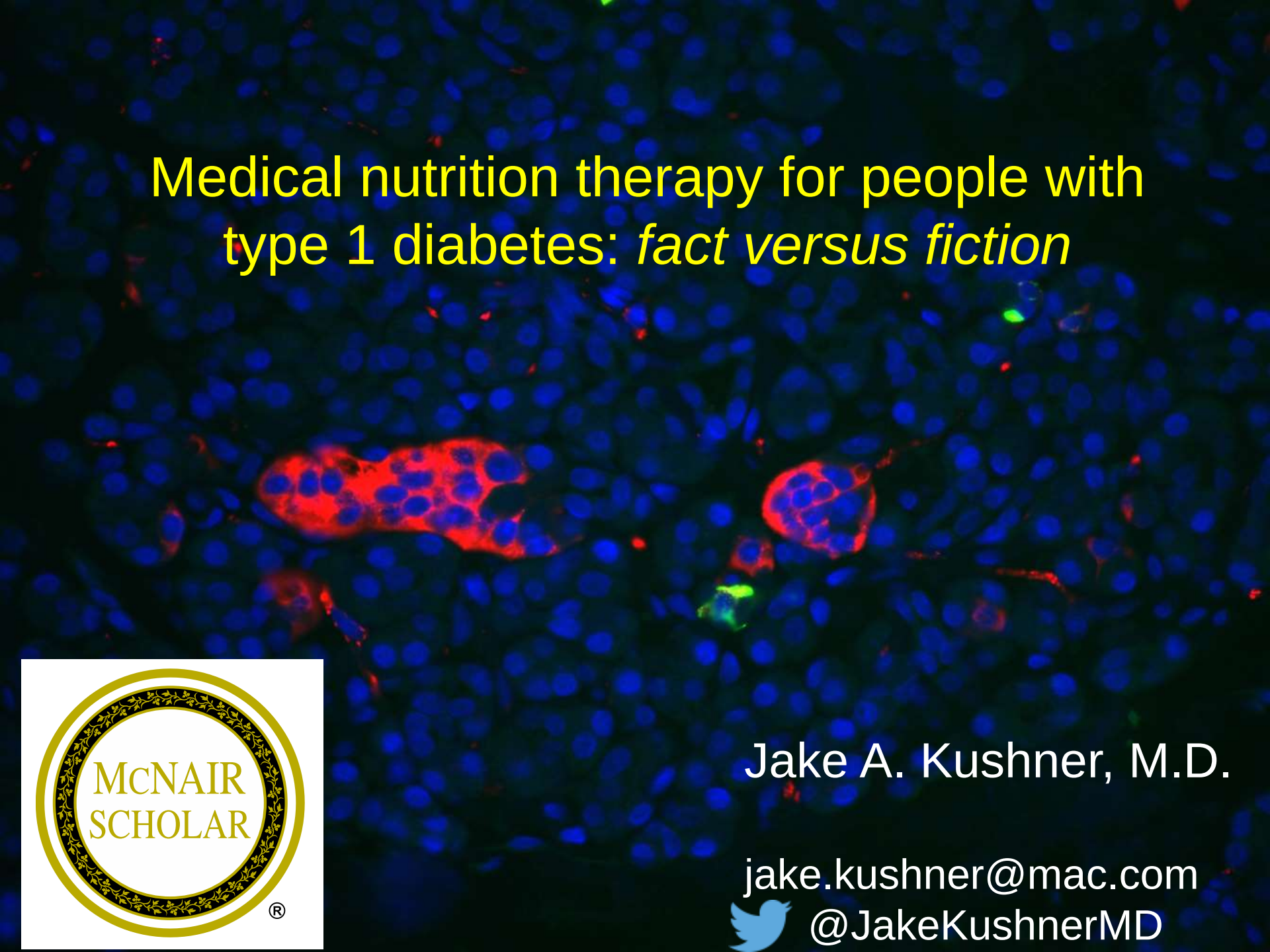
Medical nutrition therapy for people with type 1 diabetes: *fact versus fiction*



Jake A. Kushner, M.D.

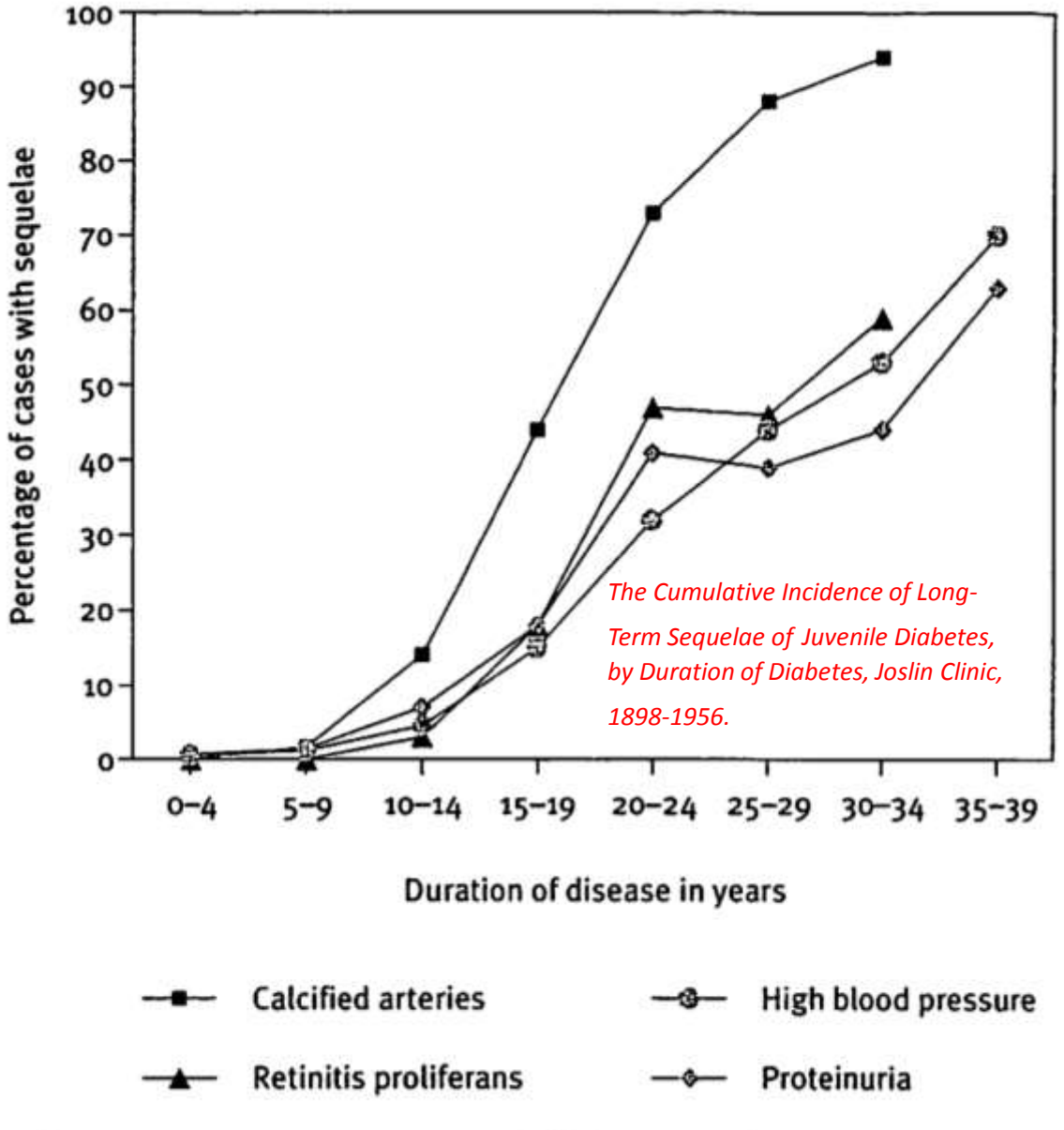
jake.kushner@mac.com
@JakeKushnerMD

Disclosure: consultant for Lexicon, Virta Health, KNOW foods, and Sanofi Aventis

Michael Bliss, *The Discovery of Insulin*,
University of Chicago Press, 1982, 2007

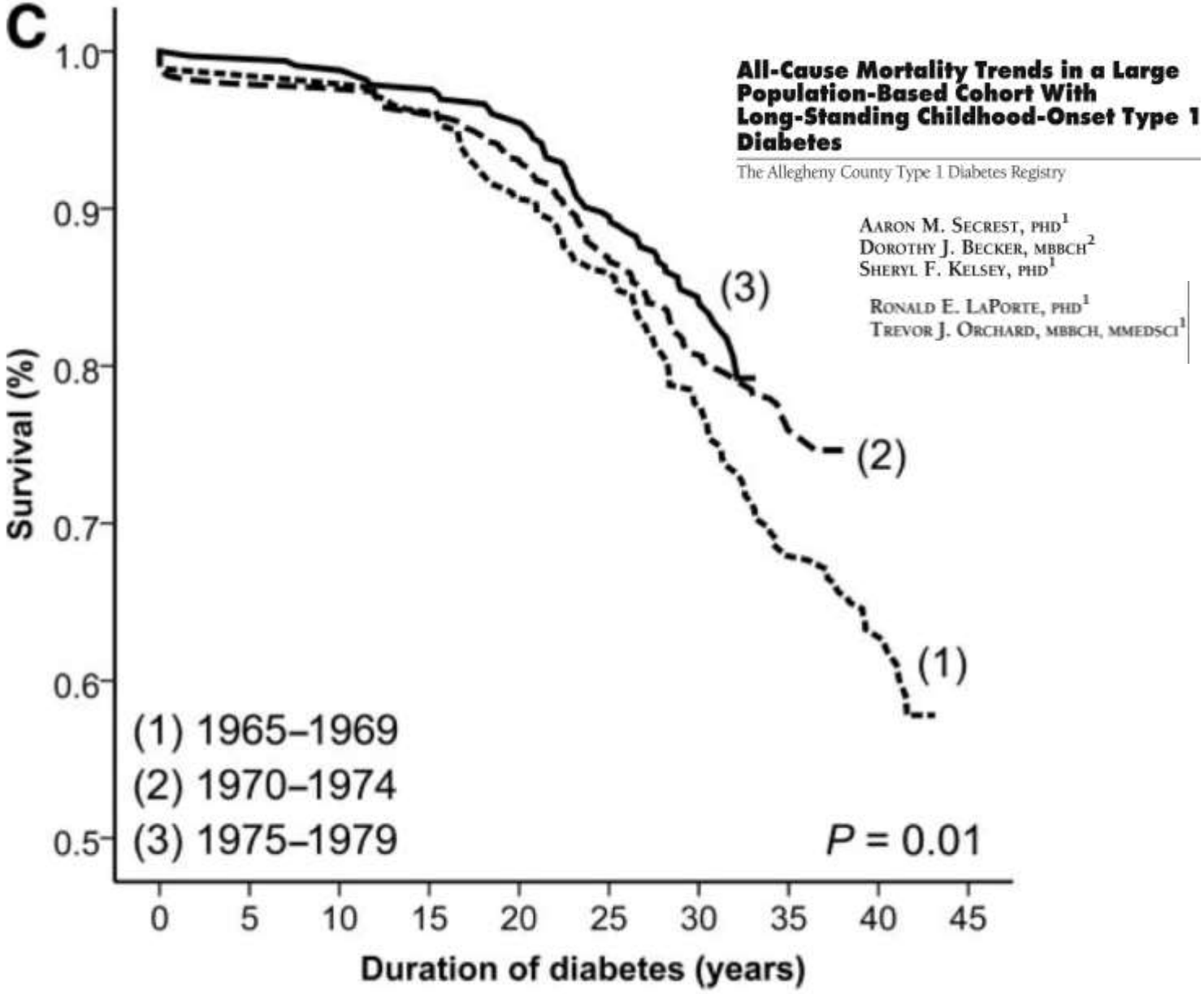
Was insulin a “cure” for type 1 diabetes?

Insulin-treated T1D patients began to suffer from unforeseen complications



The Cumulative Incidence of Long-Term Sequelae of Juvenile Diabetes, by Duration of Diabetes, Joslin Clinic, 1898-1956.

Insulin-treated T1D patients had high rates of mortality, even into the 1990s



All-Cause Mortality Trends in a Large Population-Based Cohort With Long-Standing Childhood-Onset Type 1 Diabetes

The Allegheny County Type 1 Diabetes Registry

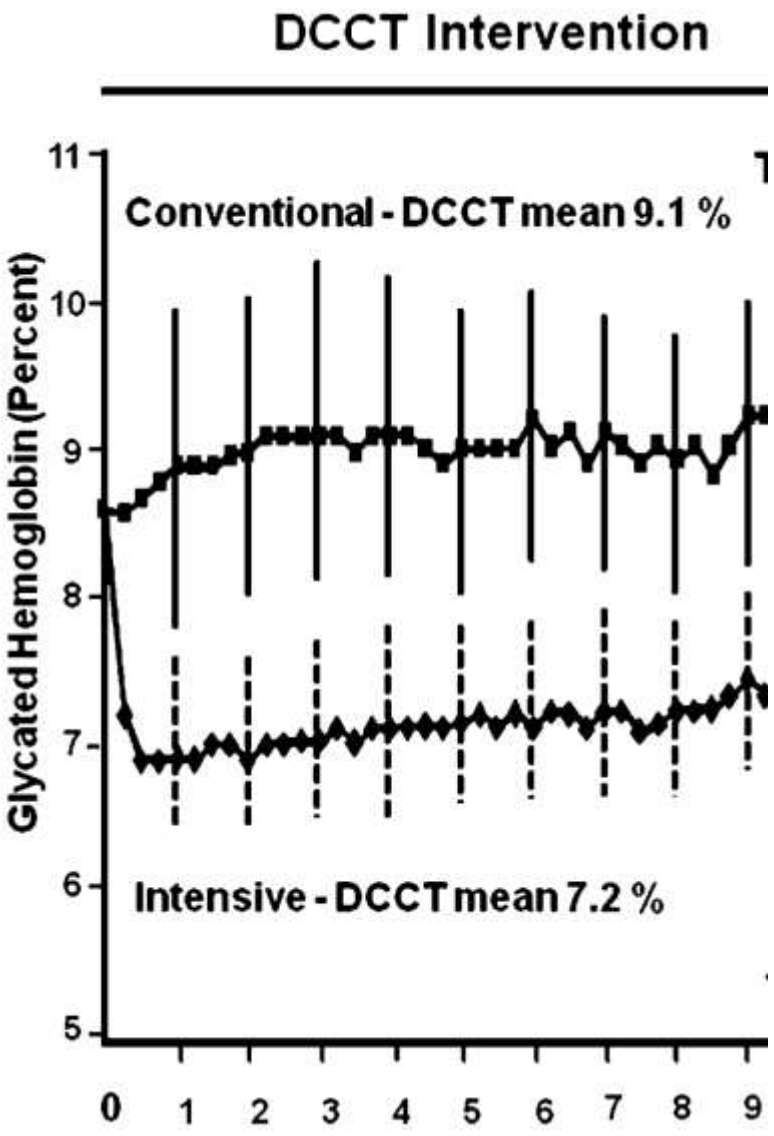
AARON M. SECREST, PHD[1]
DOROTHY J. BECKER, MBBCH[2]
SHERYL F. KELSEY, PHD[1]
RONALD E. LAPORTE, PHD[1]
TREVOR J. ORCHARD, MBBCH, MMEDSCI[1]

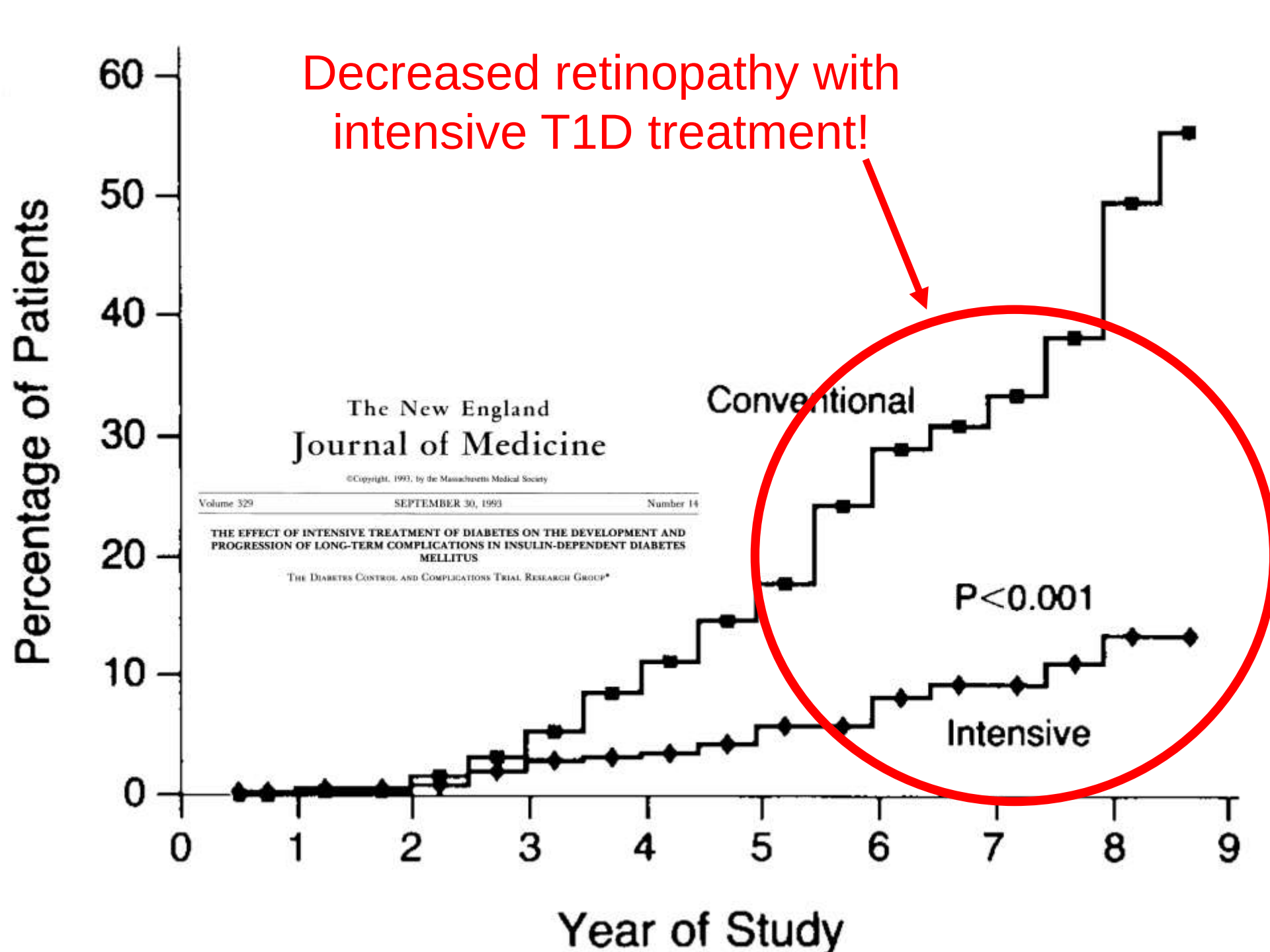
Diabetes Control Complication Trial (DCCT):

1441 T1D patients, 1-5 years duration, ages 13-39

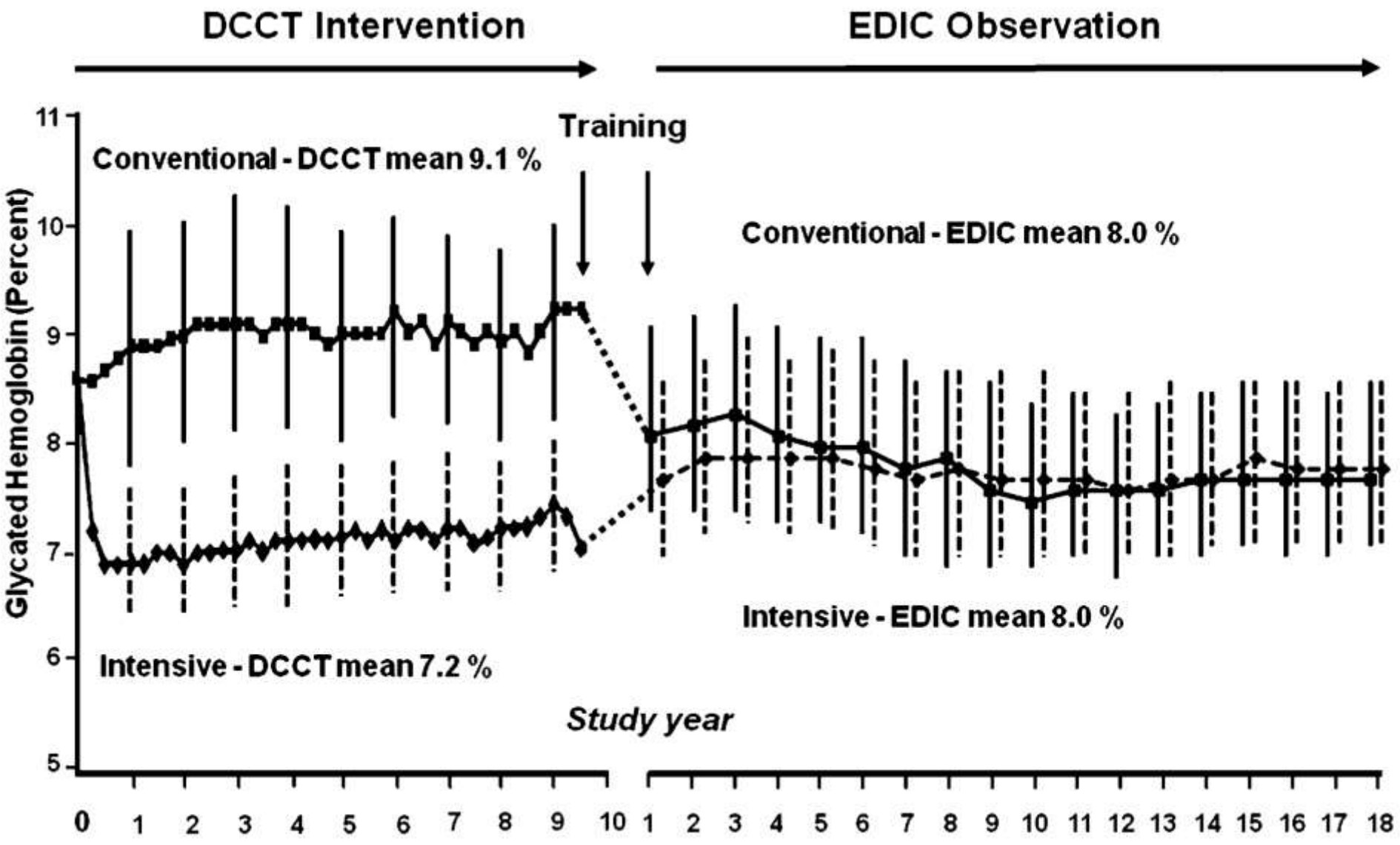
“intensive therapy” vs. “conventional therapy”

Intensive glucose control for ~6.5 years

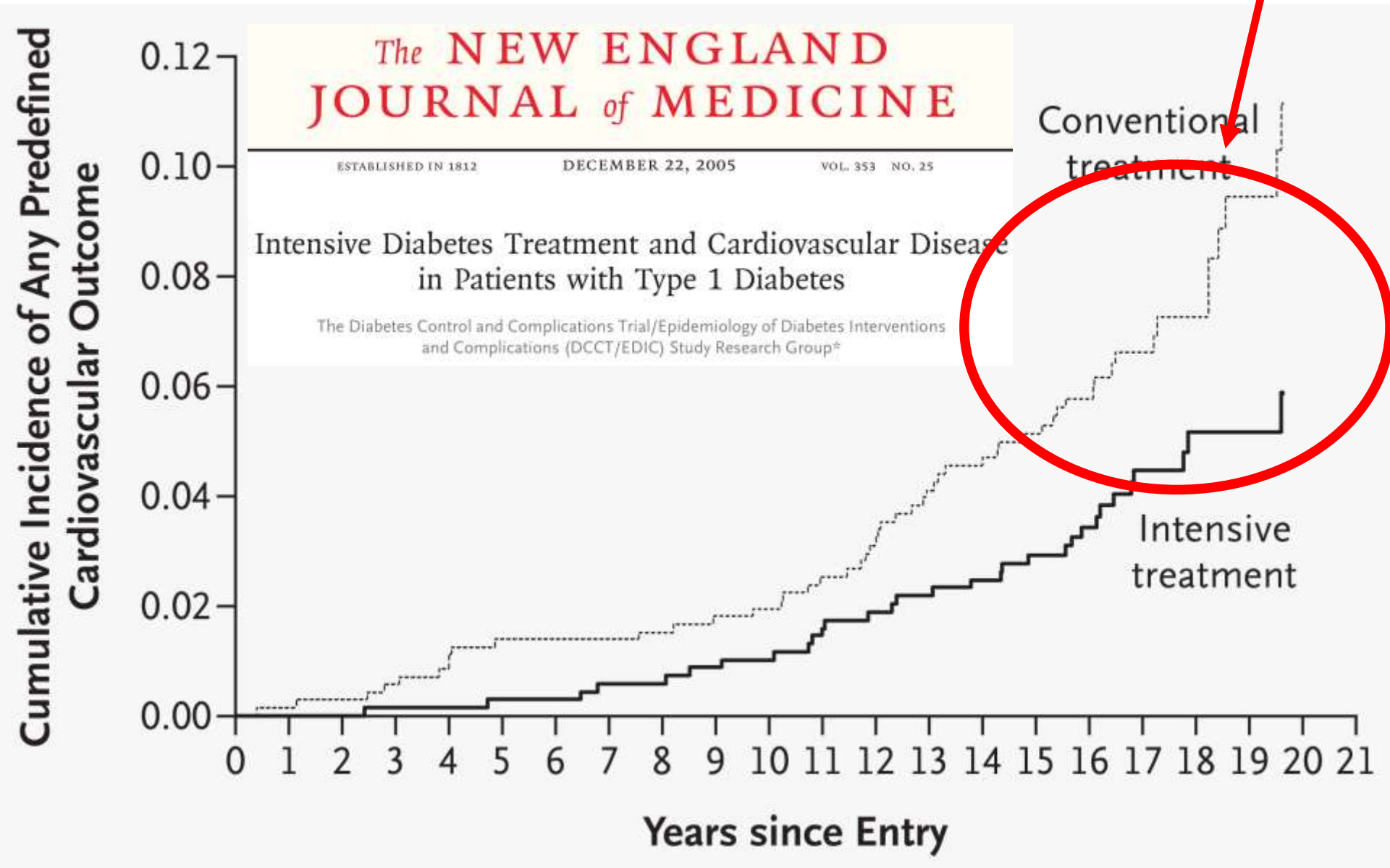



Decreased retinopathy with intensive T1D treatment!
The New England Journal of Medicine
©Copyright, 1993, by the Massachusetts Medical Society
Volume 329 SEPTEMBER 30, 1993 Number 14
THE EFFECT OF INTENSIVE TREATMENT OF DIABETES ON THE DEVELOPMENT AND PROGRESSION OF LONG-TERM COMPLICATIONS IN INSULIN-DEPENDENT DIABETES MELLITUS
The Diabetes Control and Complications Trial Research Group*
Conventional
P<0.001
Intensive
Percentage of Patients
0
10
20
30
40
50
60
0
1
2
3
4
5
6
7
8
9
Year of Study

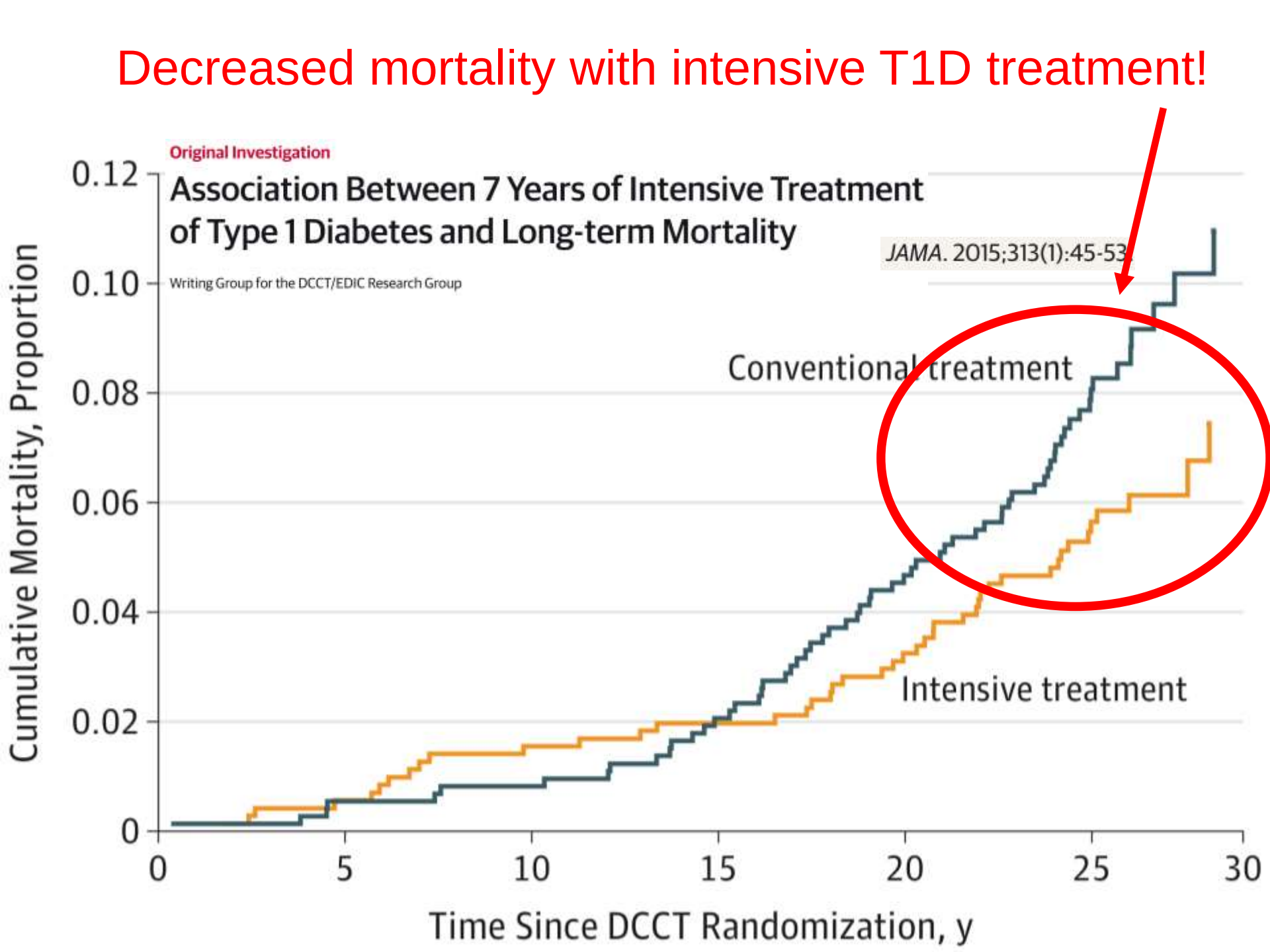
Intensive glucose control for ~10 years



Decreased CV disease with intensive T1D treatment!



Decreased mortality with intensive T1D treatment!



Goal: Help people with type 1 diabetes

Millions of people have type 1 diabetes

live long healthy lives

Are we successful in caring for people with T1D?

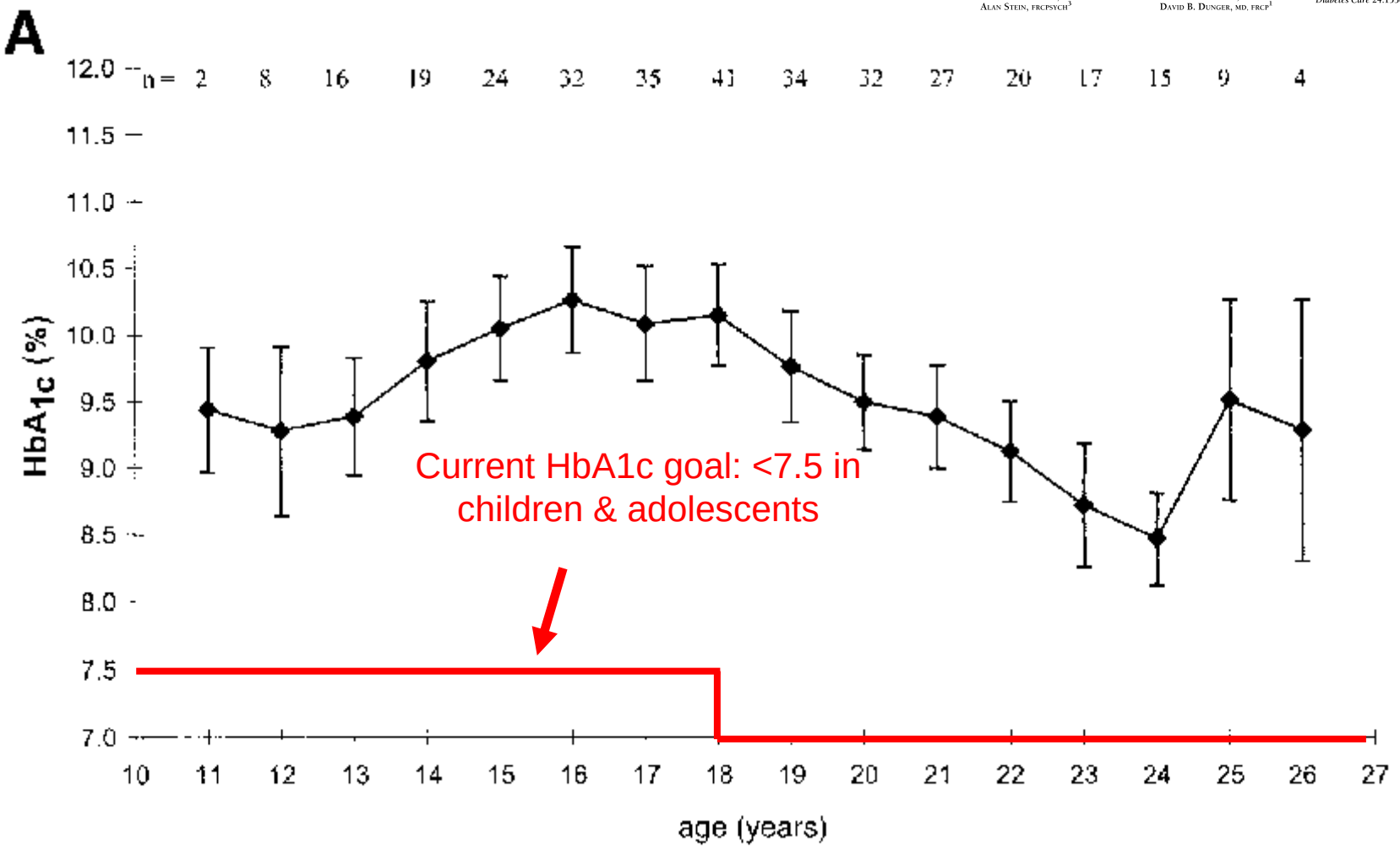
Most with T1D fail to achieve glycemic targets

Clinical and Psychological Course of Diabetes From Adolescence to Young Adulthood

A longitudinal cohort study

Kathryn S. Bryden, RN[1]
Robert C. Peveler, FRCPsych[2]
Alan Stein, FRCPsych[3]
Andrew Neil, FRCP[4]
Richard A. Mayou, FRCPsych[5]
David B. Dunger, MD, FRCP[1]

Diabetes Care 24:1536–1540, 2001



Adverse consequences of T1D
even when glycemic targets are achieved?

Near euglycemia in T1D does not prevent excess cardiovascular risk of death

ORIGINAL ARTICLE

Glycemic Control and Excess Mortality in Type 1 Diabetes

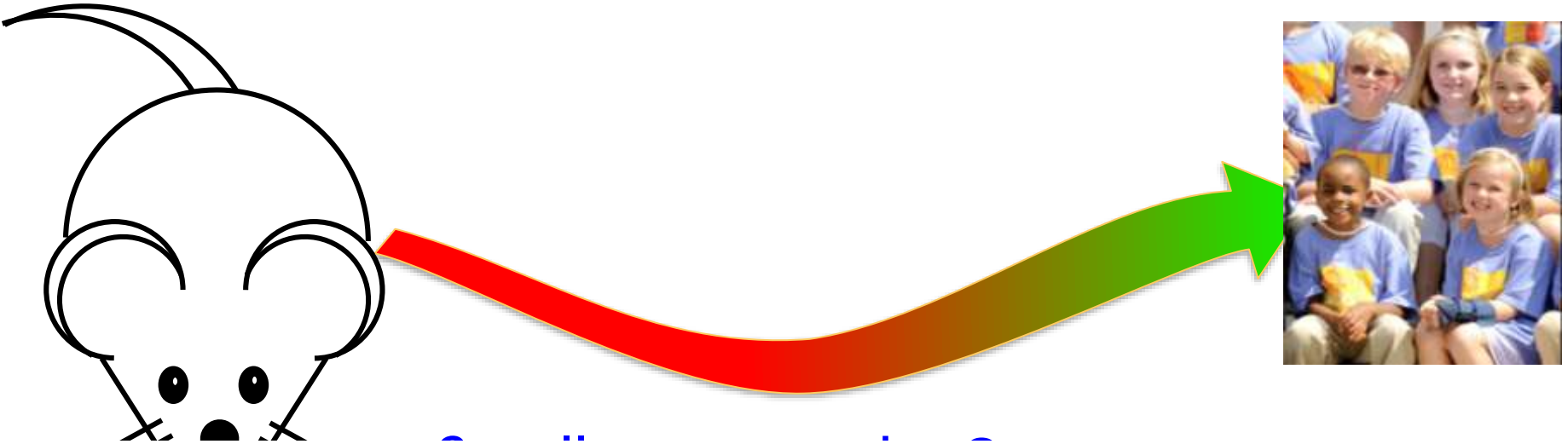
Marcus Lind, M.D., Ph.D., Ann-Marie Svensson, Ph.D., Mikhail Kosiborod, M.D., Soffia Gudbjörnsdottir, M.D., Ph.D., Aldina Pivodic, M.Sc., Hans Wedel, Ph.D., Sofia Dahlqvist, Mark Clements, M.D., Ph.D., and Annika Rosengren, M.D., Ph.D.

N ENGL J MED 371;21 NEJM.ORG NOVEMBER 20, 2014

Table 3. Adjusted Hazard Ratios for Death from Any Cause and Death from Cardiovascular Causes among Patients with Type 1 Diabetes versus Controls, According to Time-Updated Mean Glycated Hemoglobin Level and Renal Disease Status, Model 3.*

Variable	Hazard Ratio	
	Death from Any Cause	Death from Cardiovascular Disease
Time-updated mean glycated hemoglobin level — no. of events/total no.	7386/200,539	2326/200,539
Reference group (controls)	1.00	1.00
≤6.9%	2.36 (1.97–2.83)	2.92 (2.07–4.13)
7.0–7.8%	2.38 (2.02–2.80)	3.39 (2.49–4.61)
7.9–8.7%	3.11 (2.66–3.62)	4.44 (3.32–5.96)
8.8–9.6%	3.65 (3.11–4.30)	5.35 (3.94–7.26)
≥9.7%	8.51 (7.24–10.01)	10.46 (7.62–14.37)

When will basic science knowledge translate to improve T1D outcomes?

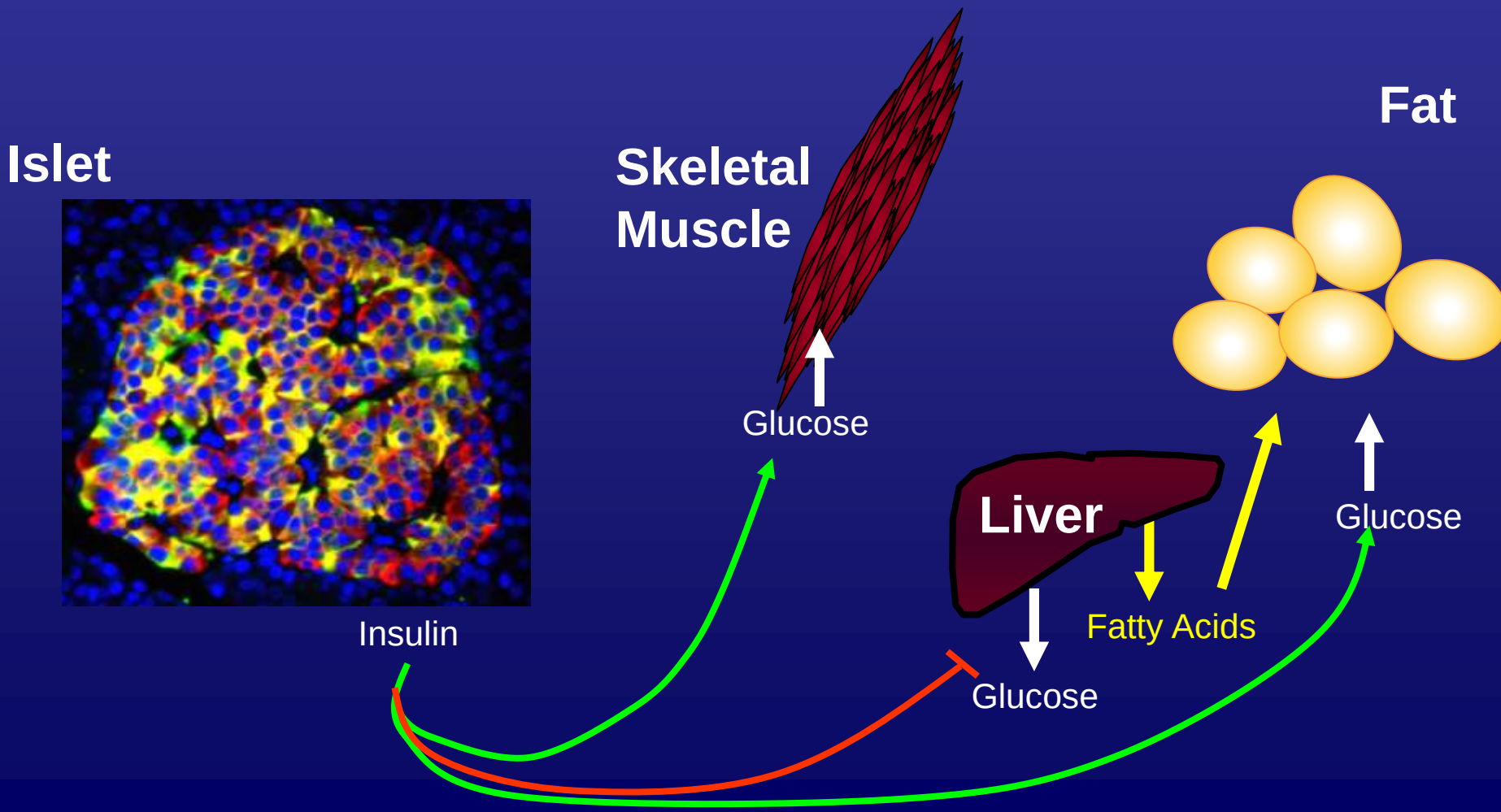


β- cells persist in T1D pancreata without evidence for ongoing β- cell turnover or neogenesis

Carol J. Lam; Daniel R. Jacobson; Matthew M. Rankin; Aaron R. Cox; Jake A. Kushner

J Clin Endocrinol Metab jc.2016-3806. **DOI:** https://doi.org/10.1210/jc.2016-3806

Insulin secretion by the pancreatic β-cell is at the center of glucose homeostasis



Carbohydrate intolerance is a central component of T1D pathophysiology

Caring for Your Child's Health

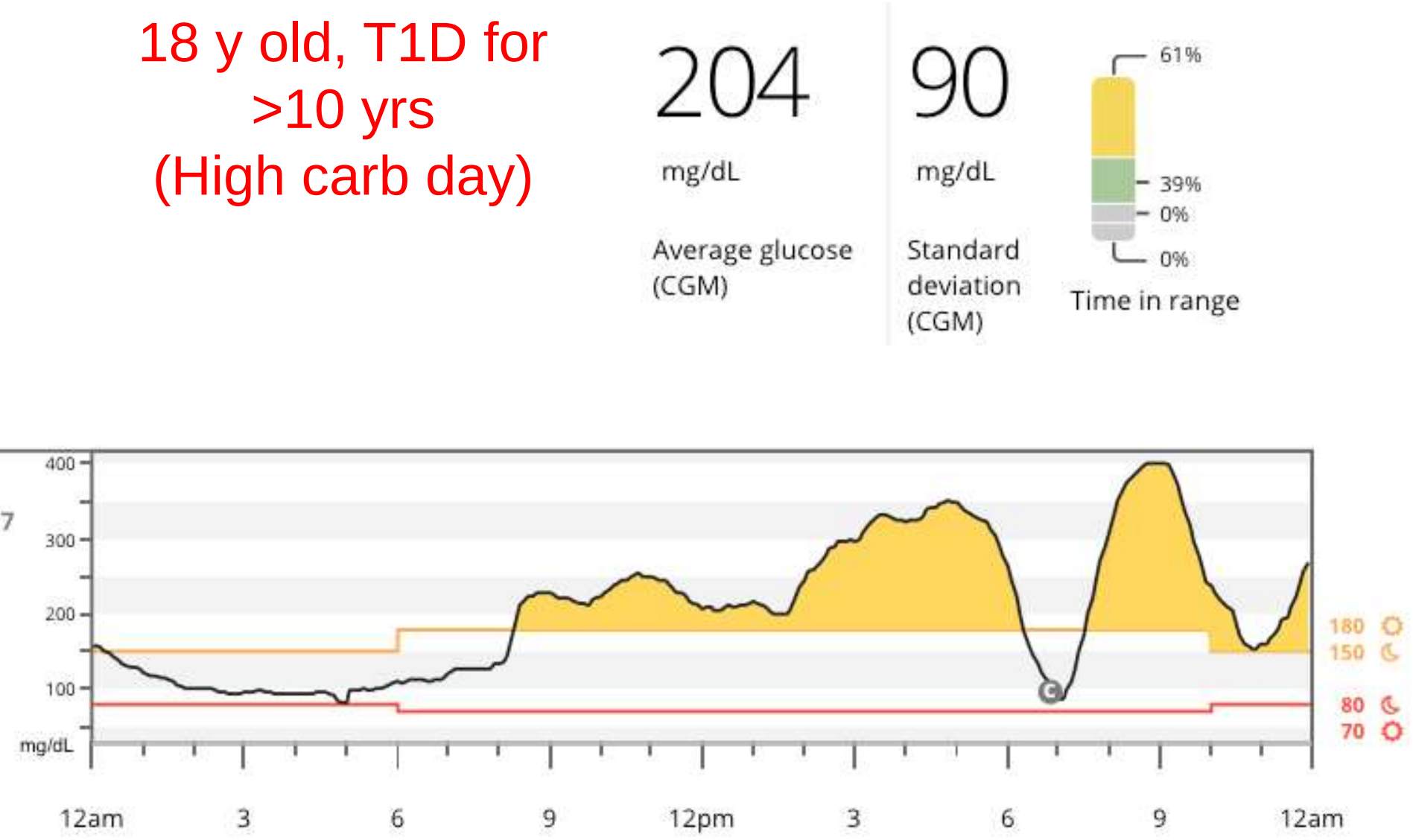
Recommended Carbohydrate Amount per Age of Child

Age	Kcal/day	Breakfast	AM snack*	Lunch	PM snack*	Dinner	evening snack
1-3	1,000	2 (30gCHO)	½ (7-8g CHO)	3 (45g CHO)	½ (7-8g CHO)	3 (45gCHO)	½ (7-8g CHO)
4-8	1,400	3 (45g CHO)	0	3 (45g CHO)	1 (15g CHO)	4 (60g CHO)	½ (7-8g CHO)
Males 9-13	1,700	4 (60g CHO)	0	5 (75 g CHO)	1 (15g CHO)	5 (75g CHO)	1 (15g CHO)
Males 14-18	2,300	5 (75g CHO)	0	7 (105g CHO)	1 (15g CHO)	8 (120g CHO)	1 (15g CHO)
Females 9-13	1,500	4 (60g CHO)	0	5 (75g CHO)	1 (15g CHO)	4 (60g CHO)	1 (15g CHO)
Females 14-18	1,700	4 (60g CHO)	0	5 (75g CHO)	1 (15g CHO)	5 (75g CHO)	1 (15g CHO)

**If the snack is to continue at home, the snack must be at least 2 1/2 - 3 hours prior to the next meal/scheduled BG check.*

1 carbohydrate servings equals 15g of Carbohydrates (CHO)
If a food item is >22 grams carbohydrate it should be rounded to 30 grams carbohydrate.
This is simply a template for starting consistent carbohydrate amounts; it can and should be individualized

18 y old, T1D for >10 yrs (High carb day)



Does macronutrient content influence T1D outcomes?

What are we eating?

What shapes macronutrient content of the foods we eat?

Standards of Medical Care in Diabetes—2014

Limited research exists concerning the ideal amount of fat for individuals with diabetes. The Institute of Medicine has defined an acceptable macronutrient distribution range (AMDR) for all adults for total fat of 20–35% of energy with no tolerable upper intake level defined. This AMDR was based on evidence for CHD risk with a low intake of fat and high intake of carbohydrate, and evidence for increased obesity and CHD with high intake of fat (166). The type of fatty acids consumed is more important than total amount of fat when looking at metabolic goals and risk of CVD (146,167,168).

DIETARY REFERENCE INTAKES

FOR

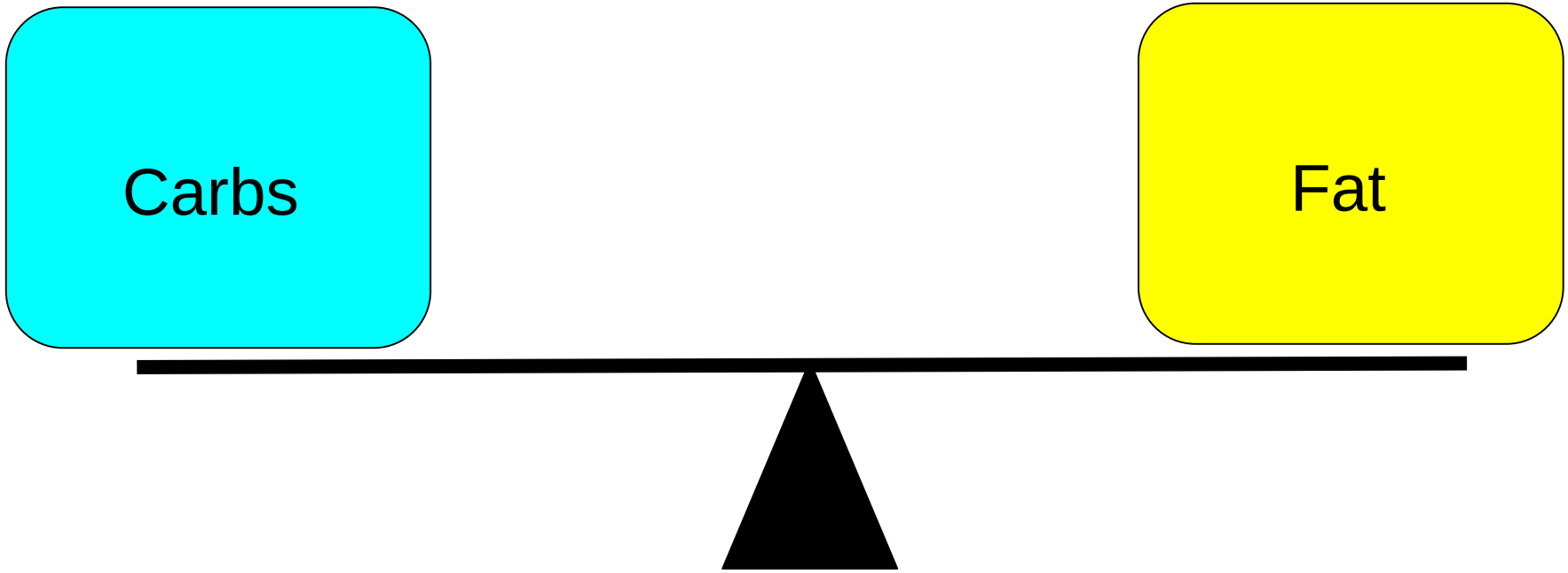
Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids

Panel on Macronutrients, Panel on the Definition of Dietary Fiber, Subcommittee on Upper Reference Levels of Nutrients, Subcommittee on Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes

Food and Nutrition Board

INSTITUTE OF MEDICINE
OF THE NATIONAL ACADEMIES

Acceptable Macronutrient Distribution Ranges (AMDR)



Acceptable Macronutrient Distribution Ranges (AMDR)

↓ High Density Lipoproteins (HDL)

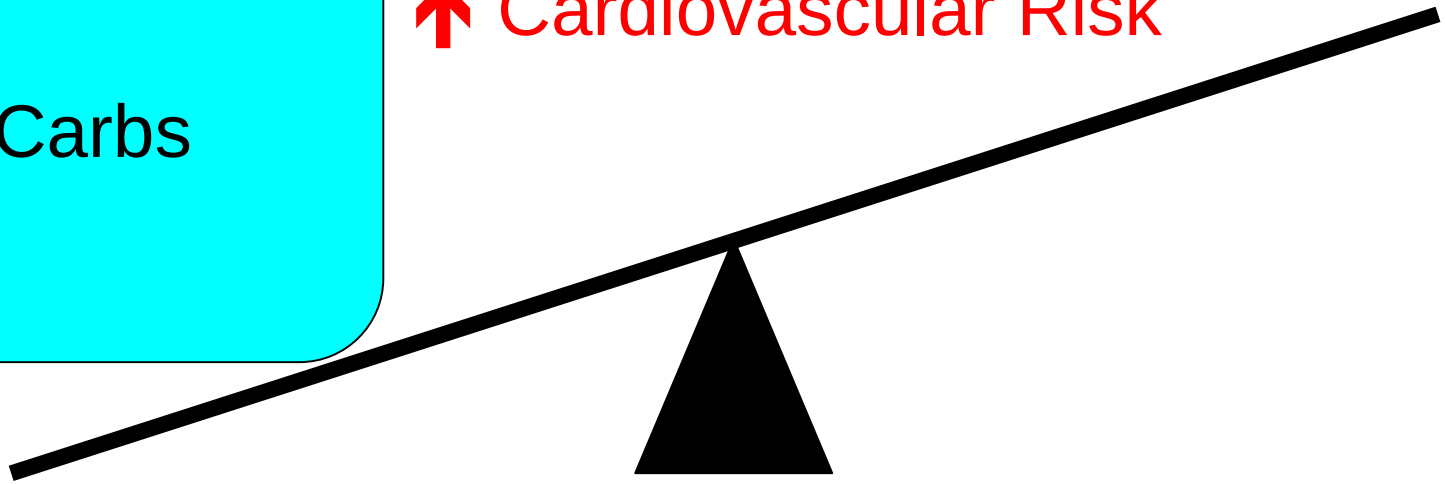
↑ Triglycerides (TG)

↑ LDL Particles

↑ Cardiovascular Risk

↓Fat

↑Carbs



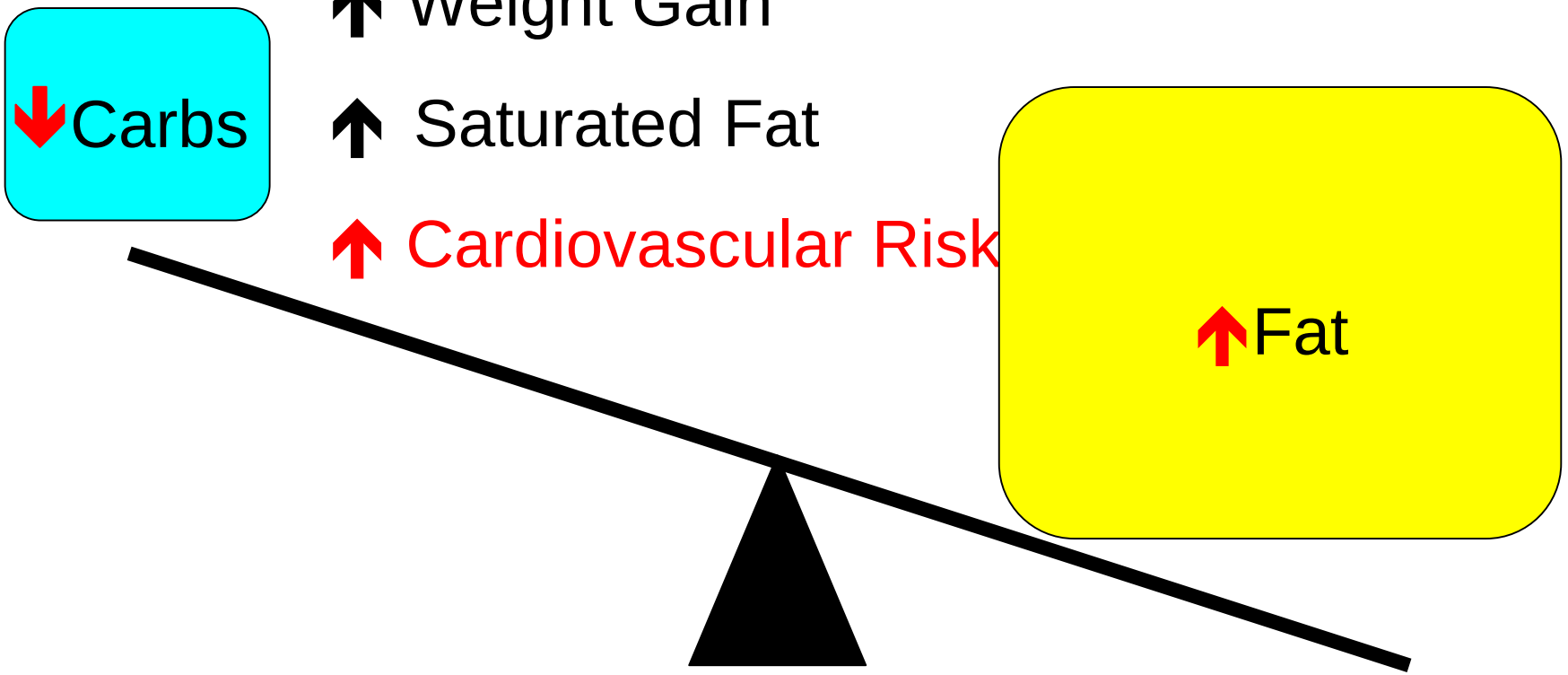
Acceptable Macronutrient Distribution Ranges (AMDR)

↑ Dietary Energy

↑ Weight Gain

↑ Saturated Fat

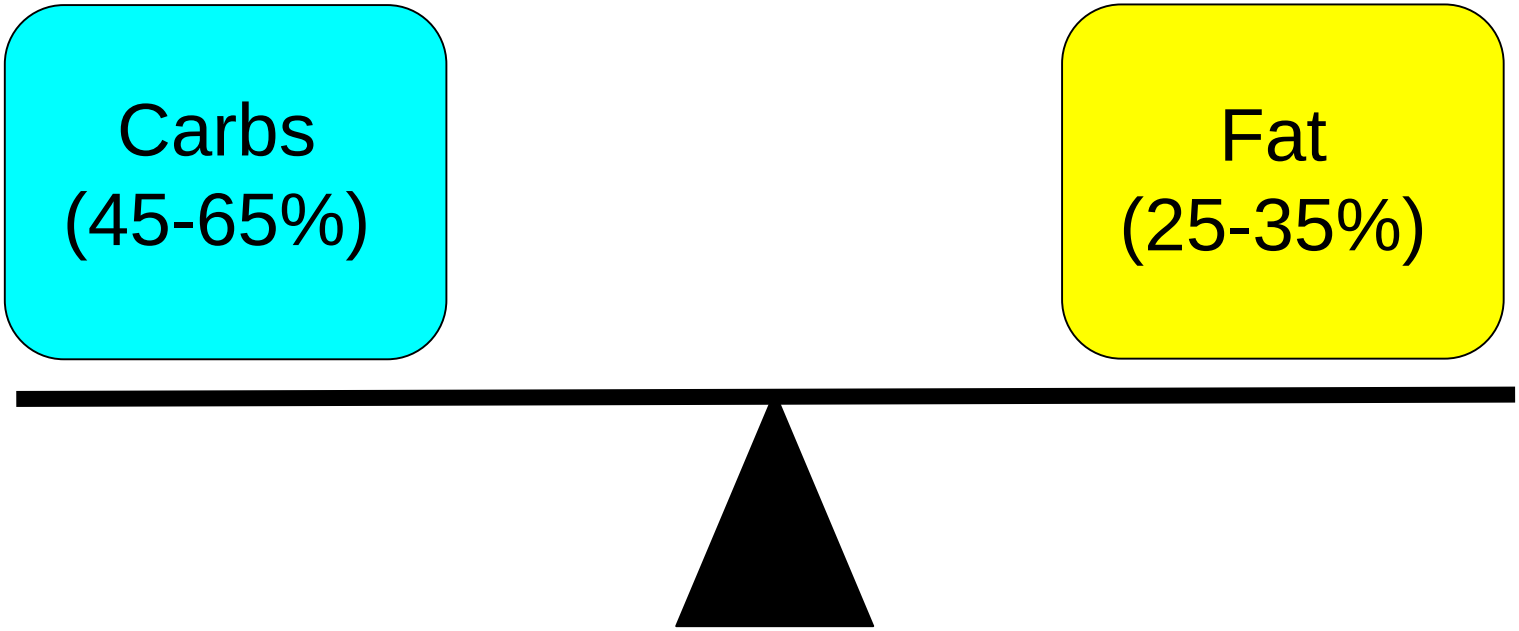
↑ Cardiovascular Risk



Acceptable Macronutrient Distribution Ranges (AMDR)

risk for CHD. Based on the apparent risk for CHD that may occur on low fat diets, and the risk for increased energy intake and therefore obesity with the consumption of high fat diets, the AMDR for fat and carbohydrate is estimated to be 20 to 35 and 45 to 65 percent of energy, respectively, for all adults. By consuming fat and carbohydrate within these ranges, the risk for obesity, as well as for CHD and diabetes, can be kept at a minimum. Furthermore, these ranges allow for sufficient intakes of essential nutrients while keeping the intake of saturated fatty acids at moderate levels.

IOM Acceptable Macronutrient Distribution Ranges (AMDR):



Do dietary fats influence human health?

Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies

Russell J de Souza,[1,2,3,4] Andrew Mente,[1,2,5] Adriana Maroleanu,[2] Adrian I Cozma,[3,4] Vanessa Ha,[1,3,4] Teruko Kishibe,[6] Elizabeth Uleryk,[7] Patrick Budylowski,[4] Holger Schünemann,[1,8] Joseph Beyene,[1,2] Sonia S Anand[1,2,5,8]

: *BMJ* 2015;351:h3978

Saturated fat: not associated with increased death

Outcome	No of studies /comparisons	No of events /participants	Relative risk (95% CI)	P	P_{het}	I^2 (%)
All cause mortality	5/7	14 090/99 906	0.99 (0.91 to 1.09)	0.91	0.17	33
CHD mortality	11/15	2970/101 712	1.15 (0.97 to 1.36)	0.10	<0.001	70
CVD mortality	3/5	3792/90 501	0.97 (0.84 to 1.12)	0.69	0.29	19
CHD total	12/17	6383/267 416	1.06 (0.95 to 1.17)	0.29	0.02	47
Ischemic stroke	12/15	6226/339 090	1.02 (0.90 to 1.15)	0.79	0.002	59
Type 2 diabetes	8/8	8739/237 454	0.95 (0.88 to 1.03)	0.20	0.61	0

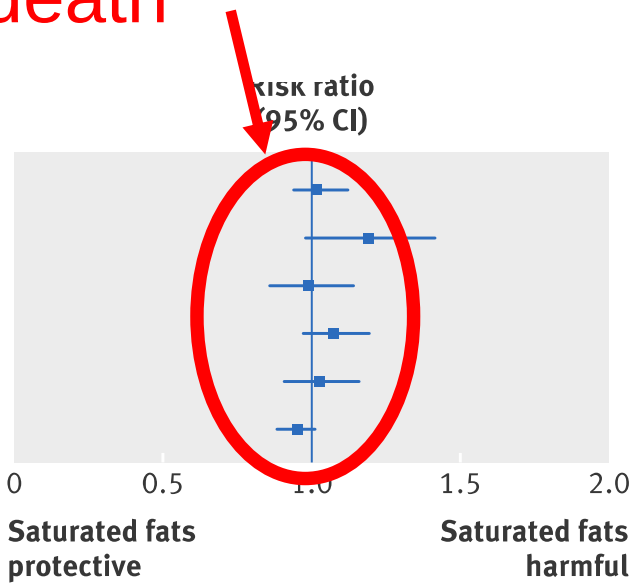


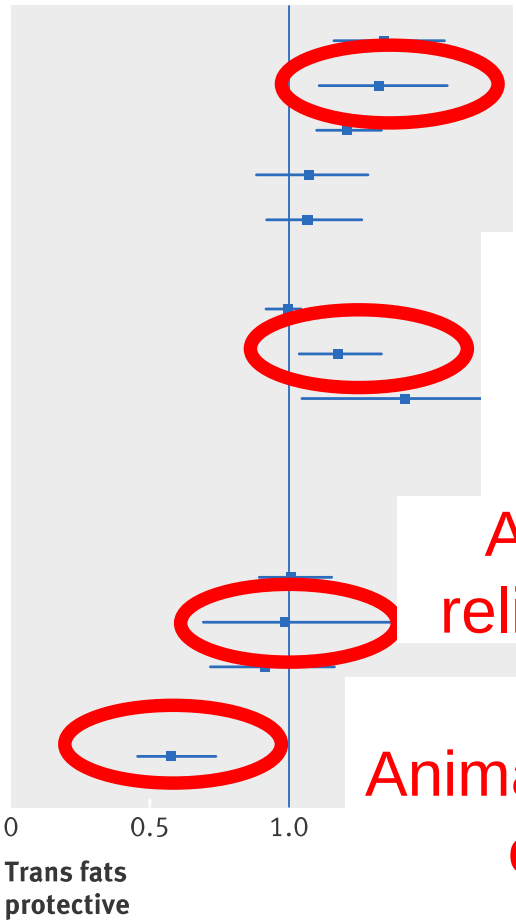
Fig 2 | Summary most adjusted relative risks for saturated fat intake and all cause mortality, CHD mortality, CVD mortality, total CHD, ischemic stroke, and type 2 diabetes. All effect estimates are from random effects analyses. P value is for Z test of no overall association between exposure and outcome; P_{het} is for test of no differences in association measure among studies; I^2 is proportion of total variation in study estimates from heterogeneity rather than sampling error

Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies

Russell J de Souza,[1,2,3,4] Andrew Mente,[1,2,5] Adriana Maroleanu,[2] Adrian I Cozma,[3,4] Vanessa Ha,[1,3,4] Teruko Kishibe,[6] Elizabeth Uleryk,[7] Patrick Budylowski,[4] Holger Schünemann,[1,8] Joseph Beyene,[1,2] Sonia S Anand[1,2,5,8]

: *BMJ* 2015;351:h3978

Outcome	No of studies /comparisons	No of events /participants	Risk ratio (95% CI)	Relative risk (95% CI)	P	P_{het}	I^2 (%)
Total trans fats							
All cause mortality	2/2	2141/20 346					
CHD mortality	5/6	1234/70 864					
CHD total	6/7	4579/145 922					
Ischemic stroke	3/4	1905/190 284					
Type 2 diabetes	6/6	8690/230 135					
Industrial trans fats							
All cause mortality	1/2	11 890/71 464					
CHD mortality	2/2	3018/93 394					
CHD total	2/2	454/69 848					
Ischemic stroke	0	0/0					
Type 2 diabetes	0	0/0					
Ruminant trans fats							
All cause mortality	1/2	11 890/71 464					
CHD mortality	2/3	3018/93 394					
CHD total	3/4	828/73 546		0.93 (0.73 to 1.18)	0.55	0.13	46
Ischemic stroke	0	0/0					
Type 2 diabetes	5/5	1153/12 942					



Trans fats increase risk of CV death

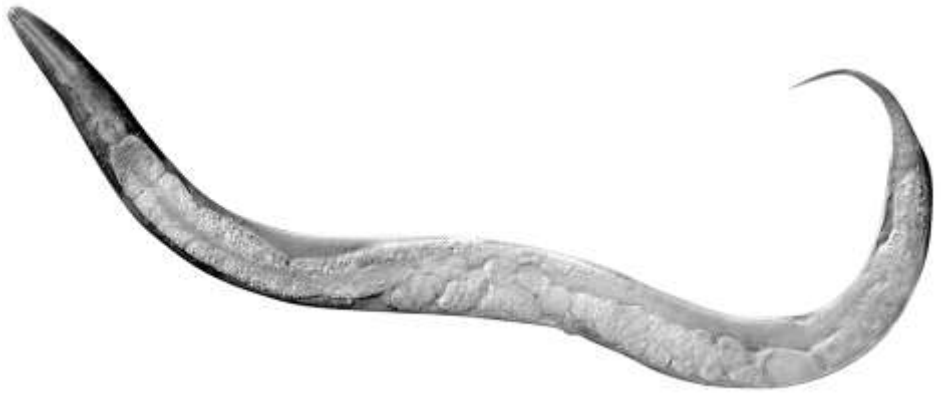
Industrial trans fats (Crisco) increase risk of CV death

Animal trans fats do not reliably influence CV death

Animal trans fats reduce risk of type 2 diabetes!

Is excess insulin signaling toxic in people with diabetes?

What happens when you decrease insulin signaling in model organisms?



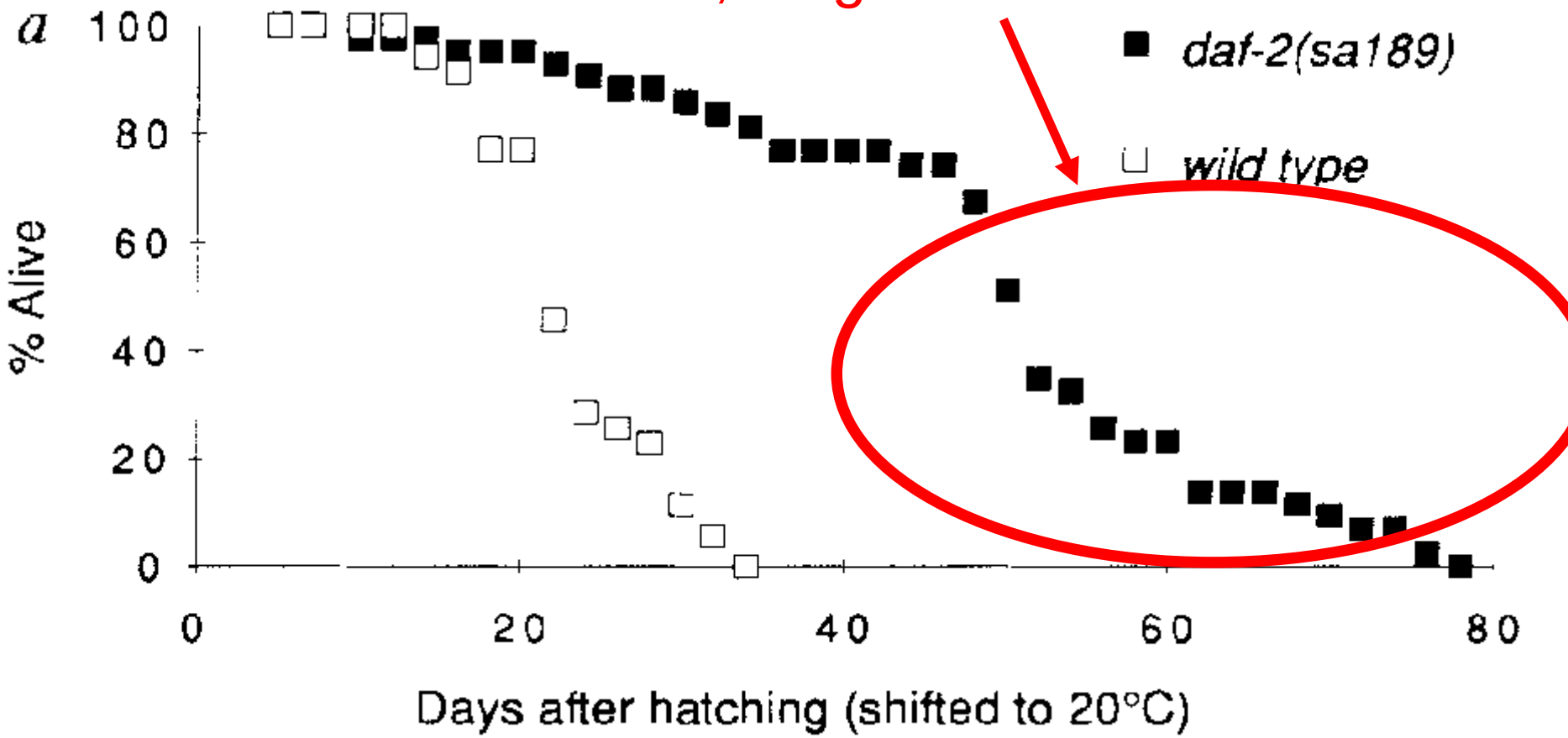
LETTERS TO NATURE

A *C. elegans* mutant that lives twice as long as wild type

Cynthia Kenyon, Jean Chang, Erin Gensch, Adam Rudner & Ramon Tabtiang

NATURE · VOL 366 · 2 DECEMBER 1993

Less insulin, longer life in roundworms



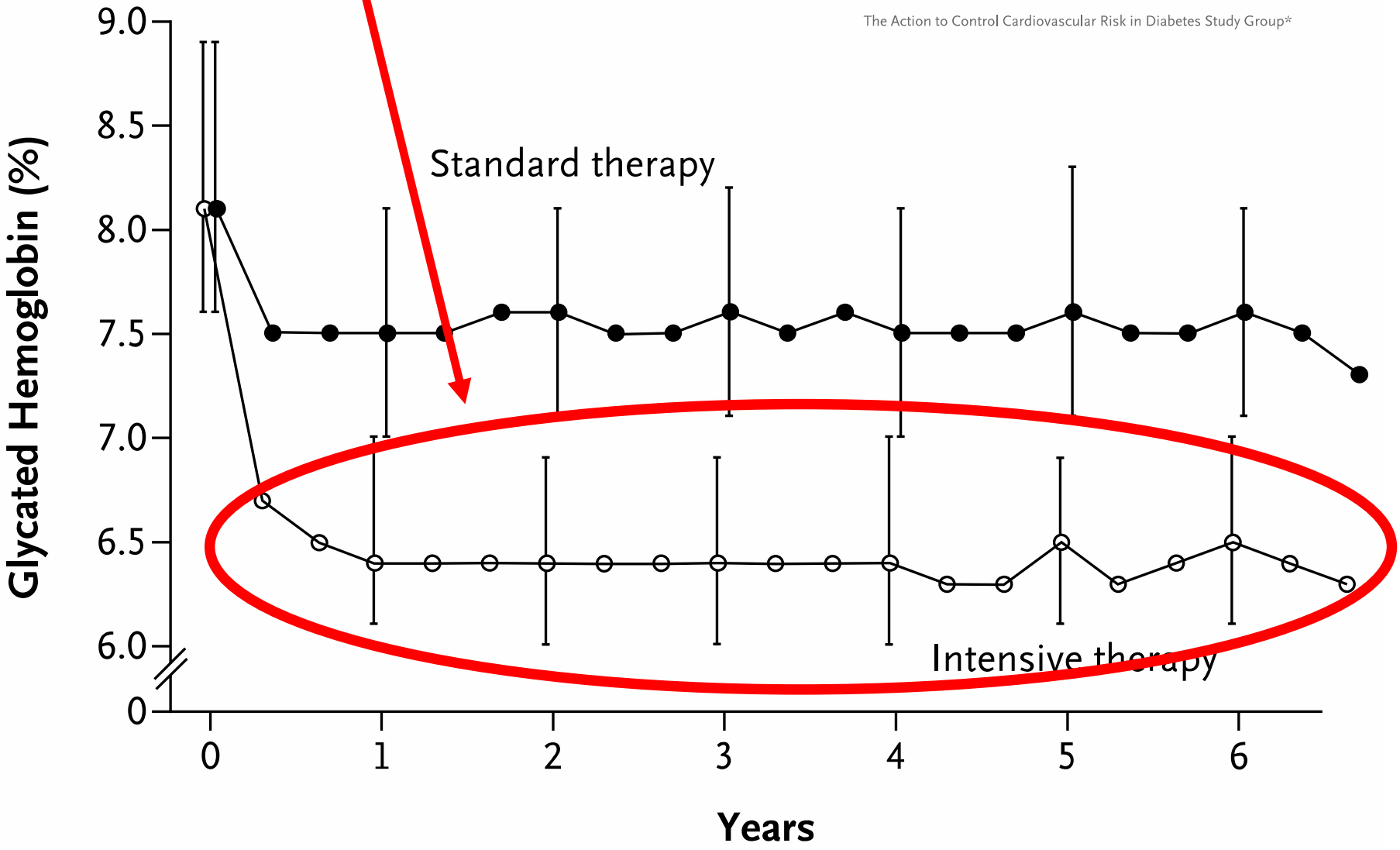
What happens if you increase insulin signaling in people with type 2 diabetes?

The NEW ENGLAND JOURNAL of MEDICINE
ESTABLISHED IN 1812 JUNE 12, 2008 VOL. 358 NO. 24

More insulin in T2D: lower blood sugars

Effects of Intensive Glucose Lowering in Type 2 Diabetes

The Action to Control Cardiovascular Risk in Diabetes Study Group*



Diabetes Care Volume 39, May 2016

More insulin in T2D: more CV death

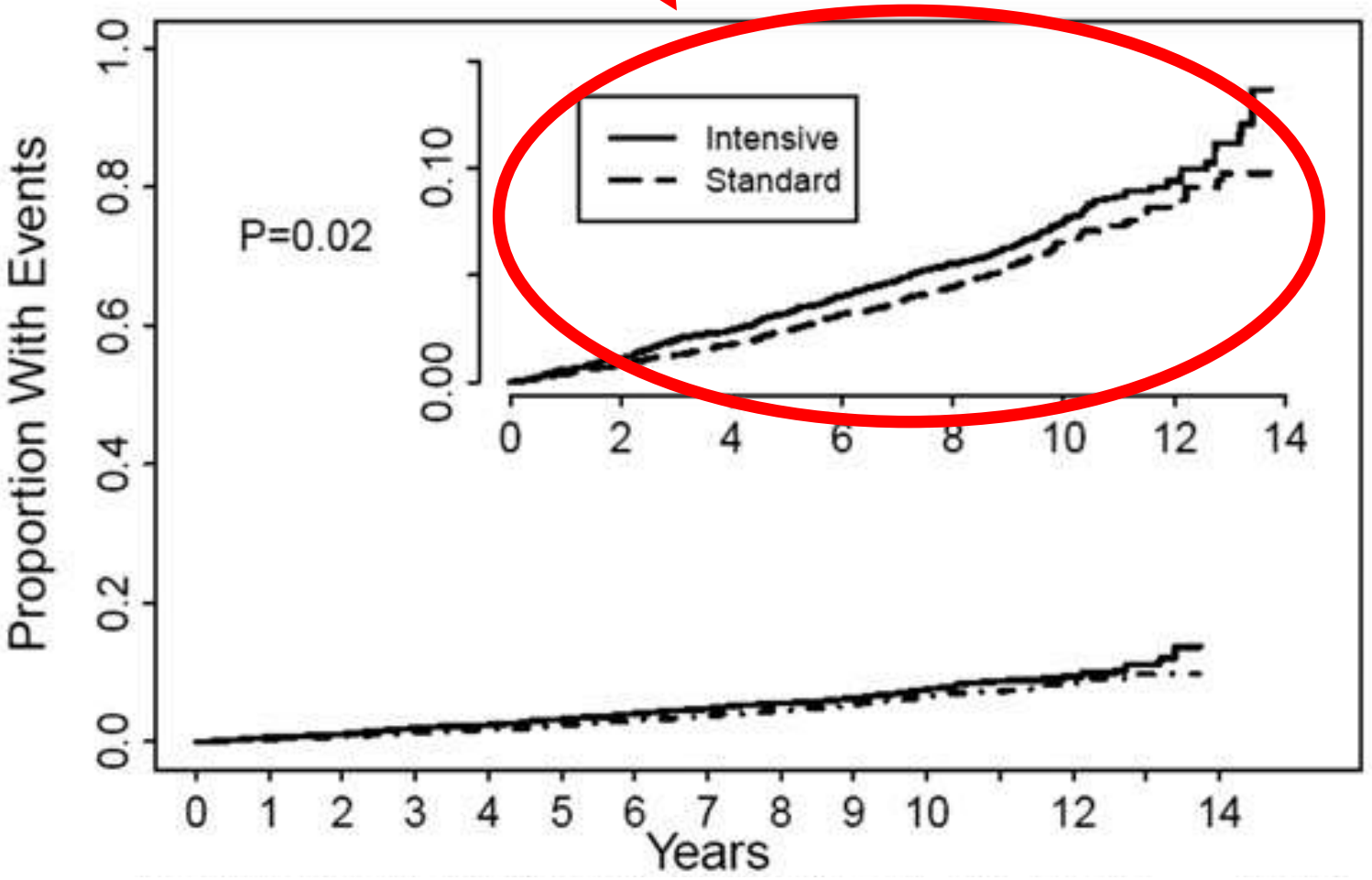
Nine-Year Effects of 3.7 Years of Intensive Glycemic Control on Cardiovascular Outcomes

The ACCORD Study Group*

Diabetes Care 2016;39:701–708 | DOI: 10.2337/dc15-2283

C

CV Death



What happens if you decrease insulin signaling in people with type 2 diabetes?

EMPA reduces insulin in T2D: less CV death

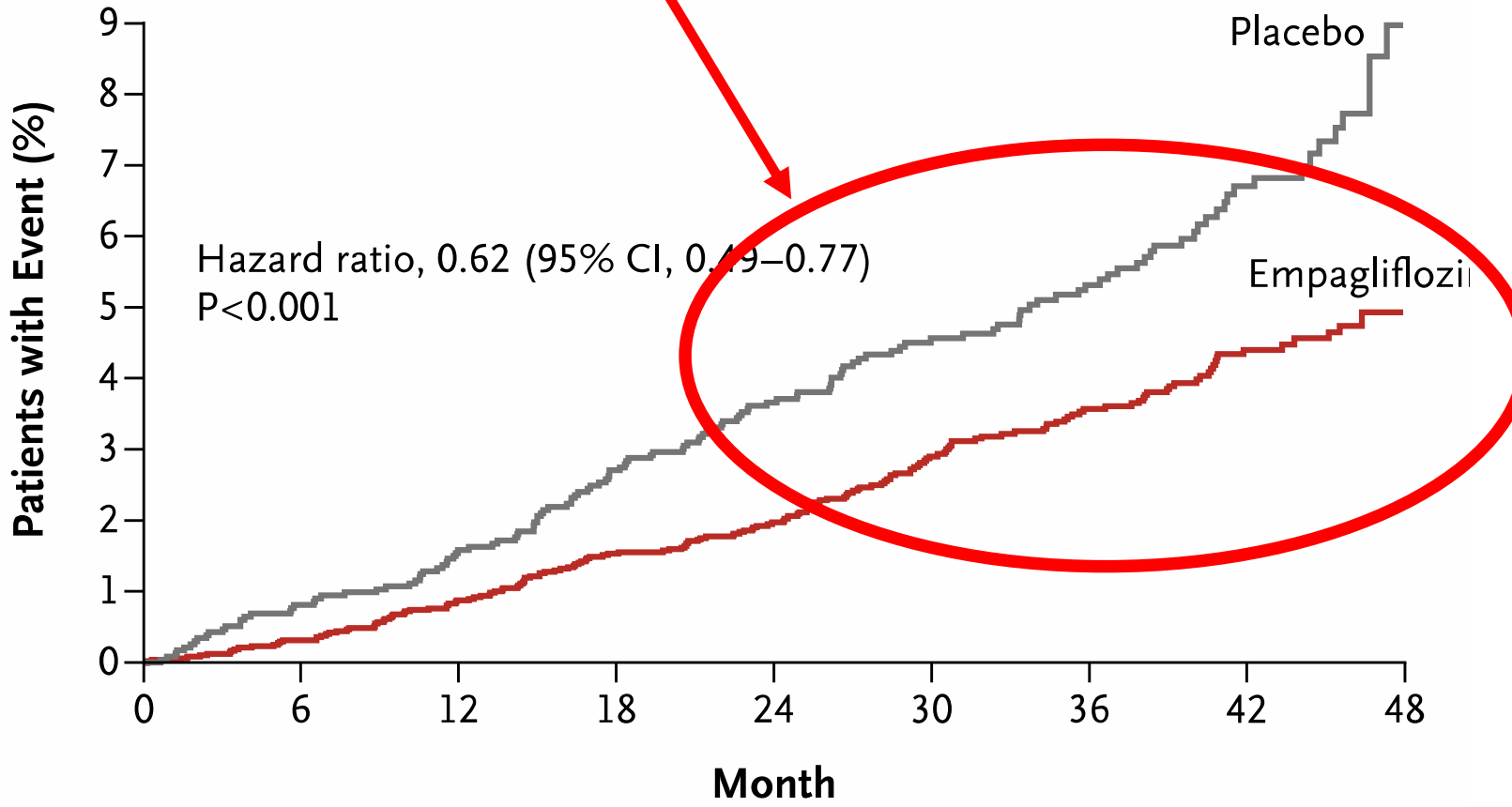
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Empagliflozin, Cardiovascular Outcomes, and Mortality in Type 2 Diabetes

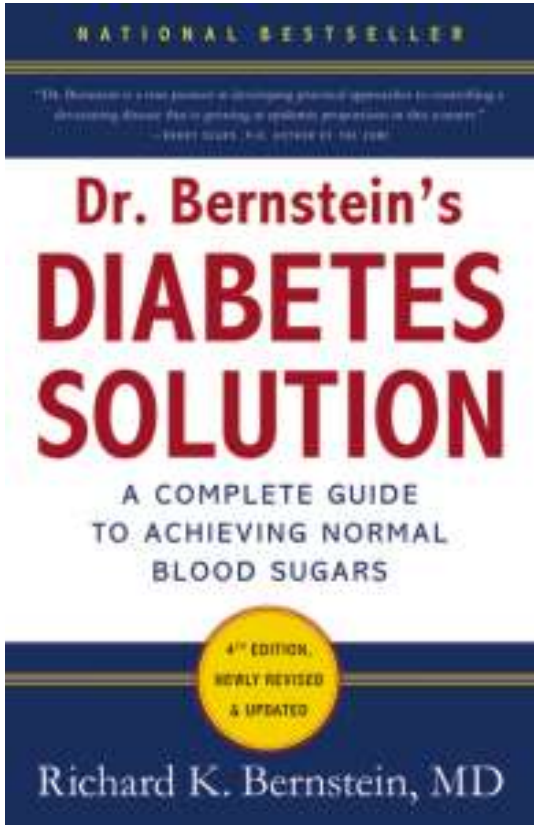
Bernard Zinman, M.D., Christoph Wanner, M.D., John M. Lachin, Sc.D., David Fitchett, M.D., Erich Bluhmki, Ph.D., Stefan Hantel, Ph.D., Michaela Mattheus, Dipl. Biomath., Theresa Devins, Dr.P.H., Odd Erik Johansen, M.D., Ph.D., Hans J. Woerle, M.D., Uli C. Broedl, M.D., and Silvio E. Inzucchi, M.D., for the EMPA-REG OUTCOME Investigators

Death from Cardiovascular Causes



Innovative dietary interventions for treatment of type 1 diabetes?

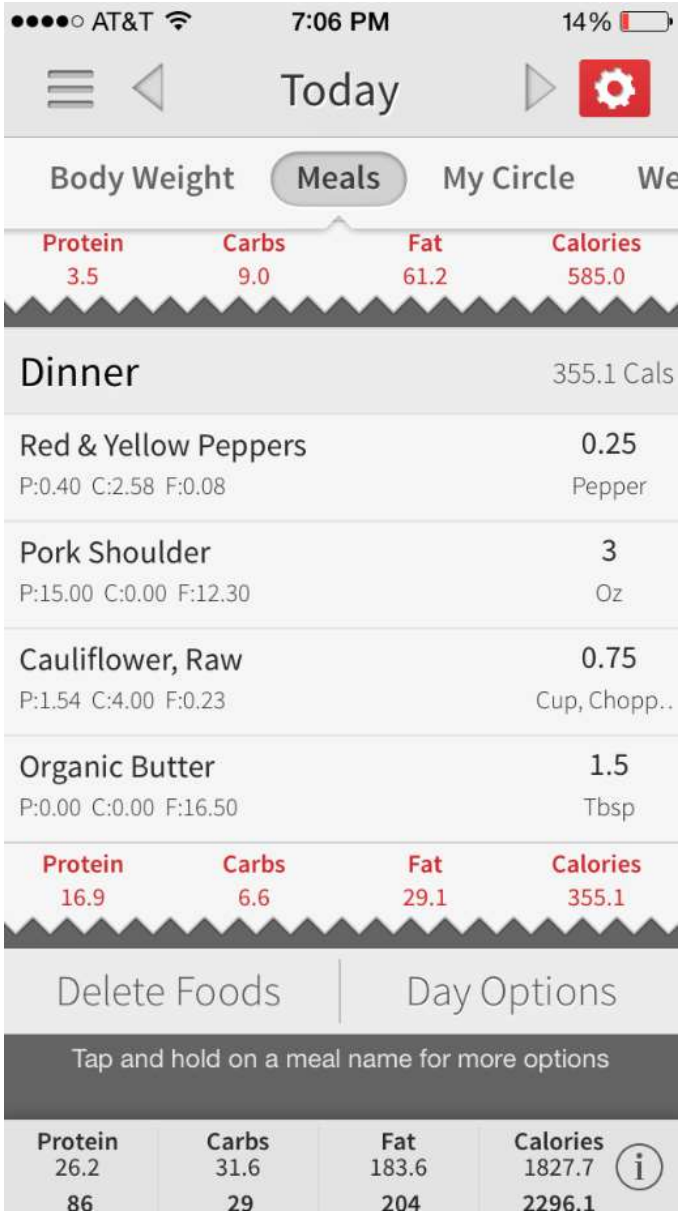
Dr. Bernstein's Amazing Story

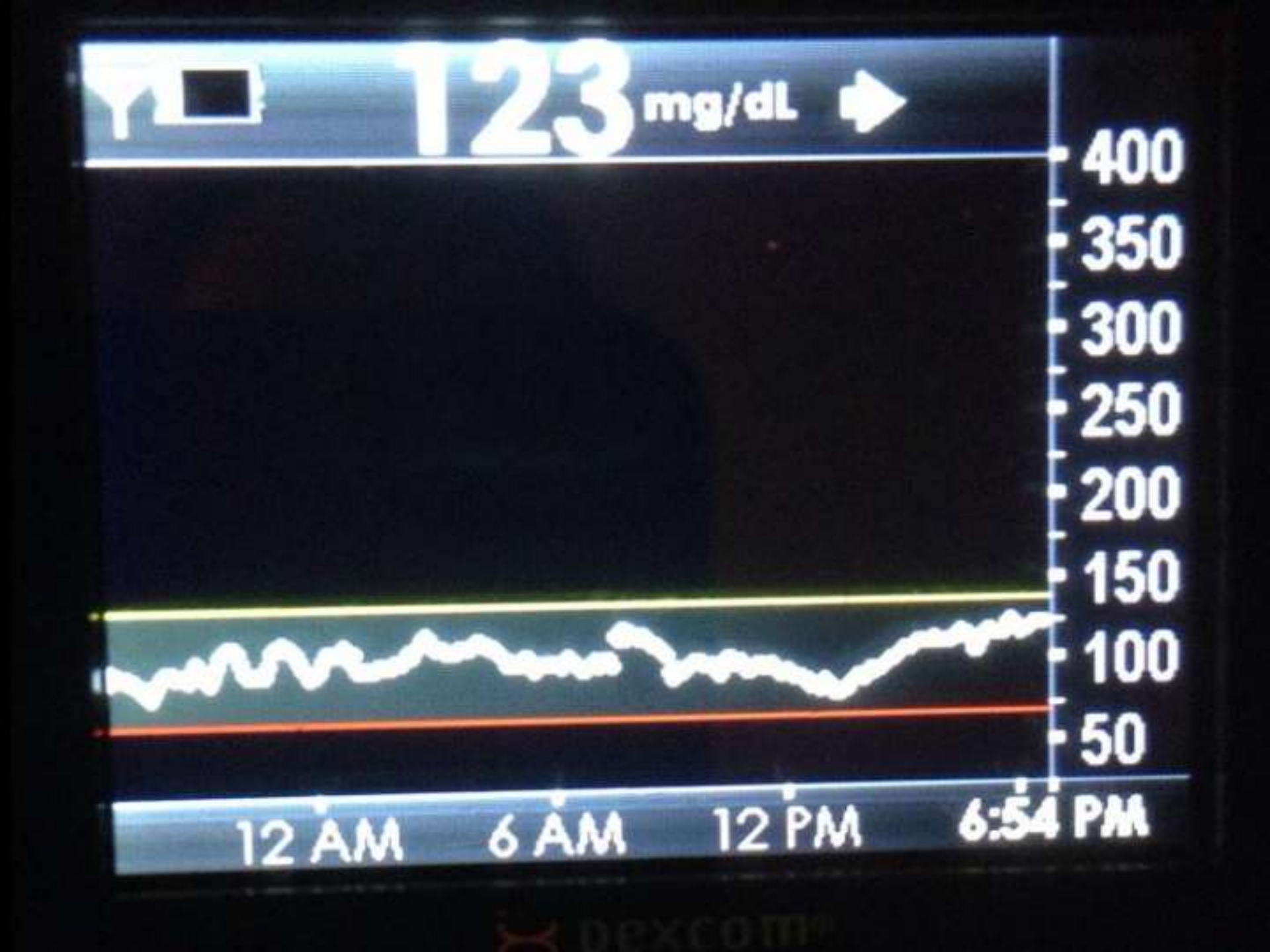


Achieving normal blood sugars for diabetics with the aid of a low carbohydrate diet and exercise is the focus of Dr. Bernstein's Diabetes Solution, and The Diabetes Diet, Dr. Bernstein's Low-Carbohydrate Solution.

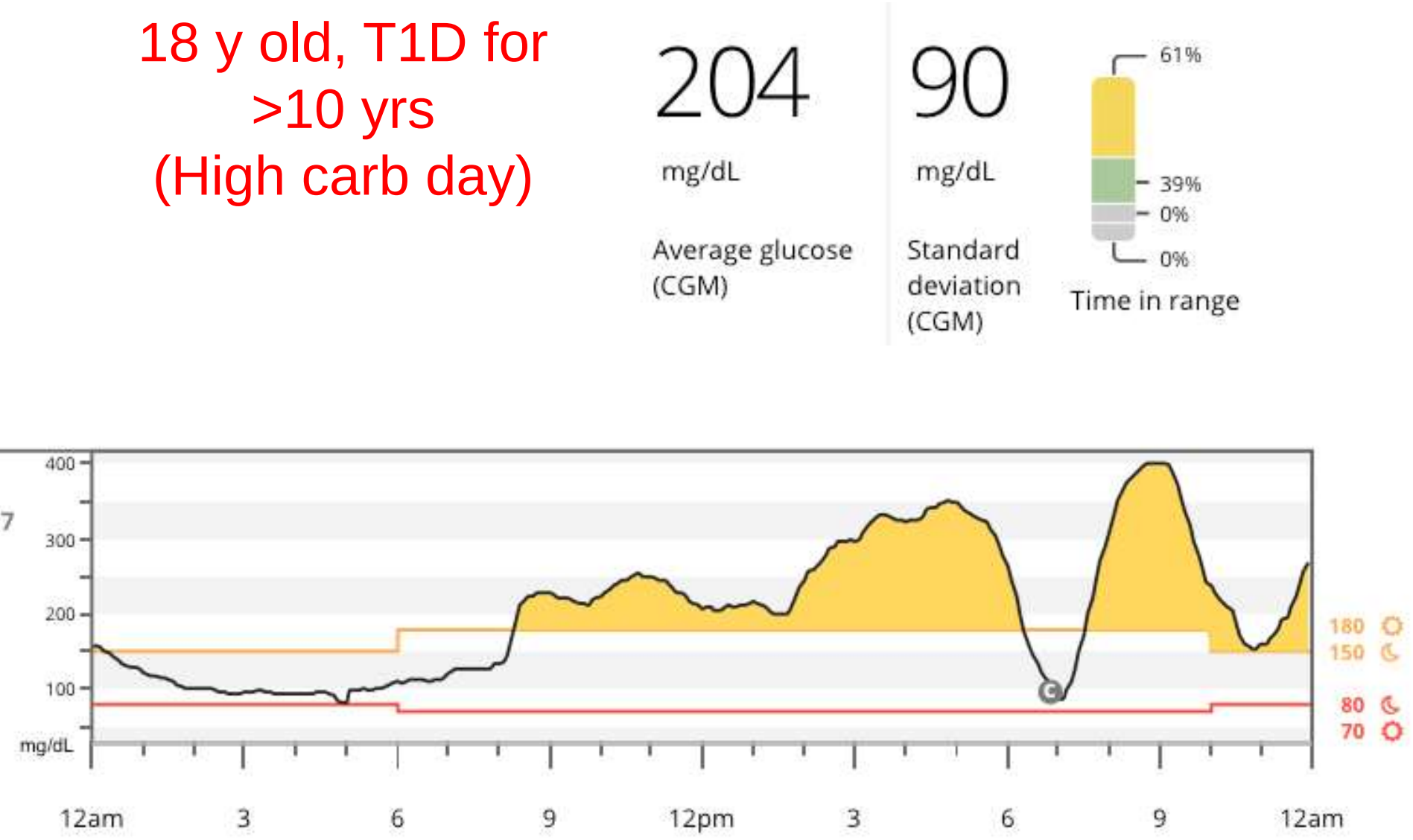
Whether you are newly diagnosed or a lifetime veteran of Type 1 or Type 2 Diabetes, Dr. Bernstein, a renowned and even revolutionary figure in diabetes treatment and diabetic himself, will show you how you could stop the roller-coaster swings in your blood sugars, steady your glucose levels, reduce your insulin intake and enjoy the same level of good health that people without diabetes have.

Books by Richard K. Bernstein, M.D., F.A.C.E., F.A.C.N., F.A.C.C.W.S.


●●●●○ AT&T
7:06 PM
14%
Today
Body Weight
Meals
My Circle
We
Protein
3.5
Carbs
9.0
Fat
61.2
Calories
585.0
Dinner
355.1 Cals
Red & Yellow Peppers
P:0.40 C:2.58 F:0.08
0.25
Pepper
Pork Shoulder
P:15.00 C:0.00 F:12.30
3
Oz
Cauliflower, Raw
P:1.54 C:4.00 F:0.23
0.75
Cup, Chopp..
Organic Butter
P:0.00 C:0.00 F:16.50
1.5
Tbsp
Protein
16.9
Carbs
6.6
Fat
29.1
Calories
355.1
Delete Foods
Day Options
Tap and hold on a meal name for more options
Protein
26.2
86
Carbs
31.6
29
Fat
183.6
204
Calories
1827.7
2296.1


123 mg/dL
400
350
300
250
200
150
100
50
12 AM
6 AM
12 PM
6:54 PM

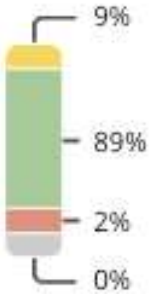
18 y old, T1D for >10 yrs (High carb day)



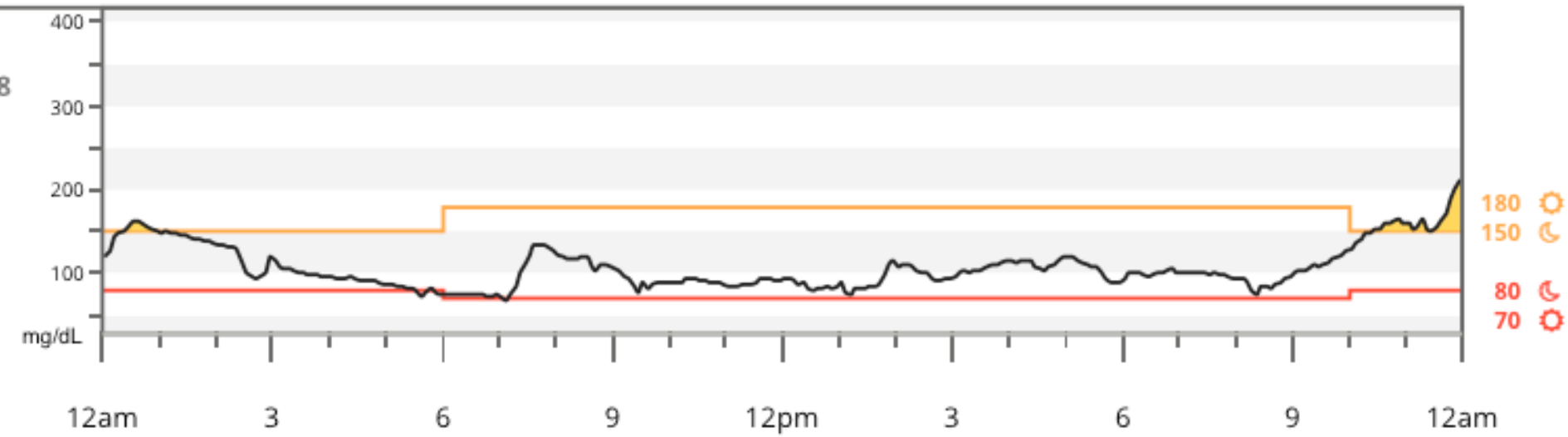
18 y old, T1D for >10 yrs (Low carb day)

107
mg/dL
Average glucose (CGM)

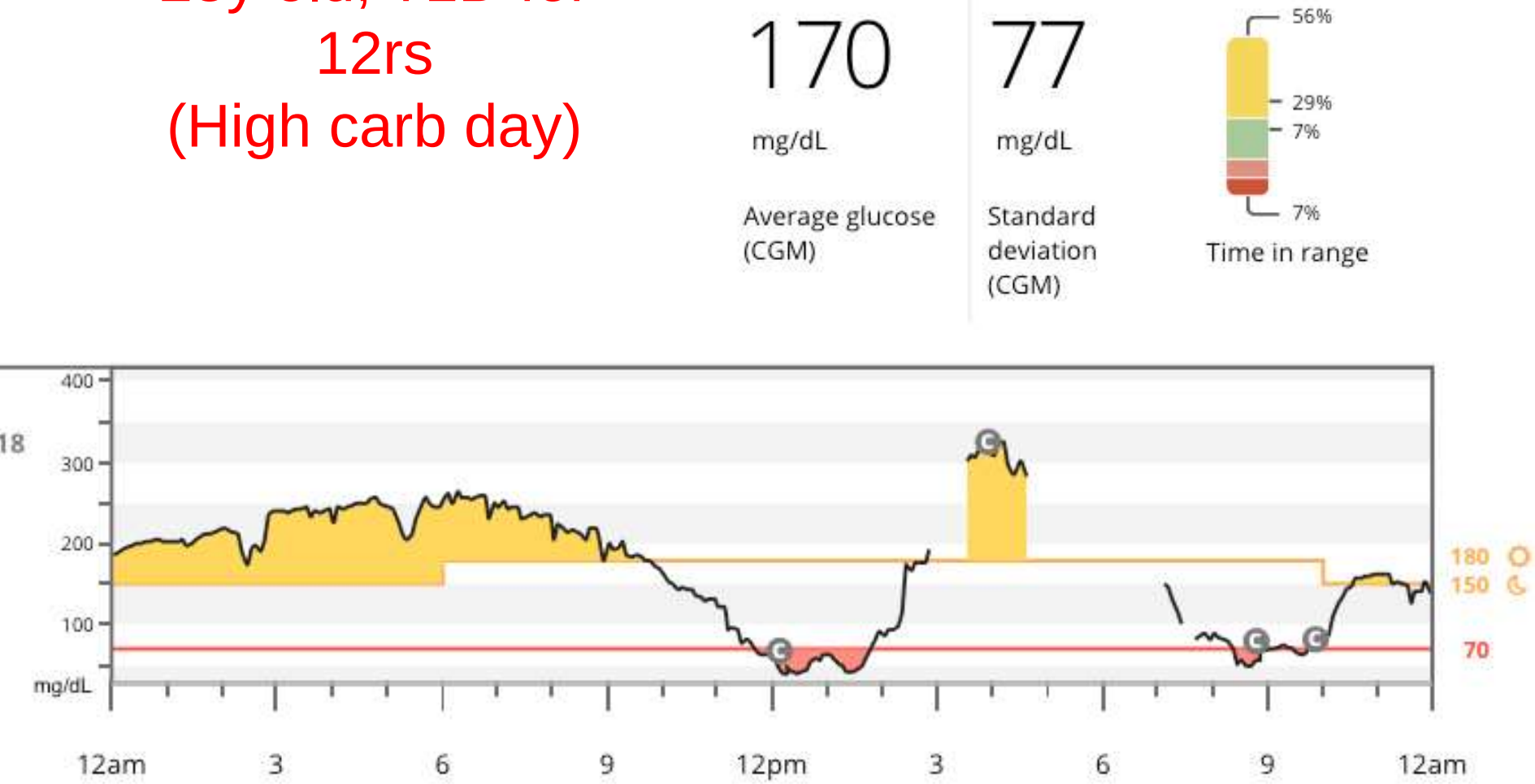
25
mg/dL
Standard deviation (CGM)



Time in range



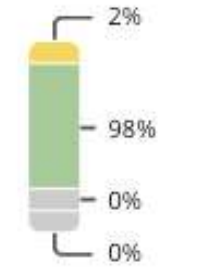
23y old, T1D for 12rs (High carb day)



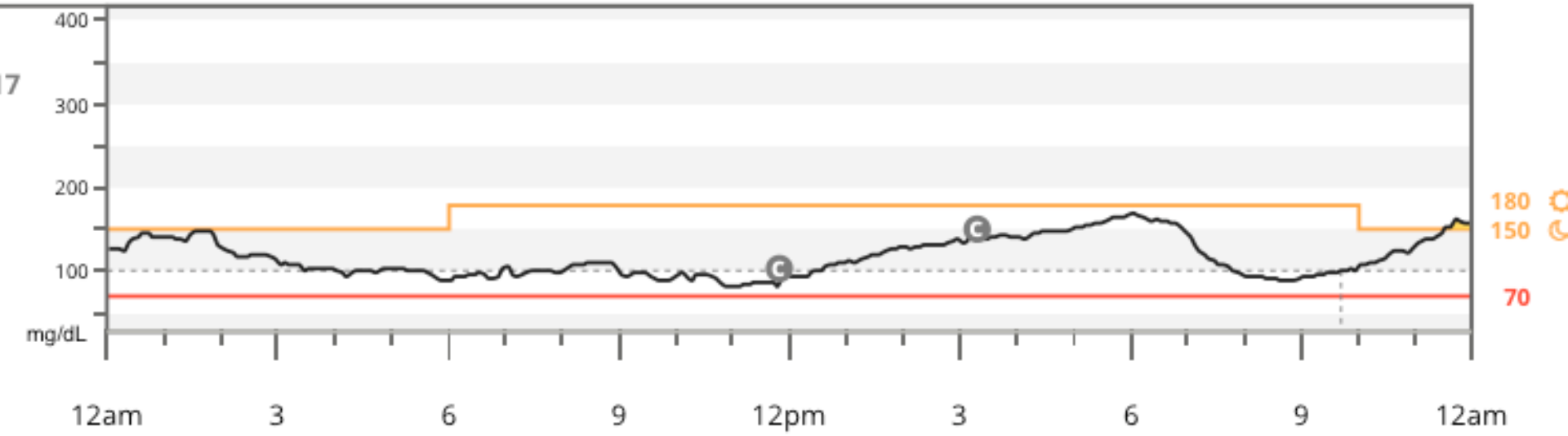
23y old, T1D for 12rs (High carb day)

117
mg/dL
Average glucose (CGM)

23
mg/dL
Standard deviation (CGM)



Time in range



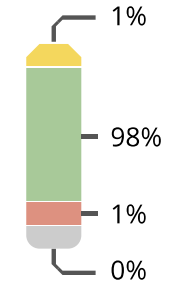
22 y old, T1D for 1 yr.

(Low carb since dx)

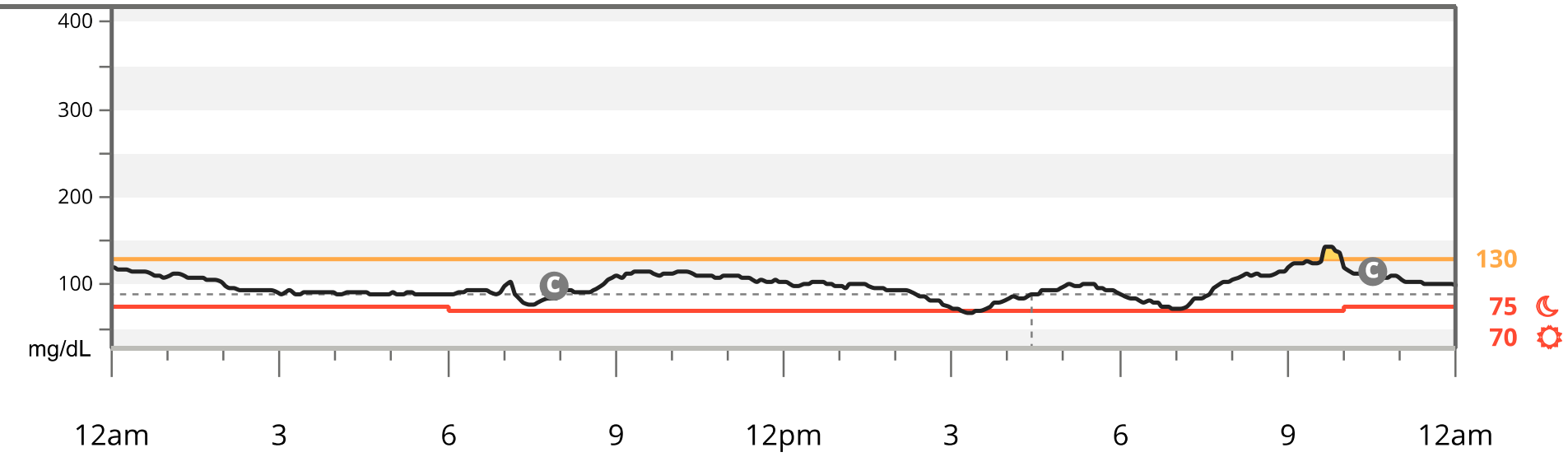
Does LCHF preserve T1D β-cell function?

98
mg/dL
Average glucose (CGM)

13
mg/dL
Standard deviation (CGM)

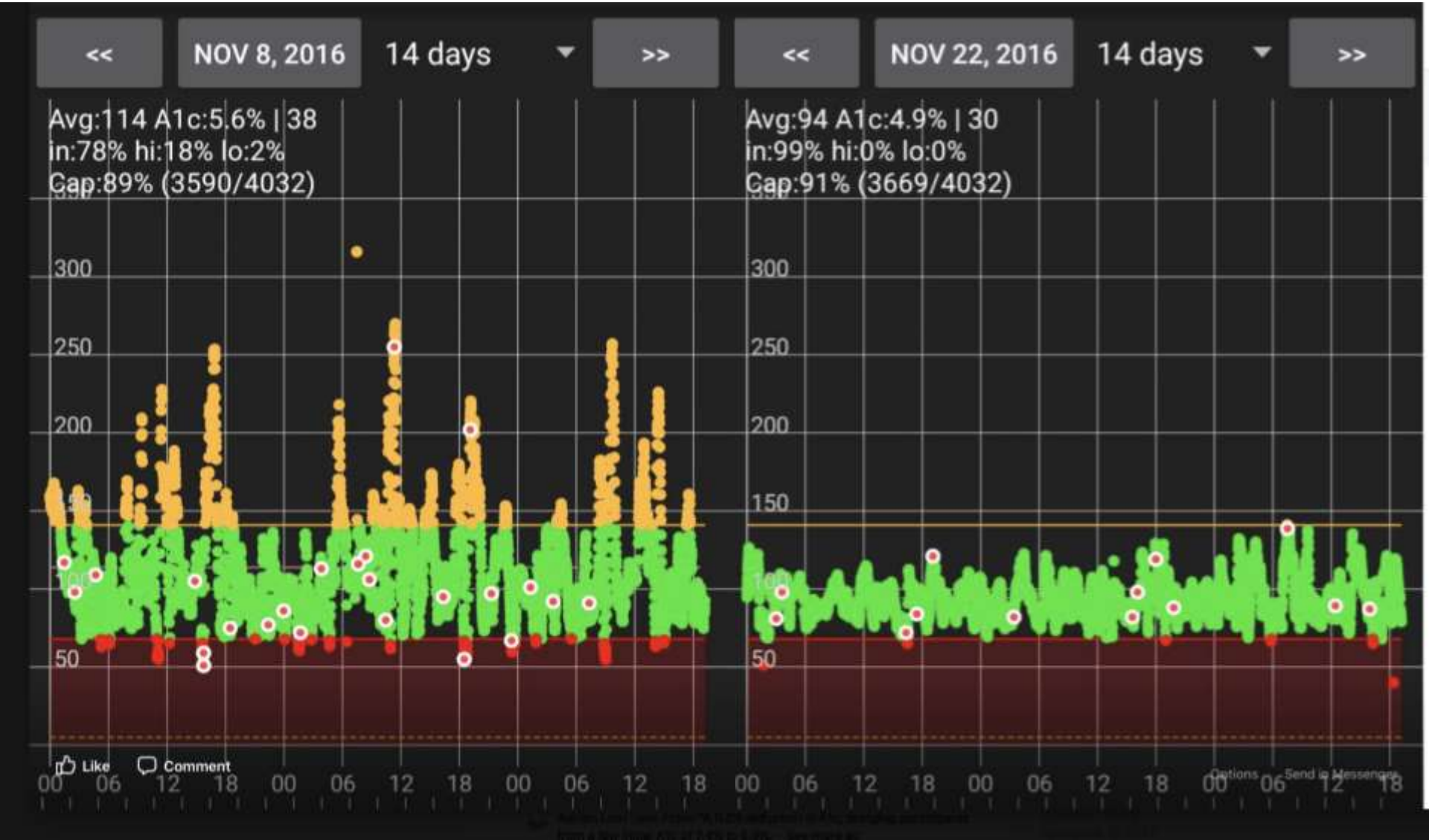


Time in range



Does LCHF synergize with automated insulin delivery?

AdrianLxM is a developer for Android APS, part of the open source DIYPS.org #OpenAPS project



Would low carb high fat nutrition violate ADA consensus guidelines?

Diabetes Care
THE JOURNAL OF CLINICAL AND APPLIED RESEARCH AND EDUCATION
January 2018 Volume 41, Supplement 1

Carbohydrates

Studies examining the ideal amount of carbohydrate intake for people with diabetes are inconclusive, although monitoring carbohydrate intake and considering the blood glucose response to dietary carbohydrate are key for improving postprandial glucose control (70,71). The literature con-

Fats

The ideal amount of dietary fat for individuals with diabetes is controversial. The National Academy of Medicine has defined an acceptable macronutrient distribution for total fat for all adults to be 20–35% of total calorie intake (92). The type of fats consumed is more important than total amount of fat when looking at metabolic goals and CVD risk, and it is recommended that the percentage of total calories from saturated fats should be limited (93–97). Multiple randomized con-

- Data on the ideal total dietary fat content for people with diabetes are inconclusive, so an eating plan emphasizing elements of a Mediterranean-style diet rich in monounsaturated and polyunsaturated fats may be considered to improve glucose metabolism and lower CVD risk and can be an effective alternative to a diet low in total fat but relatively high in carbohydrates. **B**

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Eating Patterns, Macronutrient Distribution, and Meal Planning

Evidence suggests that there is not an ideal percentage of calories from carbohydrate, protein, and fat for all people with diabetes. Therefore, macronutrient distribution should be based on an individualized assessment of current eating patterns, preferences, and metabolic goals. Consider personal preferences (e.g., tradition, culture, religion, health beliefs and goals, economics) as well as metabolic goals when working with individuals to determine the best eating pattern for them (42,51). It is

Resources for #LCHF nutrition and T1D?

BRIGHT SPOTS & LANDMINES

The **DIABETES GUIDE** *I Wish Someone Had Handed Me*

ADAM BROWN

Foreword by Kelly L. Close

What we don't know about LCHF and T1D:

- Disease burden
- Somatic growth in children
- Glucose control
- How to operationalize LCHF at scale.
- Which low carb dietary strategy?
- Synergy with other therapies including closed loop and other adjuvant therapies
- Hypoglycemia, exercise, body weight, etc..
- Health care cost
- Lipid metabolism (association in between cholesterol hyper-responder and T1D? What is the impact of SQ insulin (vs. portal) upon lipid homeostasis? Similarly, what is the impact of SQ insulin on CV disease?
- Diabetes complications (micro- & macrovascular)
- Survival

Why is our knowledge base so poor about about LCHF and T1D?

- False hope around cure
- Communication barriers between adults with T1D and stakeholders (including parents of children with T1D)
- Lack of NIH funding for innovative nutrition and T1D
- Lack of private funding for T1D health and wellness
- Impact of carb industry on nutrition guidelines and KOLs for people with T1D
- Incorrect assumptions (e.g. people with T1D need the “best nutrition” and must follow IOM AMDRs).

What can be done to implement LCHF strategies for people with type 1 diabetes:

- Get out, unite, communicate, de-stigmatize LCHF for the general population
- Advocate for better nutrition standards (Nutrition Coalition!)
- Advocate for more funding and better studies
- Build culture
- T1D peer volunteers in clinics
- Health care providers who live with T1D
- Diabetes camp volunteers (med students undergraduate students clinical fellows)
- Apprenticeships with master clinicians
- Psychology collaborations
- Philanthropy
- Technology: CGM, Ketone measurement, etc.
- Advocacy, mentoring, stewardship, local reinforcement