Can Your Apple Watch Fix How Key Lifestyle Factors Impact Your Cardiometabolic Health and Mortality Risk?

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Swiss Re

Agenda

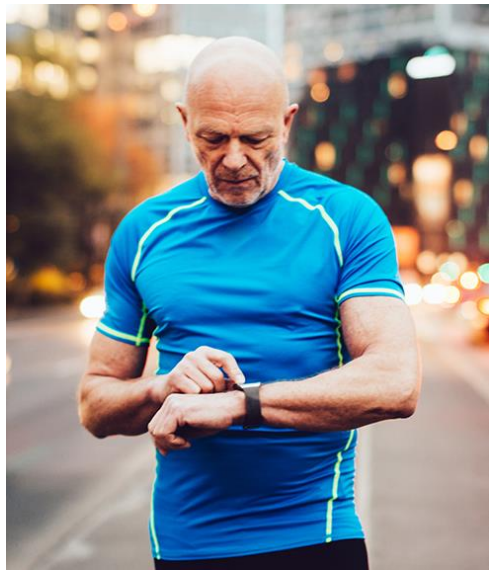
- What the apple watch represents
- Key lifestyle factors
- Cardiometabolic health
- Lifestyle impact on Cardiometabolic health
- Can wearables help to improve Cardiometabolic health?
- Cardiometabolic health beyond 2022

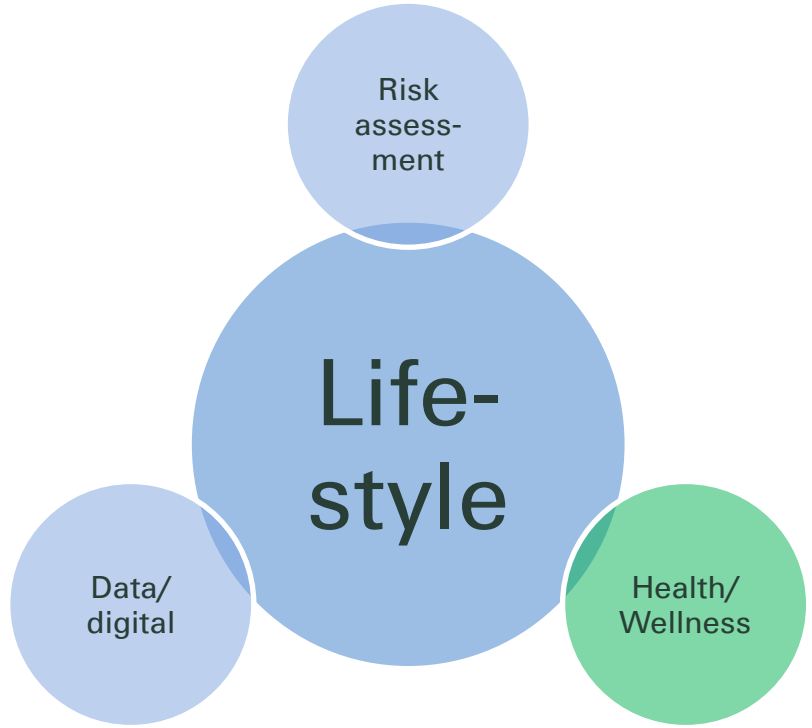
and other wearables

What the “apple watch” represents

- ubiquity of wearables
- data
- “wellness programs”

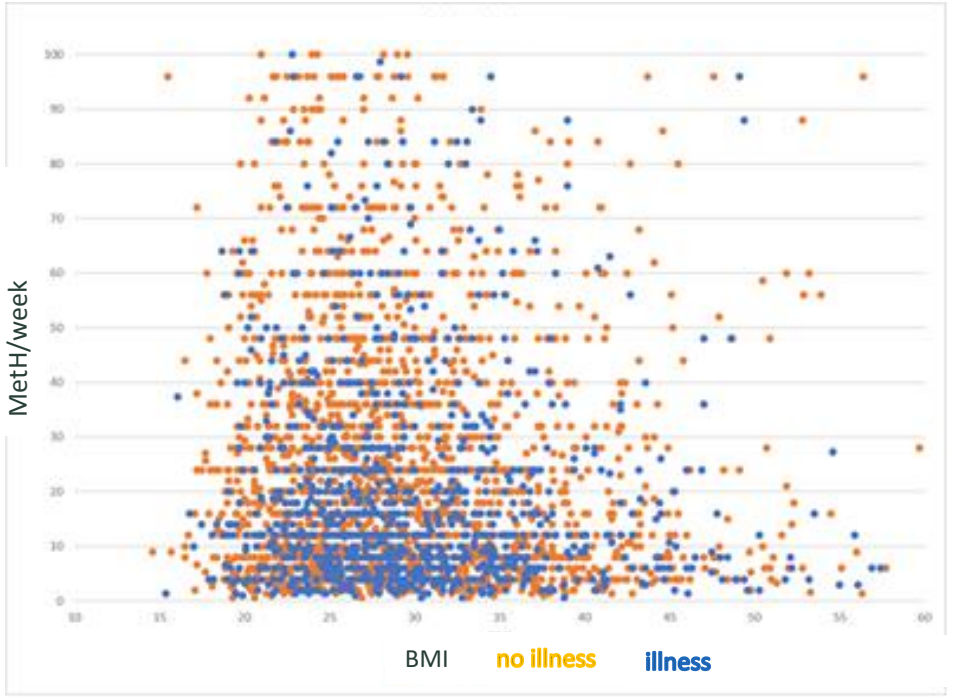
- why insurers are embracing these?
  - better engagement (reduced lapse rates)
  - more differentiated uw/risk assessment
  - ongoing (dynamic) underwriting
  - improving policyholder health





Considerations for including lifestyle data in risk assessment

- robust evidence of lifestyle impacting mortality risk
- self reporting (vs. verified) - additional questions, information
- duration of data/behavior & persistency of lifestyle behavior
- data/cost of ongoing data ingestion for dynamic underwriting* (only modifiable risk adjustment)
- policyholder benefit of dynamic underwriting
- regulation
- augmentation vs. replacement of risk assessment
- link to wellness and improved health



Key Lifestyle Factors

What are Key Lifestyle Factors?

- wearables
- “health” recommendations

Key Lifestyle Factors and mortality

- Physical Activity -

Figure 4
Relative risk of all-cause mortality per MET hours/week

MET-h/week	0	0–7.5	7.6–15	15.1–22.5	22.6–40	40+
Relative Risk (all-cause mortality)	**1.00**	**0.87**	**0.80**	**0.74**	**0.70**	**0.67**
	95% CI (Ref)	**95% CI** 0.84–0.90	**95% CI** 0.76–0.82	**95% CI** 0.70–0.78	**95% CI** 0.66–0.73	**95% CI** 0.61–0.77

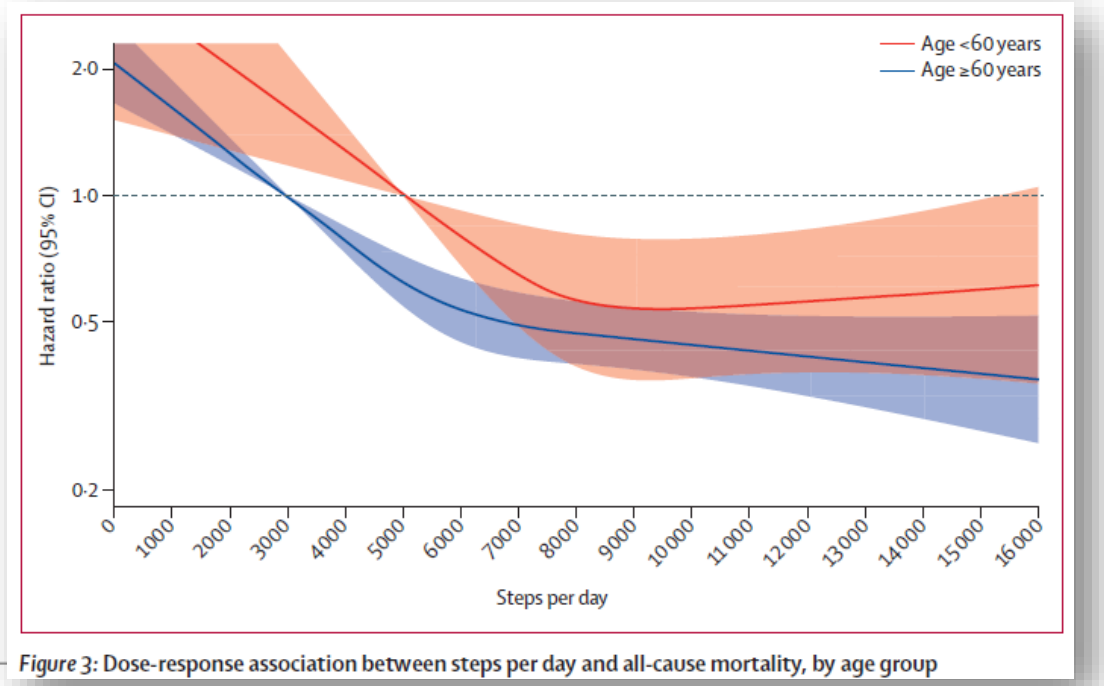


Figure 3: Dose-response association between steps per day and all-cause mortality, by age group

Paluch et al. Lancet Public Health 2022; 7: e219–28

SR analysis : Weighted averages of 4 large analyses (n>2.8 million) for different physical activity duration subgroups

Key Lifestyle Factors and mortality

- Sleep -

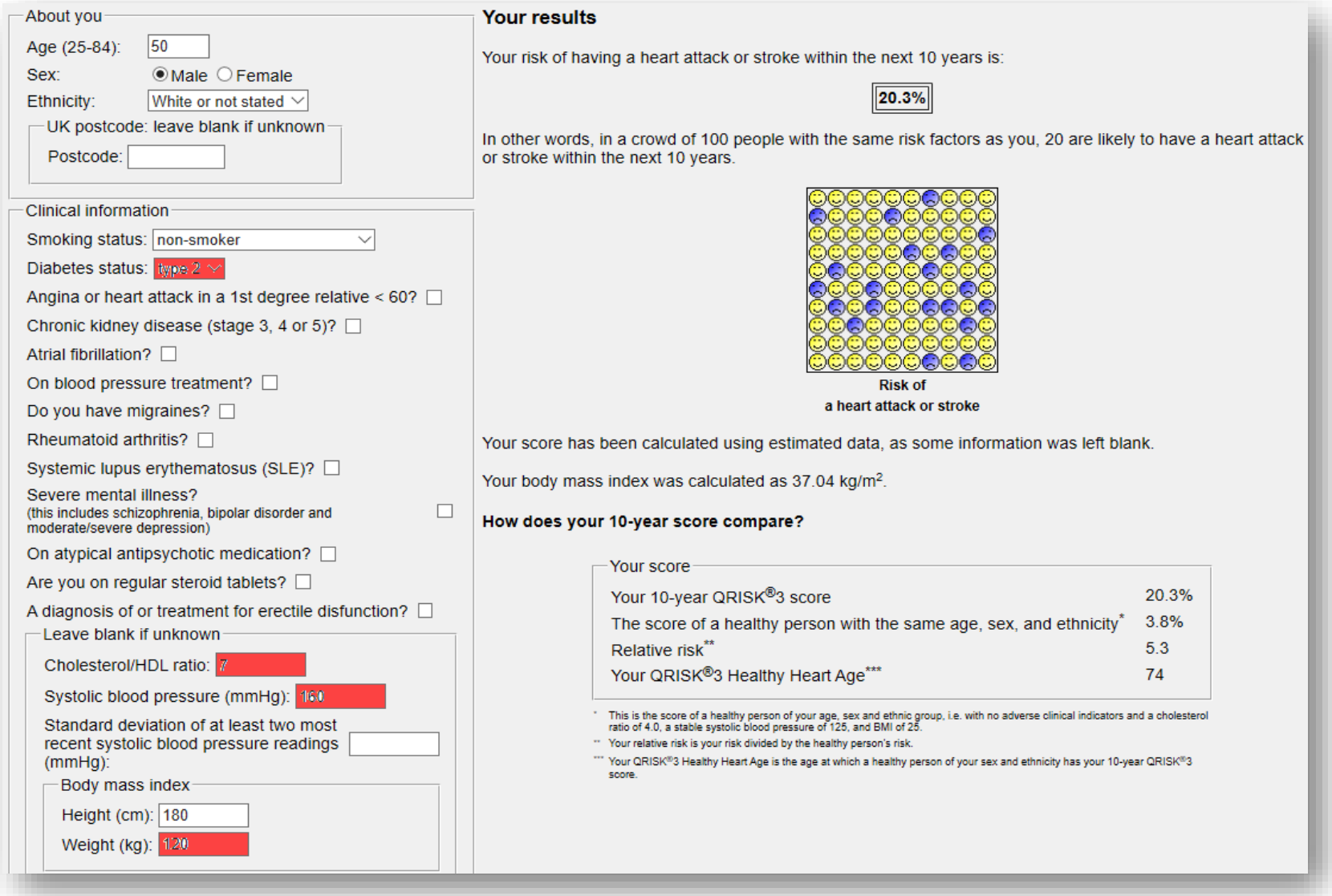
Hours	< 5	5–6	6–7	7–8	8–9	> 9
Weighted average mortality	**1.11**	**1.05**	**1.03**	**1.00**	**1.12**	**1.37**
	95% CI 1.06–1.29	**95% CI** 1.03–1.09	**95% CI** 1.00–1.10	(Ref)	**95% CI** 1.08–1.20	**95% CI** 1.18–1.62

Weighted averages of 5 large meta-analyses (n > 6 million) for different sleep duration subgroups.
(Shen 2016, Kabat 2018, Cappucio 2010, Kronholm 2011, Yin 2017)

Cardiometabolic Health

- Cardiovascular Risk
- Metabolic Risk
- Metabolic syndrome
- Hyperinsulinemia

Cardio-vascular risk



About you

Age (25-84): 50

Sex: Male Female

Ethnicity: White or not stated

UK postcode: leave blank if unknown

Postcode:

Clinical information

Smoking status: non-smoker

Diabetes status: type 2

Angina or heart attack in a 1st degree relative < 60?

Chronic kidney disease (stage 3, 4 or 5)?

Atrial fibrillation?

On blood pressure treatment?

Do you have migraines?

Rheumatoid arthritis?

Systemic lupus erythematosus (SLE)?

Severe mental illness?
(this includes schizophrenia, bipolar disorder and moderate/severe depression)

On atypical antipsychotic medication?

Are you on regular steroid tablets?

A diagnosis of or treatment for erectile disfunction?

Leave blank if unknown

Cholesterol/HDL ratio: 7

Systolic blood pressure (mmHg): 160

Standard deviation of at least two most recent systolic blood pressure readings (mmHg):

Body mass index

Height (cm): 180

Weight (kg): 120

Your results

Your risk of having a heart attack or stroke within the next 10 years is:

20.3%

In other words, in a crowd of 100 people with the same risk factors as you, 20 are likely to have a heart attack or stroke within the next 10 years.

Risk of a heart attack or stroke

Your score has been calculated using estimated data, as some information was left blank.

Your body mass index was calculated as 37.04 kg/m^2.

How does your 10-year score compare?

Your score

Your 10-year QRISK®3 score	20.3%
The score of a healthy person with the same age, sex, and ethnicity*	3.8%
Relative risk**	5.3
Your QRISK®3 Healthy Heart Age***	74

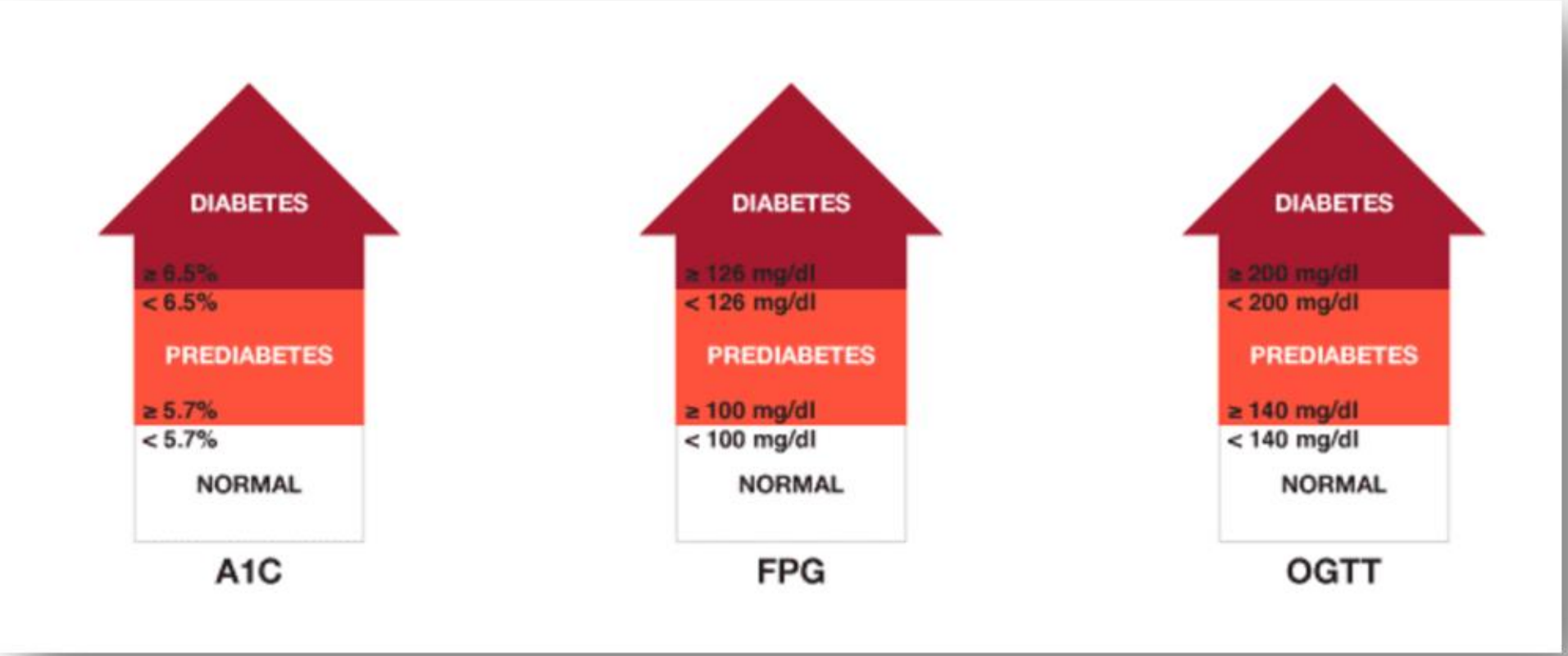
* This is the score of a healthy person of your age, sex and ethnic group, i.e. with no adverse clinical indicators and a cholesterol ratio of 4.0, a stable systolic blood pressure of 125, and BMI of 25.

** Your relative risk is your risk divided by the healthy person's risk.

*** Your QRISK®3 Healthy Heart Age is the age at which a healthy person of your sex and ethnicity has your 10-year QRISK®3 score.

R_x

Metabolic risk



R_x

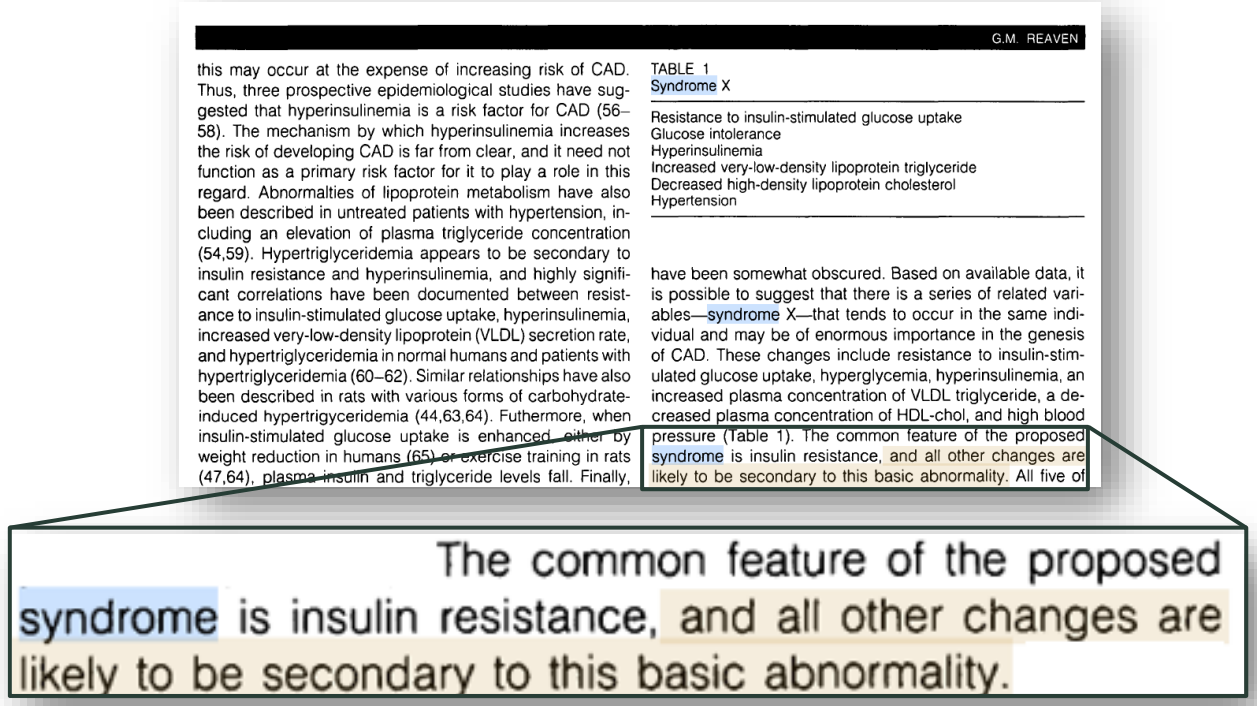
https://www.diabetes.org/diabetes/a1c/diagnosis

Metabolic risk

R_x

Metabolic syndrome

- In 1988, Gerald “Jerry” Reaven, at **American Diabetes Association** delivered the group’s prestigious Banting Lecture.
- **He methodically argued the case for a strong link between insulin resistance—the hallmark of type 2 diabetes—and high blood pressure, raised triglycerides, and other metabolic anomalies.**



G.M. REAVEN

this may occur at the expense of increasing risk of CAD. Thus, three prospective epidemiological studies have suggested that hyperinsulinemia is a risk factor for CAD (56–58). The mechanism by which hyperinsulinemia increases the risk of developing CAD is far from clear, and it need not function as a primary risk factor for it to play a role in this regard. Abnormalties of lipoprotein metabolism have also been described in untreated patients with hypertension, including an elevation of plasma triglyceride concentration (54,59). Hypertriglyceridemia appears to be secondary to insulin resistance and hyperinsulinemia, and highly significant correlations have been documented between resistance to insulin-stimulated glucose uptake, hyperinsulinemia, increased very-low-density lipoprotein (VLDL) secretion rate, and hypertriglyceridemia in normal humans and patients with hypertriglyceridemia (60–62). Similar relationships have also been described in rats with various forms of carbohydrate-induced hypertrigyceridemia (44,63,64). Futhermore, when insulin-stimulated glucose uptake is enhanced, either by weight reduction in humans (65) or exercise training in rats (47,64), plasma insulin and triglyceride levels fall. Finally,

TABLE 1
Syndrome X

Resistance to insulin-stimulated glucose uptake
Glucose intolerance
Hyperinsulinemia
Increased very-low-density lipoprotein triglyceride
Decreased high-density lipoprotein cholesterol
Hypertension

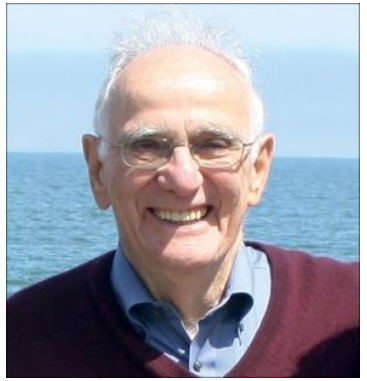
have been somewhat obscured. Based on available data, it is possible to suggest that there is a series of related variables—syndrome X—that tends to occur in the same individual and may be of enormous importance in the genesis of CAD. These changes include resistance to insulin-stimulated glucose uptake, hyperglycemia, hyperinsulinemia, an increased plasma concentration of VLDL triglyceride, a decreased plasma concentration of HDL-chol, and high blood pressure (Table 1). The common feature of the proposed syndrome is insulin resistance, and all other changes are likely to be secondary to this basic abnormality. All five of

The common feature of the proposed syndrome is insulin resistance, and all other changes are likely to be secondary to this basic abnormality.

Reaven, G. Diabetes, VOL. 37, Dec 1988

AHA (2021) Metabolic Syndrome criteria

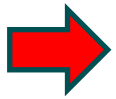
- *High blood glucose (sugar)*
- *Low levels of HDL (“good”) cholesterol in the blood*
- *High levels of triglycerides in the blood*
- *Large waist circumference or “apple-shaped” body*
- *High blood pressure*



https://doi.org/10.1016/S0140-6736(18)30906-1

Reaven became known as “the father of insulin resistance”.

Metabolic syndrome



- .. the diagnosis of the metabolic syndrome does not bring with it much in the way of pathophysiologic understanding or clinical utility
- … deciding that individuals do not have it because they fail to satisfy three of five arbitrarily chosen criteria ***may withhold relevant therapeutic intervention.***
- the ATP III focused entirely on the role of insulin resistance as increasing risk of CVD. (But).. clear…insulin-resistant individuals…(have) increased risk to develop … nonalcoholic liver disease, polycystic ovary disease, certain forms of cancer

Metabolic syndrome

Non-communicable diseases (NCDs) kill 41 million people each year, **equivalent to 71% of all deaths globally.** (*WHO*)

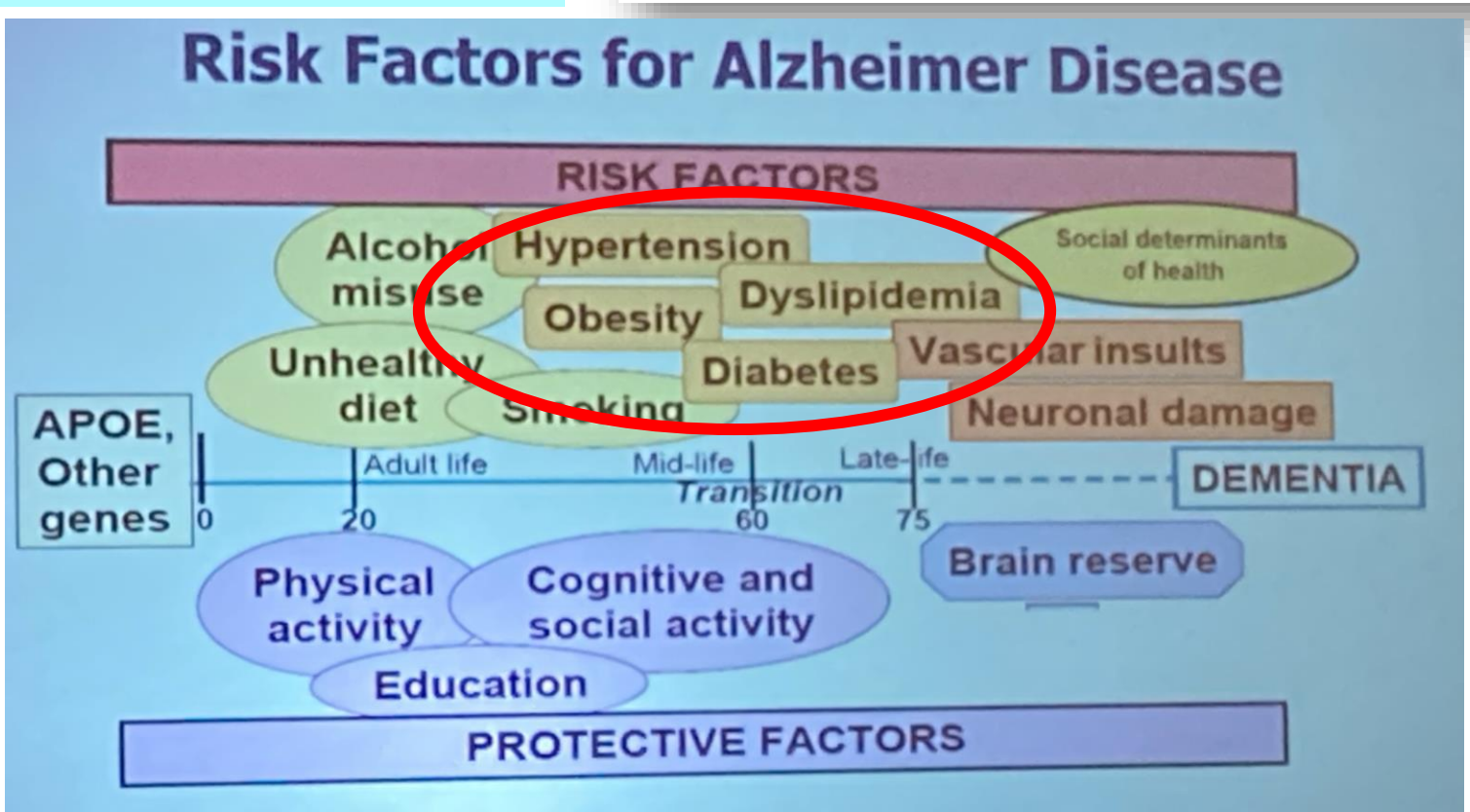
World Health Organization | Health Topics | Countries | Newsroom

Metabolic risk factors

Metabolic risk factors contribute to four key metabolic changes that increase the risk of NCDs:

- raised blood pressure;
- overweight/obesity;
- hyperglycemia (high blood glucose levels); and
- hyperlipidemia (high levels of fat in the blood).

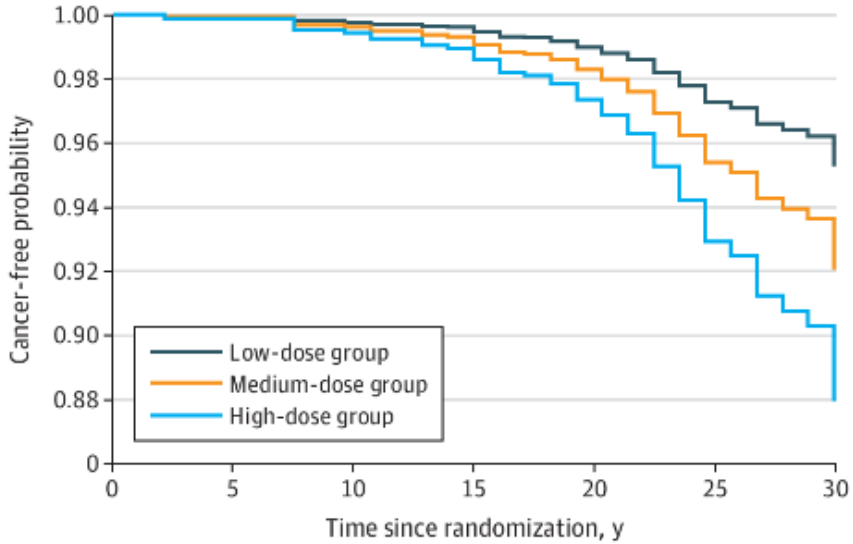
- heart failure
- stroke
- Alzheimer's
- COVID



Metabolic syndrome (and cancer)

- DCCT/EDIC studies
- 28y f/u
- 1303 T1DM patients
- low, med, high (<0.5, 0.5 to <0.8, ≥0.8u/kg)

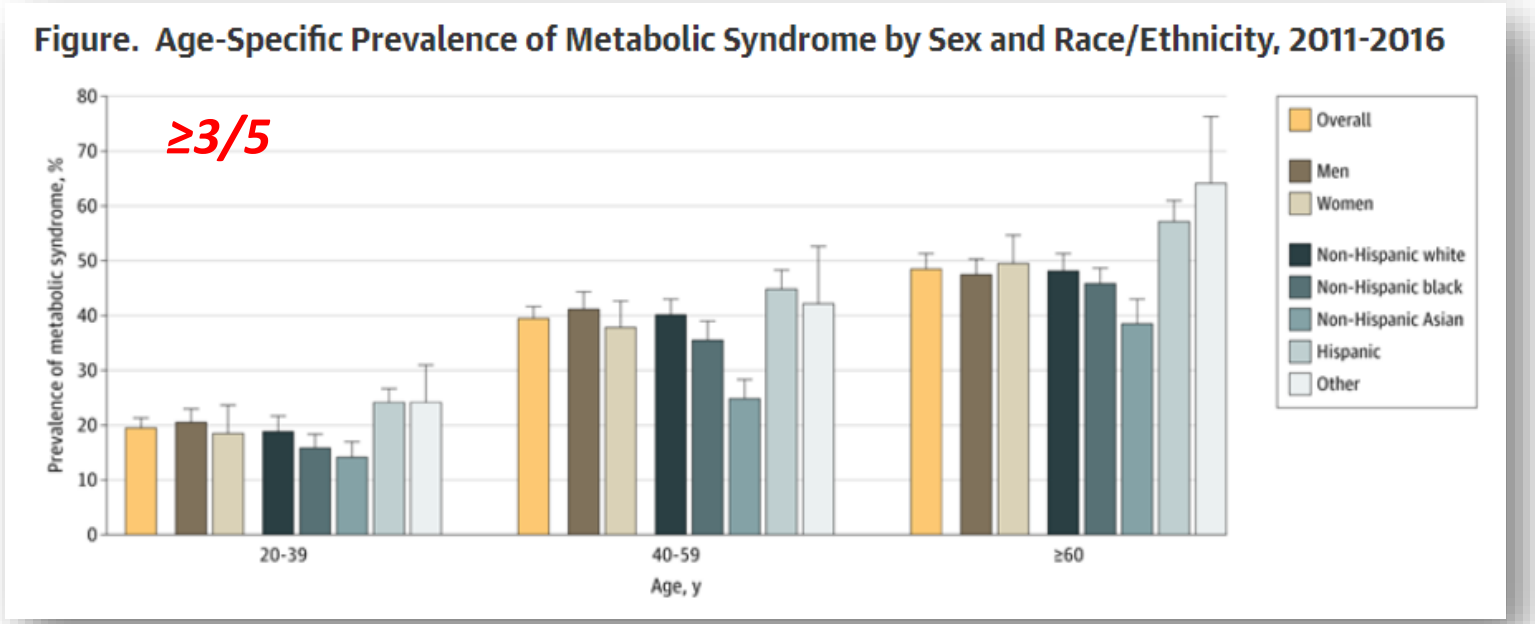
Figure. Cancer-Free Probability by Daily Insulin Dose Over 28 Years of Follow-up



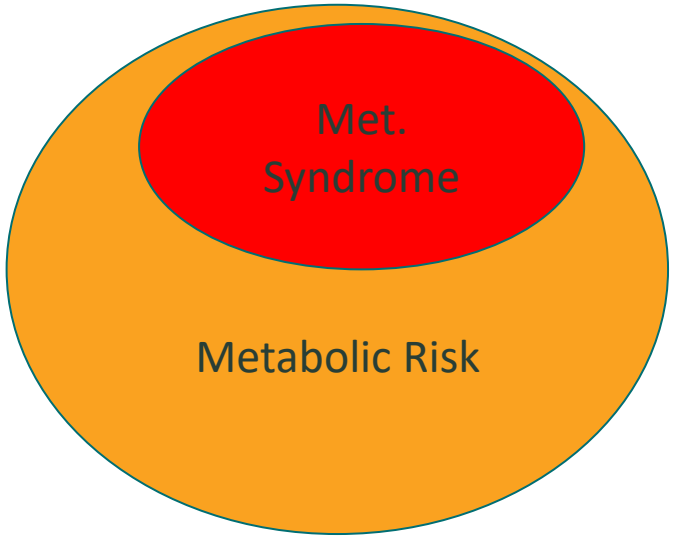
No. at risk							
Low-dose group	221	221	220	218	215	124	0
Medium-dose group	832	831	828	825	813	494	0
High dose-group	250	250	249	245	240	157	0

Wenjun Zhong, et al. JAMA Oncol. 2022;8(9):1356-1358. doi:10.1001/jamaoncol.2022.2960

Metabolic syndrome vs. **Metabolic risk**



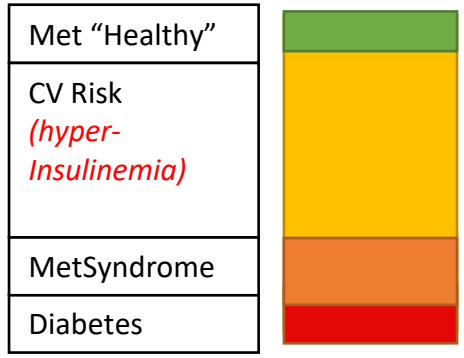
https://jamanetwork.com/journals/jama/fullarticle/2767313



"Changing from ATP III (Adult Treatment Panel III) guidelines to more recent cut points decreased the proportion of metabolically healthy Americans from 19.9% (95% confidence interval [CI]: 18.3-21.5) to 12.2% (95% CI: 10.9-13.6)."

≥1/5

Metab Syndr Relat Disord. 2019 Feb;17(1):46-52



Metabolic risk – what's "normal"

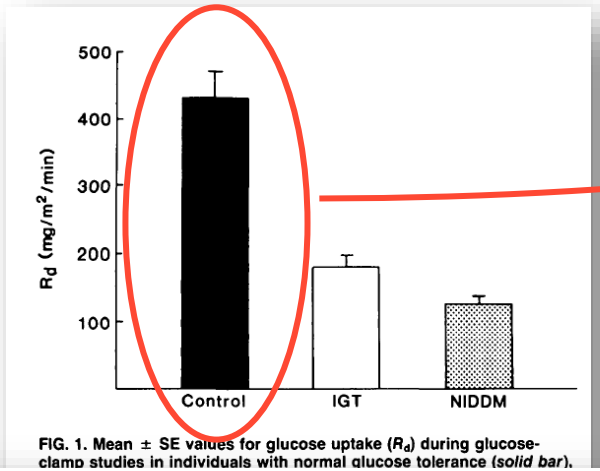


FIG. 1. Mean ± SE values for glucose uptake (R_d) during glucose-clamp studies in individuals with normal glucose tolerance (*solid bar*).

normal OGTT

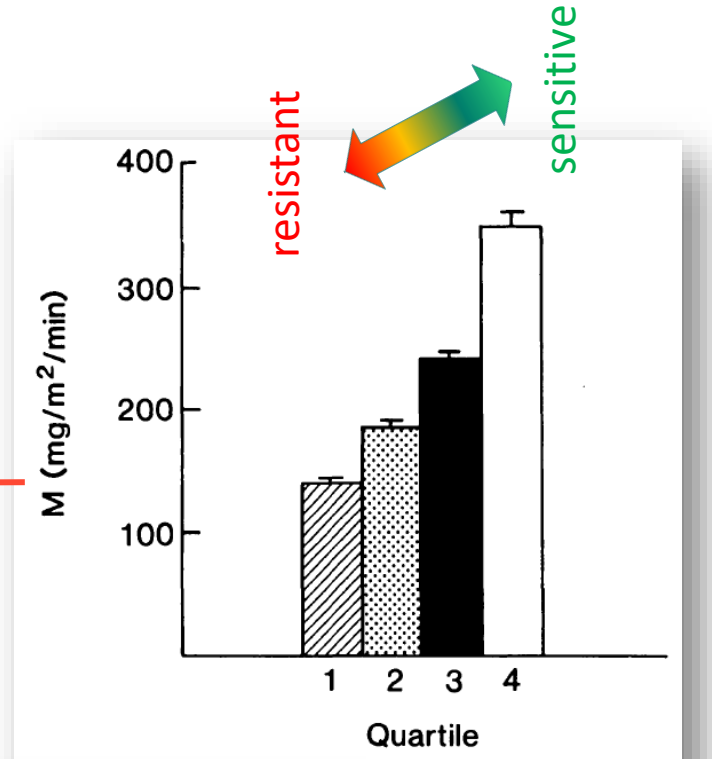


FIG. 3. Mean ± SE values for glucose uptake (M) during glucose-
th normal glucose tolerance. Study
iles on basis of glucose uptake rate
levant variables. Clamp studies
sma insulin levels of ~100 μU/ml.
with permission.

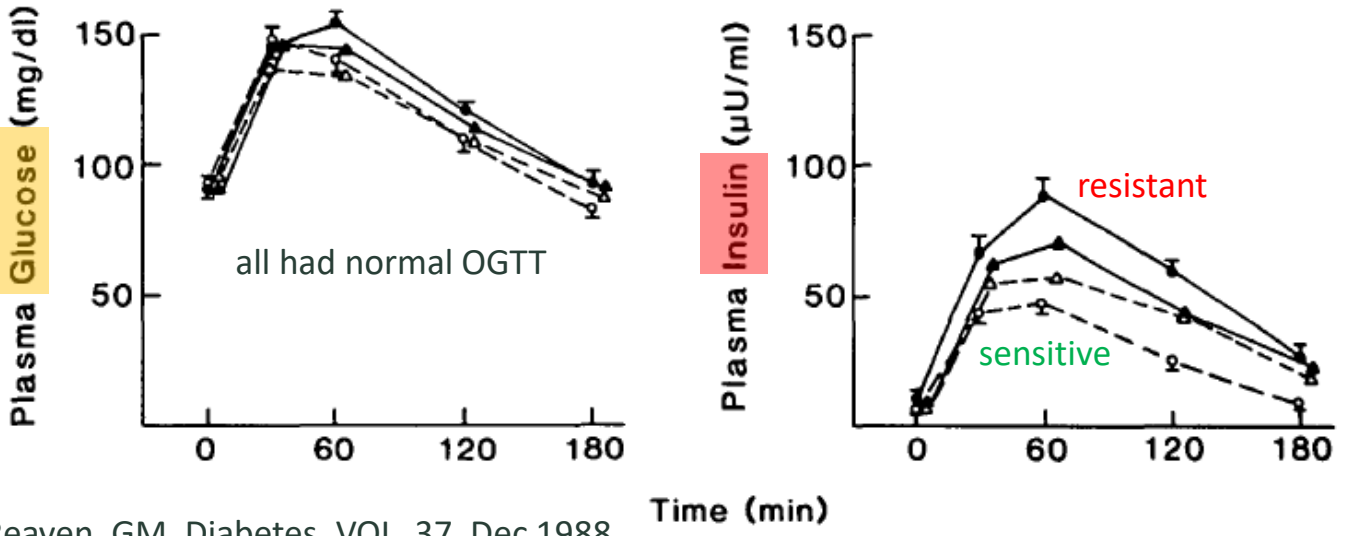
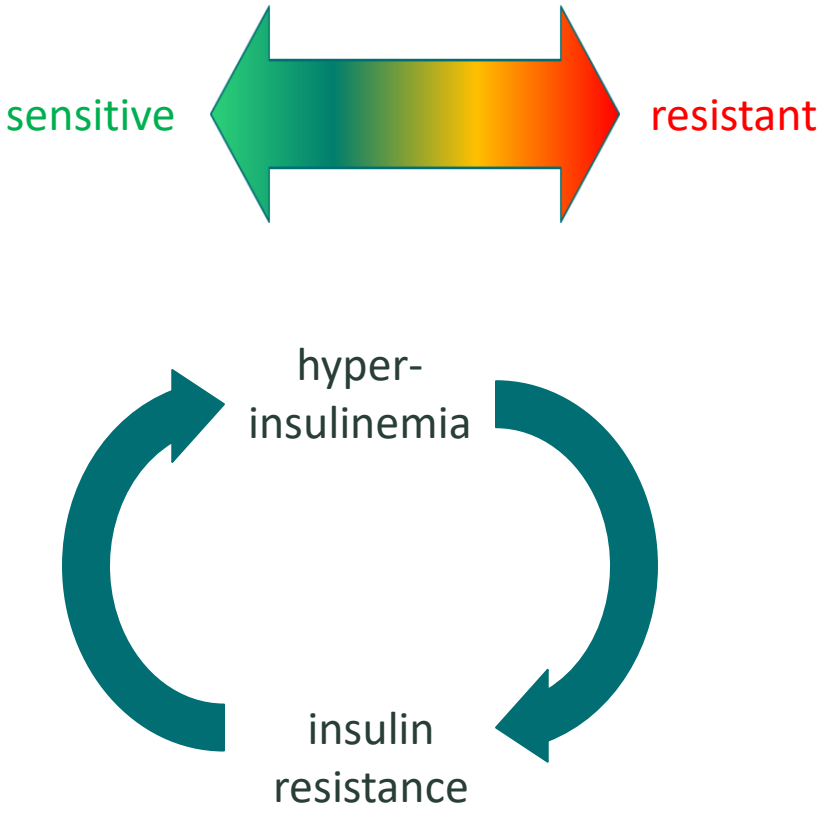


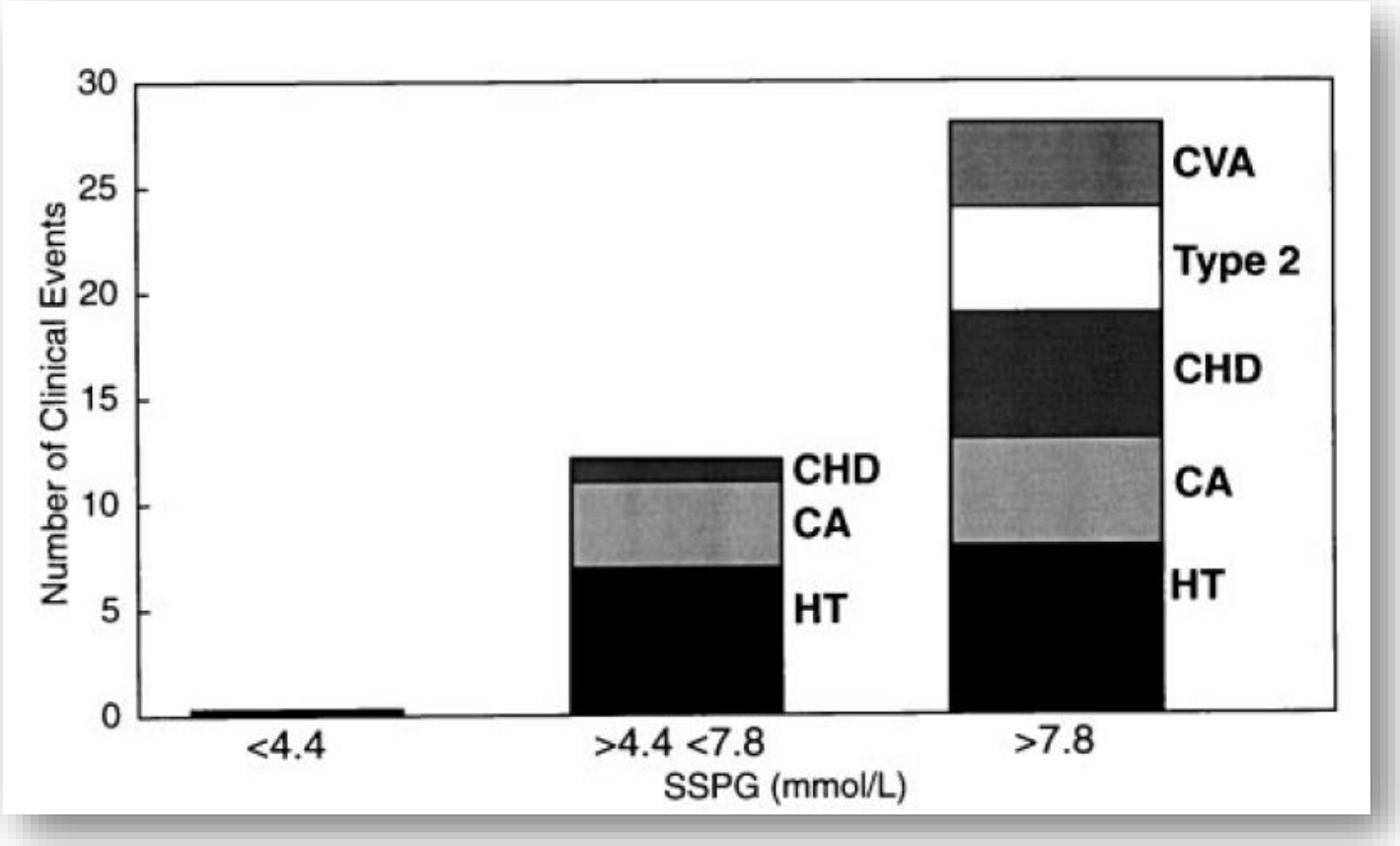
FIG. 4. Plasma glucose (*left*) and insulin (*right*) responses to 75-g oral glucose challenge in study population whose glucose-clamp results are shown in Fig. 3. Group was divided into 4 quartiles (1, ●; 2, ▲; 3, △; 4, ○) on basis of glucose-clamp determinations. From Hollenbeck and Reaven (10) with permission.

Reaven, GM. Diabetes, VOL. 37, Dec 1988

? R_x

Metabolic risk

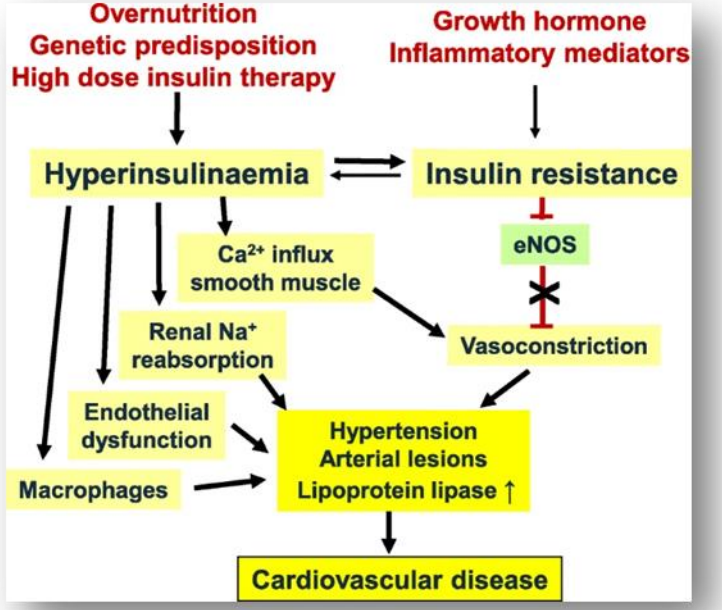




Facchini et al. The Journal of Clinical Endocrinology & Metabolism, August 2001, 86(8):3574–3578

- ~200 healthy adults, no DM, Hpt, CVD
- median age 50
- f/u ~6 years
- divided into tertiles of insulin sensitivity (resistance)

Hyperinsulinema



Crofts, CAP. et al. Diabesity 2015; 1 (4): 34-43

Biological systems and disease states affected by hyperinsulinemia, and associated mechanisms of action

Biological System	Disease	Mechanism	Direct or indirect mechanism	References	
				Mechanism of action	Epidemiology
Cancer*	Cancer (Breast, ovarian, colon, bladder, pancreas & liver)	Increased insulin-like growth factor IGF-1 enhances cellular growth and proliferation.	Direct	(5, 48)	(29)
		Enhanced glucose uptake and utilization enhances cellular growth and proliferation.	Both	(29)	(29)
		Increased production of reactive oxidative species causes derangement of DNA and enzymes involved with repair mechanisms (enhanced by hyperglycemia).	Indirect	(2, 37, 38)	(2, 37, 38)
		Increased sex-hormone production and decreased sex hormone binding globulin causes increased cellular growth and proliferation (enhanced by obesity).	Direct	(29)	(29)
Circulatory	Atherosclerosis	Arterial wall damage caused by inflammation, increased proliferation and migration of arterial smooth muscle cells. Stimulation of the mitogen-activated protein kinase pathway.	Both	(28, 40)	(28, 47, 63, 64)
		Microvascular disease, including changes to capillary permeability, microaneurysm formation, vasoconstriction and microthrombi.			
	Cardiomyopathy	Increased myocardial fibrosis by increased reactive oxidative species, deranged collagen production.	Both	(65, 66)	(65, 66)
		Diabetic neuropathy causes changes to catecholamines, which further impairs myocardial function.			
	Endothelial dysfunction	Vasoconstriction and pro-atherosclerotic effects from decreased nitric oxide bioavailability and action and increased thromboxane.	Both	(2, 41, 67)	(64)
		Enhanced by increased reactive oxidative species and advanced glycation end-products.			
	Thrombosis	Hyperinsulinemia causes increased fibrinolysis while hyperglycemia causes increased blood coagulability	Indirect	(42)	(64)

Hyperinsulinema

Gastrointestinal	Diabetes: Gestational	Pre-existing insulin resistance and increased demand for insulin.	Direct	(68)	(68)
	Diabetes: Type 2	Prolonged insulin resistance eventuating in beta-cell failure. Down-regulation of glucose transporter-4.	Direct	(3, 69, 70)	(4)
	Hyper- triglyceridemia	Increased triglyceride production.	Direct	(43, 71)	(72)
	Non-alcoholic fatty liver disease	Fatty acid production exceeds distribution capacity. Aggravated by inflammation and oxidative stress.	Direct	(71)	(72)
Endocrine	Chronic inflammation	Stimulation of mitogen-activated protein kinase pathway; glycemic variability; hyperglycemia and/or obesity influences increased cytokine production.	Indirect	(40, 48)	(73)
	Obesity	Decreased lipolysis.	Direct	(74)	(75)
		Lack of appetite suppression.	Direct	(25, 26)	(76)
Nervous	Alzheimer's disease and vascular dementia	Endothelial dysfunction resulting in microvascular disease, metabolic disturbances and neuronal damage.	Direct	(2, 67, 77)	(30, 78, 79)
		Increased blood coagulability and/or fibrinolysis cause multiple thrombotic events.	Both	(42, 80)	
		Changed regulation of beta-amyloid and tau protein (Alzheimer's disease).	Direct	(77, 81)	
		Decreased synaptic plasticity caused by dysregulated PSA-NCAM interactions (Alzheimer's disease).	Direct	(33)	
	Peripheral neuropathy	Increased production of reactive oxidative species and advanced glycation end-products enhanced by hyperglycemia.	Indirect	(2, 41)	(64, 82)
		Insulin resistance in the dorsal root ganglion neurons.	Both	(83)	
	Retinopathy	Hyperglycemia and endothelial dysfunction contribute blood-retinal barrier breakdown. Aggravated by excess advanced glycation end-products.	Direct	(41, 64, 84)	(41, 64, 84)
Skeletal	Osteoporosis	Increased reactive oxidative species and/hyperglycemia cause collagen breakdown, impairs new collagen synthesis and compromises mensenchymal cells.	Indirect	(31)	(31)
Urinary	Nephropathy	Microvascular disease, including changes to capillary permeability, microaneurysm formation, vasoconstriction and microthrombi.	Direct	(67, 85)	(64, 86)
		Increased production of reactive oxidative species and advanced glycation end-products enhanced by hyperglycemia.	Indirect	(41, 87)	

Crofts, CAP. et al. Diabesity 2015; 1 (4): 34-43

Hyperinsulinema

- 25 normal-weight adults (15 men, 10 women), 25 to 34 yrs of age, BMI 22.1 ± 0.3
- 15 days on 55% carbohydrate diet vs. 15 days on 30% carbohydrate diet

TABLE 2
Incremental areas under the curve (IAUCs) for normal-weight subjects on day 15 of a high-carbohydrate (HC) or high-fat (HF) diet[1]

	HC Diet (55%)	LC (30%)
Glucose IAUC (mmol · h/L)	35 711 ± 755	34 226 ± 867[2]
Insulin IAUC (pmol · h/L)	12 185 ± 1459	9056 ± 952[3]
Triacylglycerol IAUC (mmol · h/L)	52 438 ± 4039	52 736 ± 5082[3]
Fatty acid IAUC (g · h/L)	94 067 ± 6252	144 660 ± 7232
Glycerol IAUC (g · h/L)	28 042 ± 1605	36 318 ± 2723

[1] $\bar{x}$ ± SEM; n = 25.
[2,3] Significantly different from HC diet: [2] $P<0.01$, [3] $P<0.05$.

TABLE 1
Fasting serum or plasma values of normal-weight subjects on day 0 and day 15 of a high-carbohydrate (HC) or high-fat (HF) diet[1]

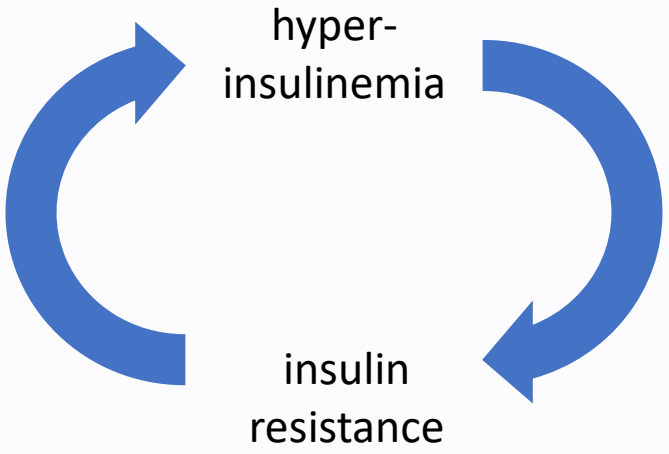
	HC diet (55%)		LC (30%)	
	Day 0	Day 15	Day 0	Day 15
Glucose (mmol/L)	4.9 ± 0.1	4.6 ± 0.1	4.8 ± 0.1	4.7 ± 0.1
Insulin (pmol/L)	43 ± 7	38 ± 3	50 ± 7	39 ± 2
Triacylglycerol (mmol/L)	1.13 ± 0.11	1.14 ± 0.11	1.20 ± 0.11	0.88 ± 0.08[2]
Fatty acids (g/L)	—	0.15 ± 0.01	—	0.14 ± 0.01
Glycerol (g/L)	—	8.93 ± 0.83	—	8.57 ± 0.83
Cholesterol (mmol/L)	3.98 ± 0.16	3.90 ± 0.16	3.85 ± 0.16	3.93 ± 0.16
HDL cholesterol (mmol/L)	1.27 ± 0.08	1.22 ± 0.06	1.27 ± 0.08	1.30 ± 0.06[3]
HDL_2 cholesterol (mmol/L)	0.23 ± 0.05	0.19 ± 0.04	0.21 ± 0.05	0.25 ± 0.04[3]

[1] $\bar{x}$ ± SEM; n = 25. Study design was as follows: diet phase 1 for 16 d (either HC or HF diet), followed by 4–6-wk washout, followed by diet phase 2 for 16 d (either HC or HF diet).
[2,3] Significantly different from day 15 of HC diet: [2] $P<0.01$, [3] $P<0.05$.

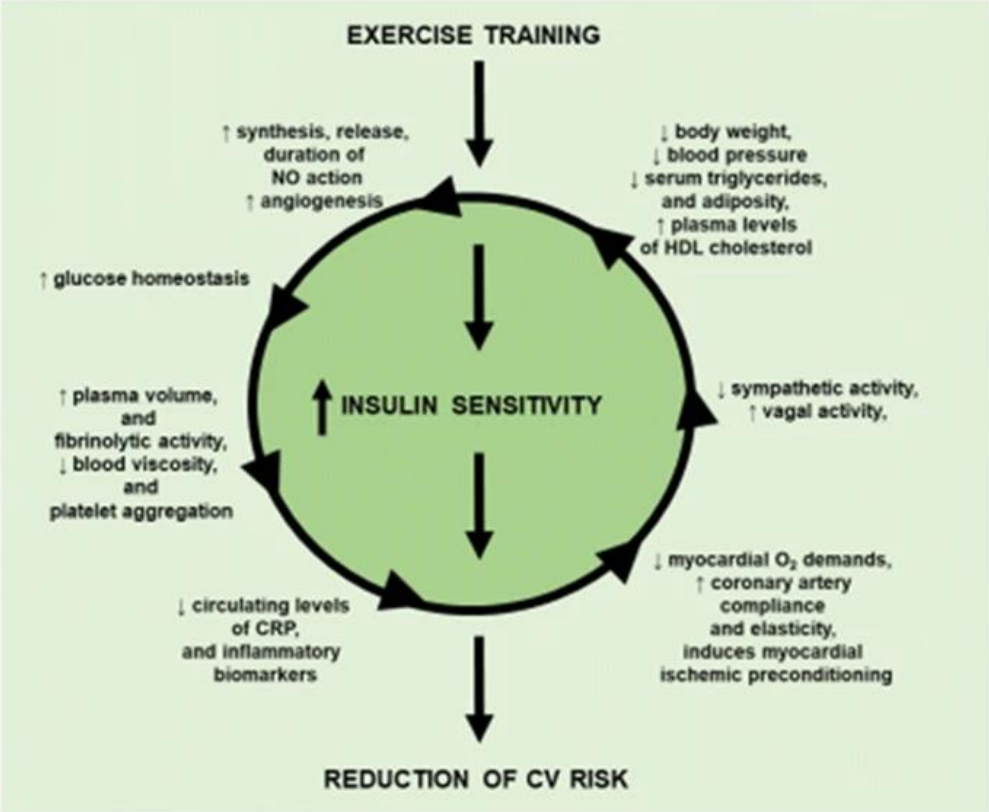
Yost, TJ. et al. Am J Clin Nutr 1998;68:296–302.

Lifestyle impact on Cardiometabolic health

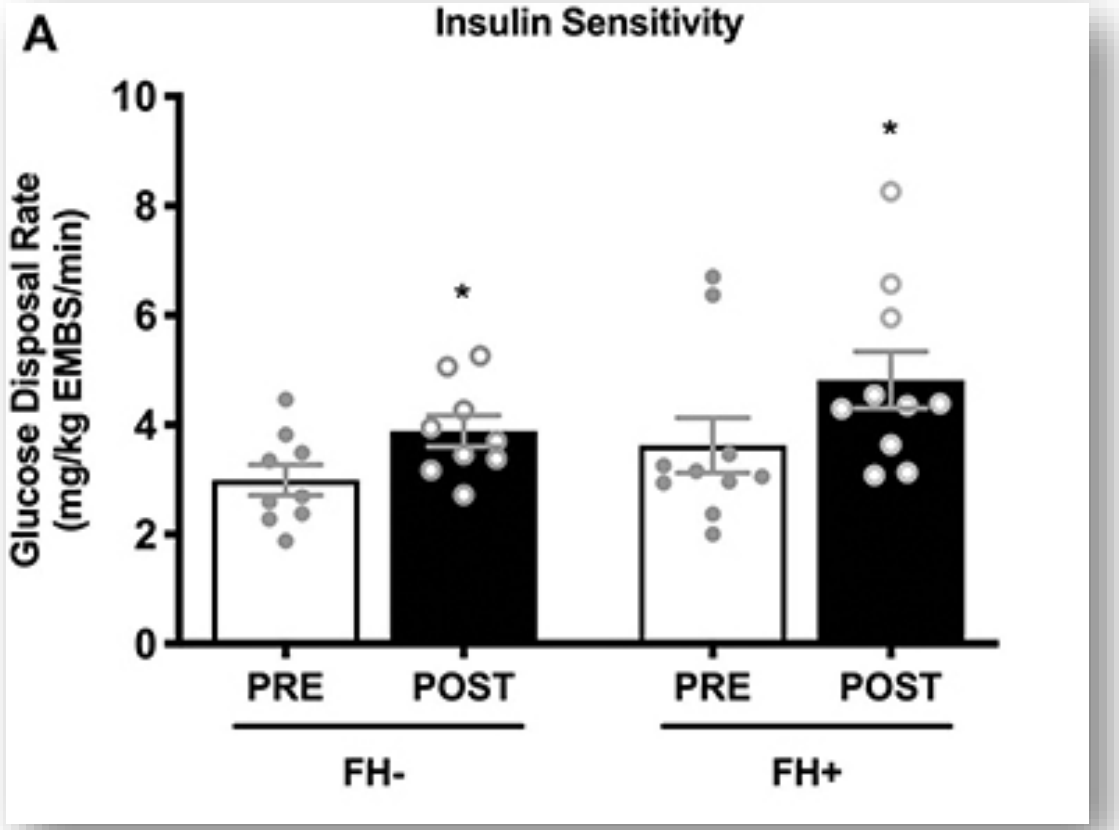
Question : can lifestyle improve insulin sensitivity/ reduce hyperinsulinemia?



Physical activity and Insulin



Iaccarino, G. et al. J of Cardiovascular Translational Research volume 14, pages256–270 (2021)



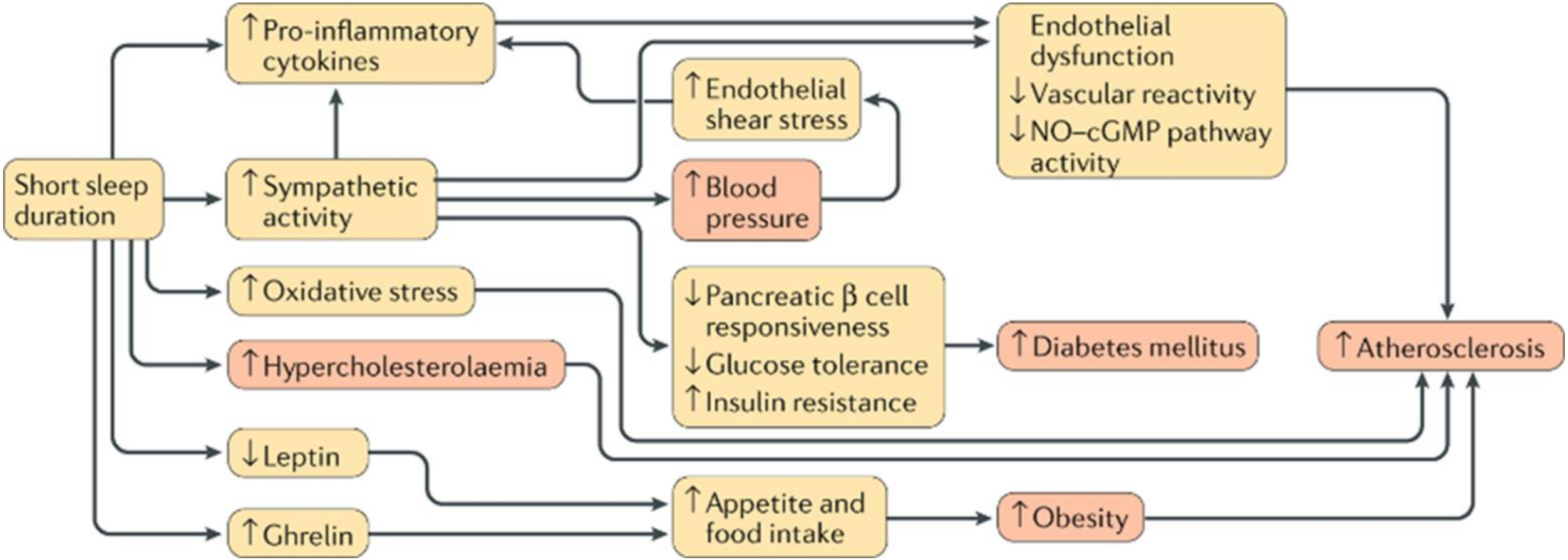
- 22 18-40yr olds
- healthy (sedentary)
- normoglycemic
- 8 weeks ex

Amador, M. et al. Front. Endocrinol. 11:120.
doi: 10.3389/fendo.2020.00120

"Being an athlete doesn't stop you from getting Atherosclerosis"

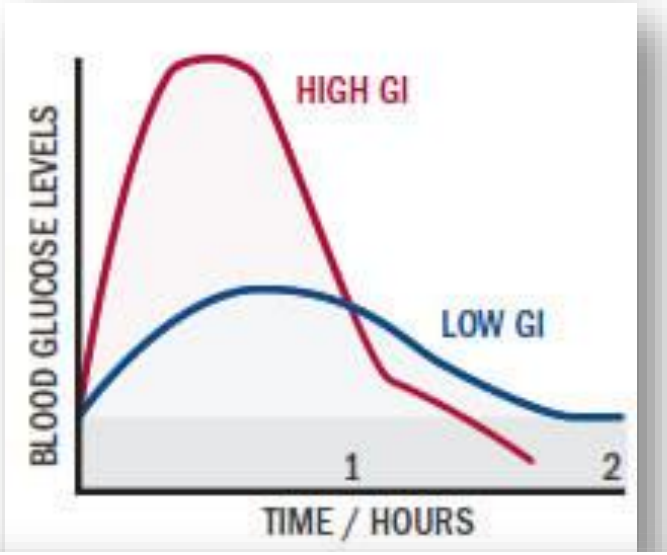
Sheldon E. Litwin

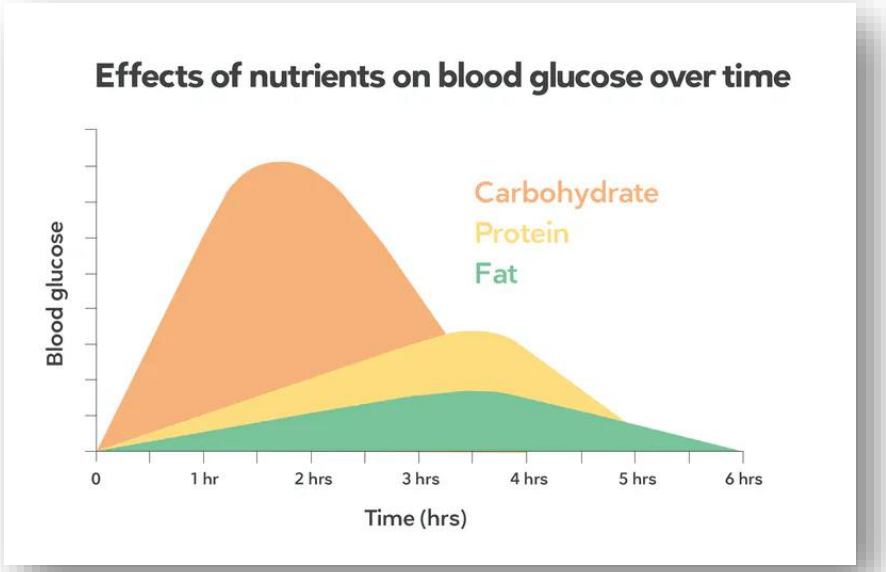
Sleep and Insulin



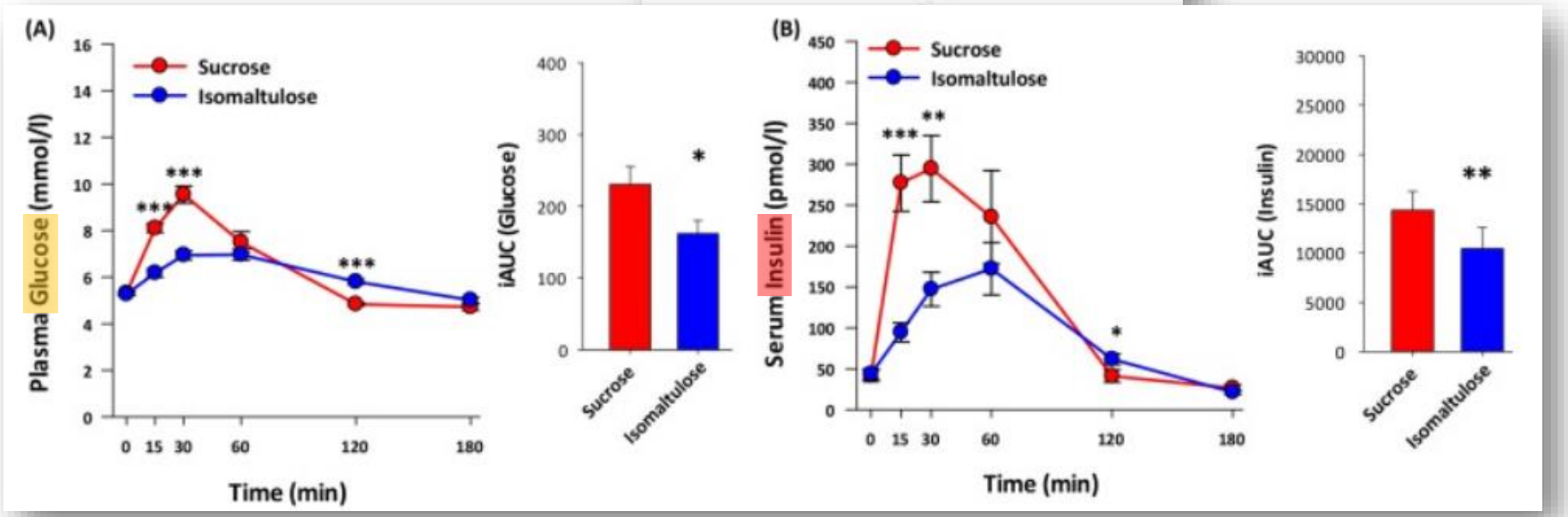
Nutrition and Insulin

- what we eat -





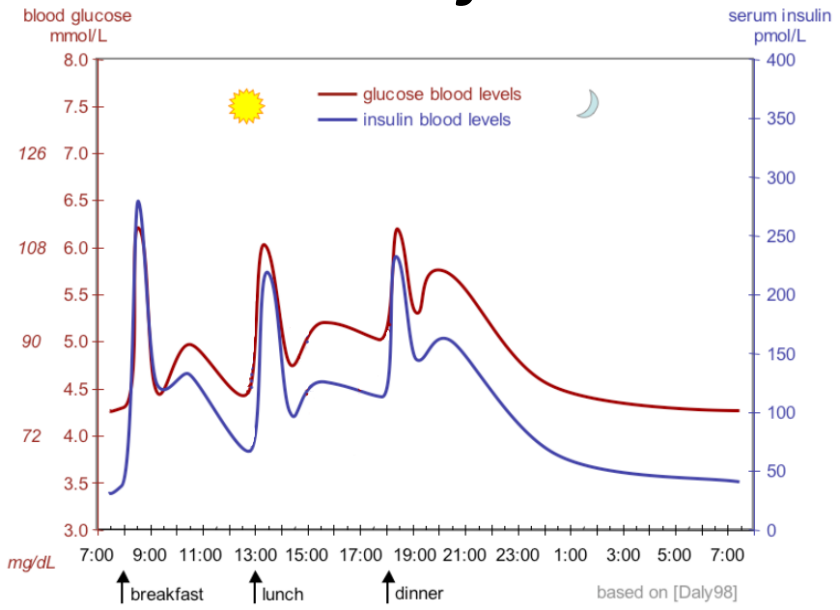
https://www.health.harvard.edu/

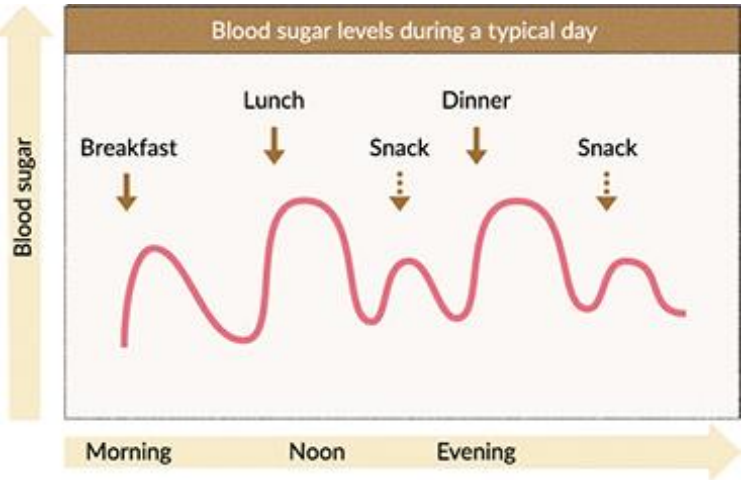


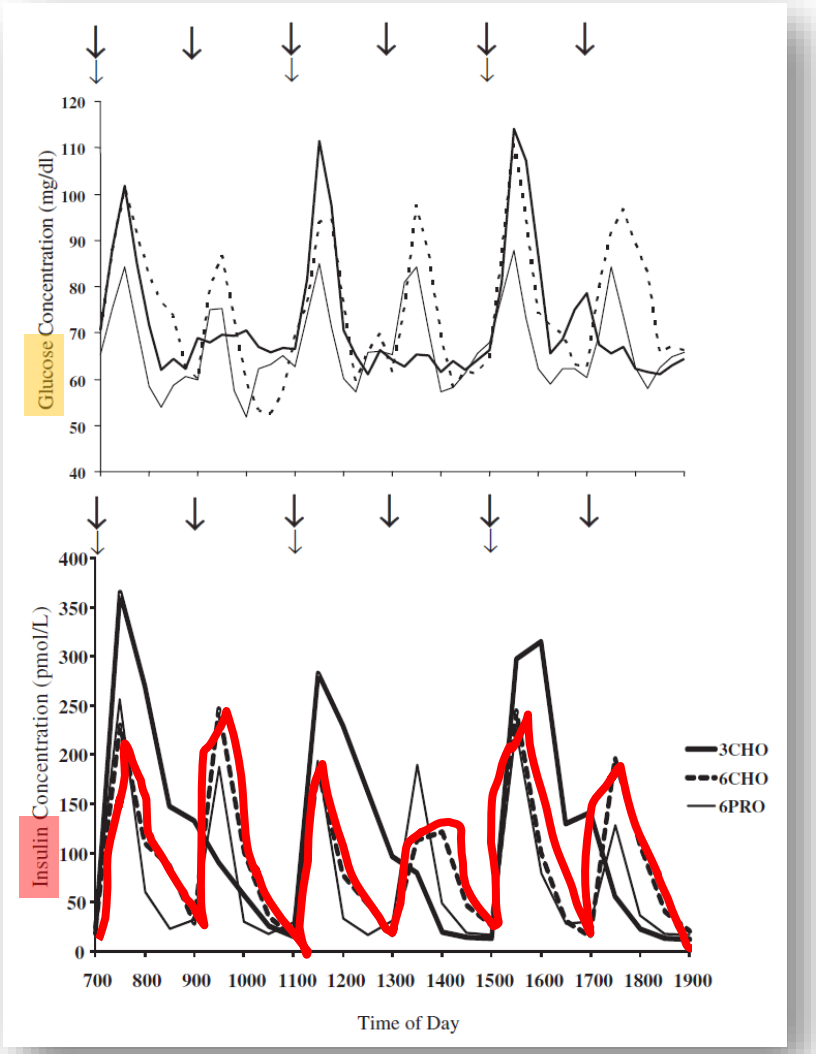
Pfeiffer, A. et al. Trends in Endocrinology & Metabolism, May 2018, Vol. 29, No. 5

Nutrition and Insulin

- how often we eat -







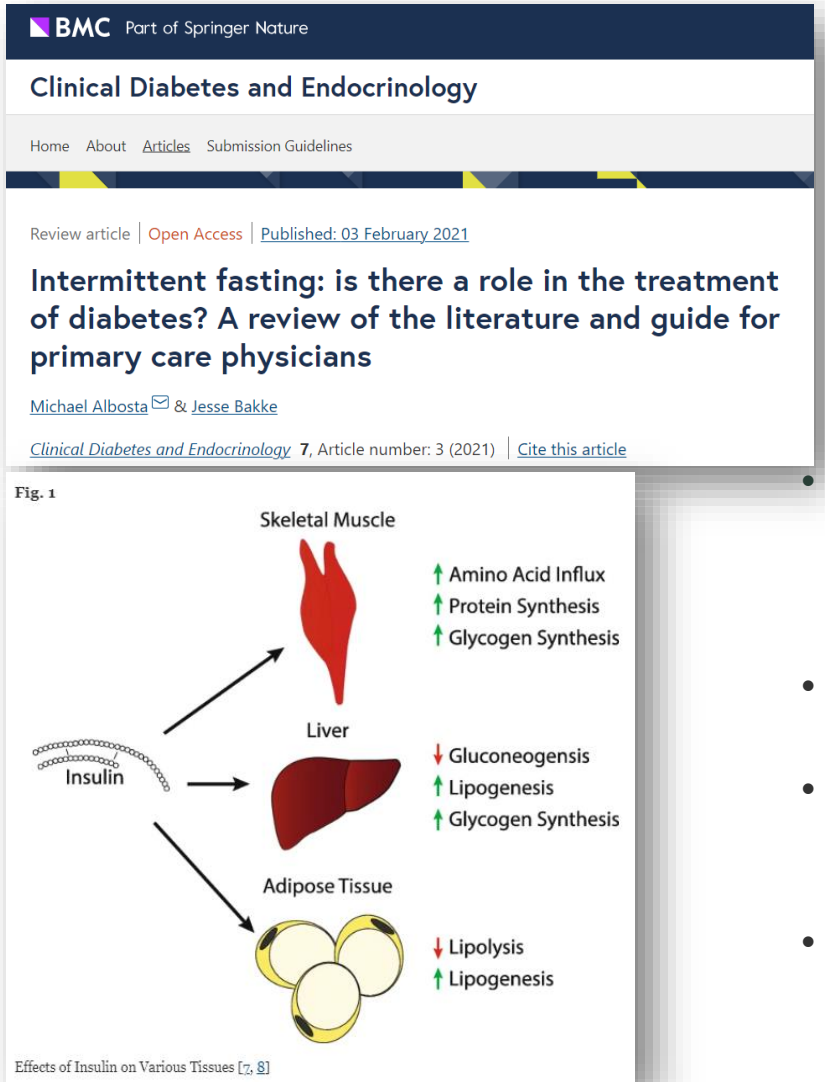
European e-Journal of Clinical Nutrition and Metabolism 5 (2010) e277ee280

Nutrition and Insulin

- how often we (don't) eat -

Soeters, MR. Endocrinologist MD PhD
Clinical Nutrition 39 (2020) 2335-2336

"The postprandial state is the period in which the largest metabolic, endocrine and inflammatory changes occur in normal, healthy, day-to-day living…. most human beings are in the postprandial state for the largest part of the day"



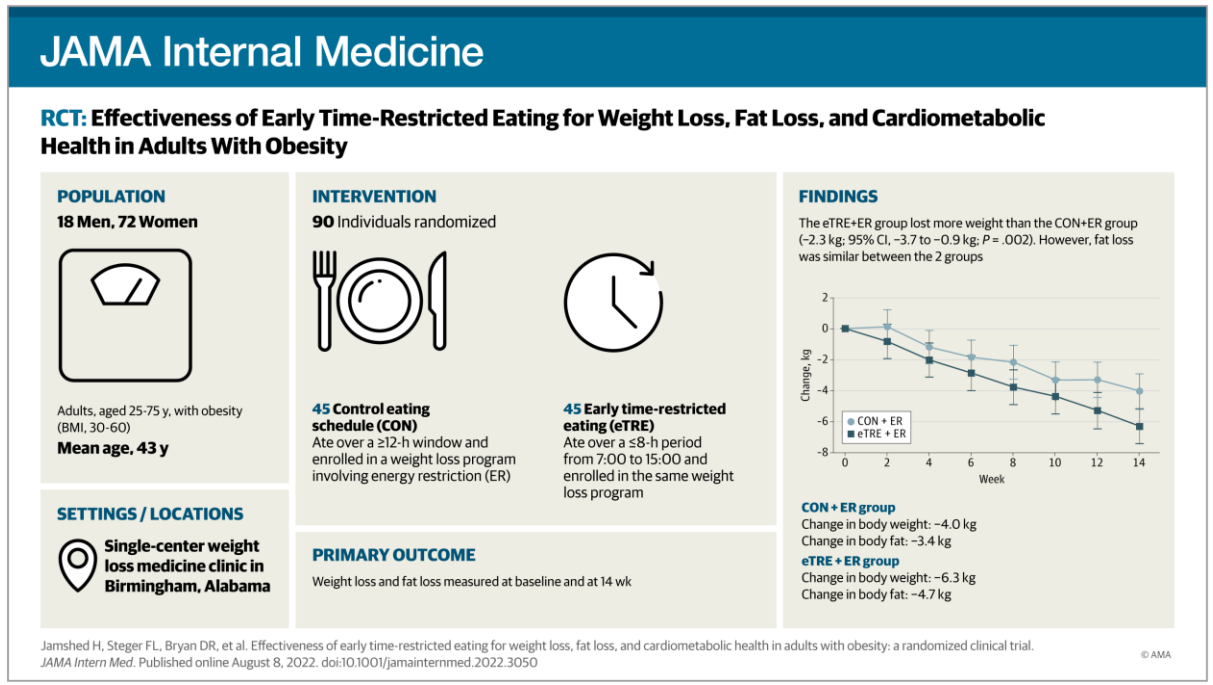
- The majority of the available research demonstrates that **intermittent fasting is effective at reducing body weight, decreasing fasting glucose, decreasing fasting insulin, reducing insulin resistance, decreasing levels of leptin, and increasing levels of adiponectin.**
- Some studies found that patients were able to reverse their need for insulin therapy during therapeutic intermittent fasting protocols.
- Current evidence suggests that intermittent fasting is an effective non-medicinal treatment option for type 2 diabetes.

- Physicians should consider educating themselves regarding the benefits of intermittent fasting.

Albosta, M. et al. Clinical Diabetes and Endocrinology volume 7, Article number: 3 (2021)

Nutrition and Insulin

- how often we (don't) eat -



JAMA Internal Medicine

RCT: Effectiveness of Early Time-Restricted Eating for Weight Loss, Fat Loss, and Cardiometabolic Health in Adults With Obesity

POPULATION

18 Men, 72 Women

Adults, aged 25-75 y, with obesity (BMI, 30-60)

Mean age, 43 y

SETTINGS / LOCATIONS

Single-center weight loss medicine clinic in Birmingham, Alabama

INTERVENTION

90 Individuals randomized

45 Control eating schedule (CON)
Ate over a ≥12-h window and enrolled in a weight loss program involving energy restriction (ER)

45 Early time-restricted eating (eTRE)
Ate over a ≤8-h period from 7:00 to 15:00 and enrolled in the same weight loss program

PRIMARY OUTCOME

Weight loss and fat loss measured at baseline and at 14 wk

FINDINGS

The eTRE+ER group lost more weight than the CON+ER group (−2.3 kg; 95% CI, −3.7 to −0.9 kg; *P* = .002). However, fat loss was similar between the 2 groups

CON + ER group
Change in body weight: −4.0 kg
Change in body fat: −3.4 kg

eTRE + ER group
Change in body weight: −6.3 kg
Change in body fat: −4.7 kg

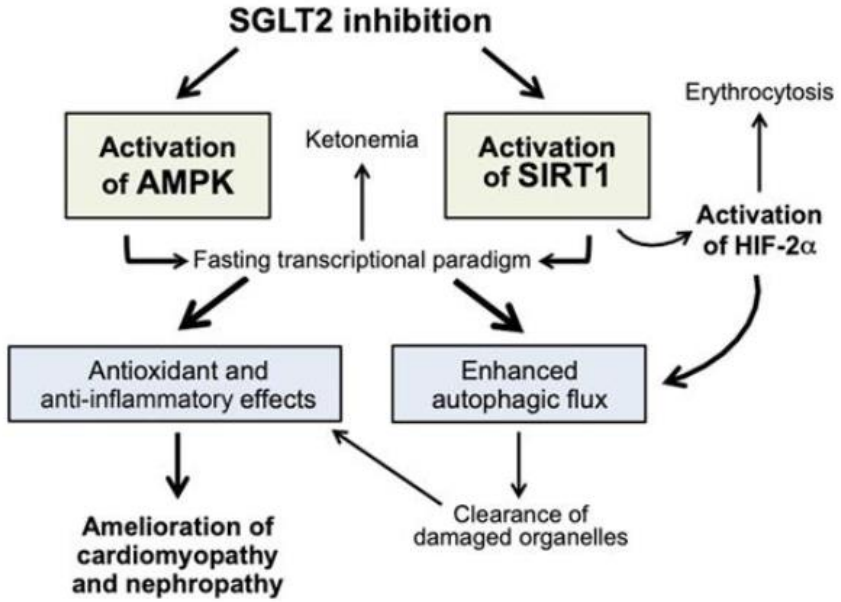
Jamshed H, Steger FL, Bryan DR, et al. Effectiveness of early time-restricted eating for weight loss, fat loss, and cardiometabolic health in adults with obesity: a randomized clinical trial. *JAMA Intern Med*. Published online August 8, 2022. doi:10.1001/jamainternmed.2022.3050

Jamshed, H. et al. *JAMA Intern Med.* Published online August 8,2022. doi:10.1001/jamainternmed.2022.3050

SGLT2 Inhibitors Produce Cardioprotective and Renoprotective Benefits by Inducing a State of Fasting Mimicry

It is therefore noteworthy that SGLT2 inhibitors induce a transcriptional paradigm that closely mimics the cellular response to starvation (Fig. 1) (27). These drugs activate SIRT1/AMPK and suppress Akt/mTOR signaling and, consequently, they can promote autophagy, independent of their effects on glucose or insulin (28–31). Importantly, the effect of SGLT2 to stimulate the activity of low-energy sensors is not mediated by interference with SGLT2 protein on an individual cellular level, since it is seen in organs that do not express SGLT2 (30,32).

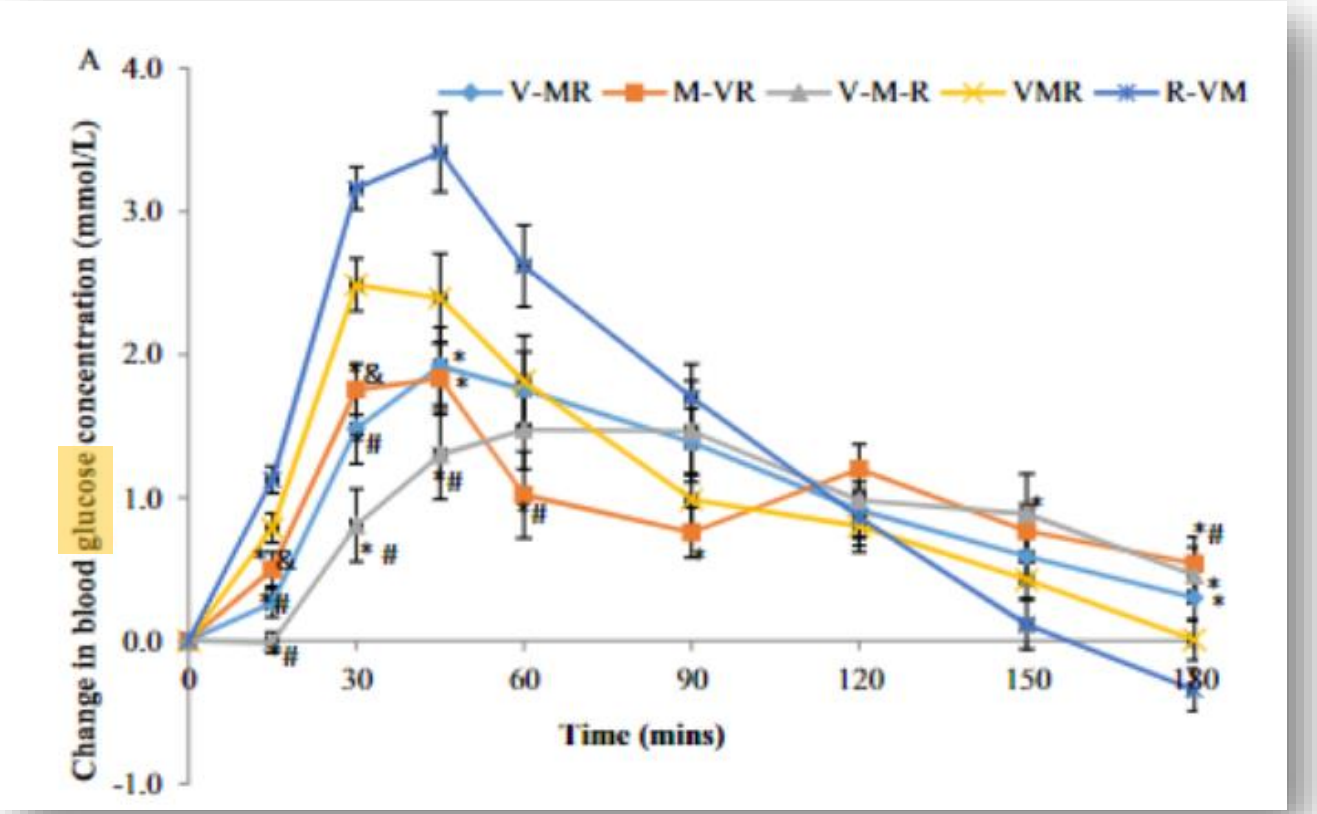
Figure 1

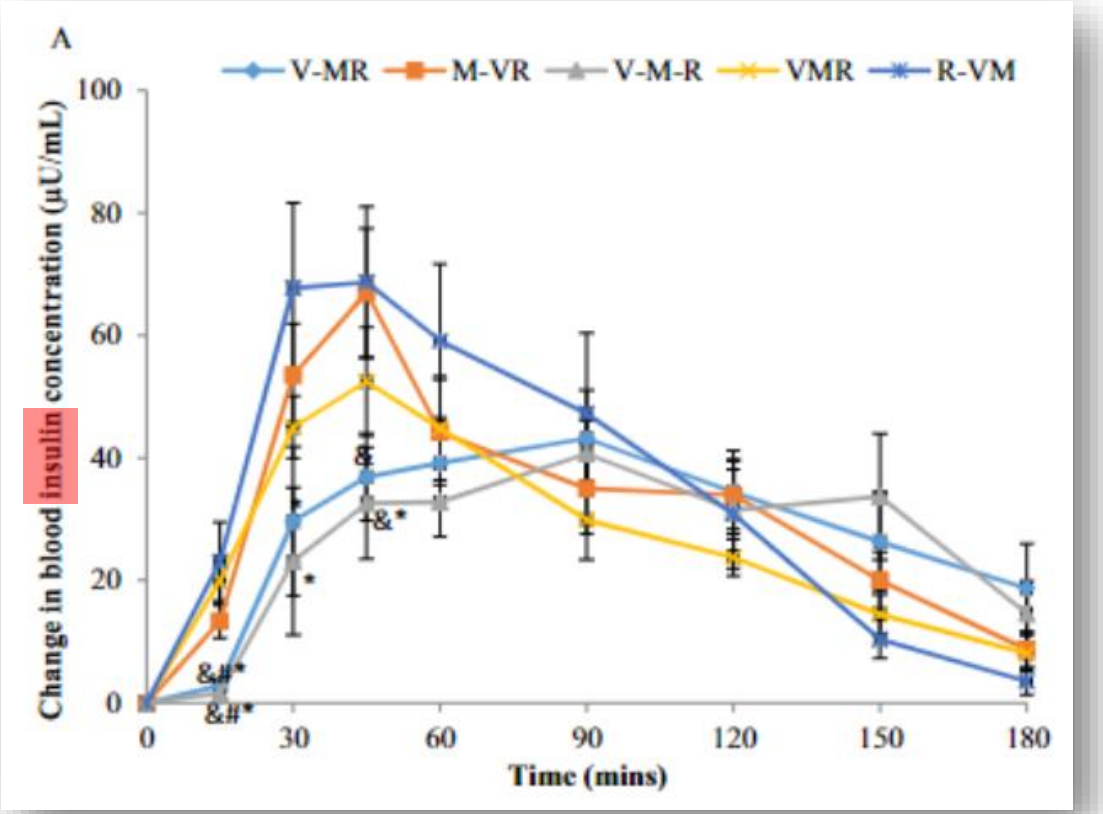


Packer, M. *Diabetes Care* 2020;43(3):508–511

Nutrition and Insulin

- order of what we eat -

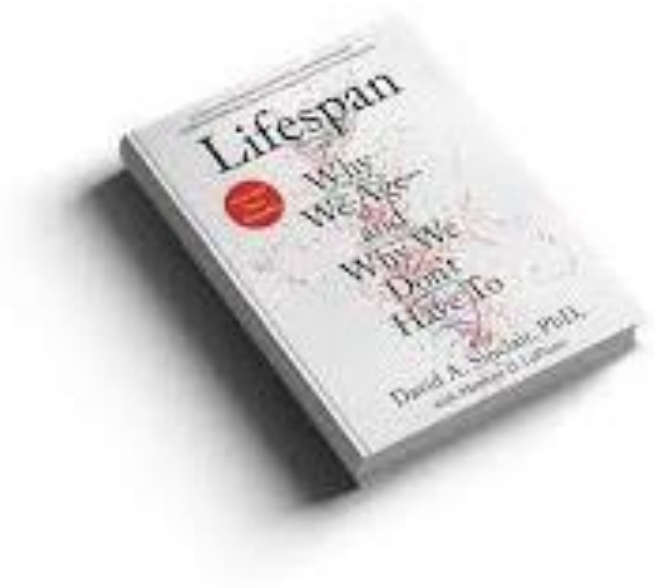




Sun, L. et al. Clinical Nutrition 39 (2020) 950-957

Nutrition and Insulin

- avoids sugar
- intermittent fasting
- CGM



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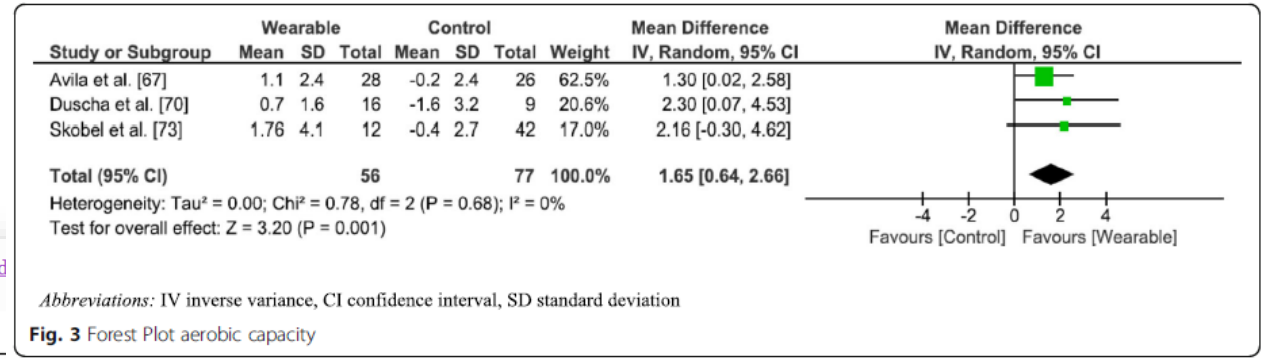
People

David Sinclair, A.O., Ph.D.
Professor
Principal Investigator
Co-Director

David A. Sinclair, A.O., Ph.D. is a Professor in the Department of Genetics and co-Director of the Paul F. Glenn Center for Biology of Aging Research at Harvard Medical School. He is best known for his work on understanding why we age and how to slow its effects. He obtained his Ph.D. in Molecular Genetics at the University of New South Wales, Sydney in 1995. He worked as a postdoctoral researcher at M.I.T. with Dr. Leonard Guarente where he co discovered a cause of aging for yeast as well as the role of Sir2 in epigenetic changes driven by genome instability. In 1999 he was recruited to Harvard Medical School where he has been teaching aging biology and translational medicine for aging for the past 16 years. His research has been primarily focused on the sirtuins, protein-modifying enzymes that respond to changing NAD+ levels and to caloric restriction (CR) with... read more

Can wearables help to improve Cardiometabolic health?

Physical activity

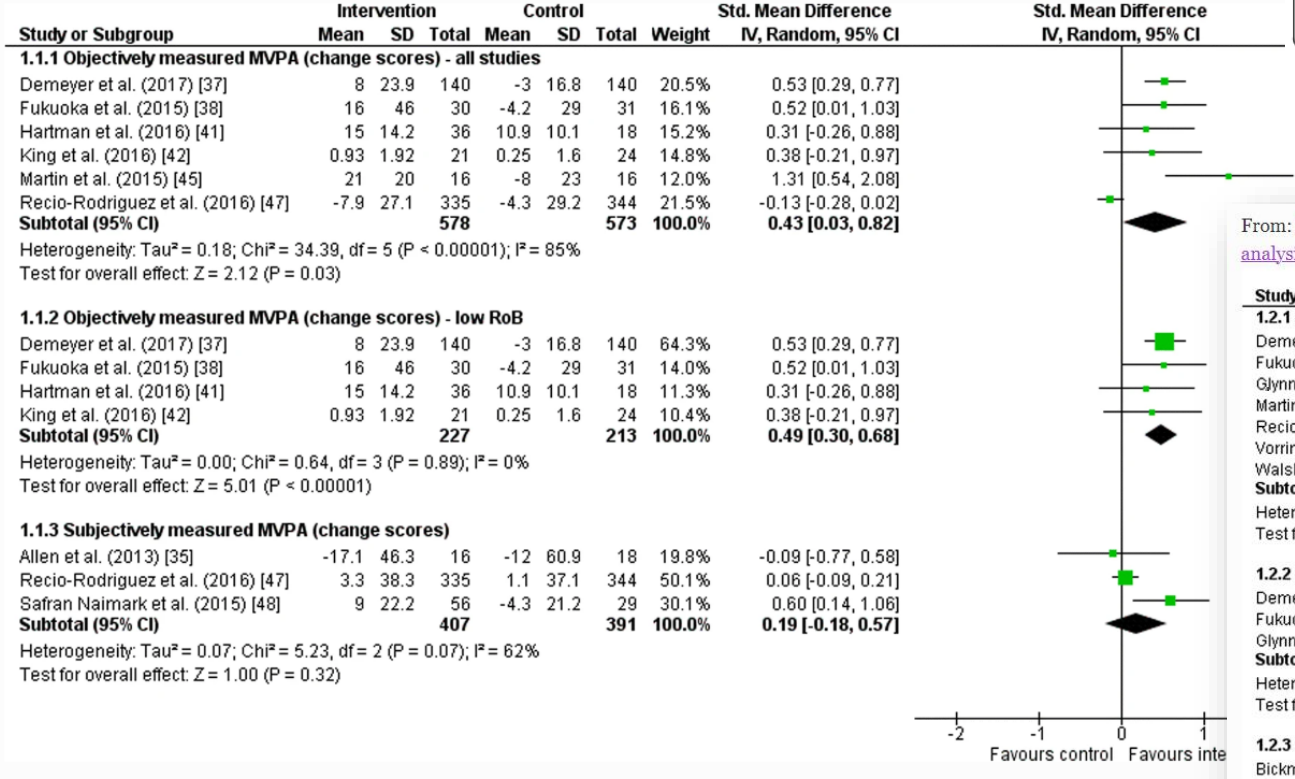


Abbreviations: IV inverse variance, CI confidence interval, SD standard deviation

Fig. 3 Forest Plot aerobic capacity

Hannan A et al. BMC Sports Science, Medicine and Rehabilitation volume 11, Article number: 14 (2019)
(9 studies, 1300 participants)

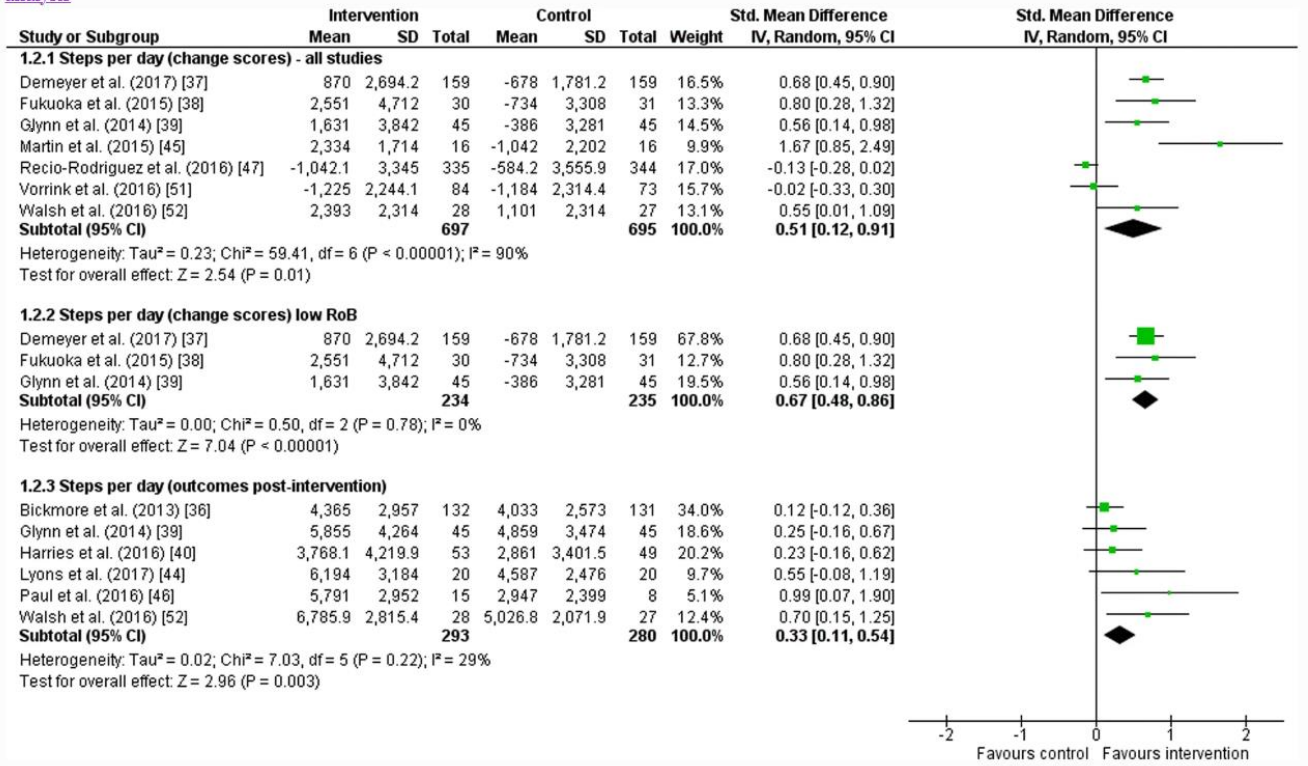
From: The Effect of Physical Activity Interventions Comprising Wearables and Smartphone Applications on Physical Activity: a Systematic Review and analysis



Forest plot of the effect of wearables and smartphone applications versus control on moderate-to-vigorous physical activity (MVPA) in minutes
confidence interval, *IV* inverse variance, *RoB* risk of bias, *SD* standard deviation, *Std* standardized

Gal, R et al. Sports Med - Open 4, 42 (2018)
(18 RCTs, 2700 participants)

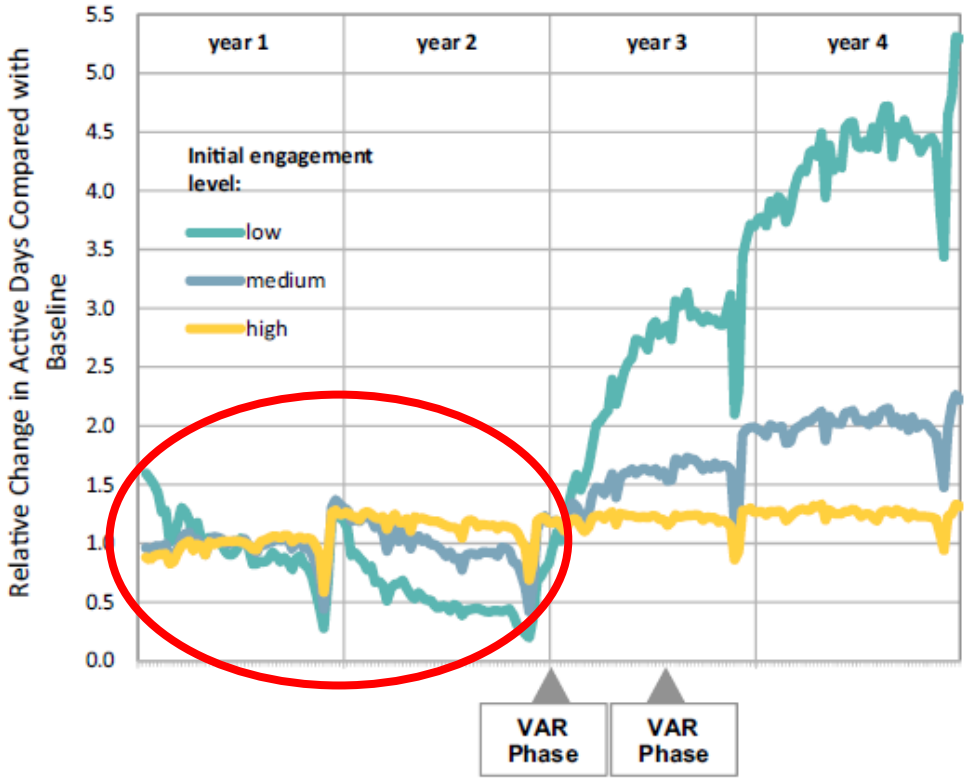
From: The Effect of Physical Activity Interventions Comprising Wearables and Smartphone Applications on Physical Activity: a Systematic Review and Meta-analysis



Forest plot of the effect of wearables and smartphone applications versus control on daily step count. *CI* confidence interval, *IV* inverse variance, *RoB* risk of bias, *SD* standard deviation, *Std* standardized

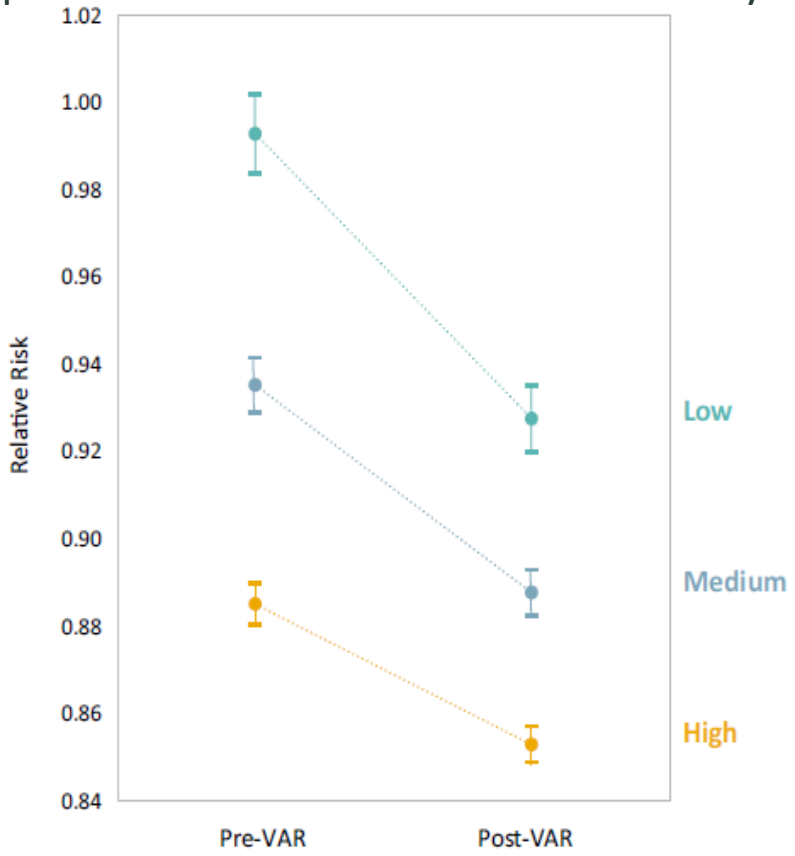
Physical activity

- Self-reported mean minutes of weekly physical activity were 20, 110 and 291
- Mean verified active days of physical activity annually were 9, 40 and 132 for the low, medium and high physical activity groups,



Hajat, C et al. npj Digital Medicine (2019) 2:91

Impact of VAR on relative risk of mortality due



Sleep

Nutrition and Insulin* (CGM) – a case study

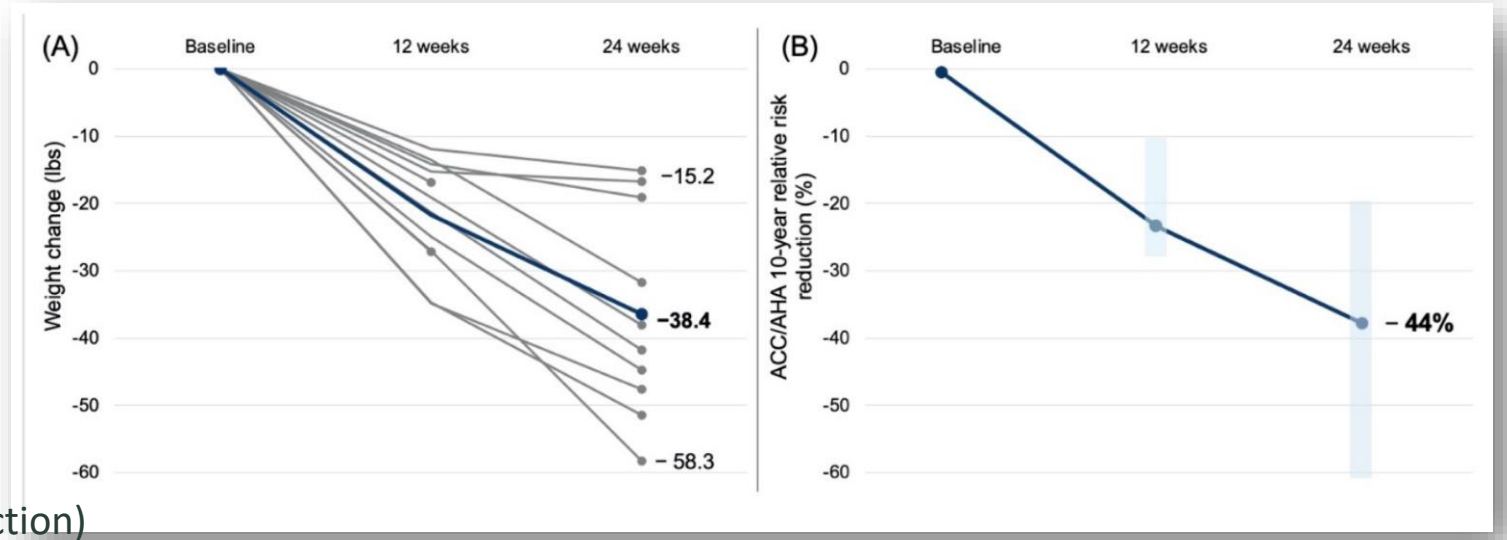
- BM : “I had a treadmill desk when I turned 42, so I was standing all day and walking 1 to 2 miles per hour, all day, including during meetings. **And still I was unable to drop weight**.”
- BM : “I just kept at it…I thought, “There’s got to be an answer to this, I’ve just not found it.”
- BM : “For the 20 years between 40 and 60 years old, **anything that anybody threw my way I engaged in, and it did not work.”**
- BM : “ There was concern that the glucose was rising and that I was pre-diabetic, but nobody had answers because the answers were lifestyle.”
- *“In order for you to get these numbers in order, you need to follow this program.”* It didn’t matter if the doctor was in Europe or Asia or the US. … I was told to
  - “Make sure (you) exercise three times a week.” Okay, I exercise six times a week.
  - “You need to get your heart rate above X amount for 20 min.” Okay. I do that.
  - “You have to do 10,000 steps.” I do that.
  - “Have you considered a plant-based diet?” I’ve been on a plant-based diet for three years.
  - “How much alcohol do you drink?” I stopped drinking alcohol.
  - “How much processed sugar do you have?” I stopped that 20 years ago.
- BM : “Then everybody just goes, ‘We have no idea. We don’t know what to tell you.’”

Nutrition and Insulin* (CGM) – a case study

- HEALTH COACH : “For two weeks, I just want you to monitor and eat, and let’s just see what’s happening with your glucose.”
- BM : “I don’t have a glucose problem. I don’t have an insulin problem. I don’t have a diabetes problem. I’ve watched this, my dad has diabetes, so I’m a little sensitive to it…”
- **Quinoa, cooked (then chilled) sweetpotato….everything caused a glucose spike. Eventually got to 30 foods that didn’t cause a glucose spike….**
- BM : ….”I have found is that there are foods I can eat **if I eat them in the right order**. If I eat my protein first, and then I have a carb, it absorbs much better for me.”
- BM : “And then I went to have my checkup. Usually you get the text from the guys who run your blood that says, your numbers are there, you can look them up. I received a text from my internist who just said, ‘Holy shit, I’ve never seen anything like this.’ It was crazy. My A1C went from 6.1 to 5.2, my glucose went from 117 to 84. My insulin went from 30 to 5. It was nuts ...”
- **Lost 80 pounds 7 months in…**

Obese PreD and T2D

- 10 individuals selected at employer
- all obese; 3 x pre diabetic, 1 x T1D, 6 x T2DM
- all CGMs and dietary guidance (no quantity restriction)



	Baseline		12 Weeks (Q1)		24 Weeks		Change	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age	52.9	5.7						
BMI	48.3	6.8	44.5	6.3	41.7	4.8	−6.5 ***	2.9
Weight, lbs (kg)	290.5 (131.8)	44.9 (20.4)	268.0 (121.6)	44.2 (20.2)	252.1 (114.4)	41.1 (18.6)	−38.4 *** (−17.4)	14.8 (6.7)
Fasting glucose	145	45	119	19	110	21	−35 **	35
HbA1C	7.1	1.4	6.4	0.9	6.0	0.6	−1.1 ***	1.4
Total cholesterol	182	32	155	20	175	51	−7	47
HDL-C	44	10	40	8	42	7	−2	6
LDL-C	107	31	97	22	114	49	−6.3	43
Trigs	165	75	110	39	105	36	−60 **	64
Systolic BP	141	13	130	9	124	10	−17 *	17
Diastolic BP	83	7	81	8	78	8	−6	12
ACC/AHA 10-year risk (absolute, %)	9.2	9.7	7.2	6.9	5.2	7.3	−4.0 *	5.6
ACC/AHA 10-year risk (relative change, %)							−44 *	24

Norwitz, N et al. Metabolites 2022, 12(9), 848

Medical student experiment

- A cohort of 13 first-year medical (n = 10) and dental (n = 3) students participated
- CGMs to monitor BG

“

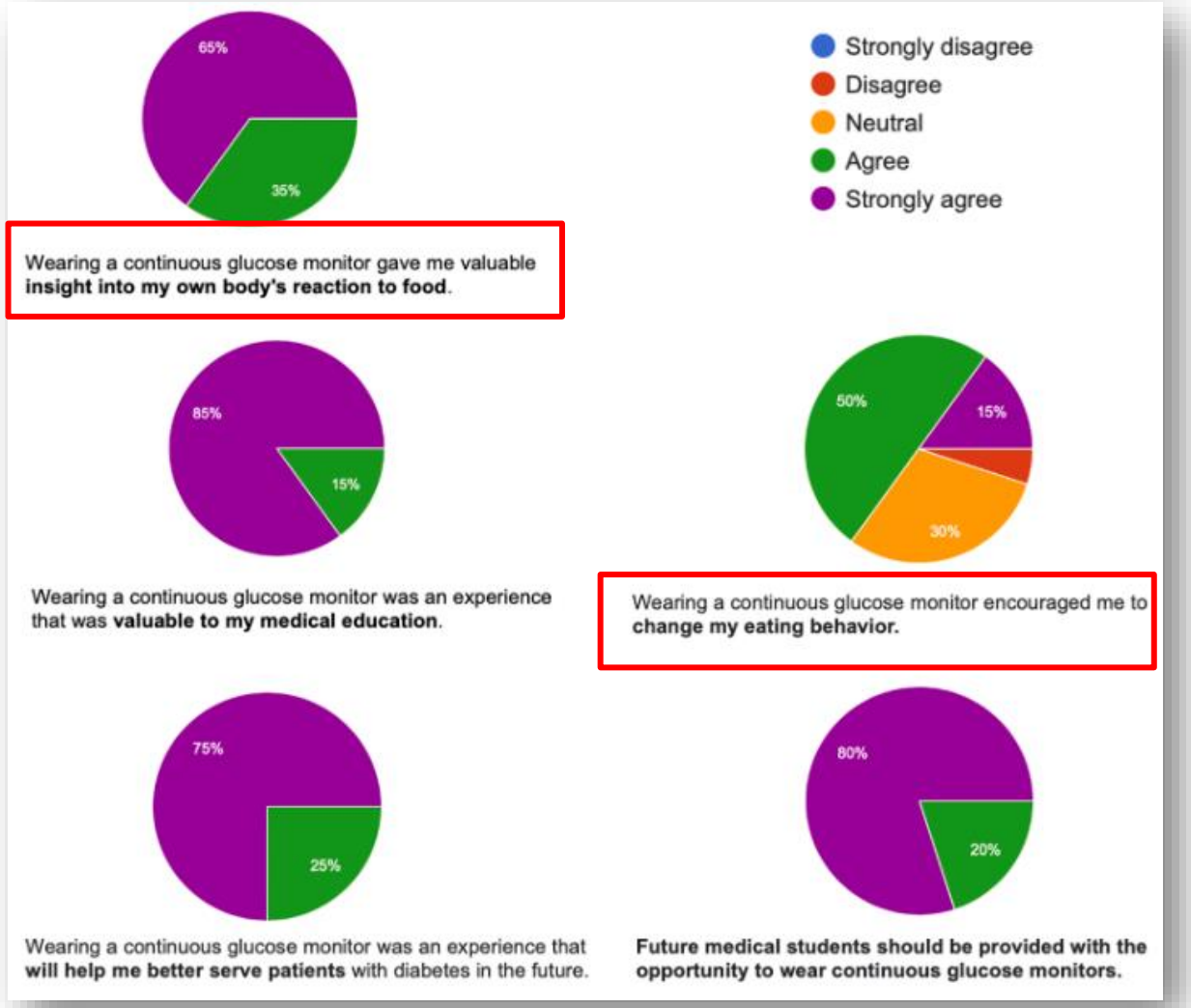
• *I’m more cognizant of reducing snacking and drinking sugary drinks ..during the night as it causes huge spikes and variability throughout my day.*

• *I’m trying to snack less and eat more protein in meals to keep myself satiated for a longer period and have more stable glucose levels.*

• *I have also sought to decrease by simple carb intake, as I found that I had dramatic glucose spikes, and subsequent fatigue, when consuming large amounts of simple carbs.*

• *[My] habits have changed since starting this experience …. The major change has been to avoid high-glycemic load carbohydrates*

“



Norwitz NG, et al. American Journal of Lifestyle Medicine. 2022;0(0)

Cardiometabolic health beyond 2022

Metabolic ill health and COVID-19


BMJ Nutrition, Prevention & Health

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Article info

Original research

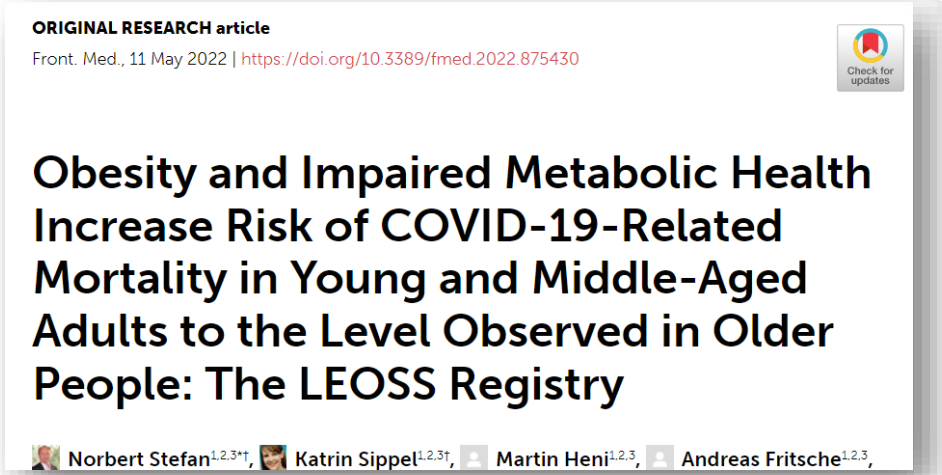
Overweight and obesity as risk factors for COVID-19-associated hospitalisations and death: systematic review and meta-analysis

PDF

PDF + Supplementary Material

Wendemi Sawadogo , Medhin Tsegaye , Andinet Gizaw and Tilahun Adera

Sawadogo W, et al. BMJ Nutrition, Prevention & Health 2022;5


ORIGINAL RESEARCH article

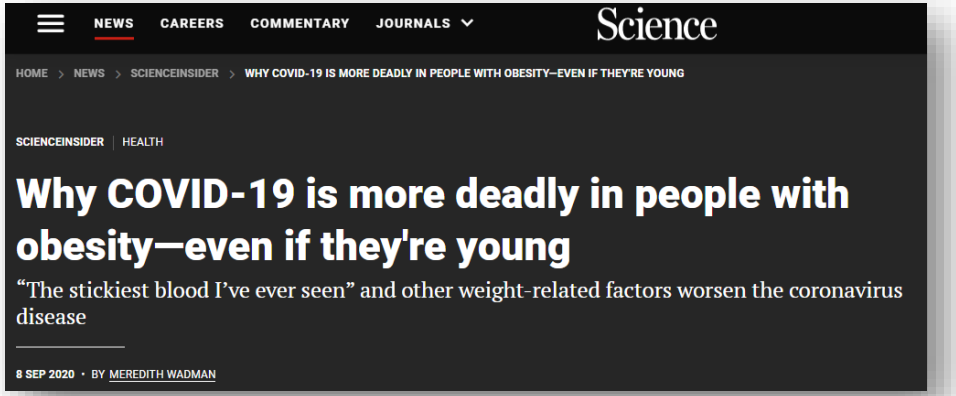
Front. Med., 11 May 2022 | https://doi.org/10.3389/fmed.2022.875430

Check for updates

Obesity and Impaired Metabolic Health Increase Risk of COVID-19-Related Mortality in Young and Middle-Aged Adults to the Level Observed in Older People: The LEOSS Registry

Norbert Stefan[1,2,3*†], Katrin Sippel[1,2,3†], Martin Heni[1,2,3], Andreas Fritsche[1,2,3],

Stefan, N. et al. Front Med (Lausanne). 2022 May 11;9:875430.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HOME > NEWS > SCIENCEINSIDER > WHY COVID-19 IS MORE DEADLY IN PEOPLE WITH OBESITY—EVEN IF THEY'RE YOUNG

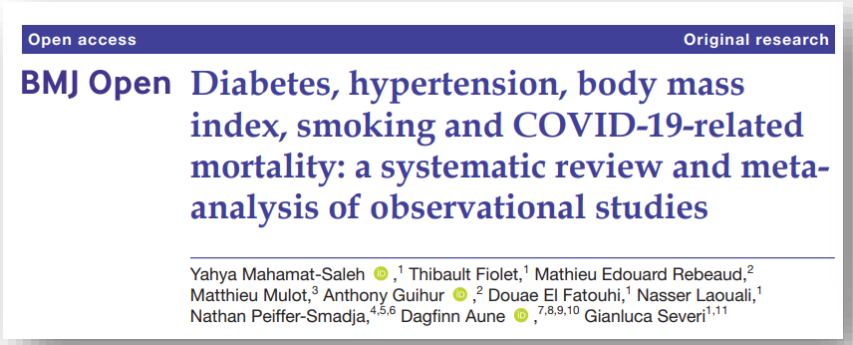
SCIENCEINSIDER | HEALTH

Why COVID-19 is more deadly in people with obesity—even if they're young

"The stickiest blood I've ever seen" and other weight-related factors worsen the coronavirus disease

8 SEP 2020 • BY MEREDITH WADMAN

doi: 10.1126/science.abe7010


Open access Original research

BMJ Open **Diabetes, hypertension, body mass index, smoking and COVID-19-related mortality: a systematic review and meta-analysis of observational studies**

Yahya Mahamat-Saleh ,[1] Thibault Fiolet,[1] Mathieu Edouard Rebeaud,[2] Matthieu Mulot,[3] Anthony Guihur ,[2] Douae El Fatouhi,[1] Nasser Laouali,[1] Nathan Peiffer-Smadja,[4,5,6] Dagfinn Aune ,[7,8,9,10] Gianluca Severi[1,11]

Conclusion Our findings suggest that diabetes, hypertension, obesity and smoking were associated with higher COVID-19 mortality, contributing to nearly 30% of COVID-19 deaths.

Mahamat-Saleh Y, et al. BMJ Open 2021;11:e052777

Metabolic ill health and COVID-19

Review

COVID-19 and metabolic disease: mechanisms and clinical management

Charlotte Steenblock, Peter E H Schwarz, Barbara Ludwig, Andreas Linkermann, Paul Zimmet, Konstantin Kulebyakin, Vsevolod A Tkachuk, Alexander G Markov, Hendrik Lehnert, Martin Hrabě de Angelis, Hannes Rietzsch, Roman N Rodionov, Kamlesh Khunti, David Hopkins, Andreas L Birkenfeld, Bernhard Boehm, Richard I G Holt, Jay S Skyler, J Hans DeVries, Eric Renard, Robert H Eckel, K George M M Alberti, Bruno Geloneze, Juliana C Chan, Jean Claude Mbanya, Henry C Onyegbutulem, Ambady Ramachandran, Abdul Basit, Mohamed Hassanein, Gavin Bewick, Giatgen A Spinas, Felix Beuschlein, Rüdiger Landgraf, Francesco Rubino, Geltrude Mingrone, Stefan R Bornstein

Lancet Diabetes Endocrinol 2021; 9:786–98
Published Online October 4, 2021
https://doi.org/10.1016/S2213-8587(21)00244-8

Department of Internal Medicine III, University Hospital Carl Gustav Carus
(C Steenblock PhD,

Up to 50% of the people who have died from COVID-19 had metabolic and vascular disorders. Notably, there are many direct links between COVID-19 and the metabolic and endocrine systems. Thus, not only are patients with metabolic dysfunction (eg, obesity, hypertension, non-alcoholic fatty liver disease, and diabetes) at an increased risk of developing severe COVID-19 but also infection with SARS-CoV-2 might lead to new-onset diabetes or aggravation of pre-existing metabolic disorders. In this Review, we provide an update on the mechanisms of how metabolic and endocrine disorders might predispose patients to develop severe COVID-19. Additionally, we update the practical recommendations and management of patients with COVID-19 and post-pandemic. Furthermore, we summarise new treatment options for patients with both COVID-19 and diabetes, and highlight current challenges in clinical management.

Steenblock C, et al. Lancet Diabetes Endocrinol. 2021;9 :786-98

METABOLIC SYNDROME AND RELATED DISORDERS
Volume 20, Number 4, 2022
© Mary Ann Liebert, Inc.
Pp. 191–198
DOI: 10.1089/met.2021.0102

ORIGINAL ARTICLES

Open camera or QR reader and scan code to access this article and other resources online.

Impact of Metabolic Syndrome on Severity of COVID-19 Illness

Shannon Wu, BA,[1] Keren Zhou, MD,[2] Anita Misra-Hebert, MD, MPH,[3] James Bena, MS,[4] and Sangeeta R. Kashyap, MD[2]

On multivariable analysis, patients with metabolic syndrome had an increased risk of 77% for hospitalization, 56% for ICU admission, and 81% for death ($P < 0.001$).

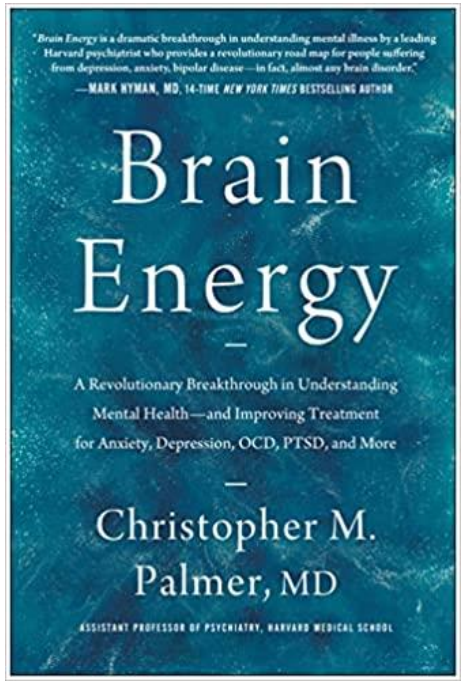
Wu S, et al. Metabolic Syndrome and Related Disorders. Vol 20 No4, 2022

Will COVID-19 put the spotlight on MetS?

Metabolism (and psych conditions)

"it is not clear how much the pathophysiology of psychiatric disorders contributes to metabolic syndrome or vice versa"

Deng, C et al. Front. Neurosci., 29 January 2020 Sec. Neuroendocrine Science
https://doi.org/10.3389/fnins.2020.00021

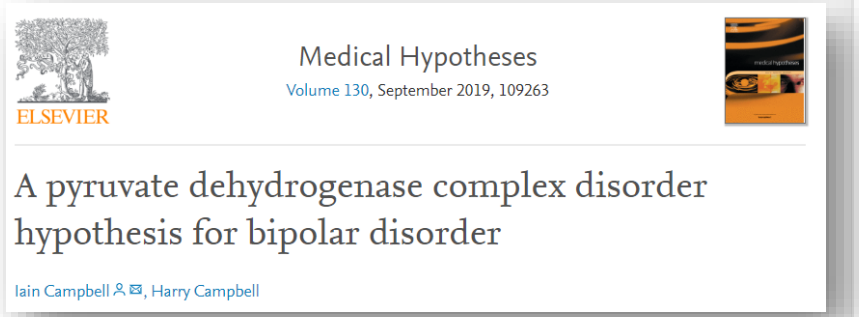


"Mental disorders are metabolic disorders of the brain."

Excess weight*		p < 0.001	p < 0.001
No	35 (7.4)	Ref	Ref
Yes	211 (39.1)	3.71 (2.75-4.99)	4.58 (3.27-6.40)
Non-HDL cholesterol		p < 0.001	p < 0.001
Normal	35 (9.6)	Ref	Ref
High	214 (32.6)	3.40 (2.44-4.76)	2.65 (1.92-3.68)
Current psychotropic medication		p = 0.475	-
No	177(23.1)	Ref	
Yes	72 (27.9)	1.09 (0.86-1.39)	
Current anxiety disorders		p < 0.001	p = 0.044
No	196 (22.1)	Ref	Ref
Yes	53 (38.4)	1.69 (1.31-2.17)	1.33 (1.01-1.78)
Current mood disorders		p < 0.001	p = 0.006
None	200 (22.1)	Ref	Ref
Depression	30 (40.0)	1.82 (1.33-2.45)	1.57 (1.15-1.89)
Bipolar disorder	19 (46.3)	2.11 (1.47-2.98)	1.62 (1.19-2.08)
Suicide risk		p < 0.001	p = 0.021
No	207 (22.5)	Ref	Ref
Yes	42 (40.8)	1.74 (1.32-2.28)	1.58 (1.18-2.03)

Moreira, F. et al. Brazilian Journal of Psychiatry. 2019 Jan-Feb;41(1):38–43

Metabolism (and psych conditions)



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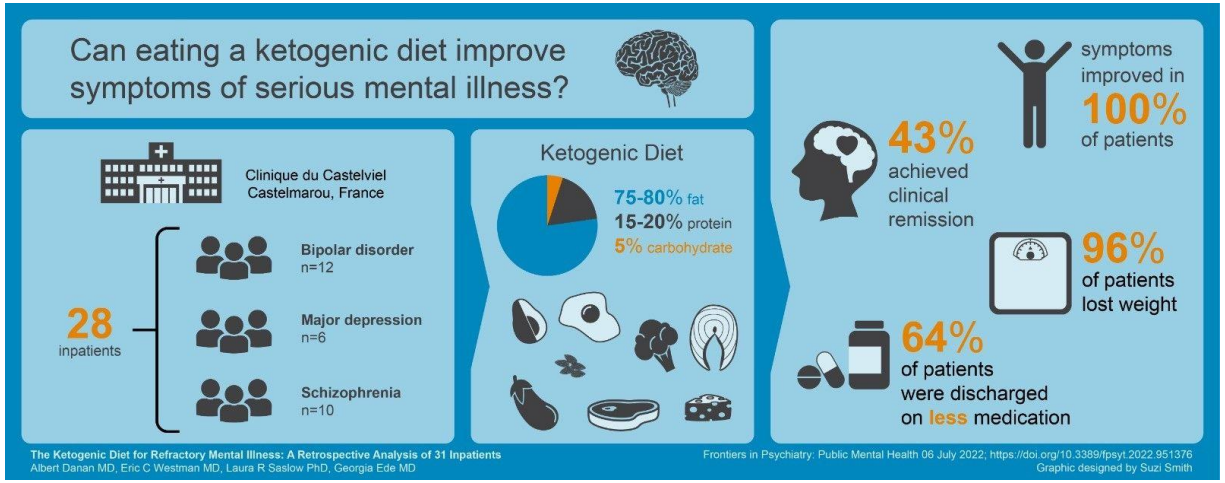

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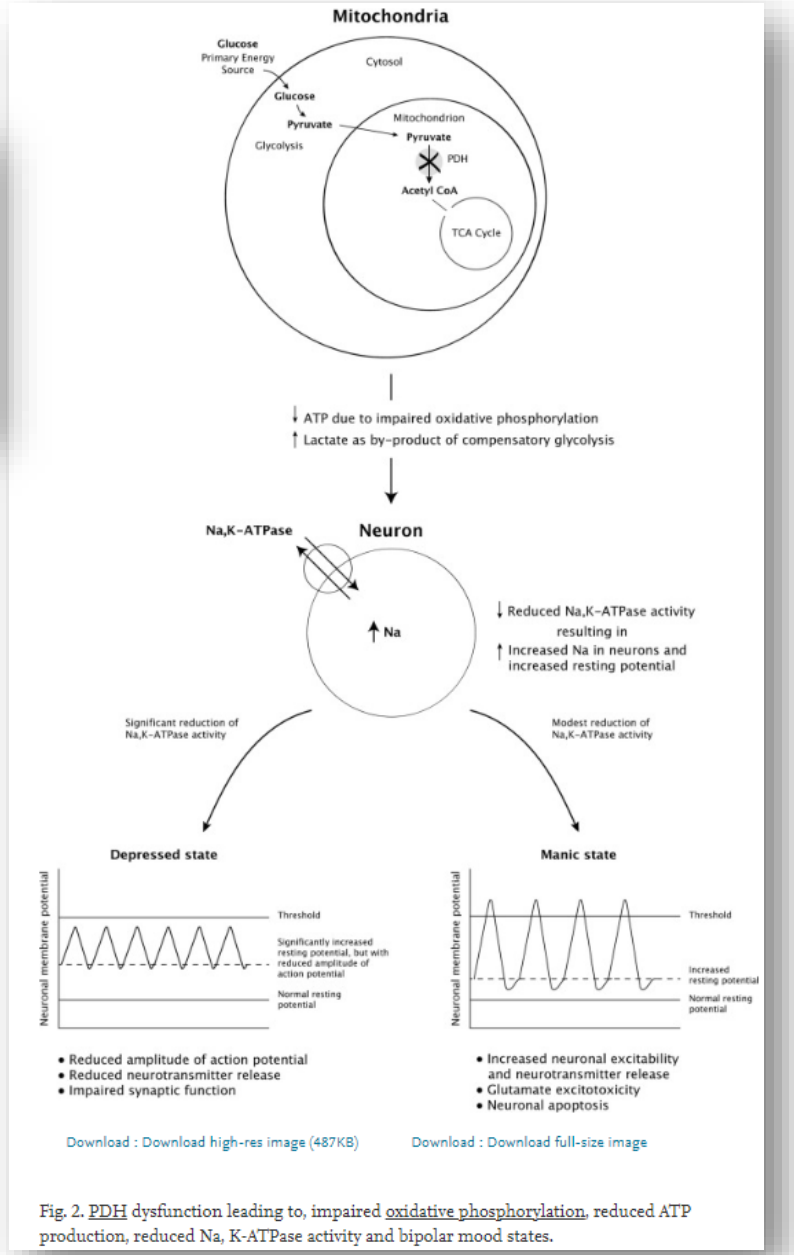

Fig. 2. PDH dysfunction leading to, impaired oxidative phosphorylation, reduced ATP production, reduced Na, K-ATPase activity and bipolar mood states.

Q: Can Your Apple Watch Fix How Key Lifestyle Factors Impact Your Cardiometabolic Health and Mortality Risk?

A: not yet, but it will

Continuous glucose (insulin) monitoring for all

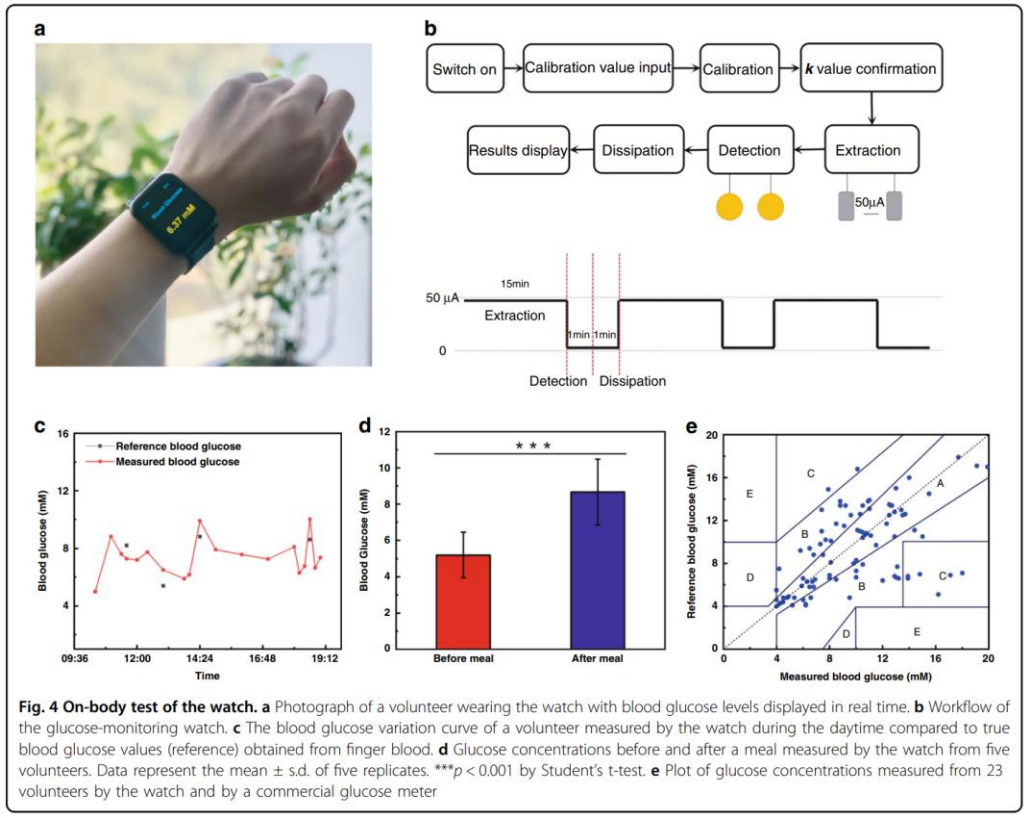

Fig. 4 On-body test of the watch. a Photograph of a volunteer wearing the watch with blood glucose levels displayed in real time. **b** Workflow of the glucose-monitoring watch. **c** The blood glucose variation curve of a volunteer measured by the watch during the daytime compared to true blood glucose values (reference) obtained from finger blood. **d** Glucose concentrations before and after a meal measured by the watch from five volunteers. Data represent the mean ± s.d. of five replicates. ***$p < 0.001$ by Student's t-test. **e** Plot of glucose concentrations measured from 23 volunteers by the watch and by a commercial glucose meter

Chang et al. *Microsystems & Nanoengineering* (2022)8:25
https://doi.org/10.1038/s41378-022-00355-5

Microsystems & Nanoengineering
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Highly integrated watch for noninvasive continual glucose monitoring

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Chang, T. et al. Nature Microsystems & Nanoengineering (2022)8:25


Questions

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