 14;36(19):1171-7. doi: 10.1093/eurheartj/ehv008. Epub 2015 Feb 9.

WHAT IS DRIVING CORONARY ARTERY DISEASE?

Therefore, of all patients with MI, at least 76% had glucose dysmetabolism (i.e. pre-diabetes or diabetes).

Therefore, DM is TOTALLY undiagnosed in patients with any type of vascular disease. Diabetes is the main driving force of vascular disease.

Diabetes IS A VASCULAR DISEASE.

WHAT IS DRIVING CORONARY ARTERY DISEASE?

Of the 24% of MI victims that were deemed normal, ONLY glucose was measured, NO insulin levels were done! Had insulin levels been performed, I guarantee you that every MI victim had an abnormality in glucose metabolism: HYPERINSULINEMIA

So let's look at how accurate glucose measurements are in telling the whole GLYCEMIC PICTURE

My Proposition: The hidden parameter is really INSULIN – NOT GLUCOSE levels

DEFINING DIABETES WITH FASTING GLUCOSE IS EXTREMELY INACCURATE

Fasting Glucose	Normal Glucose Tolerance	Impaired Glucose Tolerance	Diabetic Glucose Tolerance
Number of Patients	9598	2775	2011
<110 mg/dL	99%	84%	40%
<100 mg/dL	93%	60%	20%
<90mg/dL	71%	32%	13%
<80mg/dL	32%	12%	4%
<70mg/dL	9%	9%	2%
<60 mg/dL	2%	2%	<1%

- 40% of diabetics had a fasting glucose <110
- 20% of diabetics had a fasting glucose of <100
- 60% of Impaired Glucose Tolerance patients had a fasting glucose of <100.
- FASTING GLUCOSE IS HIGHLY INACCURATE AS IT MISSES THE MAJORITY OF PATIENTS THAT NEED INTERVENTION.

Kraft, J. R. 1974. "Glucose/insulin intolerance: A routine clinical laboratory tool enhancing diabetes detection." In O. B. Hunter, ed. *Radioassay: Clinical Concepts.* (Skokie, Illinois: G.D. Searle), 91-106.

FASTING INSULIN LEVELS

Fasting Insulin (μ units/mL)	Normal Glucose Tolerance	Impaired Glucose Tolerance	Diabetic Glucose Tolerance
Number of Patients	9598	2775	2011
0-10	53%	43%	33%
11-15	23%	19%	19%
16-20	11%	14%	14%
22-30	8%	14%	18%
>30	5%	10%	16%

Kraft, J. R. 1974. "Glucose/insulin intolerance: A routine clinical laboratory tool enhancing diabetes detection." In O. B. Hunter, ed. *Radioassay: Clinical Concepts*. (Skokie, Illinois: G.D. Searle), 91-106.

Only 16% of Diabetics had a fasting insulin level of >30 so therefore a fasting insulin level is NOT a good way to diagnose Diabetes or Insulin Resistance.

It is the Insulin response to food that is critical.

GLUCOSE/INSULIN TOLERANCE TEST

EUINSULINEMIA PATTERN I

Time	Fasting	½ hour	1 hour	2 hours	3 hours	4 hours	5 hours
Insulin Level	8	59	61	30	13	7	6

2-hour + 3-hour sum = 43 µU/mL (normal is <60)
Insulin level peaks at ½ hour

Kraft, J. R. 1974. "Glucose/insulin intolerance: A routine clinical laboratory tool enhancing diabetes detection." In O. B. Hunter, ed. *Radioassay: Clinical Concepts*. (Skokie, Illinois: G.D. Searle), 91-106.

GLUCOSE/INSULIN TOLERANCE TEST

HYPERINSULINEMIA PATTERN II

Time	Fasting	½ hour	1 hour	2 hours	3 hours	4 hours	5 hours
Insulin Level	13	93	116	80	46	20	11

2-hour + 3-hour sum = 126 µU/mL (normal is <60)

44% of Normal GTT had this pattern

27% of Impaired GTT had this pattern

8% of Diabetic patients had this pattern

Kraft, J. R. 1974. "Glucose/insulin intolerance: A routine clinical laboratory tool enhancing diabetes detection." In O. B. Hunter, ed. *Radioassay: Clinical Concepts*. (Skokie, Illinois: G.D. Searle), 91-106.

GLUCOSE/INSULIN TOLERANCE TEST

HYPERINSULINEMIA PATTERN III

Time	Fasting	½ hour	1 hour	2 hours	3 hours	4 hours	5 hours
Insulin Level	13	64	93	133	80	34	17

2-hour + 3-hour sum = 213 µU/mL (normal is <60)

Insulin level peaks at 2 hours

24% of Normal GTT had this pattern

58% of Impaired GTT had this pattern

66% of Diabetic patients had this pattern

Kraft, J. R. 1974. "Glucose/insulin intolerance: A routine clinical laboratory tool enhancing diabetes detection." In O. B. Hunter, ed. *Radioassay: Clinical Concepts*. (Skokie, Illinois: G.D. Searle), 91-106.

GLUCOSE/INSULIN TOLERANCE TEST

HYPERINSULINEMIA PATTERN IV

Time	Fasting	½ hour	1 hour	2 hours	3 hours	4 hours	5 hours
Insulin Level	56	147	165	185	135	75	47

2-hour + 3-hour sum = >320 µU/mL (normal is <60)

Fasting Insulin level is >30

6% of Normal GTT had this pattern

10% of Impaired GTT had this pattern

16% of Diabetic patients had this pattern

Kraft, J. R. 1974. "Glucose/insulin intolerance: A routine clinical laboratory tool enhancing diabetes detection." In O. B. Hunter, ed. *Radioassay: Clinical Concepts*. (Skokie, Illinois: G.D. Searle), 91-106.

GLUCOSE/INSULIN TOLERANCE TEST

HYPOINSULINEMIA, LOW INSULIN RESPONSE, PATTERN V

Time	Fasting	½ hour	1 hour	2 hours	3 hours	4 hours	5 hours
Insulin Level	5	15	16	15	10	7	6

2-hour + 3-hour sum = 25 µU/mL (normal is <60)

4% of Normal GTT had this pattern

2% of Impaired GTT had this pattern

8% of Diabetic patients had this pattern

Kraft, J. R. 1974. "Glucose/insulin intolerance: A routine clinical laboratory tool enhancing diabetes detection." In O. B. Hunter, ed. *Radioassay: Clinical Concepts*. (Skokie, Illinois: G.D. Searle), 91-106.

DIABETIC GLUCOSE TOLERANCE TEST

1% had normal insulin test
8% had Pattern II
66% had Pattern III
16% had Pattern IV
8% had Pattern V

Almost 10% of Diabetics are insulin deficient but don't know it!

Hence, they get even more Insulin which makes things worse!

NORMAL GLUCOSE TOLERANCE TEST

22% had normal insulin test
44% had Pattern II
24% had Pattern III
6% had Pattern IV
4% had Pattern V

Thus, normal GTT does not predict hyperinsulinemia!
24% have Pattern III !

IMPAIRED GLUCOSE TOLERANCE TEST

3% had normal insulin test
27% had Pattern II
58% had Pattern III
10% had Pattern IV
2% had Pattern V

Thus, 68% of patients with impaired GTT have severe hyperinsulinemia!

FUNCTIONAL HYPOGLYCEMIA

>2 hours later in GTT – Glucose is 20-50 mg/dL
- 75% have increased insulin levels! (i.e. early Diabetes)

ALIMENTARY HYPOGLYCEMIA

- Dumping Syndrome
- Blood sugar <50 mg/dL before 2 hours post-prandial!

- Postprandial hypoglycemia also identifies DM

SO, WHAT IS THE PATHOLOGY OF DIABETES?

IT IS ATHEROSCLEROSIS

Patients have high insulin levels years before their blood glucose levels increase.

Atherosclerosis starts when the Insulin levels rise and by the time the glucose levels rise, it is TOO LATE; they already have extensive disease.

WHAT IS THE PATHOLOGY OF DIABETES?

Based on current evidence, the difference between diabetes and pre-diabetes is ARBITRARY and MISLEADING and current work up and treatment are antiquated, outdated, and misses the boat!

We have been chasing the wrong parameter; glucose levels instead of insulin levels!

INSULIN RESISTANCE AS A PREDICTOR OF AGE-RELATED DISEASE

Degree of Insulin Resistance	# of Dead or Diseased
High Insulin Resistance	28
Medium Insulin Resistance	12
Low Insulin Resistance	0

This study followed 208 healthy subjects (not obese) for 6 years.

Outcome: Only insulin status was the MOST predictor of patient's health

J Clin Endocrinol Metab. 2001 Aug;86(8):3574-8.

ALL-CAUSE DEATH RATES IS ALSO RELATED TO HEMOGLOBIN A1C (DUE TO INSULIN LEVELS)

In CAD, cancer, respiratory disease, infectious disease, and strokes

Ann Intern Med. 2004 Sep 21;141(6):413-20.

HOW DOES INSULIN/GLUCOSE DYSMETABOLISM CAUSE DISEASE?

Dr. Stout – in 1970s showed that increased INSULIN causes endothelial dysfunction

- Which then causes microangiopathy of the retina and glomerulus, CAD, microvascular disease of the heart, and Central Nervous System (CNS) disease

HOW DOES INSULIN/GLUCOSE DYSMETABOLISM CAUSE DISEASE?

Dr. Stout said that there is little evidence that high glucose ALONE causes vascular disease – that atherosclerosis is stimulated by INSULIN.

- Endothelial dysfunction
- Smooth muscle proliferation
- Lipid deposition
- Lack of repair (Decreased growth hormone)
- Increased catabolism causing arterial disease

DIABETES MELLITUS INCREASES CARDIOVASCULAR DISEASE BY 500%

BUT, it appears that it is the insulin level "THE HIDDEN PARAMETER".

SO HOW DID INSULIN LEVELS GET SO HIGH?

INSULIN RESISTANCE

- Frequent meals
- Sugar intake is MASSIVE (50% Glucose and 50% Fructose)
- Fructose intake is HIGH (HFCS is 55-60% Fructose and 40-45% glucose)

SO HOW DID INSULIN LEVELS GET SO HIGH?

SUGAR INTAKE TRENDS

1977	228 Calories	57g sugar/day
2000	340 Calories	85g sugar/day
2004	388 Calories	97g sugar/day
NOW	600 Calories	>120g sugar/day

We obeyed the "LOW FAT DIET" mantra

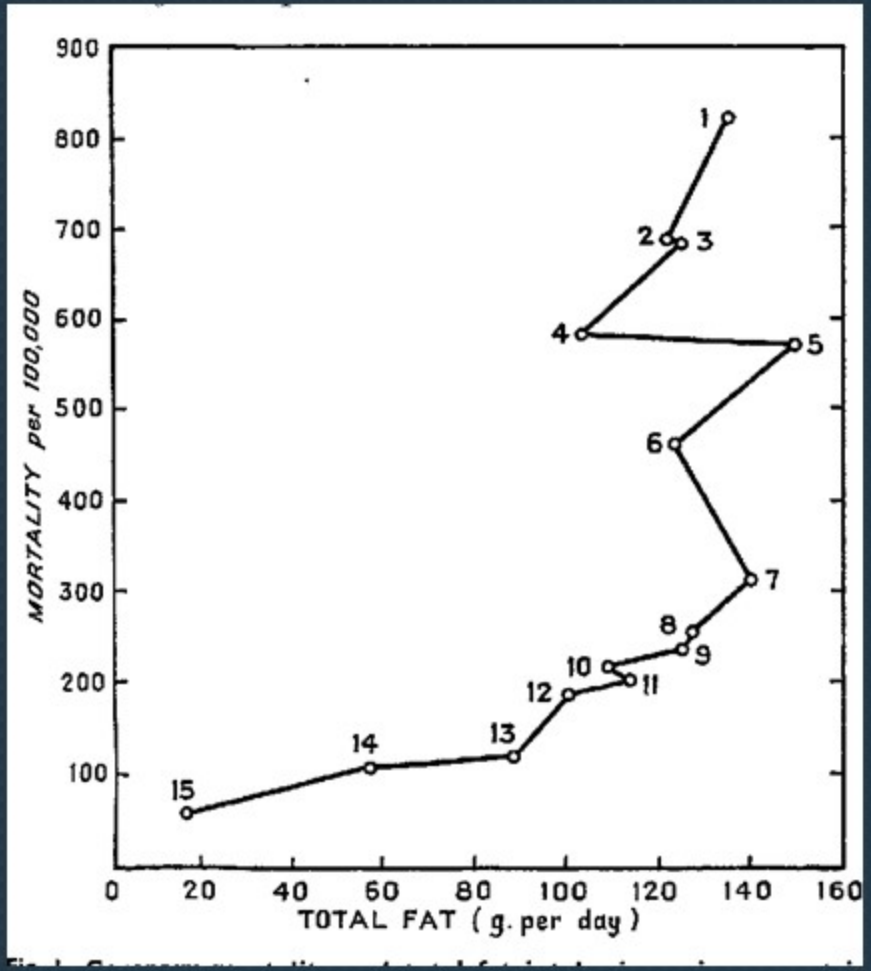
American Food Pyramid emphasized to eat CARBOHYDRATES and to avoid FAT.

"A Brief History of USDA Food Guides." *Choose MyPlate*, 30 Nov. 2018, www.choosemyplate.gov/brief-history-usda-food-guides.

KEY

1. USA
2. Australia
3. Canada
4. Finland
5. New Zealand
6. UK
7. Denmark
8. Sweden
9. Norway
10. Netherlands
11. Switzerland
12. W. Germany
13. France
14. Italy
15. Japan

Fig. – 1 Coronary mortality and total fat intake in various countries.



No significant association with fat
P > 0.05

Fig. 9 – Coronary mortality and sugar intake in various countries

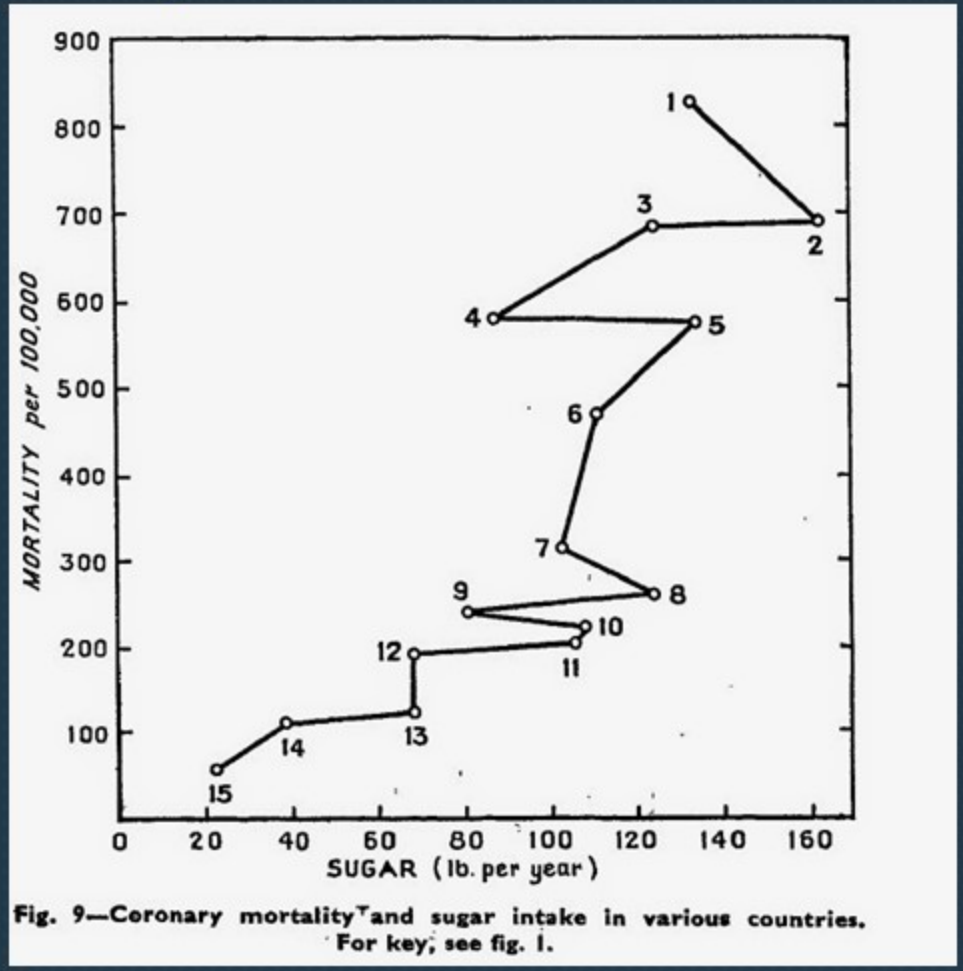


Fig. 9—Coronary mortality and sugar intake in various countries. For key, see fig. 1.

Significant association with sugar
P < 0.05

Cummins, Ivor [Ivor Cummins]. (2014, January 14). *Sugar as a Primary Root Cause of Metabolic Syndrome and the Obesity/Diabetes Ep*

Lancet. 1957 Jul 27;273(6987):155-62. from https://www.youtube.com/watch?v=xrsHqIKtXNw

Annu Rev Nutr. 1996;16:523-57.

Regulation of hepatic de novo lipogenesis in humans.

Hellerstein MK[1], Schwarz JM, Neese RA.

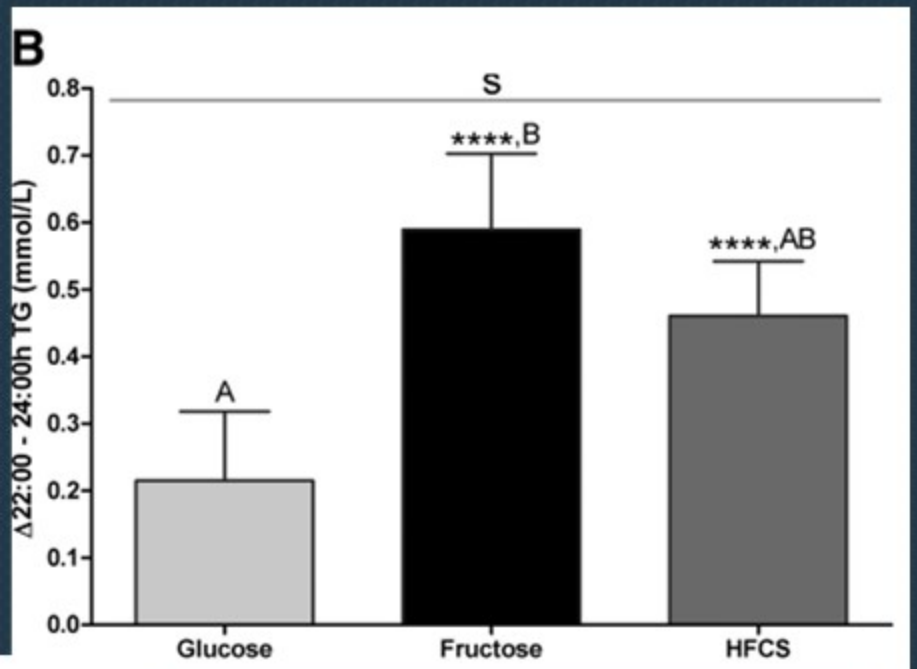
Ann Intern Med. 1965 Jun;62:1199-212.

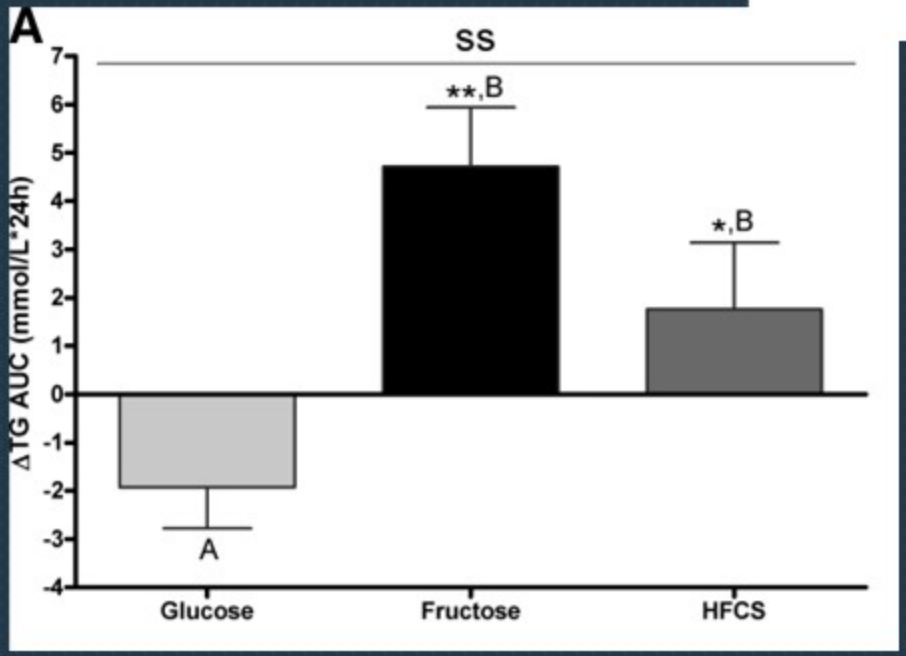
DIETARY SUGAR IN THE PRODUCTION OF HYPERGLYCERIDEMIA.

KUO PT, BASSETT DR.

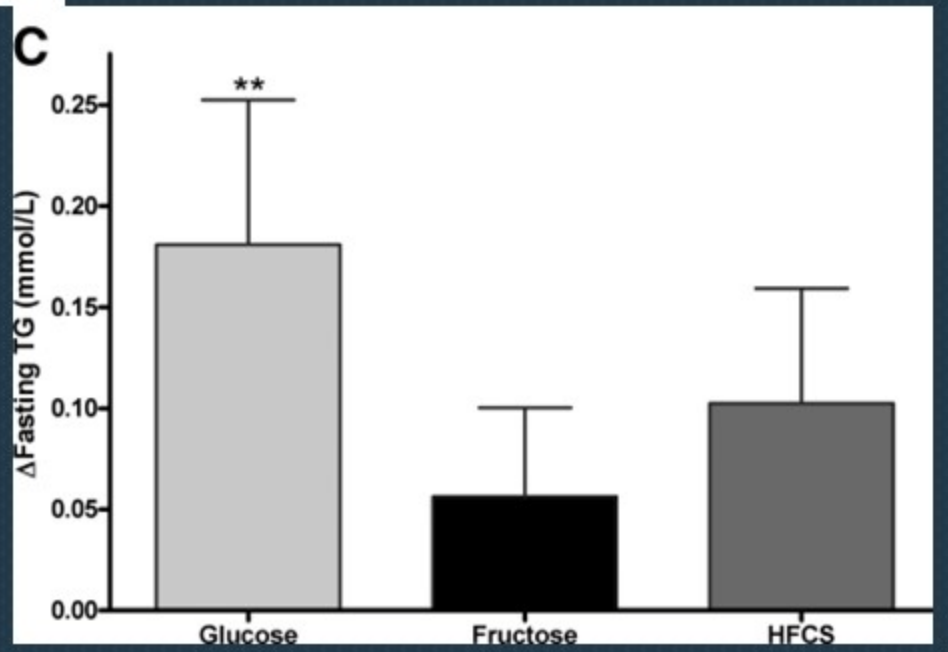
These studies demonstrated that excess carbohydrates directly feed into new fat formation by the liver and this is now very well established.

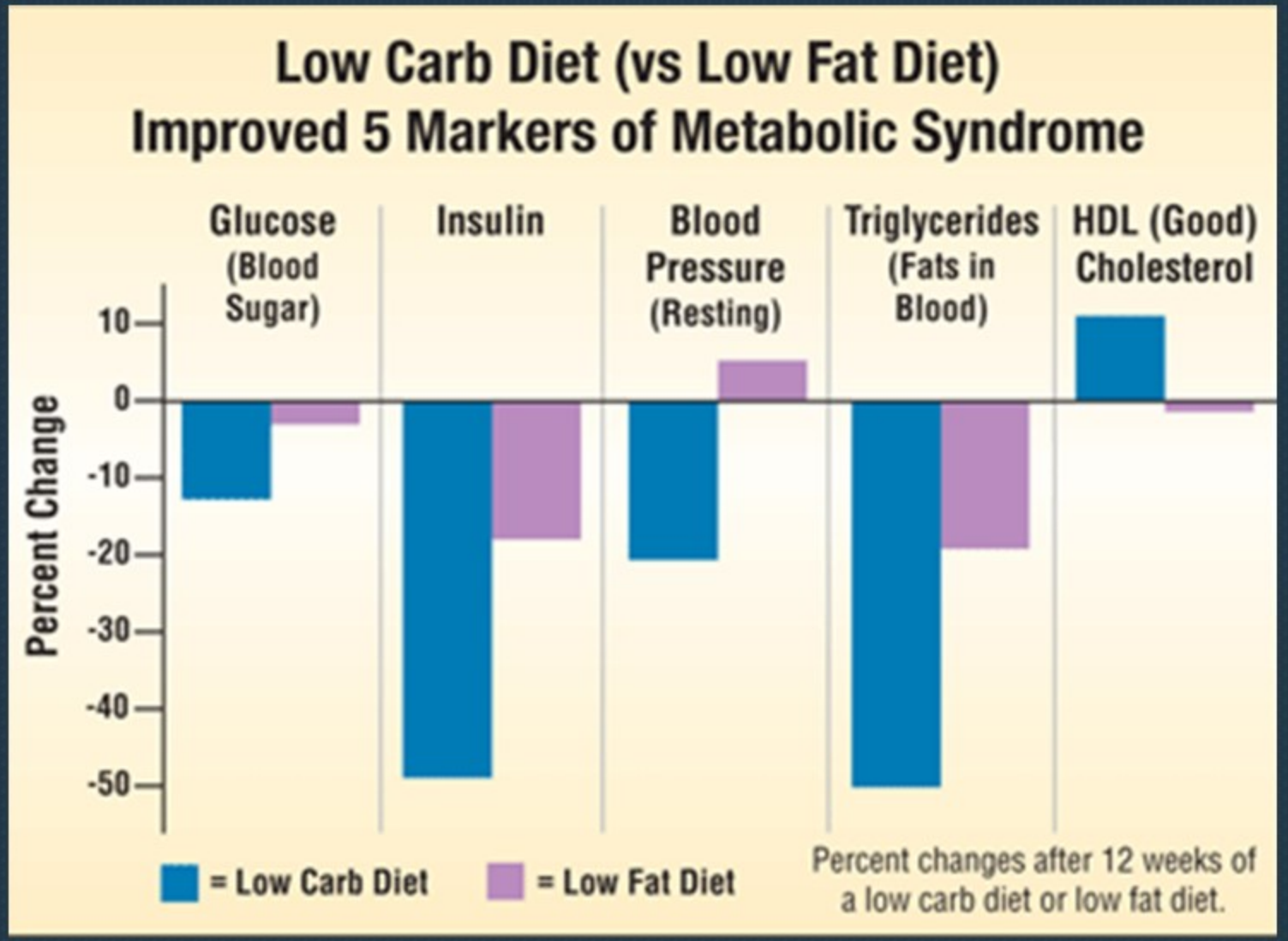
2011: FRUCTOSE DRIVES POST-PRANDIAL TRIGLYCERIDES





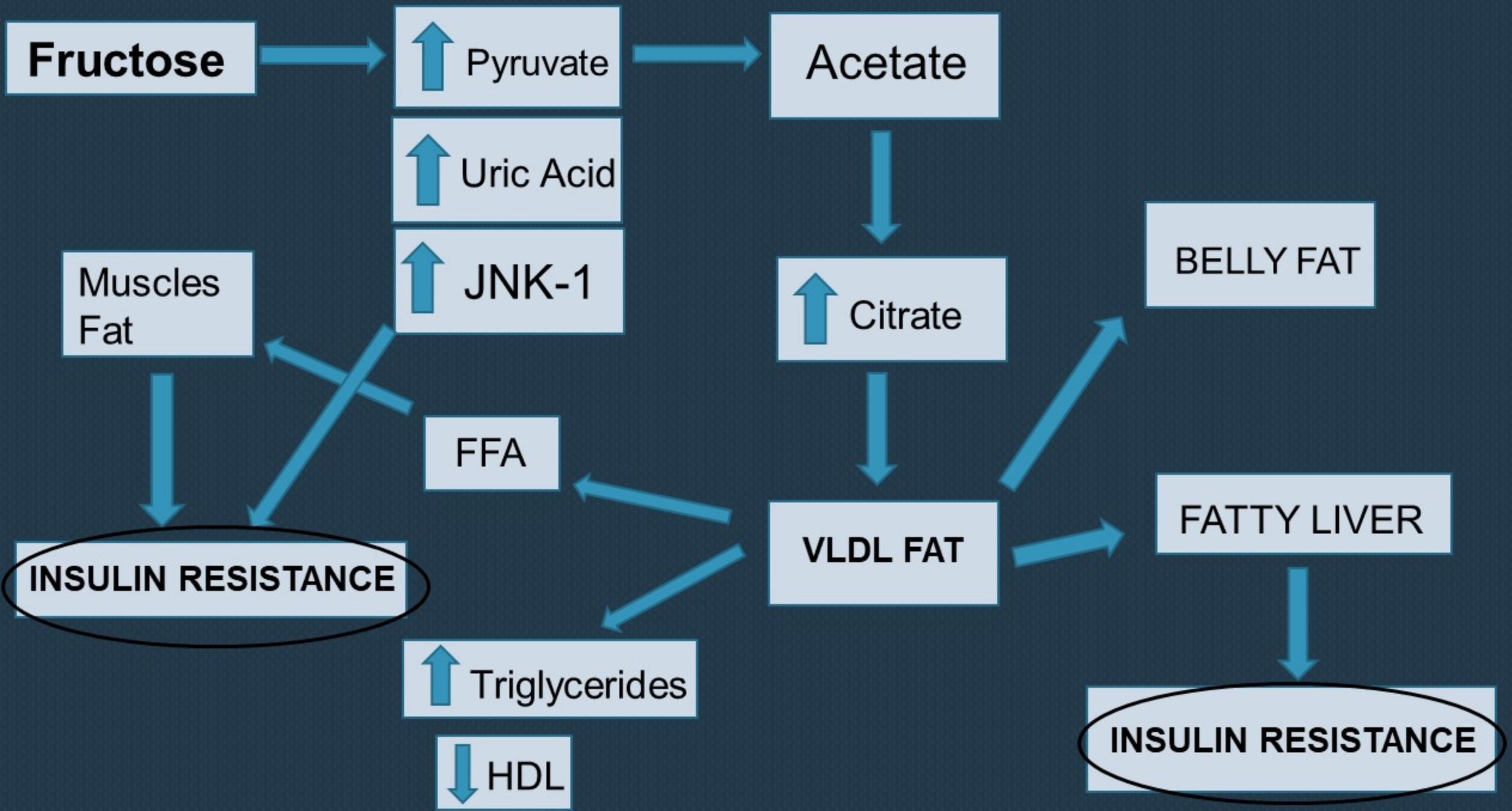
J Clin Endocrinol Metab. 2011 Oct;96(10):E1596-605. doi: 10.1210/jc.2011-1251. Epub 2011 Aug 17.





The Bittersweet Truth: It is our craving of carbohydrates that has altered us hormonally and metabolically. It has caused an explosion of obesity, hypertension, atherosclerosis, Alzheimer's, premature death, probably contributing to cancer, and endless morbidity.

Jeff S. Volek, Ph.D., R.D.
Cummins, Ivor [Ivor Cummins]. (2014, January 14). *Sugar as a Primary Root Cause of Metabolic Syndrome and the Obesity/Diabetes Epidemic.* Retrieved from https://www.youtube.com/watch?v=xrsHqIKtXNw


Fructose
Pyruvate
Acetate
Uric Acid
JNK-1
Muscles Fat
Citrate
BELLY FAT
FFA
INSULIN RESISTANCE
VLDL FAT
FATTY LIVER
Triglycerides
HDL
INSULIN RESISTANCE

Fructose is not glucose

- Fructose is 7 times more likely than glucose to form Advanced Glycation End-Products (AGE's)
- Fructose does not suppress ghrelin
- Acute fructose does not stimulate insulin (or leptin)
- Hepatic fructose metabolism is different
- **Chronic fructose exposure promotes the metabolic syndrome**

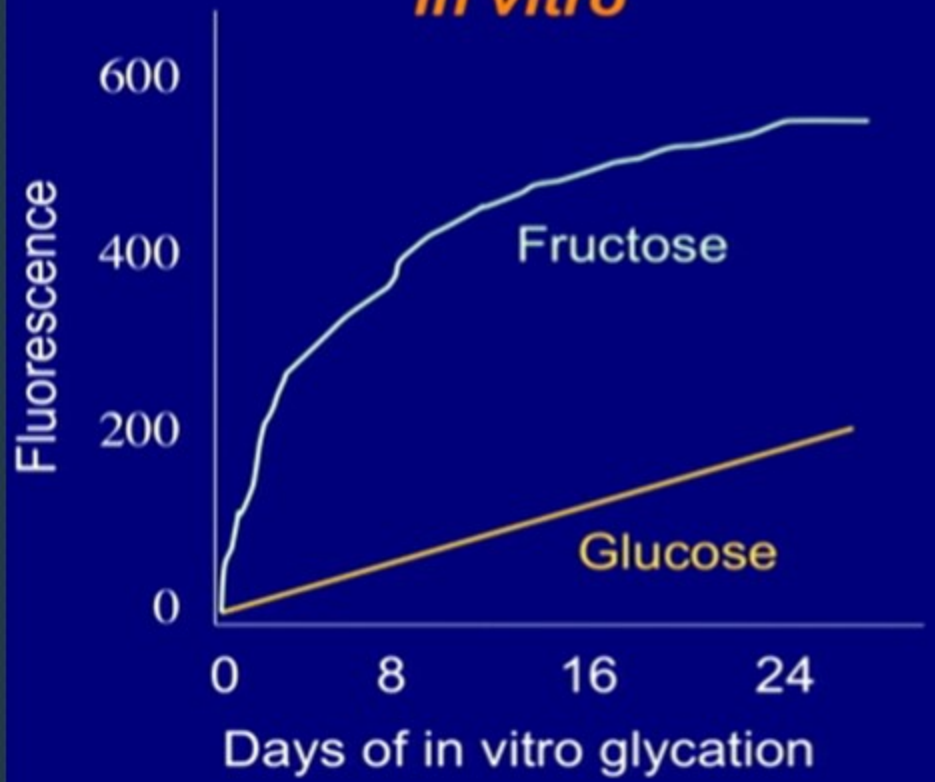
Elliot et al. Am J Clin Nutr, 2002
Bray et al. Am J Clin Nutr, 2004
Teff et al. J Clin Endocrinol Metab, 2004
Gaby, Alt Med Rev, 2005

Le and Tappy, Curr Opin Clin Nutr Metab Care, 2006
Wei et al. J Nutr Biochem, 2006
Johnson et al. Am J Clin Nutr 2007
Rutledge and Adeli, Nutr Rev, 2007
Brown et al. Int. J. Obes, 2008

Lustig, Robert [JumpStartMD]. (2019, February 1). *What is Metabolic Syndrome Anyway?* Retrieved from https://www.youtube.com/watch?v=zx-QriIOoSM

Non-enzymatic glycation: fructose >> glucose

Fructose and glycation *in vitro*



Ahmed and Furth, Clin Chem 38:1301, 1992

Rates of reactivity

	Rate (/mM/hr)	Carbonyl %
Glucose	0.6	0.002
Galactose	2.8	0.02
Fructose	4.5	0.7

Bunn and Higgins, Science 213:222, 1981

Clin Chem. 1992 Jul;38(7):1301-

Lustig, Robert [JumpStartMD]. (2019, February 1). *What is Metabolic Syndrome Anyway?* Retrieved from https://www.youtube.com/watch?v=zx-QrilOoSM

Obesity (Silver Spring). 2016 Feb;24(2):453-60. doi: 10.1002/oby.21371. Epub 2015 Oct 26.

Isocaloric fructose restriction and metabolic improvement in children with obesity and metabolic syndrome.

Lustig RH[1], Mulligan K[2,3], Noworolski SM[4], Tai VW[2], Wen MJ[2], Erkin-Cakmak A[1], Gugliucci A[3], Schwarz JM[5].

Strategy

- Isocaloric fructose restriction x 9 days in children who are habitual sugar consumers
- No change in weight
- Substitute complex carbs for sugar
- Maintain baseline macronutrient composition of the the diet
- Study in PCRC at Day 0 and Day 10
- Assess changes in organ fat, *de novo* lipogenesis, and metabolic health

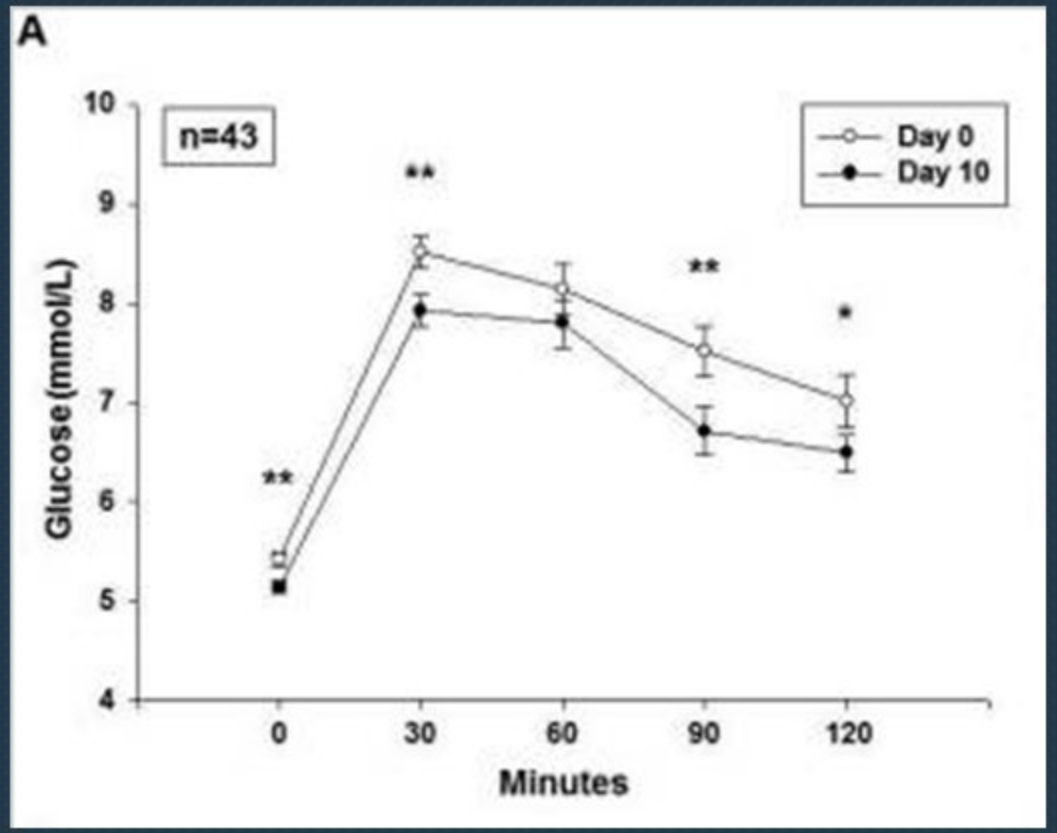
Lustig, Robert [JumpStartMD]. (2019, February 1). *What is Metabolic Syndrome Anyway?* Retrieved from https://www.youtube.com/watch?v=zx-QrilOoSM

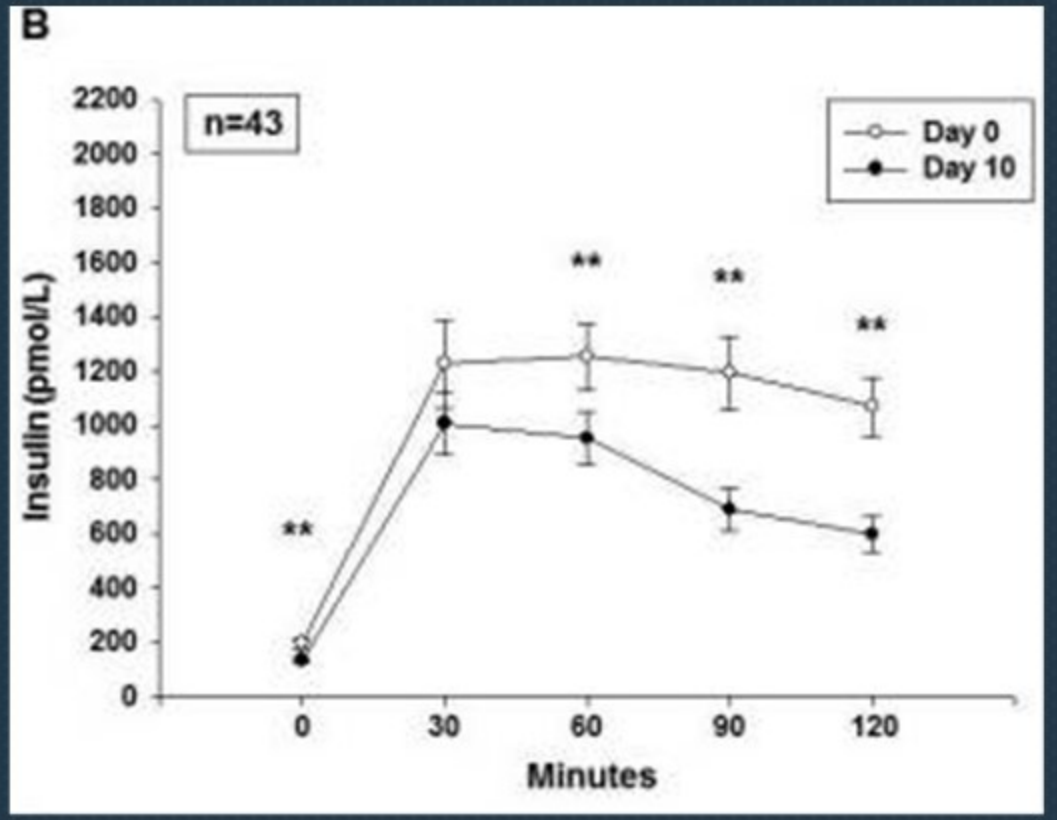
Fasting Labs

	Day 0	Day 10	β−coefficient (Adjusted Change) [95% CI]	p value
Heart rate (bpm)	83.1 ± 10.7	80.1 ± 11.3	-2.8 [-6.5, +0.9]	0.13
Systolic BP (mmHg)	122.6 ± 10.5	121.1 ± 9.9	- 1.39 [-4.9, +2.1]	0.43
Diastolic BP	68.8 ± 8.9	63.7 ± 7.5	- 4.9 [-8.1, -1.8]	0.003
Fasting lactate (mmol/L)	1.2 ± 0.4	0.9 ± 0.3	-0.3 [-0.5, -0.2]	<0.001
Lactate AUC (mM/120 min)	160.0 ± 34.5	129.0 ± 34.5	-31.2 [-41.9, -20.5]	<0.001
HOMA-IR[Y]	7.9 ± 4.8	5.2 ± 2.6	-2.7 [-3.8, -1.5]	<0.001
AST (U/L) *	27.4 ± 14.1	23.8 ± 8.9		0.02
ALT (U/L) [Y]	28.9 ± 22.8	26.7 ± 19.6	-2.2 [-4.7, +0.3]	0.09
Fasting TG (mM)	1.4 ± 0.9	1.0 ± 0.5	-0.4 [-0.6, -0.2]	0.002
Fasting LDL-C (mM)	2.4 ± 0.6	2.1 ± 0.6	-0.3 [-0.4, -0.1]	<0.001
Fasting HDL-C (mM)	1.2 ± 0.2	1.0 ± 0.2	-0.1 [-0.2, -0.1]	<0.001
Fasting FFA (mM)	0.6 ± 0.2	0.7 ± 0.2	+0.1 [+0.1, +0.2]	<0.001

Lustig, Robert [JumpStartMD]. (2019, February 1). *What is Metabolic Syndrome Anyway?* Retrieved from https://www.youtube.com/watch?v=zx-QrilOoSM

Oral glucose tolerance test before and after isocaloric fructose restriction





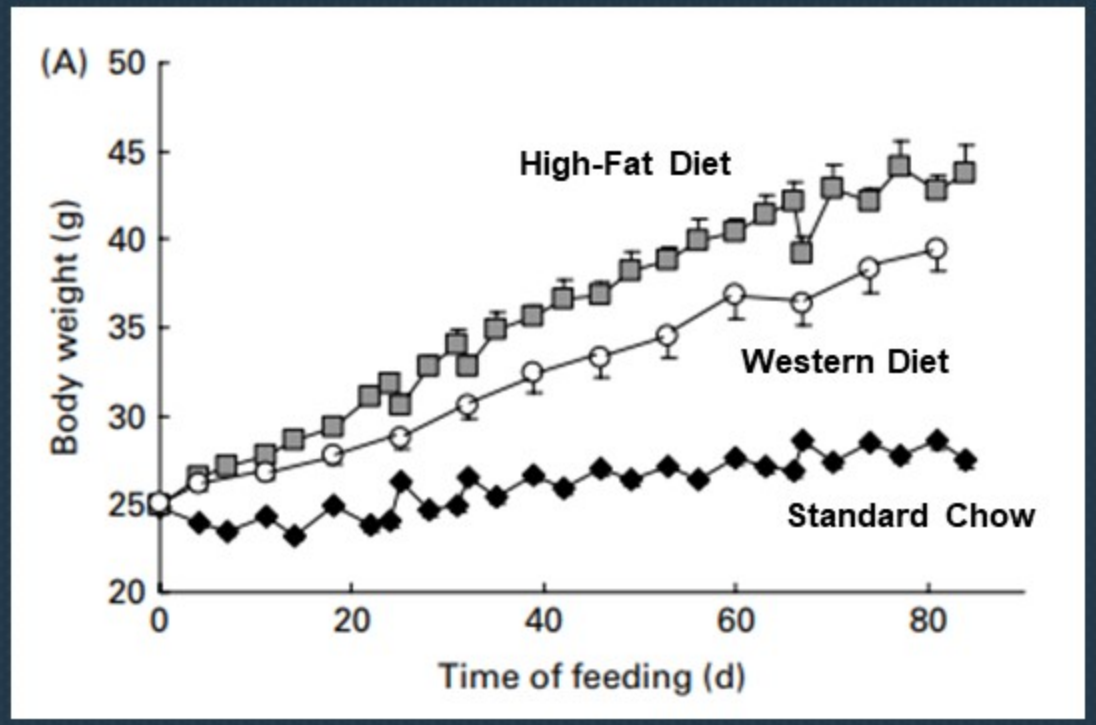
Obesity (Silver Spring). 2016 Feb;24(2):453-60. doi: 10.1002/oby.21371. Epub 2015 Oct 26.

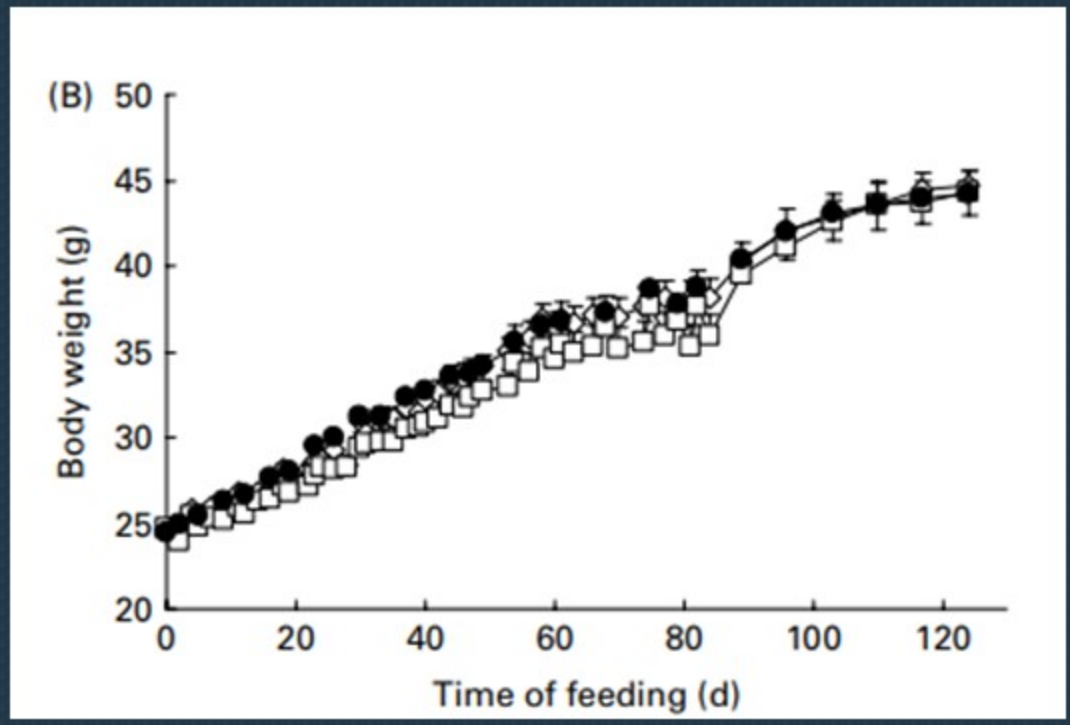
Lustig, Robert [JumpStartMD]. (2019, February 1). *What is Metabolic Syndrome Anyway?* Retrieved from https://www.youtube.com/watch?v=zx-QrilOoSM

Br J Nutr. 2013 Apr 28;109(8):1518-27. doi: 10.1017/S0007114512003340. Epub 2012 Aug 6.

Diet-induced obesity in ad libitum-fed mice: food texture overrides the effect of macronutrient composition.

Desmarchelier C[1], Ludwig T, Scheundel R, Rink N, Bader BL, Klingenspor M, Daniel H.



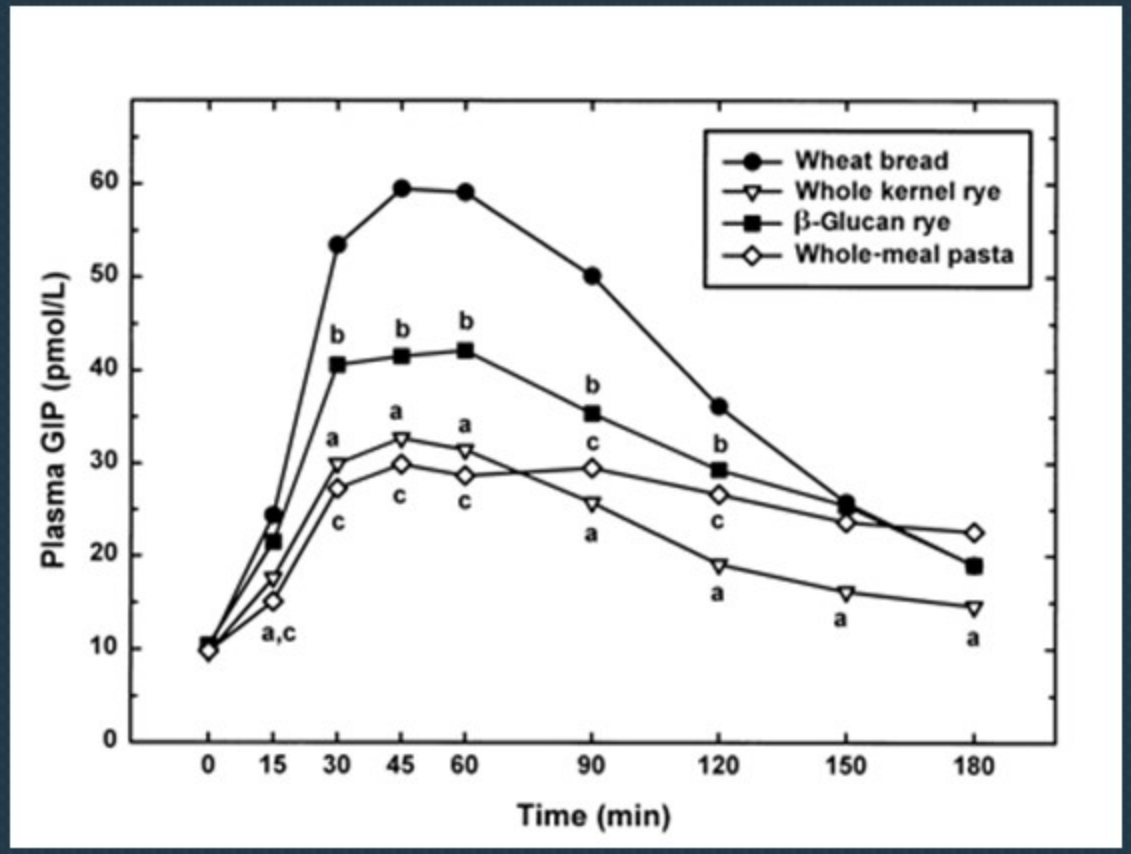


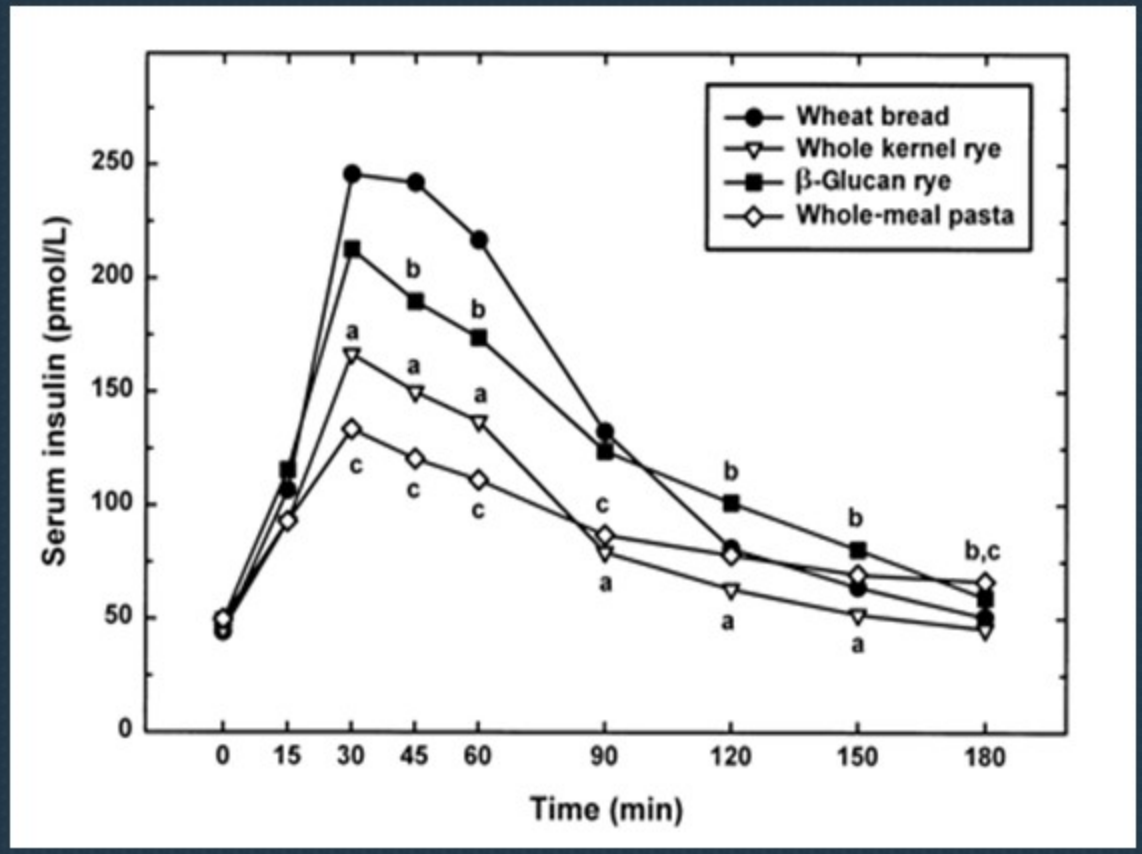
Changing the standard chow diet to a pelleted form makes it as bad as a western diet and high-fat diet in terms of weight gain

Am J Clin Nutr. 2002 Feb;75(2):254-62.

Postprandial glucose, insulin, and incretin responses to grain products in healthy subjects.

Juntunen KS[1], Niskanen LK, Liukkonen KH, Poutanen KS, Holst JJ, Mykkänen HM.





Similarly, Insulin and GIP responses are greatest to refined bread products

Excess Exposure to Insulin Is the Primary Cause of Insulin Resistance and its Associated Atherosclerosis

Authors: Cao, Wenhong; Ning, Jie; Yang, Xuefeng; Liu, Zhenqi
Source: Current Molecular Pharmacology, Volume 4, Number 3, 2011, pp. 154-166(13)
Publisher: Bentham Science Publishers

Int J Clin Pract Suppl. 2004 Oct;(143):9-21.

Dysfunctional fat cells, lipotoxicity and type 2 diabetes.

DeFronzo RA[1].

Nutr Metab (Lond). 2005; 2: 5.
Published online 2005 Feb 21. doi: 10.1186/1743-7075-2-5

Fructose, insulin resistance, and metabolic dyslipidemia

Heather Basciano,[1] Lisa Federico,[1] and Khosrow Adeli[1]

HIGH sugar intake causes Insulin Resistance in FAT CELLS

FAT CELL DYSMETABOLISM then causes insulin resistance in other organs

LIVER insulin resistance is caused mainly by FRUCTOSE

INSULIN RESISTANCE

CAUSED BY: Excess insulin levels

HOW TO CORRECT HYPERINSULINEMIA?

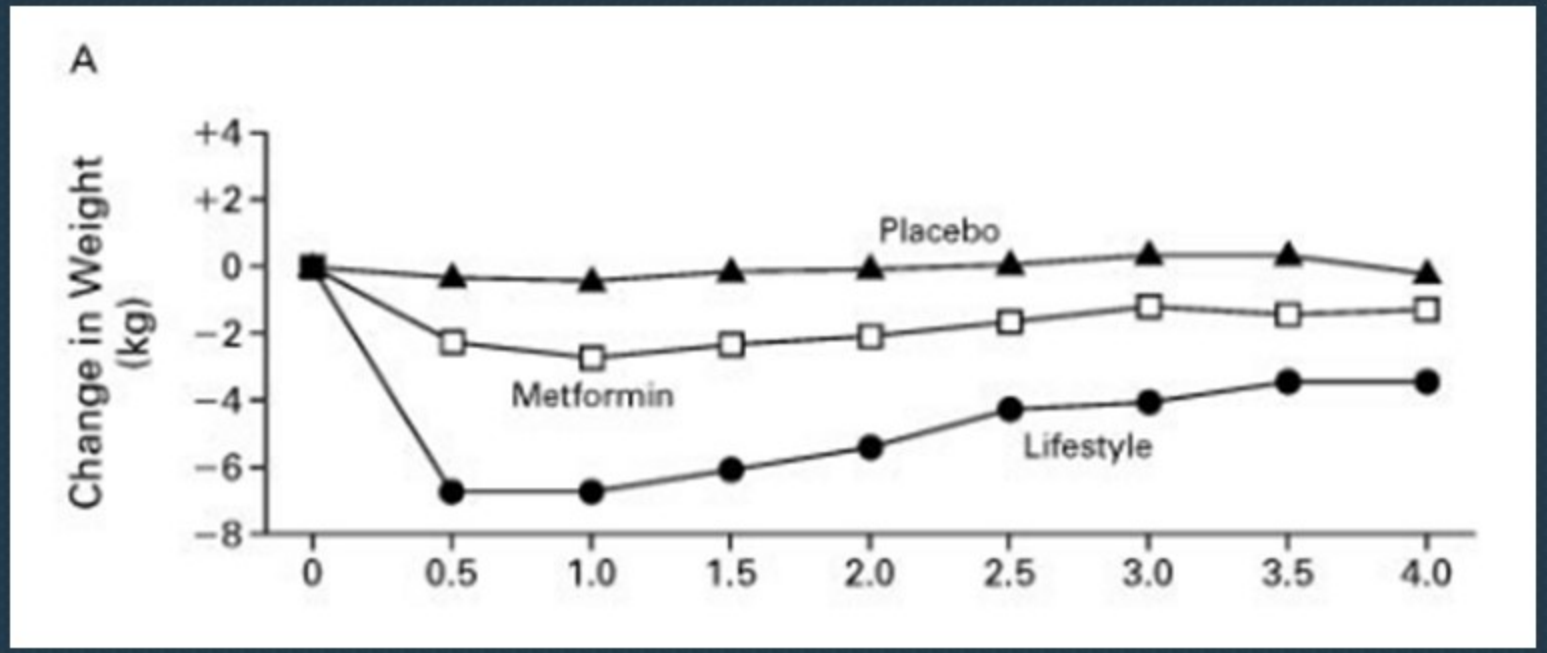
Create low levels of Insulin

HOW TO CREATE LOW LEVELS OF INSULIN?

- Without drugs, it is through diet and fasting which restores sensitivity of insulin to cells again
- The day after low levels of Insulin, i.e. a fast, your body becomes more sensitive to Insulin and Glucose levels fall the next day in response to a meal because of better sensitivity.

Reduction in the Incidence of Type 2 Diabetes with Lifestyle Intervention or Metformin

Diabetes Prevention Program Research Group*



Lifestyle changes resulted in initial weight loss, but eventually weight was gained back

N Engl J Med 2002; 346:393-403

Fung, Jason. "5: Fasting for Weight Loss." *The Complete Guide to Fasting: Heal Your Body through Intermittent, Alternate-Day, and Extended Fasting*, by Jason Fung and Jimmy Moore, Victory Belt Publishing, 2016, pp. 115–120.

The effects of intermittent or continuous energy restriction on weight loss and metabolic disease risk markers: a randomised trial in young overweight women

Michelle N. Harvie, Mary Pegington, Mark P. Mattson, Jan Frystyk, Bernice Dillon, Gareth Evans, Jack Cuzick, Susan A Jebb, Bronwen Martin, Roy G. Cutler, Tae G. Son, Stuart Maudsley, Olga D. Carlson, Josephine M. Egan, Allan Flyvbjerg, and Anthony Howell

Parameter		Baseline	1 Month	3 Month	6 Month	P value[4]
Insulin (μU/ml)[2]	IER	7.3 (6.3 to 8.4)	6.4 (5.7 to 7.3)	5.6 (4.7 to 6.5)	5.2[3] (4.5 to 6.0)	0.04
	CER	7.4 (6.4 to 8.6)	6.5 (5.7 to 7.5)	6.3 (5.4 to 7.3)	6.3[3] (5.4 to 7.4)	

IER – Intermittent Energy Restriction

CER – Complete Energy Restriction

Over time, IER reduced Insulin levels more than CER did.

South Med J. 1988 Jan;81(1):61-3.

Reducing the serum cholesterol level with a diet high in animal fat.

Newbold HL.

Abstract

Multiple food allergies required a group of seven patients with elevated serum cholesterol levels to follow a diet in which most of the calories came from beef fat. Their diets contained no sucrose, milk, or grains. They were given nutritional supplements. This is the only group of people in recent times to follow such a diet. During the study, the patients' triglyceride levels decreased from an average of 113 mg/dl to an average of 74 mg/dl; at the same time, their serum cholesterol levels fell from an average of 263 mg/dl to an average of 189 mg/dl. At the beginning of the study, six of the patients had an average high-density lipoprotein percentage of 21%. At the end of the study, the average had risen to 32%. These findings raise an interesting question: are elevated serum cholesterol levels caused in part not by eating animal fat (an extremely "old food"), but by some factor in grains, sucrose, or milk ("new foods") that interferes with cholesterol metabolism?

Cholesterol	**Fell by 27.5% from an average of 263 mg/dL to 189 mg/dL**
HDL	**Increased from 21% of the total to 32%**
Triglyceride	**Decreased from average of 1.13g/L to a more healthy average of 0.74g/L**

Impressive lipid changes by changing to "unhealthy" diet with more fat and no sugar, milk, or grains.

Low-Carbohydrate-Diet Score and the Risk of Coronary Heart Disease in Women

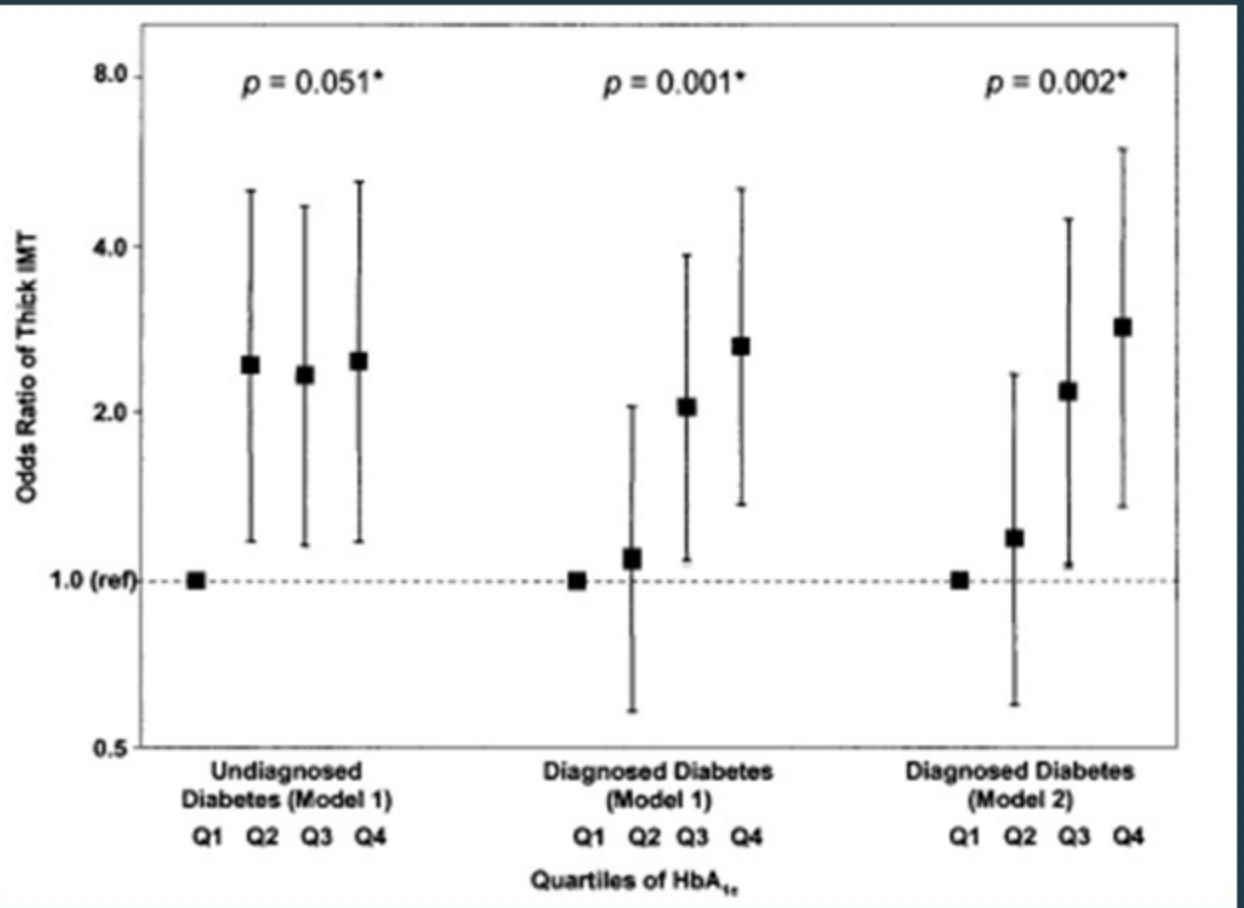
Thomas L. Halton, Sc.D., Walter C. Willett, M.D., Dr.P.H., Simin Liu, M.D., Sc.D., JoAnn E. Manson, M.D., Dr.P.H., Christine M. Albert, M.D., M.P.H., Kathryn Rexrode, M.D., and Frank B. Hu, M.D., Ph.D.

In conclusion, diets lower in carbohydrate and higher in protein and fat were not associated with an increased risk of coronary heart disease in this cohort of women. When vegetable sources of fat and protein were chosen, these diets were related to a lower risk of coronary heart disease.

Glycemic Control, Atherosclerosis, and Risk Factors for Cardiovascular Disease in Individuals With Diabetes

The Atherosclerosis Risk in Communities study

Elizabeth Selvin, PHD, MPH12, Josef Coresh, MD, PHD123, Sherita H. Golden, MD, MHS123, Lori L. Boland, MPH4, Frederick L. Brancati, MD, MHS123 and Michael W. Steffes, MD, PHD5



High glucose levels correlate to atherosclerosis

Am J Cardiol. 2000 Jan 1;85(1):45-8.

High carbohydrate diets, triglyceride-rich lipoproteins, and coronary heart disease risk.

Abbasi F[1], McLaughlin T, Lamendola C, Kim HS, Tanaka A, Wang T, Nakajima K, Reaven GM.

Abstract

In this study we compared the effects of variations in dietary fat and carbohydrate (CHO) content on concentrations of triglyceride-rich lipoproteins in 8, healthy, nondiabetic volunteers. The diets contained, as a percentage of total calories, either 60% CHO, 25% fat, and 15% protein, or 40% CHO, 45% fat, and 15% protein. They were consumed in random order for 2 weeks, with a 2-week washout period in between. Measurements were obtained at the end of each dietary period of plasma triglyceride, cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, remnant lipoprotein (RLP) cholesterol, and RLP triglyceride concentrations, both after an overnight fast and throughout an 8-hour period (8 A.M. to 4 P.M.) in response to breakfast and lunch. The 60% CHO diet resulted in higher (mean +/- SEM) fasting plasma triglycerides (206 +/- 50 vs 113 +/- 19 mg/dl, p = 0.03), RLP cholesterol (15 +/- 6 vs 6 +/- 1 mg/dl, p = 0.005), RLP triglyceride (56 +/- 25 vs 16 +/- 3 mg/dl, p = 0.003), and lower HDL cholesterol (39 +/- 3 vs 44 +/- 3 mg/dl, p = 0.003) concentrations, without any change in LDL cholesterol concentration. Furthermore, the changes in plasma triglyceride, RLP cholesterol, and RLP triglyceride persisted throughout the day in response to breakfast and lunch. These results indicate that the effects of lowfat diets on lipoprotein metabolism are not limited to higher fasting plasma triglyceride and lower HDL cholesterol concentrations, but also include a persistent elevation in RLPs. Given the atherogenic potential of these changes in lipoprotein metabolism, it seems appropriate to question the wisdom of recommending that all Americans should replace dietary saturated fat with CHO.

"Given the atherogenic potential of these changes in Lipoprotein metabolism, it seems appropriate to question the wisdom of recommending that all Americans should replace dietary saturated fat with carbohydrates"

Lancet. 2002 Jun 22;359(9324):2140-4.

Glucose metabolism in patients with acute myocardial infarction and no previous diagnosis of diabetes mellitus: a prospective study.

Norhammar A[1], Tenerz A, Nilsson G, Hamsten A, Efendíc S, Rydén L, Malmberg K.

Abstract

BACKGROUND: Glycometabolic state at hospital admission is an important risk marker for long-term mortality in patients with acute myocardial infarction, whether or not they have known diabetes mellitus. Our aim was to ascertain the prevalence of impaired glucose metabolism in patients without diagnosed diabetes but with myocardial infarction, and to assess whether such abnormalities can be identified in the early course of a myocardial infarction.

METHODS: We did a prospective study, in which we enrolled 181 consecutive patients admitted to the coronary care units of two hospitals in Sweden with acute myocardial infarction, no diagnosis of diabetes, and a blood glucose concentration of less than 11.1 mmol/L. We recorded glucose concentrations during the hospital stay, and did standardised oral glucose tolerance tests with 75 g of glucose at discharge and again 3 months later.

FINDINGS: The mean age of our cohort was 63.5 years (SD 9) and the mean blood glucose concentration at admission was 6.5 mmol/L (1.4). The mean 2-h postload blood glucose concentration was 9.2 mmol/L (2.9) at hospital discharge, and 9.0 mmol/L (3.0) 3 months later. 58 of 164 (35%, 95% CI 28-43) and 58 of 144 (40%, 32-48) individuals had impaired glucose tolerance at discharge and after 3 months, respectively, and 51 of 164 (31%, 24-38) and 36 of 144 (25%, 18-32) had previously undiagnosed diabetes mellitus. Independent predictors of abnormal glucose tolerance at 3 months were concentrations of HbA(1c) at admission (p=0.024) and fasting blood glucose concentrations on day 4 (p=0.044).

INTERPRETATION: Previously undiagnosed diabetes and impaired glucose tolerance are common in patients with an acute myocardial infarction. These abnormalities can be detected early in the postinfarction period. Our results suggest that fasting and postchallenge hyperglycaemia in the early phase of an acute myocardial infarction could be used as early markers of high-risk individuals.

Acute heart attack patients have abnormal glucose metabolism.

J Nutr. 2002 Jul;132(7):1879-85.

A ketogenic diet favorably affects serum biomarkers for cardiovascular disease in normal-weight men.

Sharman MJ[1], Kraemer WJ, Love DM, Avery NG, Gómez AL, Scheett TP, Volek JS.

	Ketogenic group ($n = 12$)				Control group ($n = 8$)			
		Wk 0	Wk 3	Wk 6	Percent Δ[2]	Wk 0	Wk 6	Percent Δ
TC, *mmol/L*	4.27 ± 0.8[b]	4.78 ± 0.9[a]	4.47 ± 0.81[b]	4.7%	4.24 ± 1.0[b]	4.10 ± 1.2[b]	−3.3%	
TAG, *mmol/L*	1.09 ± 0.5[a]	0.75 ± 0.3[b]	0.73 ± 0.3[b]	−33.0%	1.14 ± 0.3[a]	1.08 ± 0.7[a]	−5.3%	
HDL-C, *mmol/L*	1.22 ± 0.2[b]	1.43 ± 0.3[a]	1.36 ± 0.4[b]	11.5%	1.16 ± 0.2[b]	1.16 ± 0.5[b]	0.0%	
LDL-C, *mmol/L*	2.87 ± 0.8[b]	3.22 ± 0.9[a]	2.99 ± 0.8[b]	4.2%	2.89 ± 0.9[b]	2.74 ± 1.1[b]	−5.2%	
VLDL-C, *mmol/L*	0.17 ± 0.1[a]	0.12 ± 0.0[b]	0.12 ± 0.0[b]	−29.4%	0.18 ± 0.0[a]	0.20 ± 0.1[a]	11.1%	
TC/HDL	3.60 ± 0.9	3.45 ± 0.88	3.45 ± 0.9	−4.2%	3.67 ± 0.7	3.59 ± 0.8	−2.2%	
Insulin, *pmol/L*	23.7 ± 16.3[a]	19.1 ± 12.2[b]	15.6 ± 8.9[b]	−34.2%	21.5 ± 6.7[a]	24.3 ± 9.9[a]	13.0%	
Glucose, *mmol/L*	5.00 ± 0.4	4.84 ± 0.4	4.99 ± 0.3	−0.2%	5.00 ± 0.3	5.09 ± 0.3	1.8%	
β-HBA, *mmol/L*	0.08 ± 0.1[b]	0.40 ± 0.3[a]	0.28 ± 0.09[a]	250.0%	0.09 ± 0.1[b]	0.10 ± 0.1[b]	11.1%	

There are no medicines that can have such a profound effect on risk biomarkers

Am J Med. 2002 Jul;113(1):30-6.

Effect of 6-month adherence to a very low carbohydrate diet program.

Westman EC[1], Yancy WS, Edman JS, Tomlin KF, Perkins CE.

Results

Forty-one (80%) of the 51 subjects attended visits through 6 months. In these subjects, the mean (± SD) body weight decreased 10.3% ± 5.9% (P <0.001) from baseline to 6 months (body weight reduction of 9.0 ± 5.3 kg and body mass index reduction of 3.2 ± 1.9 kg/m^2). The mean percentage of body weight that was fat decreased 2.9% ± 3.2% from baseline to 6 months (P <0.001). The mean serum bicarbonate level decreased 2 ± 2.4 mmol/L (P <0.001) and blood urea nitrogen level increased 2 ± 4 mg/dL (P <0.001). Serum total cholesterol level decreased 11 ± 26 mg/dL (P = 0.006), low-density lipoprotein cholesterol level decreased 10 ± 25 mg/dL (P = 0.01), triglyceride level decreased 56 ± 45 mg/dL (P <0.001), high-density lipoprotein (HDL) cholesterol level increased 10 ± 8 mg/dL (P <0.001), and the cholesterol/HDL cholesterol ratio decreased 0.9 ± 0.6 units (P <0.001). There were no serious adverse effects, but the possibility of adverse effects in the 10 subjects who did not adhere to the program cannot be eliminated.

Total Cholesterol	**Decreased 11± 26 mg/dL**
LDL	**Decreased 10 ± 25 mg/dL**
Triglycerides	**Decreased 56 ± 45 mg/dL**
HDL	**Increased 10 ± 8 mg/dL**

Atherosclerosis. 2005 Jun;180(2):389-97. Epub 2005 Jan 25.

Risk factors for peripheral arterial disease incidence in persons with diabetes: the Atherosclerosis Risk in Communities (ARIC) Study.

Wattanakit K[1], Folsom AR, Selvin E, Weatherley BD, Pankow JS, Brancati FL, Hirsch AT.

Abstract

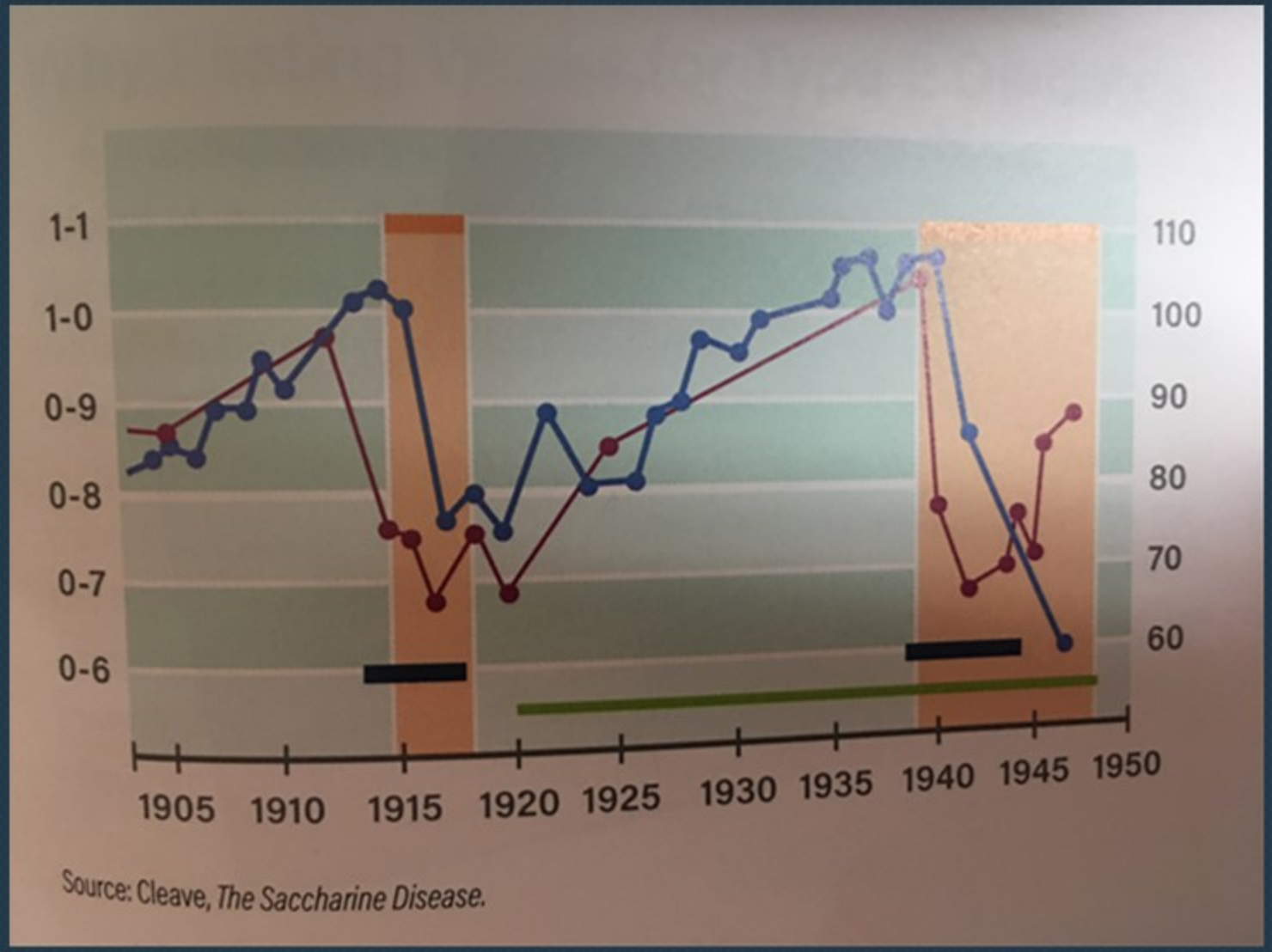
BACKGROUND: Some risk factors for peripheral arterial disease (PAD) have been identified, but little information is available on PAD risk factors in individuals with diabetes.

METHODS: Using data from the Atherosclerosis Risk in Communities (ARIC) Study, we assessed the relation of traditional and non-traditional risk factors with the risk of PAD in 1651 participants with diabetes, but not PAD, at baseline. Incident PAD was defined as an ankle-brachial index (ABI)<0.9 assessed at regular examinations; hospital discharge codes for PAD, amputation, or leg revascularization; or claudication assessed by annual questionnaire.

RESULTS: Over a mean of 10.3 years of follow-up, 238 persons developed incident PAD identified, yielding a PAD event rate of 13.9 per 1000 person years. Adjusted for sex, age, race, and center, the risk of developing PAD was increased 1.87-fold (95% confidence interval (95% CI): 1.36-2.57) in persons who were current smokers versus non-smokers, 2.27-fold (95% CI: 1.57-3.26) for baseline coronary heart disease (CHD) versus no baseline CHD, and 1.75-fold (95% CI: 1.18-2.60) for the highest quartile versus lowest quartile of triglycerides. We found no evidence of an association with other blood lipids or hypertension. Compared with the lowest quartiles, comparably-adjusted relative risks for the highest quartiles were 1.60 (95% CI: 1.10-2.33) for waist-to-hip ratio, 2.52 (95% CI: 1.70-3.73) for fibrinogen, 1.70 (95% CI: 1.17-2.47) for factor VIII, 1.73 (95% CI: 1.18-2.54) for von Willebrand factor, 2.15 (95% CI: 1.43-3.24) for white blood cell count, 1.81 (95% CI: 1.19-2.74) for serum creatinine, 0.55 (95% CI: 0.37-0.83) for serum albumin, and 2.73 (95% CI: 1.77-4.22) for carotid intima-media thickness. Persons who had a prior history of diabetes and were taking insulin had a relative risk of 1.97 (95% CI: 1.35-2.87) for future PAD events, compared with those with newly identified diabetes at baseline. In our final multivariable model, current smoking, prevalent CHD, elevated fibrinogen and carotid IMT, and a prior history of diabetes with insulin treatment were independently associated with greater PAD incidence.

CONCLUSION: These markers might be useful to identify individuals with diabetes at particular risk for PAD.

Cholesterol was not a marker and insulin treatment is a definite marker because insulin makes it WORSE



See the Diabetes Mortality trend (blue) tracks the sugar consumption (red) perfectly, especially the drops during food rationing and war

Fung, Jason. "5: Fasting for Weight Loss." *The Complete Guide to Fasting: Heal Your Body through Intermittent, Alternate-Day, and Extended Fasting*, by Jason Fung and Jimmy Moore, Victory Belt Publishing, 2016, pp. 115–120.

The Galen Foundation

Galen is a federally recognized 501(c)(3) non-profit organization through which donations are tax deductible.
Please donate generously

www.Galen.OrlandoCVI.com

Cash or Check to one of our volunteers just outside of the auditorium

Please join us at the next talk on

Tuesday June 18th, 2019 At 7am & 7pm

FASTING FOR SURVIVAL