Please indicate how much you agree or disagree with each of the following statements:

1. Fat people are less physically attractive than thin

2 I would never date a fat

3. On average, fat people are lazier than thin people

4. Fat people only have themselves to blame for their weight

5. It is disgusting when a fat person wears a bathing suit at the beach

Do you think that stereotypes about obese persons could affect the way that they are treated by health care professionals? If yes, in what ways do you think their care may be compromised? If no, why not?

Ending the Blame Game:
A Physiologic Approach to Treating Obesity

JESSE BITTMAN MD FRCPC ABIM

Disclosures

Speaker fees from Novo Nortis and Bausch

I practice in obesity and hypertension clinics

Acknowledgment

I would like to thank Dr. Zentner for help preparing slides and her mentorship

My patients, from whom I learn every day

Objectives

Review the relevance of obesity to kidney disease

Review the pathophysiology of obesity

Review evidence based strategies for obesity treatment

NOTE: this presentation will focus on patients with CKD not on dialysis

But First, A Story

40 y/o M,

On Medicine ward, after ICU admission for Sepsis, AKI requiring dialysis.

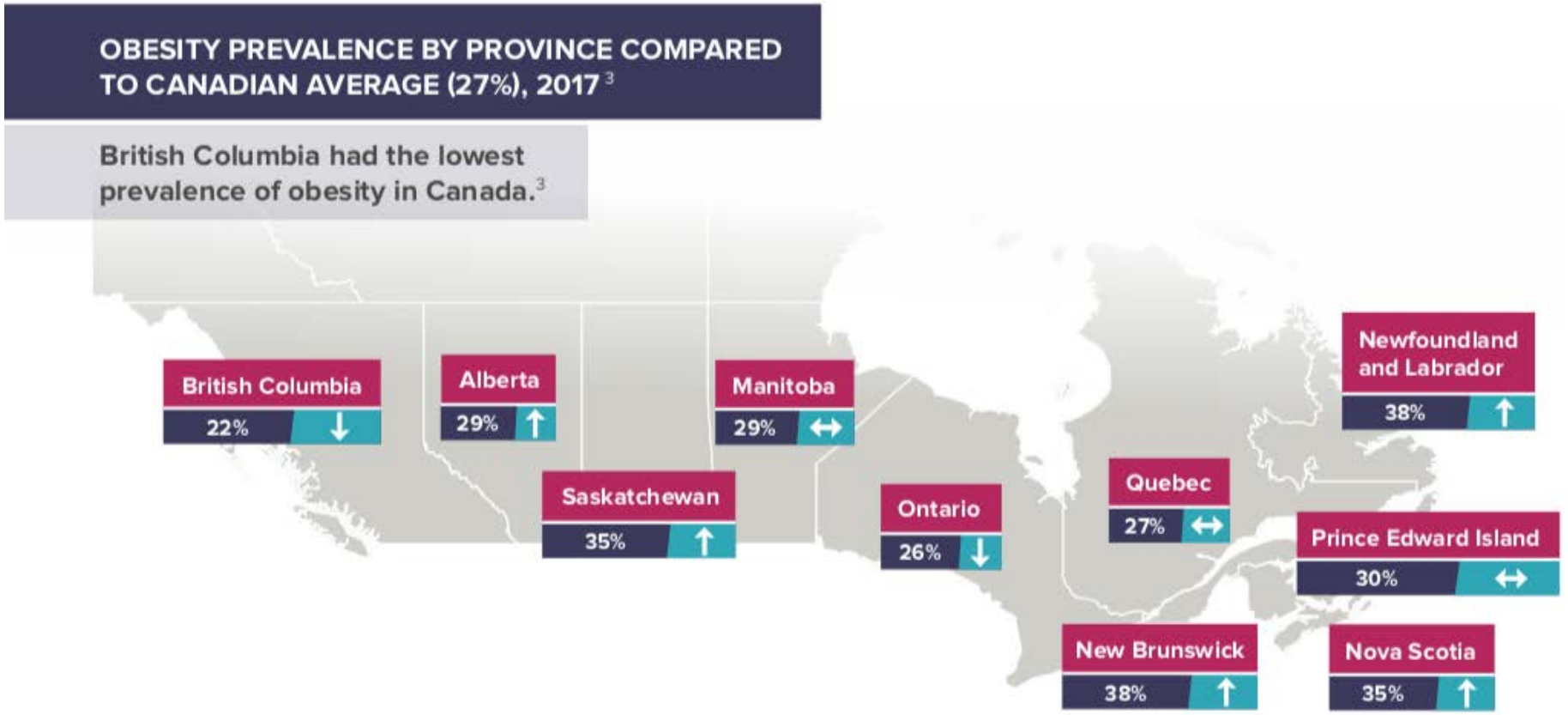
Now kidneys recovered, eGFR 50.

BMI 65, diabetes, hypertension, OSA

He tells me he is a dieting expert, he has lost hundreds of pounds…

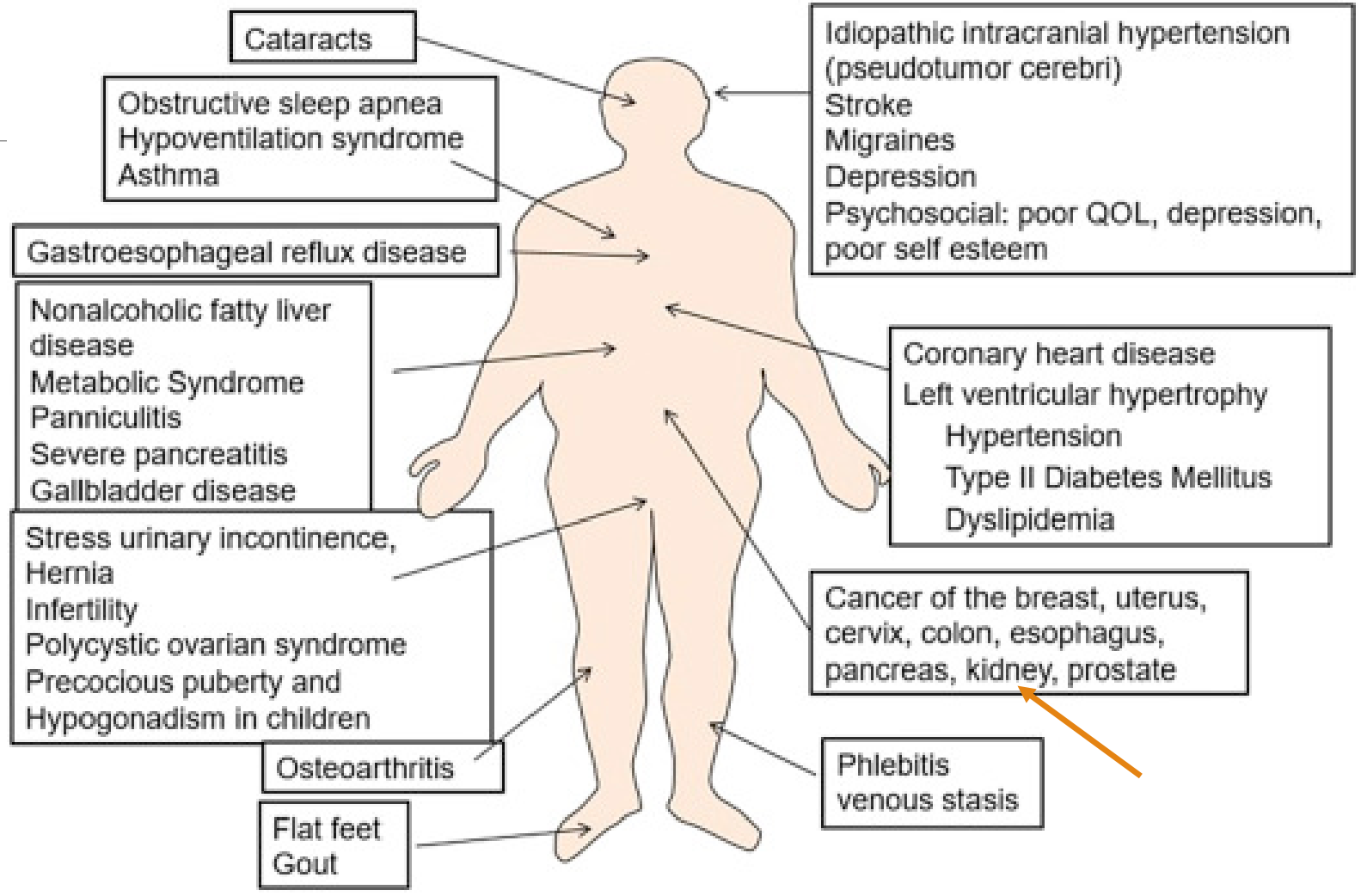
Did his weight contribute to renal deterioration and now what, if anything, can I do…

Adapted from Statistics Canada, Obesity in Canadian Adults, 2016 and 2017, October 24, 2018.



Adapted from Statistics Canada, Obesity in Canadian Adults, 2016 and 2017, October 24, 2018.

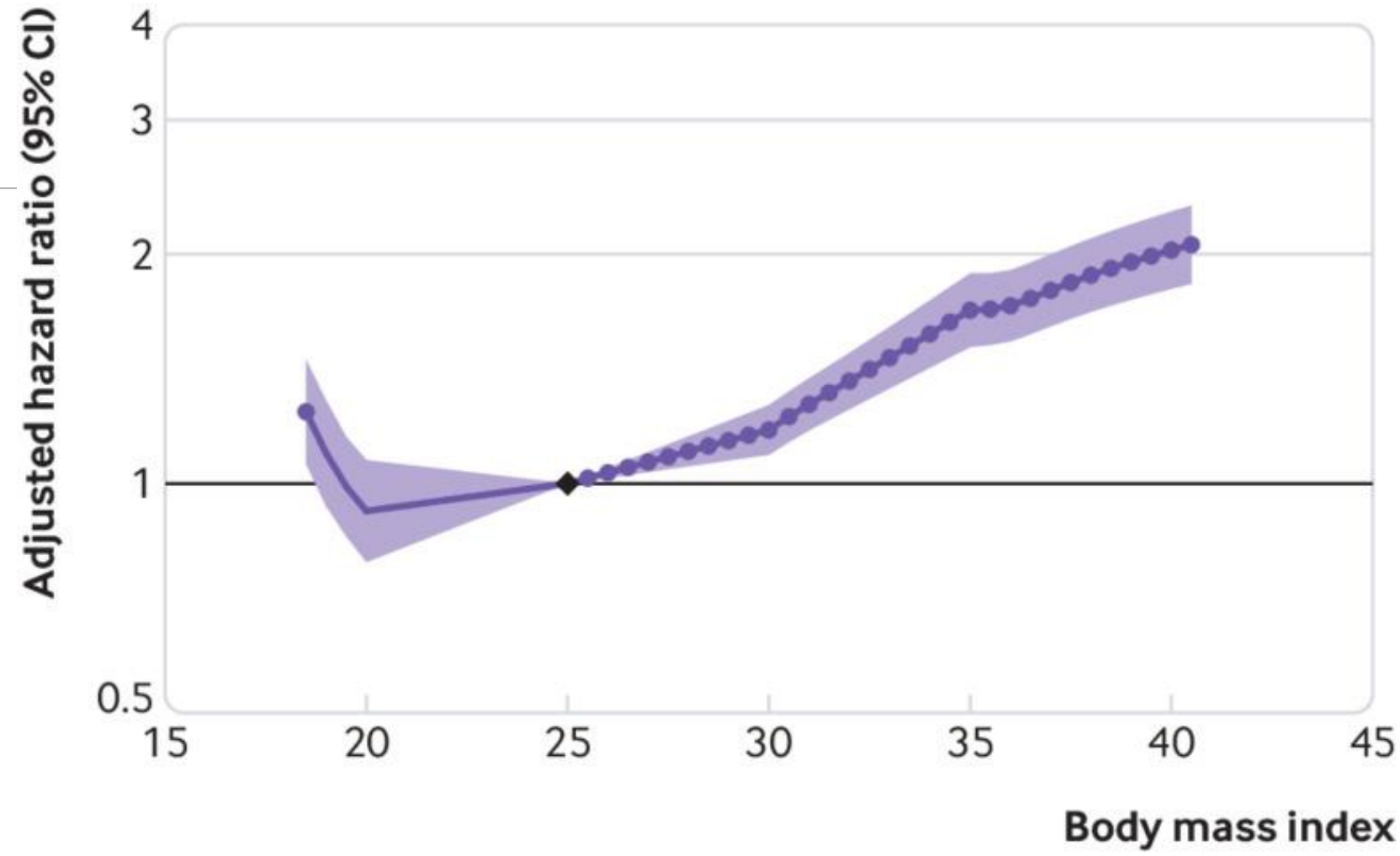
Comorbidities associated with obesity.



Obesity as a Disease
Upadhyay, Jagriti, MD, Medical Clinics of North America, Volume 102, Issue 1, 13-33

BMI and risk of decline in GFR --General Populations

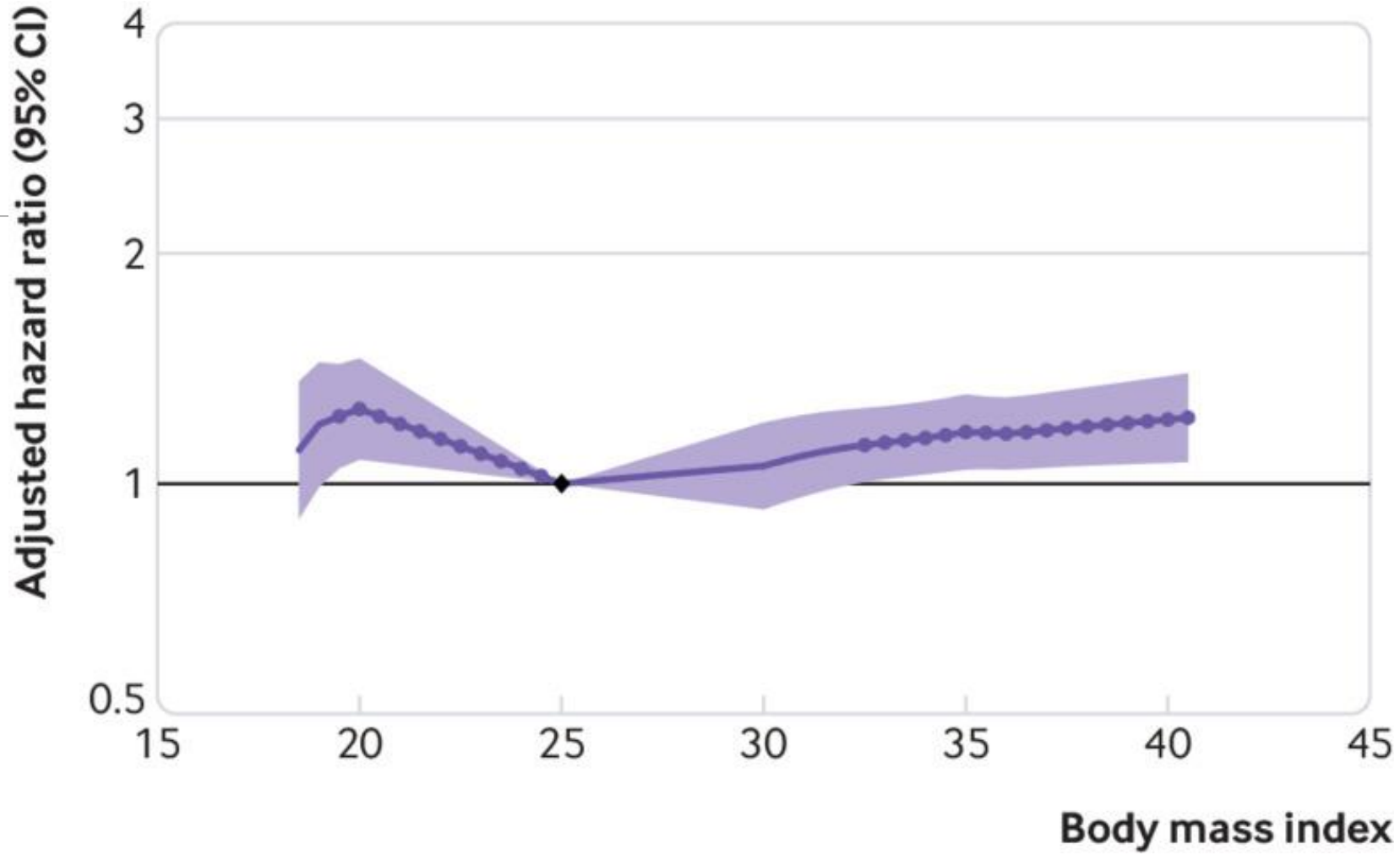
Association between body mass index and risk of decline in glomerular filtration rate in general population cohorts, as shown by meta-analysed hazard ratios and 95% confidence intervals related to body mass index



Alex R Chang et al. BMJ 2019;364:bmj.k5301

BMI and risk of decline in GFR --CKD cohorts

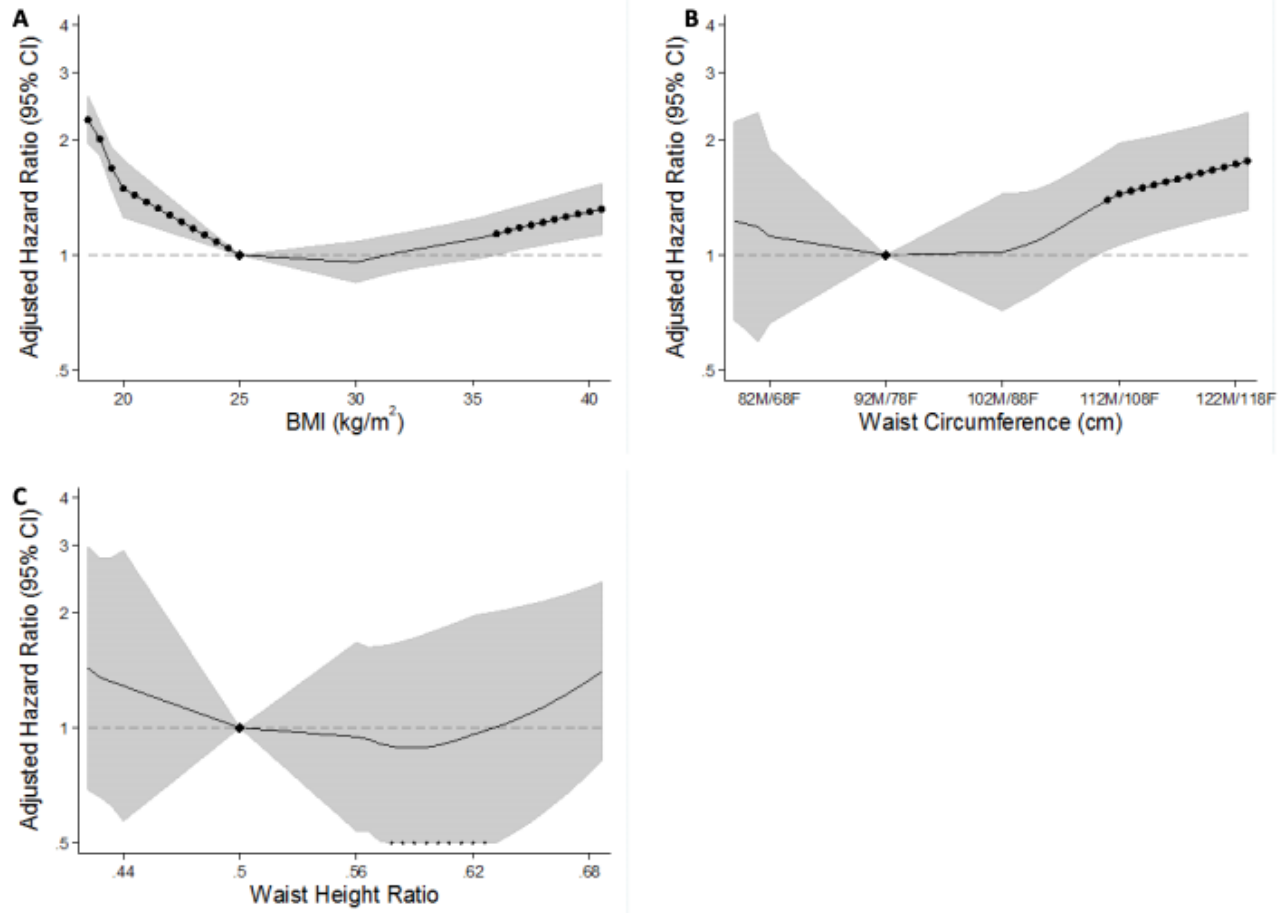
Association of body mass index with risk of decline in glomerular filtration rate in cohorts with chronic kidney disease, as shown by meta-analysed hazard ratios and 95% confidence interval related to body mass index, modelled by linear splines with knots at body mass indices 20, 25, 30, and 35.



Alex R Chang et al. BMJ 2019;364:bmj.k5301

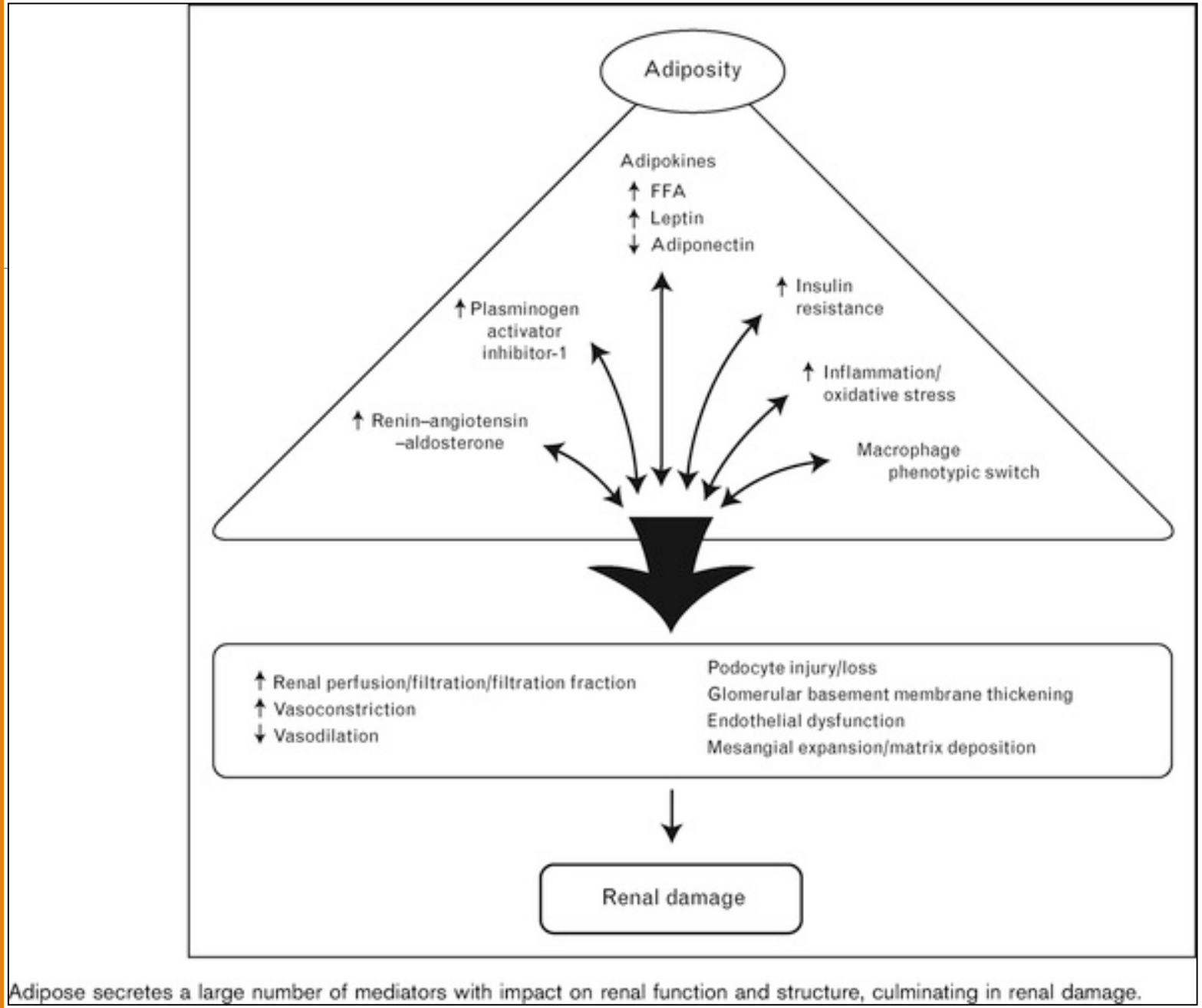
BMI and risk of Mortality --CKD cohorts

eFigure 13. Association of Adiposity Measures with All-Cause Mortality in CKD Cohorts



Alex R Chang et al. BMJ 2019;364:bmj.k5301

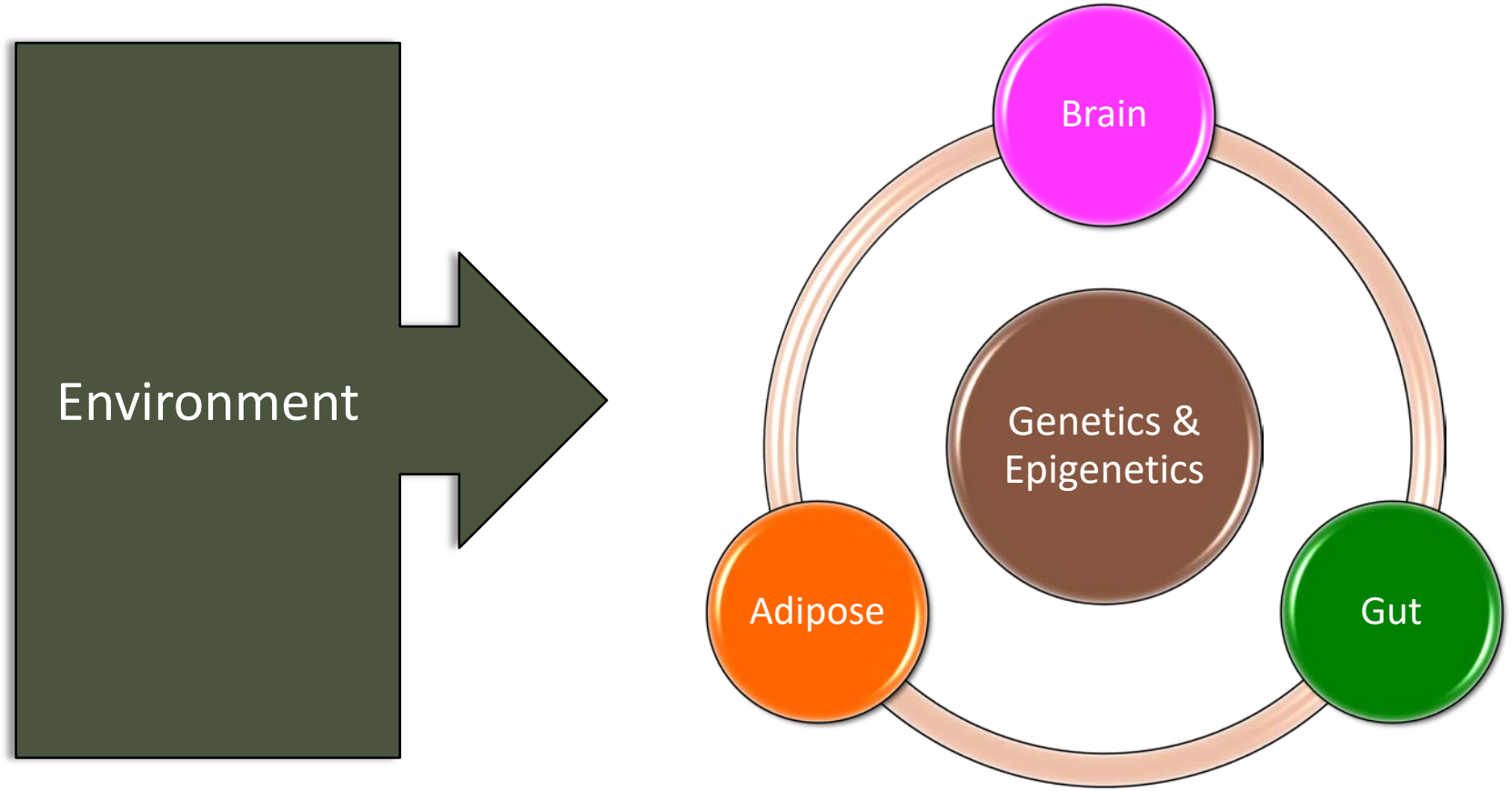
Mechanisms of Obesity-Related Renal Disease



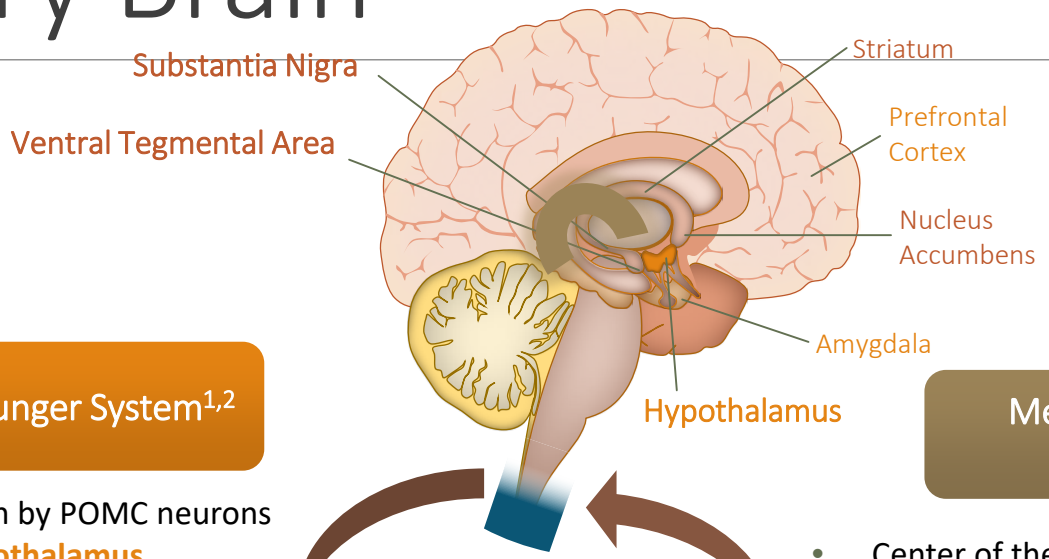
Adipose secretes a large number of mediators with impact on renal function and structure, culminating in renal damage.

Hunley, Tracy; Ma, Li-Jun; Kon, Valentina **Scope and mechanisms of obesity-related renal disease.** Current Opinion in Nephrology & Hypertension. 19(3):227-234, May 2010.

Pathophysiology of Obesity



The Hungry Brain



Hypothalamic Hunger System[1,2]

- Primarily driven by POMC neurons within the **hypothalamus**
- Detection and integration of energy state information (e.g., hunger, fullness) based on peripheral signals
- Altered function in obesity (e.g., leptin resistance)

Bidirectional Interaction to Control Food Intake

Mesolimbic Reward System[3]

- Center of the brain that mediates **motivation**, **reward**, and **desire** associated with activities needed for survival
- **Dopamine** and **opioid** signaling known to play important roles

Mesolimbic Reward System can typically override the Hypothalamic Hunger System,

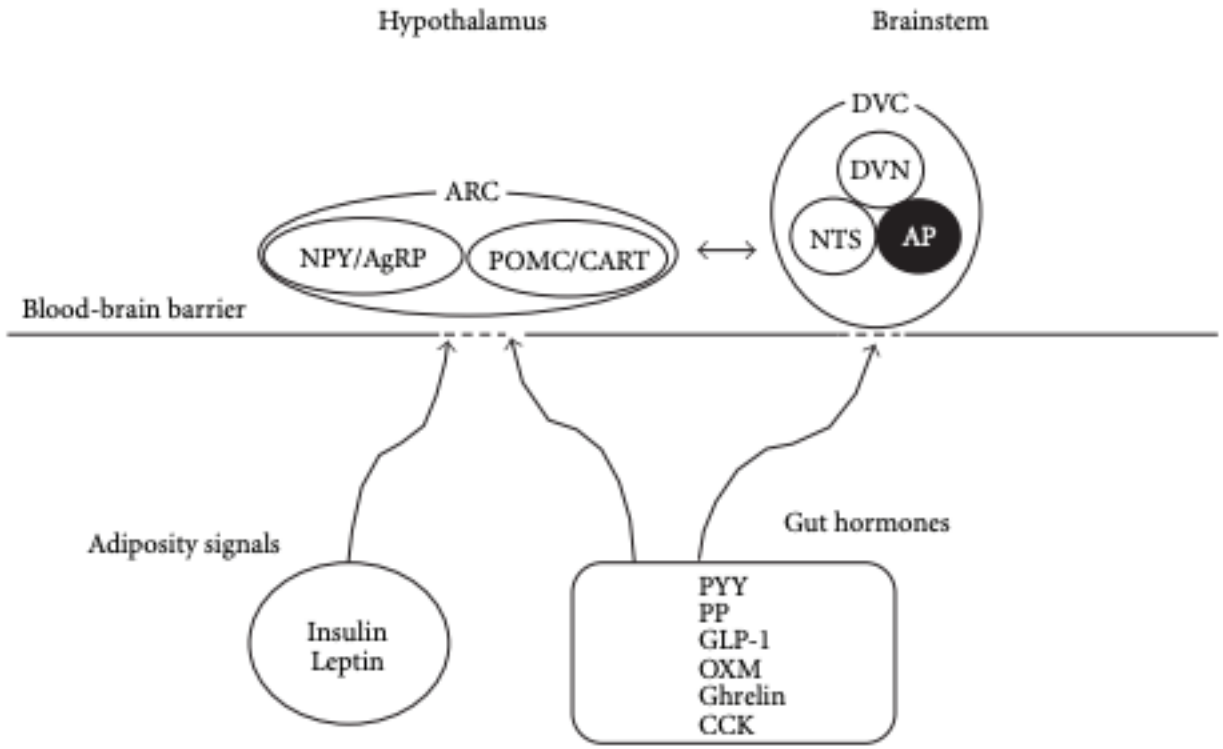
increasing the consumption of highly palatable foods[4]

CNS=central nervous system**; POMC=**proopiomelanocortin.

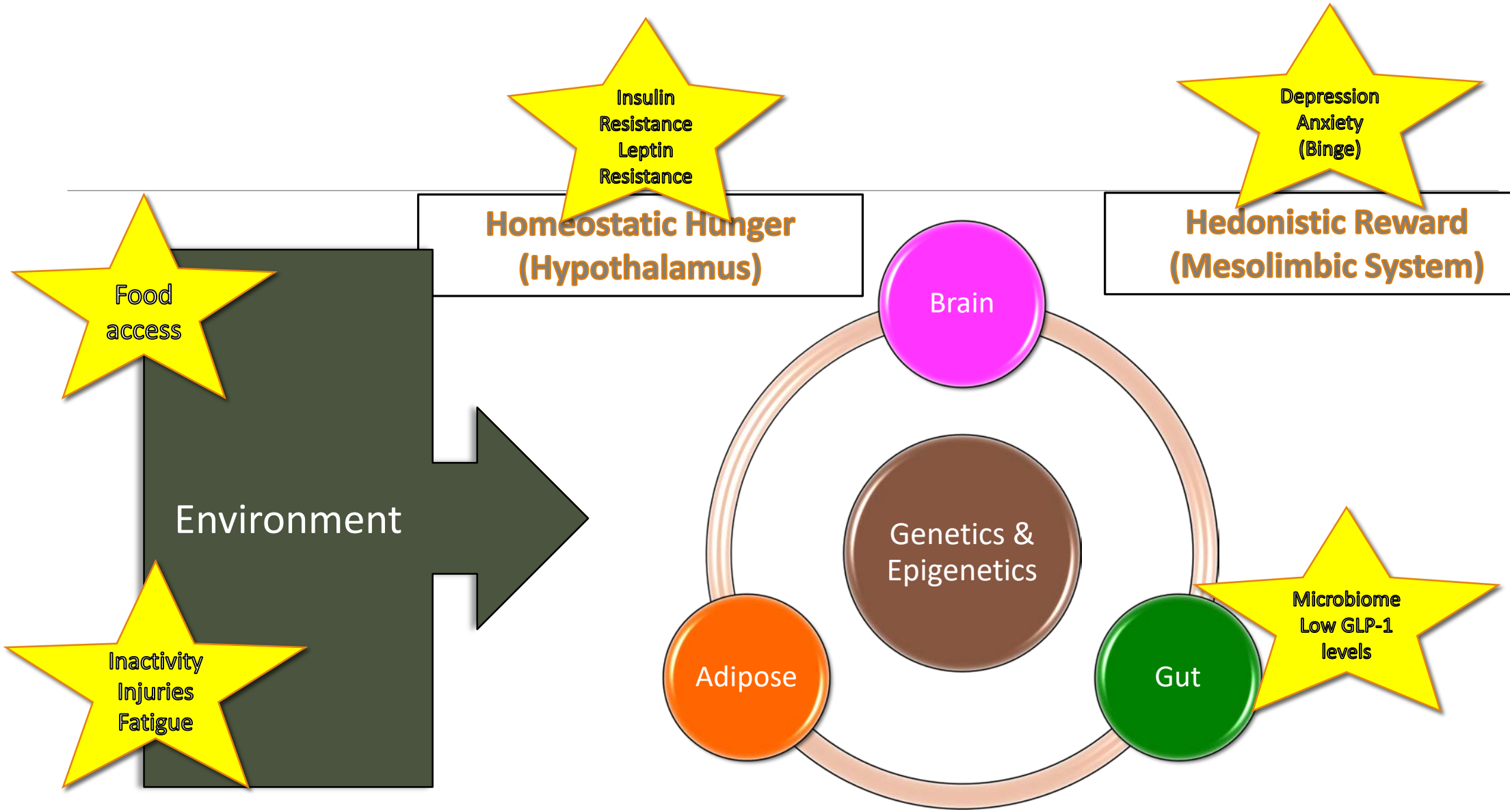
1. Billes SK et al. *Pharmacol Res*. 2014;84:1-11. 2. Yu JH et al. *Diabetes Metab J*. 2012;36:391-398 3. Elliott R et al. *Nature*. 2000;443(7109):287-291.
4. Volkow ND et al. *Obes Rev*. 2013;14:2-18.

Figure adapted from Billes et al.[1] © 2014, with permission from Elsevier.

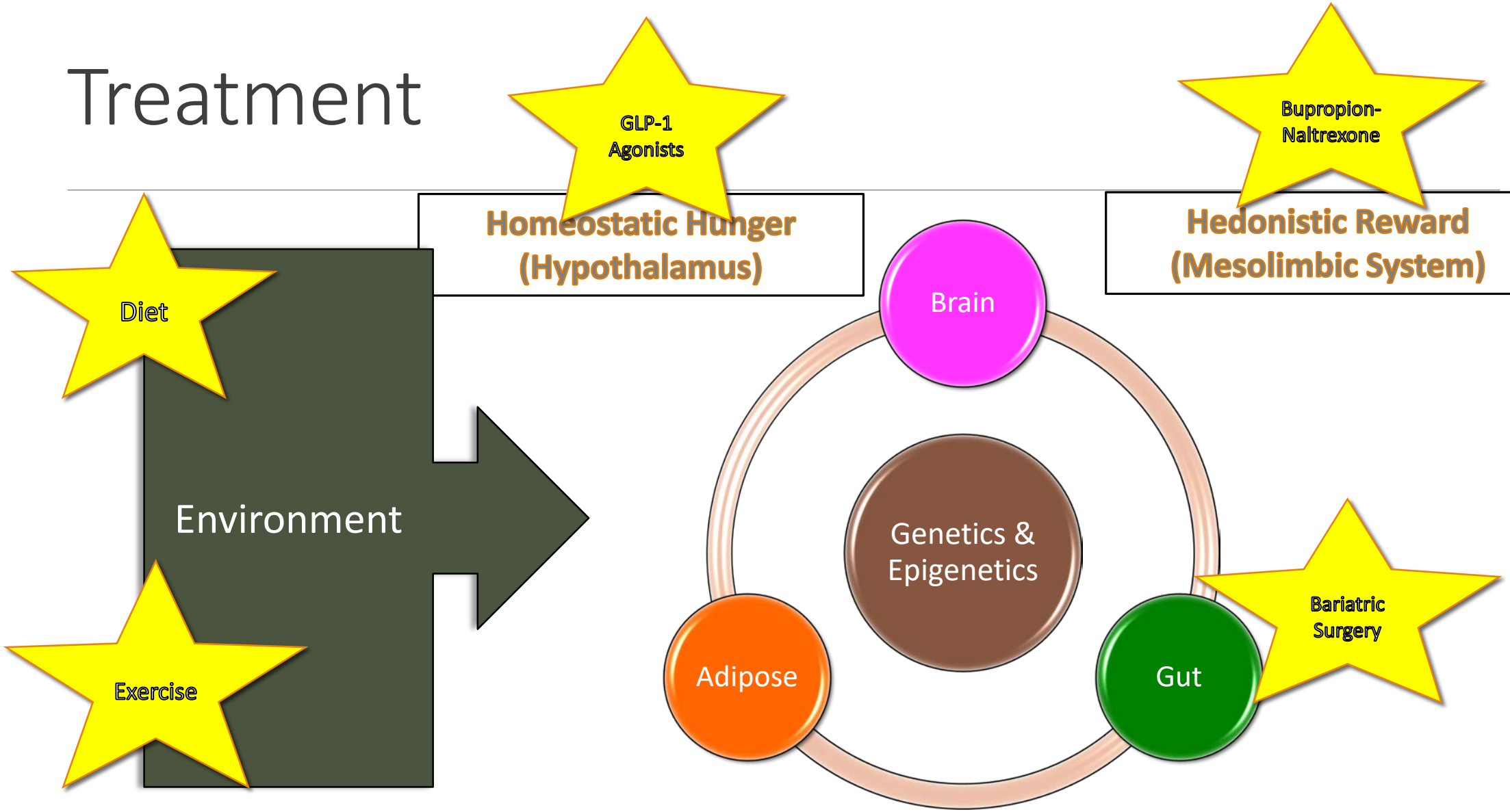
Gut Brain Interaction



Suzuki K, Jayasena CN, Bloom SR. The gut hormones in appetite regulation. Journal of obesity. 2011 Sep 22;2011.


Insulin
Resistance
Leptin
Resistance
Depression
Anxiety
(Binge)
Homeostatic Hunger
(Hypothalamus)
Hedonistic Reward
(Mesolimbic System)
Food
access
Environment
Inactivity
Injuries
Fatigue
Brain
Genetics &
Epigenetics
Adipose
Gut
Microbiome
Low GLP-1
levels

Treatment


GLP-1 Agonists
Bupropion-Naltrexone
Homeostatic Hunger (Hypothalamus)
Hedonistic Reward (Mesolimbic System)
Diet
Environment
Exercise
Brain
Genetics & Epigenetics
Adipose
Gut
Bariatric Surgery

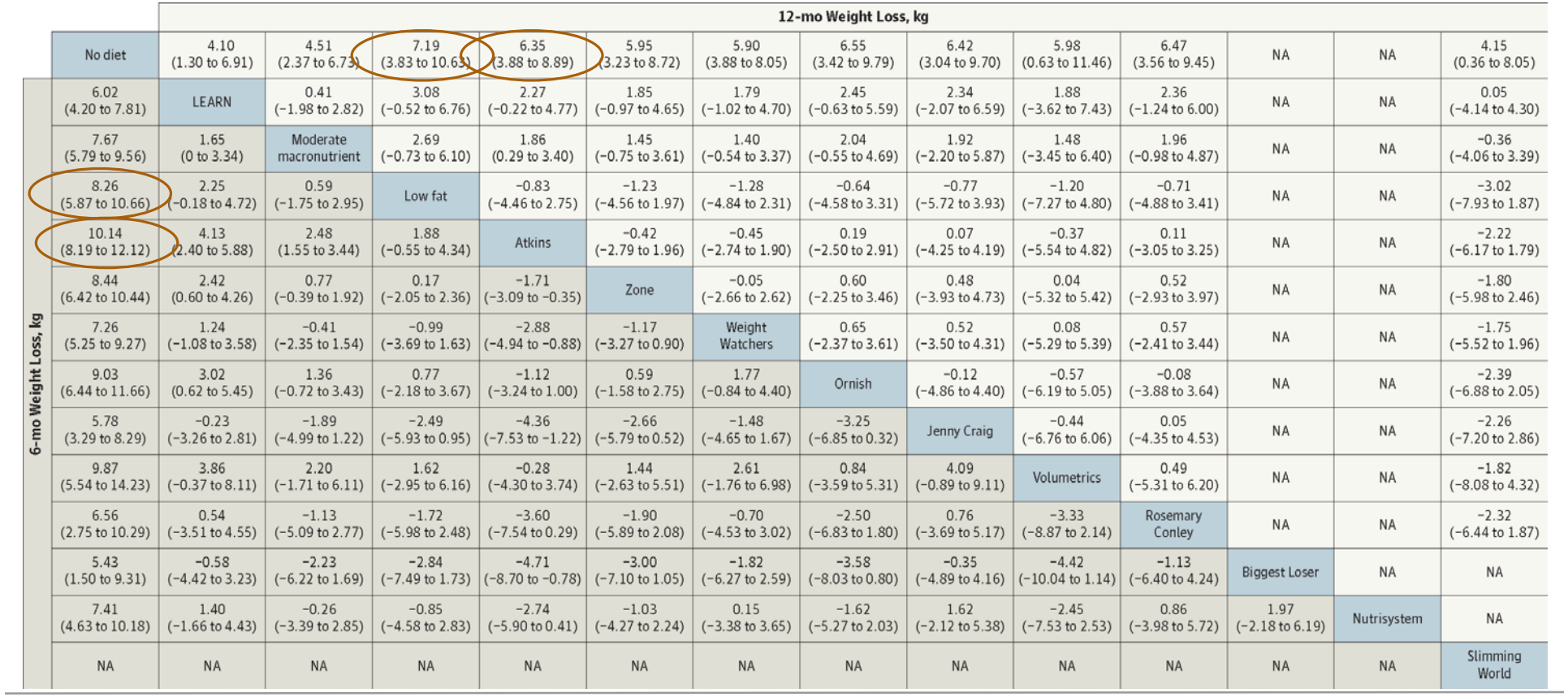
Treatment: Just Go On A Diet

Put the fork down

Use some will power

Media Messaging

Comparison of Weight Loss Among Named Diet Programs in Overweight and Obese Adults: A Meta-analysis



6-mo Weight Loss, kg \ 12-mo Weight Loss, kg	No diet	LEARN	Moderate macronutrient	Low fat	Atkins	Zone	Weight Watchers	Ornish	Jenny Craig	Volumetrics	Rosemary Conley	Biggest Loser	Nutrisystem	Slimming World
No diet		4.10 (1.30 to 6.91)	4.51 (2.37 to 6.73)	7.19 (3.83 to 10.63)	6.35 (3.88 to 8.89)	5.95 (3.23 to 8.72)	5.90 (3.88 to 8.05)	6.55 (3.42 to 9.79)	6.42 (3.04 to 9.70)	5.98 (0.63 to 11.46)	6.47 (3.56 to 9.45)	NA	NA	4.15 (0.36 to 8.05)
6.02 (4.20 to 7.81)	LEARN	0.41 (−1.98 to 2.82)	3.08 (−0.52 to 6.76)	2.27 (−0.22 to 4.77)	1.85 (−0.97 to 4.65)	1.79 (−1.02 to 4.70)	2.45 (−0.63 to 5.59)	2.34 (−2.07 to 6.59)	1.88 (−3.62 to 7.43)	2.36 (−1.24 to 6.00)	NA	NA	0.05 (−4.14 to 4.30)
7.67 (5.79 to 9.56)	1.65 (0 to 3.34)	Moderate macronutrient	2.69 (−0.73 to 6.10)	1.86 (0.29 to 3.40)	1.45 (−0.75 to 3.61)	1.40 (−0.54 to 3.37)	2.04 (−0.55 to 4.69)	1.92 (−2.20 to 5.87)	1.48 (−3.45 to 6.40)	1.96 (−0.98 to 4.87)	NA	NA	−0.36 (−4.06 to 3.39)
8.26 (5.87 to 10.66)	2.25 (−0.18 to 4.72)	0.59 (−1.75 to 2.95)	Low fat	−0.83 (−4.46 to 2.75)	−1.23 (−4.56 to 1.97)	−1.28 (−4.84 to 2.31)	−0.64 (−4.58 to 3.31)	−0.77 (−5.72 to 3.93)	−1.20 (−7.27 to 4.80)	−0.71 (−4.88 to 3.41)	NA	NA	−3.02 (−7.93 to 1.87)
10.14 (8.19 to 12.12)	4.13 (2.40 to 5.88)	2.48 (1.55 to 3.44)	1.88 (−0.55 to 4.34)	Atkins	−0.42 (−2.79 to 1.96)	−0.45 (−2.74 to 1.90)	0.19 (−2.50 to 2.91)	0.07 (−4.25 to 4.19)	−0.37 (−5.54 to 4.82)	0.11 (−3.05 to 3.25)	NA	NA	−2.22 (−6.17 to 1.79)
8.44 (6.42 to 10.44)	2.42 (0.60 to 4.26)	0.77 (−0.39 to 1.92)	0.17 (−2.05 to 2.36)	−1.71 (−3.09 to −0.35)	Zone	−0.05 (−2.66 to 2.62)	0.60 (−2.25 to 3.46)	0.48 (−3.93 to 4.73)	0.04 (−5.32 to 5.42)	0.52 (−2.93 to 3.97)	NA	NA	−1.80 (−5.98 to 2.46)
7.26 (5.25 to 9.27)	1.24 (−1.08 to 3.58)	−0.41 (−2.35 to 1.54)	−0.99 (−3.69 to 1.63)	−2.88 (−4.94 to −0.88)	−1.17 (−3.27 to 0.90)	Weight Watchers	0.65 (−2.37 to 3.61)	0.52 (−3.50 to 4.31)	0.08 (−5.29 to 5.39)	0.57 (−2.41 to 3.44)	NA	NA	−1.75 (−5.52 to 1.96)
9.03 (6.44 to 11.66)	3.02 (0.62 to 5.45)	1.36 (−0.72 to 3.43)	0.77 (−2.18 to 3.67)	−1.12 (−3.24 to 1.00)	0.59 (−1.58 to 2.75)	1.77 (−0.84 to 4.40)	Ornish	−0.12 (−4.86 to 4.40)	−0.57 (−6.19 to 5.05)	−0.08 (−3.88 to 3.64)	NA	NA	−2.39 (−6.88 to 2.05)
5.78 (3.29 to 8.29)	−0.23 (−3.26 to 2.81)	−1.89 (−4.99 to 1.22)	−2.49 (−5.93 to 0.95)	−4.36 (−7.53 to −1.22)	−2.66 (−5.79 to 0.52)	−1.48 (−4.65 to 1.67)	−3.25 (−6.85 to 0.32)	Jenny Craig	−0.44 (−6.76 to 6.06)	0.05 (−4.35 to 4.53)	NA	NA	−2.26 (−7.20 to 2.86)
9.87 (5.54 to 14.23)	3.86 (−0.37 to 8.11)	2.20 (−1.71 to 6.11)	1.62 (−2.95 to 6.16)	−0.28 (−4.30 to 3.74)	1.44 (−2.63 to 5.51)	2.61 (−1.76 to 6.98)	0.84 (−3.59 to 5.31)	4.09 (−0.89 to 9.11)	Volumetrics	0.49 (−5.31 to 6.20)	NA	NA	−1.82 (−8.08 to 4.32)
6.56 (2.75 to 10.29)	0.54 (−3.51 to 4.55)	−1.13 (−5.09 to 2.77)	−1.72 (−5.98 to 2.48)	−3.60 (−7.54 to 0.29)	−1.90 (−5.89 to 2.08)	−0.70 (−4.53 to 3.02)	−2.50 (−6.83 to 1.80)	0.76 (−3.69 to 5.17)	−3.33 (−8.87 to 2.14)	Rosemary Conley	NA	NA	−2.32 (−6.44 to 1.87)
5.43 (1.50 to 9.31)	−0.58 (−4.42 to 3.23)	−2.23 (−6.22 to 1.69)	−2.84 (−7.49 to 1.73)	−4.71 (−8.70 to −0.78)	−3.00 (−7.10 to 1.05)	−1.82 (−6.27 to 2.59)	−3.58 (−8.03 to 0.80)	−0.35 (−4.89 to 4.16)	−4.42 (−10.04 to 1.14)	−1.13 (−6.40 to 4.24)	Biggest Loser	NA	NA
7.41 (4.63 to 10.18)	1.40 (−1.66 to 4.43)	−0.26 (−3.39 to 2.85)	−0.85 (−4.58 to 2.83)	−2.74 (−5.90 to 0.41)	−1.03 (−4.27 to 2.24)	0.15 (−3.38 to 3.65)	−1.62 (−5.27 to 2.03)	1.62 (−2.12 to 5.38)	−2.45 (−7.53 to 2.53)	0.86 (−3.98 to 5.72)	1.97 (−2.18 to 6.19)	Nutrisystem	NA
NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	Slimming World

JAMA. 2014;312(9):923-933. doi:10.1001/jama.2014.10397

Comparison of Weight Loss Among Named Diet Programs in Overweight and Obese Adults: A Meta-analysis

		12-mo Weight Loss, kg			
6-mo Weight Loss, kg	No diet (6 mo: 0; 12 mo: 0)[a]	5.16 (2.68 to 7.63)	5.70 (4.14 to 7.35)	7.25 (5.33 to 9.25)	7.27 (5.26 to 9.34)
	6.07 (4.23 to 7.84)	LEARN (6 mo: 0; 12 mo: 0.02)[a]	0.55 (−1.71 to 2.87)	2.10 (−0.20 to 4.47)	2.12 (−0.33 to 4.59)
	6.78 (5.50 to 8.05)	0.71 (−0.97 to 2.44)	Moderate macronutrients (6 mo: 0; 12 mo: 0)[a]	1.55 (0.13 to 2.95)	1.56 (−0.17 to 3.30)
	8.73 (7.27 to 10.20)	2.66 (0.93 to 4.44)	1.95 (1.13 to 2.79)	Low carbohydrate (6 mo: 0.83; 12 mo: 0.48)[a]	0.02 (−1.78 to 1.79)
	7.99 (6.01 to 9.92)	1.92 (−0.19 to 4.06)	1.20 (−0.42 to 2.79)	−0.74 (−2.31 to 0.78)	Low fat (6 mo: 0.17; 12 mo: 0.50)[a]

JAMA. 2014;312(9):923-933. doi:10.1001/jama.2014.10397

Diets in CKD

KDIGO 2012

3.1.13: We suggest lowering protein intake to 0.8 g/kg/day in adults with diabetes (2C) or without diabetes

(2B) and GFR o30 ml/min/ 1.73 m2 (GFR categories G4-G5), with appropriate education. 3.1.14: We suggest avoiding high protein intake (41.3 g/ kg/day) in adults with CKD at risk of progression. (2C)

2 Year Change in eGFR with diet change

A. Change in eGFR by baseline CKD stage

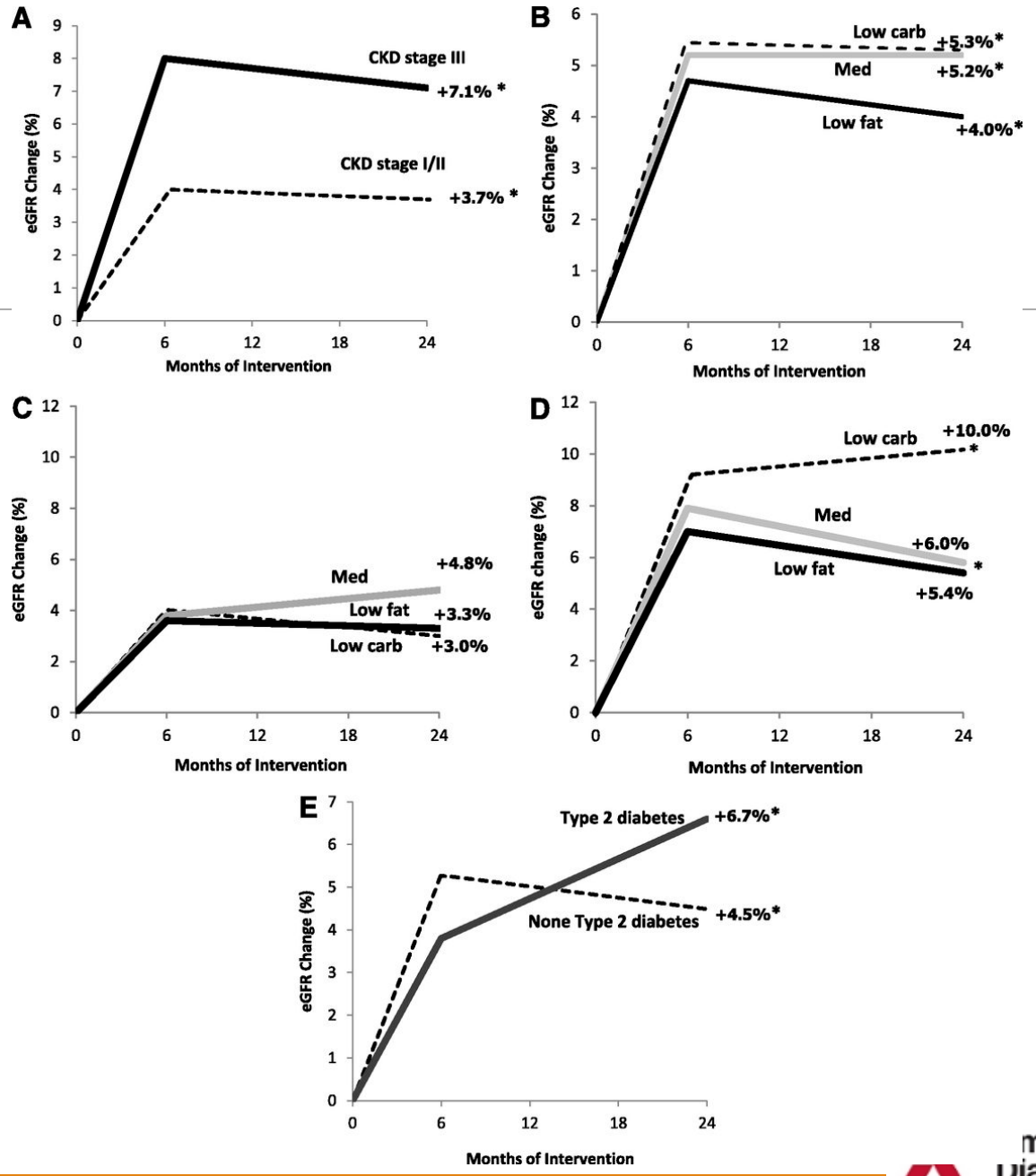
B. Change in eGFR by diet all eGFR

C. Change in eGFR by diet, baseline eGFR>60.

D. Change in eGFR by diet type baseline eGFR <60

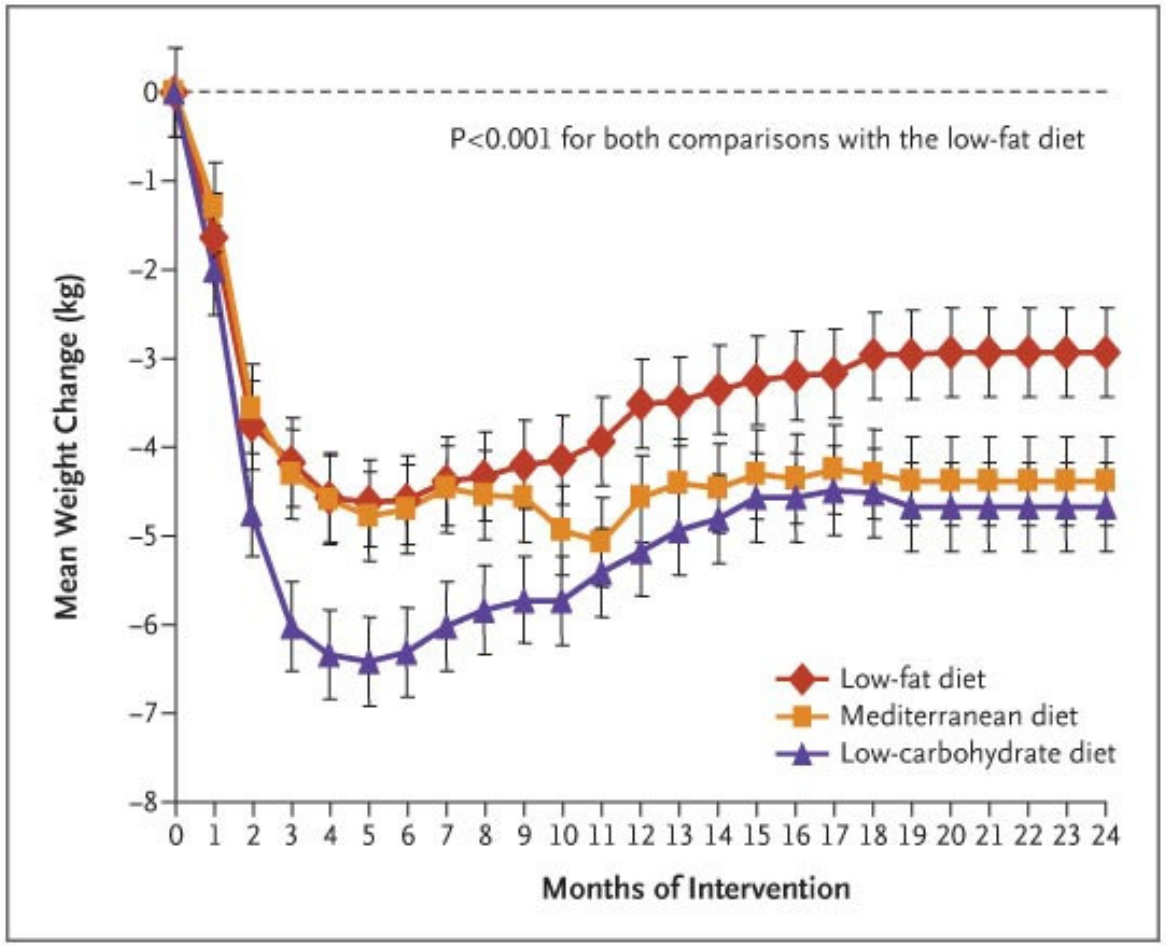
E. Change in eGFR by T2DM status

Amir Tirosh et al. Dia Care 2013;36:2225-2232



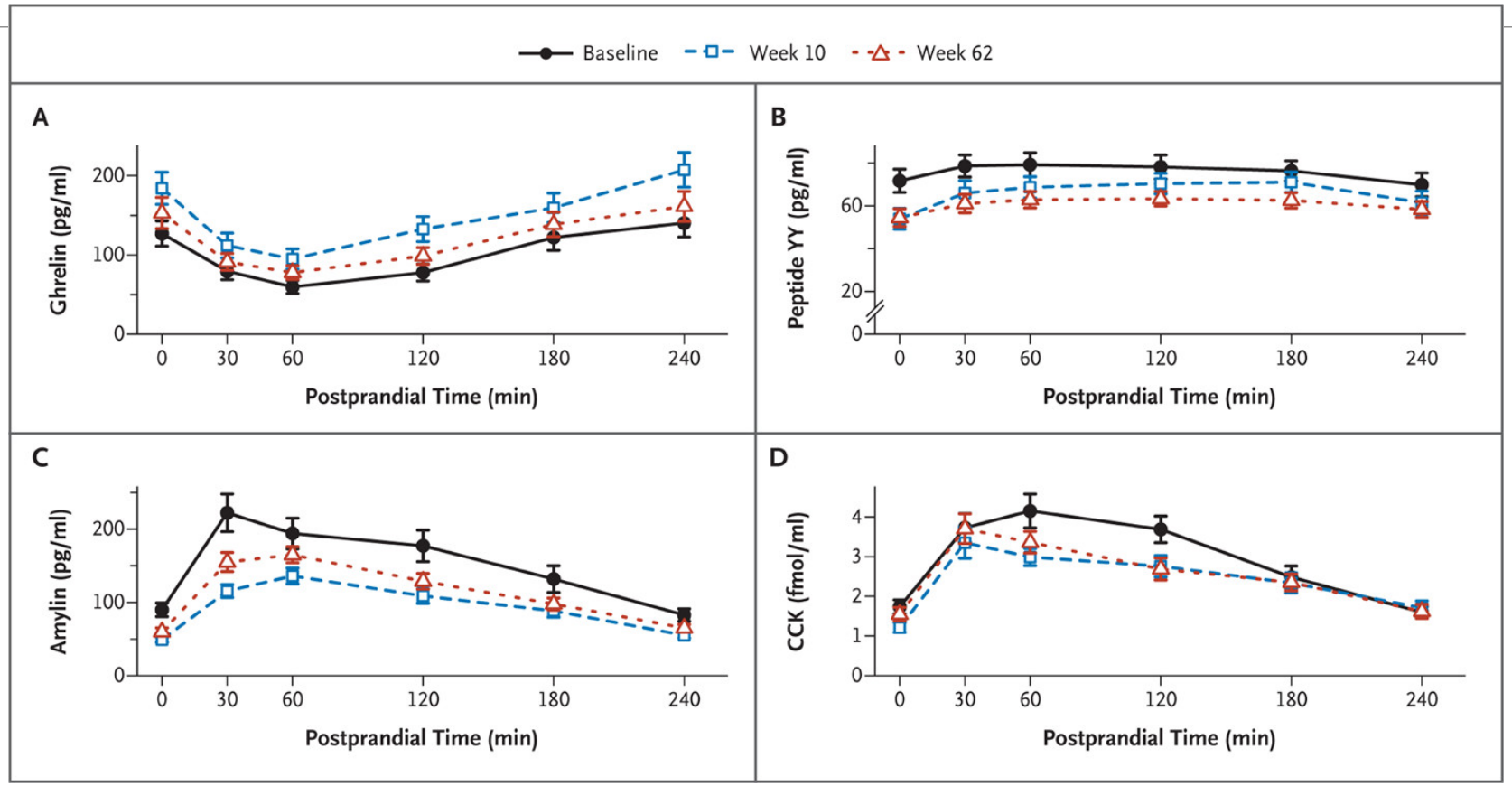
merican Diabetes Association.

Weight Changes during 2 Years According to Diet Group.



Shai I et al. N Engl J Med 2008;359:229-241.

Long-Term Persistence of Hormonal Adaptations to Weight Loss



Sumithran P et al. N Engl J Med 2011;365:1597-1604.

The NEW ENGLAND JOURNAL of MEDICINE

Treatment: Diets

Diet alone, across multiple studies: 5-8Kg (11-17lb)

I wouldn’t use DASH Diet alone for BP 200/120

I wouldn’t use Profile diet alone for LDL 5

AND I would explain my disagreement if that is the treatment they wanted!

Drugs for Long-Term Weight Management

Drug (trade name)	Health Canada Approval	Mechanism of Action	1-year weigth loss, placebo-substracted	Major Safety Issues	Tolerability
Orlistat (Xenical)	1999	Gastrointestinal lipase inhibitor	~3%	Fat-soluble vitamin malabsorption	Fecal urgency, fecal incontinence, flatus with discharge, oily spotting
Liraglutide (Saxenda)	2015	GLP-1 receptor agonist	4.0-5.4%	Gallstones, acute pancreatitis	Nausea, vomiting, diarrhea, constipation, dyspepsia, abdominal pain, headache, fatigue, hypoglycemia, increased lipase
Naltrexone hydrochloride/Bupropion hydrochloride (Contrave)	2018	Opioid receptor antagonist / aminoketone antidepressant	3.3-4.8%	Use in controlled hypertension only	Nausea, vomiting, constipation, diarrhea, dizziness, dry mouth

Orlistat

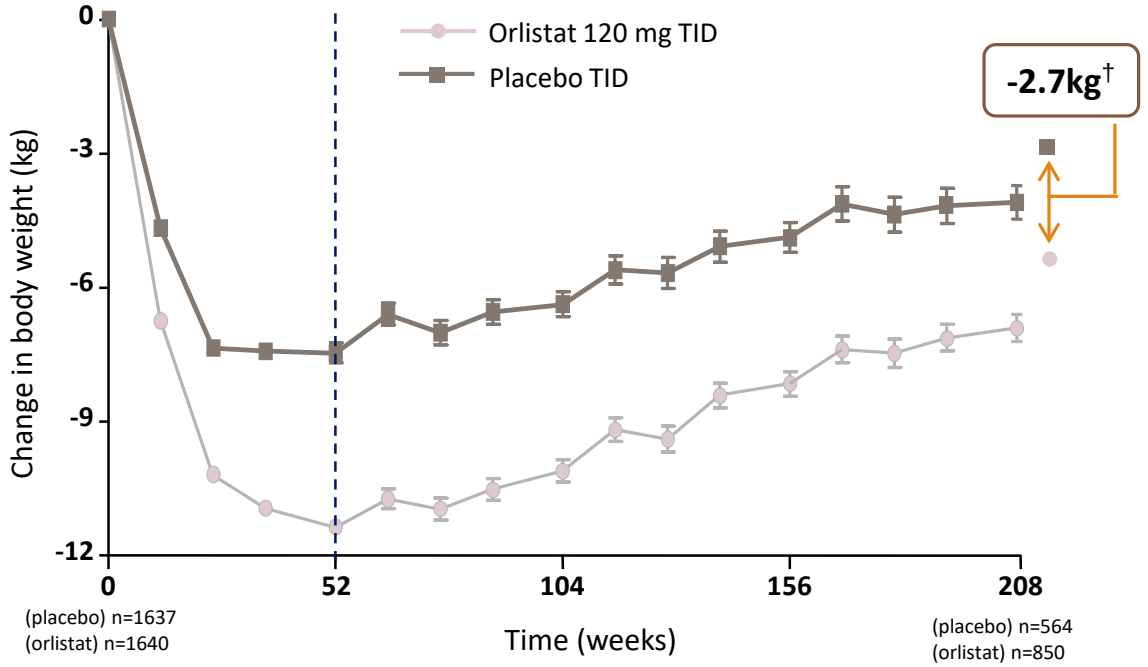
Selective Lipase inhibitor

Prevents digestion and absorption of fat in the duodenum

20% reduction in calories

Orlistat XENDOS trial

ORLISTAT TID[1,2]



YEAR 1

110.4–110.6 kg

-11.4 kg

vs. 7.5 kg* (completers)

≥5% weight loss

73%

vs. 45%*

>10% weight loss

41%

vs. 21%*

YEAR 4

110.4–110.6 kg

-6.9 kg

vs. -4.1 kg* (completers)

≥5% weight loss

44.8%

vs. 28.0%*

>10% weight loss

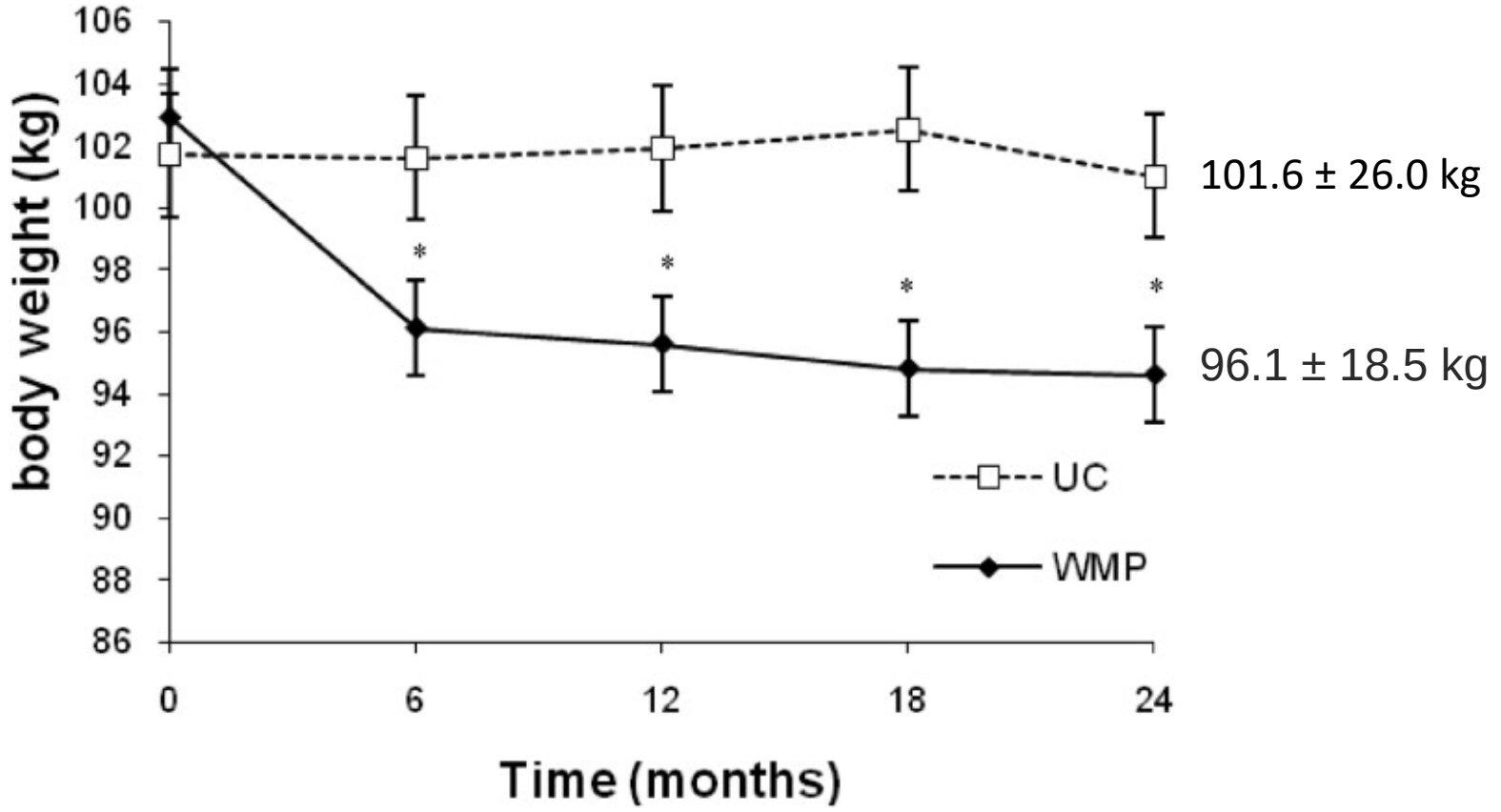
21%

vs. 10%*

*p<0.001; †p<0.001 by LOCF analysis (last observation carried forward)

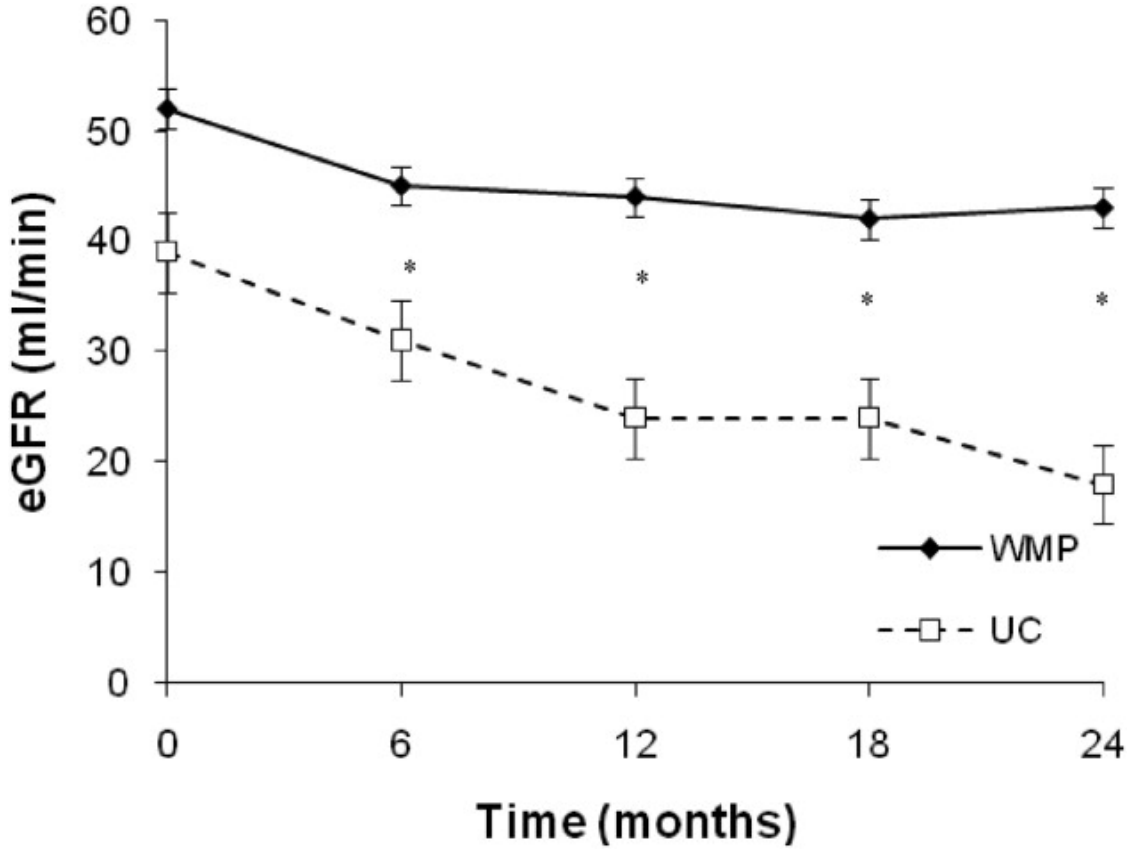
1. TORGERSON JS ET AL. DIABETES CARE. 2004;27:155-61; 2. ORLISTAT PRODUCT MONOGRAPH. HOFFMAN-LA ROCHE, LTD. 2012

Orlistat and Kidney Disease: Weight



MacLaughlin HL, Cook SA, Kariyawasam D, Roseke M, van Niekerk M, Macdougall IC. Nonrandomized trial of weight loss with orlistat, nutrition education, diet, and exercise in obese patients with CKD: 2-year follow-up. American Journal of Kidney Diseases. 2010 Jan 1;55(1):69-76.

Orlistat and Kidney Disease: eGFR



MacLaughlin HL, Cook SA, Kariyawasam D, Roseke M, van Niekerk M, Macdougall IC. Nonrandomized trial of weight loss with orlistat, nutrition education, diet, and exercise in obese patients with CKD: 2-year follow-up. American Journal of Kidney Diseases. 2010 Jan 1;55(1):69-76.

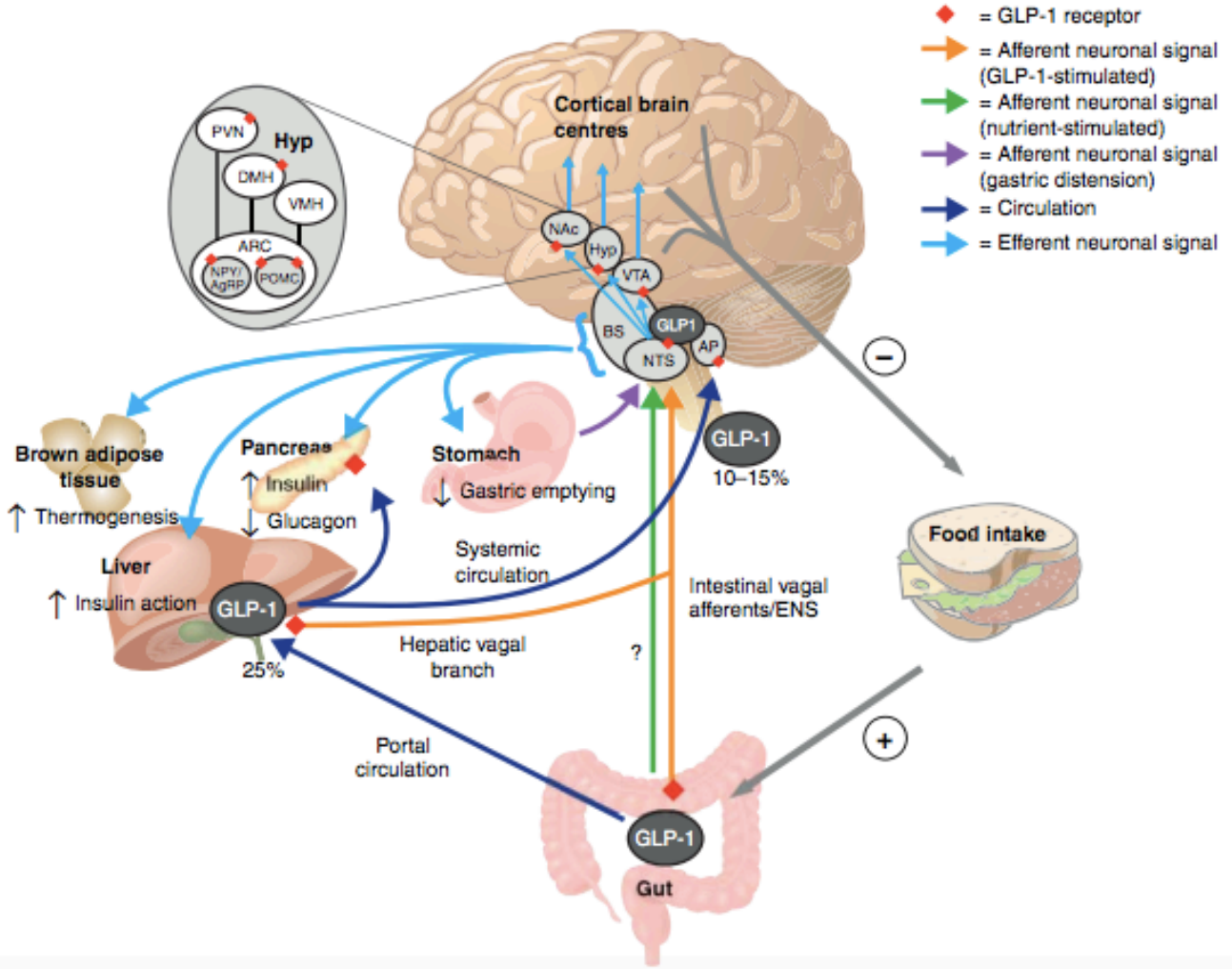
Let’s Talk Liraglutide

The compound

Physiological effects

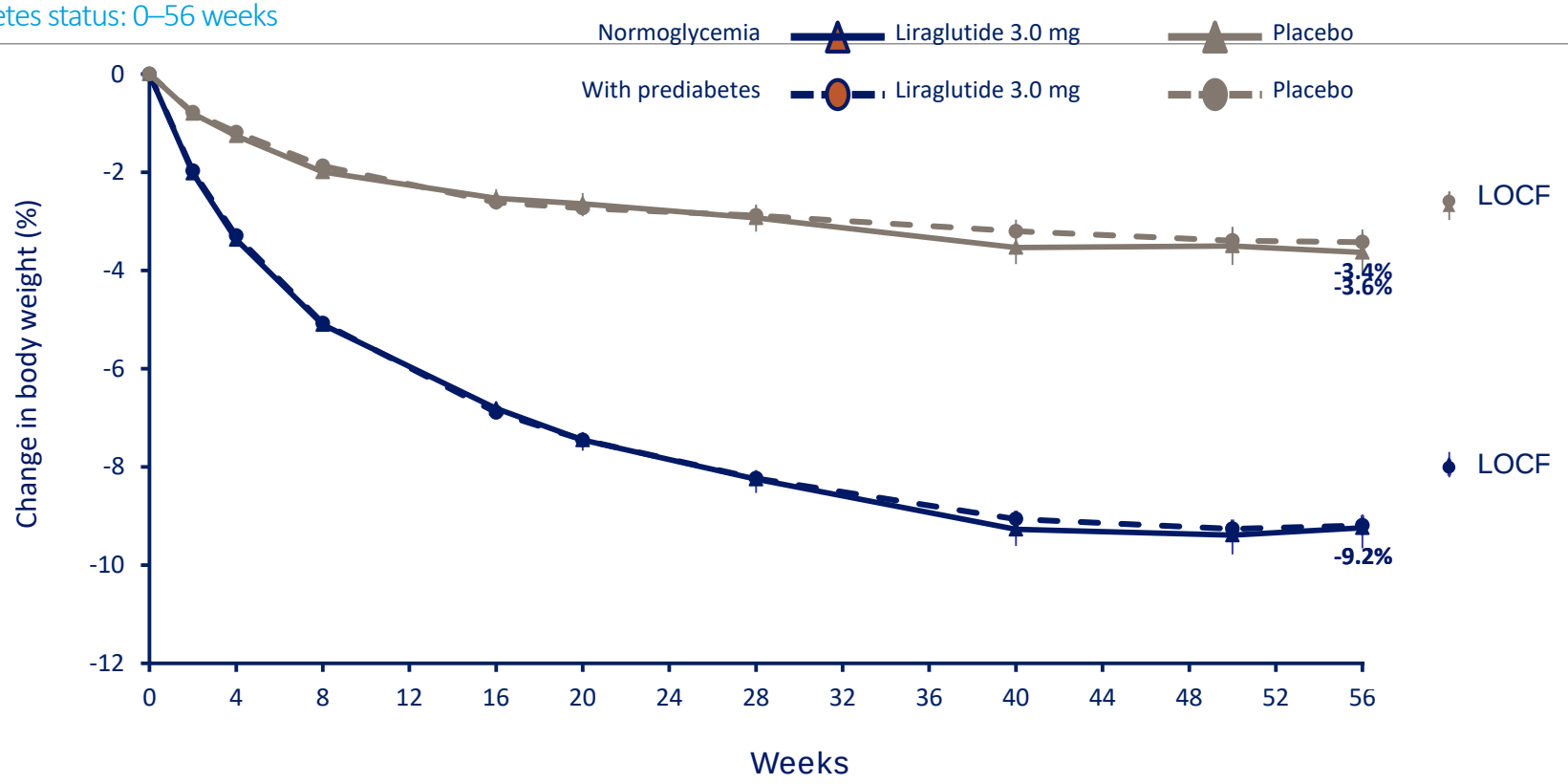
Clinical Studies

GLP-1 Receptors and the Hypothalamus



SCALE Trial: Mean change in body weight (%)

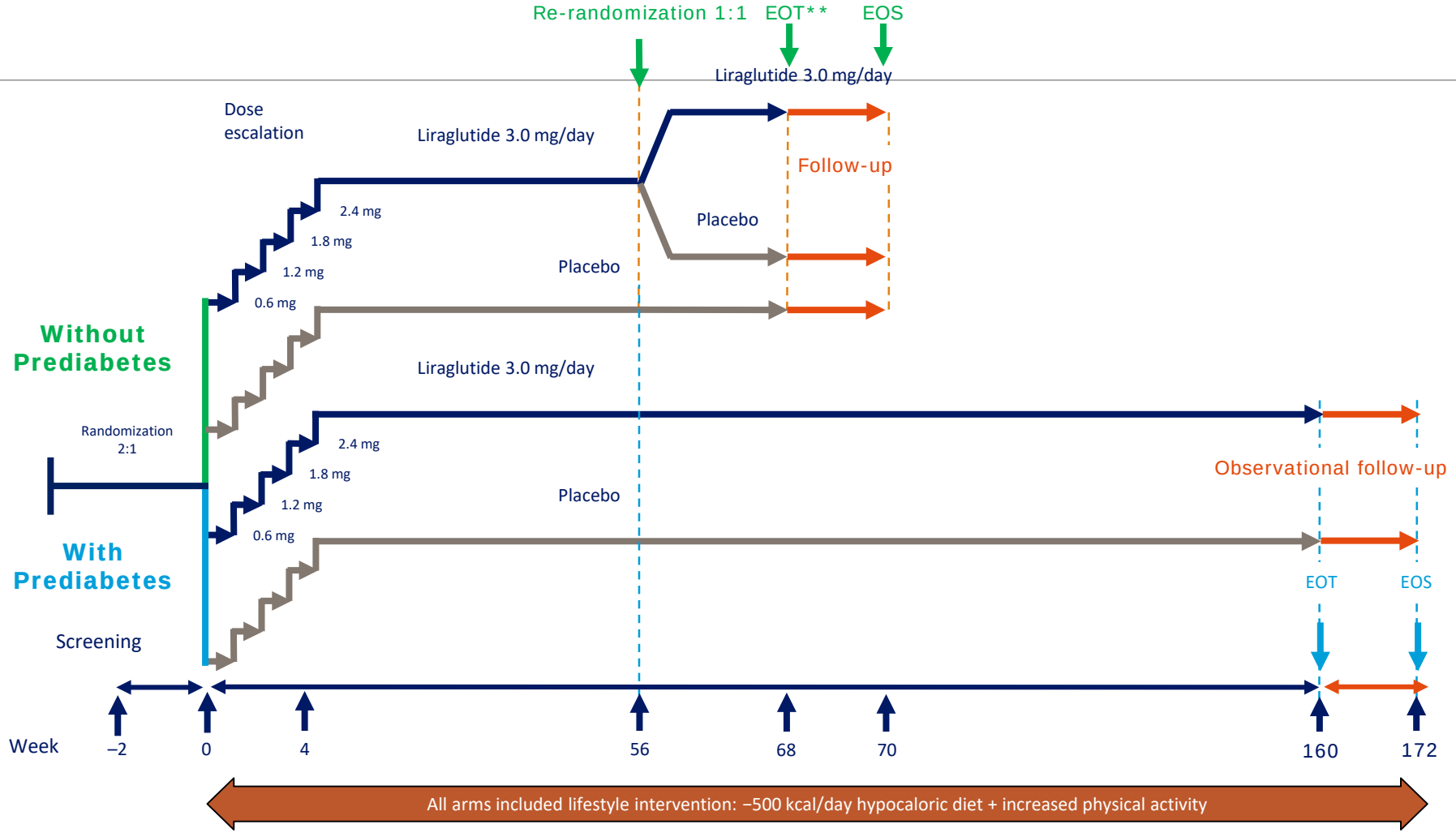
By prediabetes status: 0–56 weeks



- Patients treated with Saxenda® experienced an observed mean **waist circumference change of -8.2 cm** vs. -3.9 cm with placebo (p<0.001)

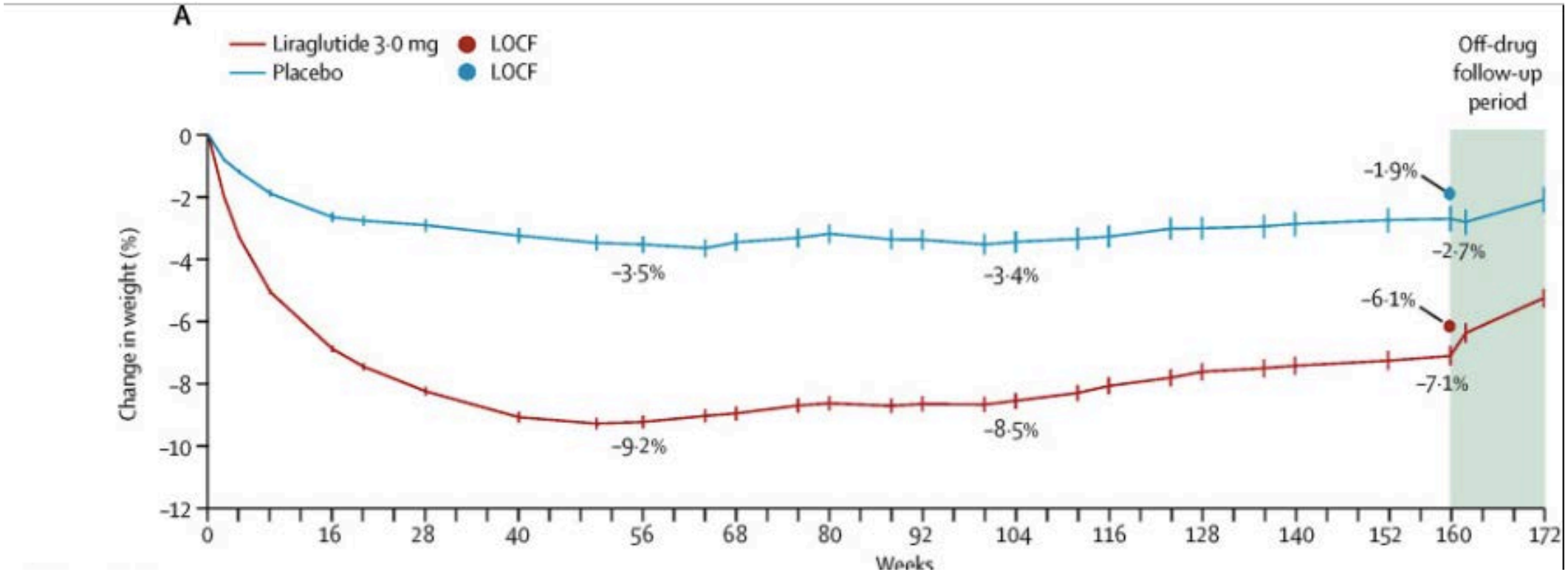
Data are observed means with standard error bars, and the symbols at the right represent the 56-week weight change using LOCF imputation

Further 3 year follow up after the study



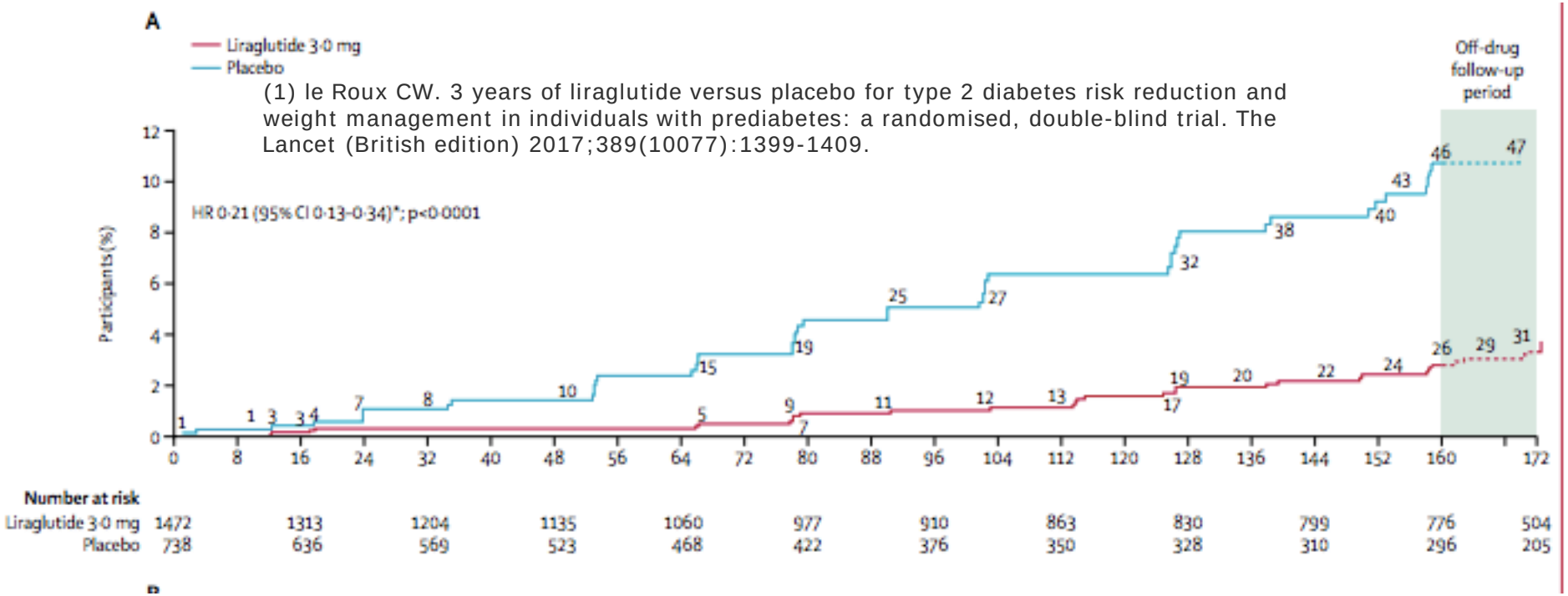
*Treated or untreated hypertension or dyslipidaemia according to ATP-III; **Treatment ends at week 68 for individuals without prediabetes and is followed by an off-treatment follow-up period of 2 weeks. EOT, end of treatment; EOS, end of study

Weight loss



le Roux CW. 3 years of liraglutide versus placebo for type 2 diabetes risk reduction and weight management in individuals with prediabetes: a randomised, double-blind trial. The Lancet (British edition) 2017;389(10077):1399-1409.

Progression to Diabetes at 3 yrs



le Roux CW. 3 years of liraglutide versus placebo for type 2 diabetes risk reduction and weight management in individuals with prediabetes: a randomised, double-blind trial. The Lancet (British edition) 2017;389(10077):1399-1409.

Liraglutide and Renal Outcomes in T2DM

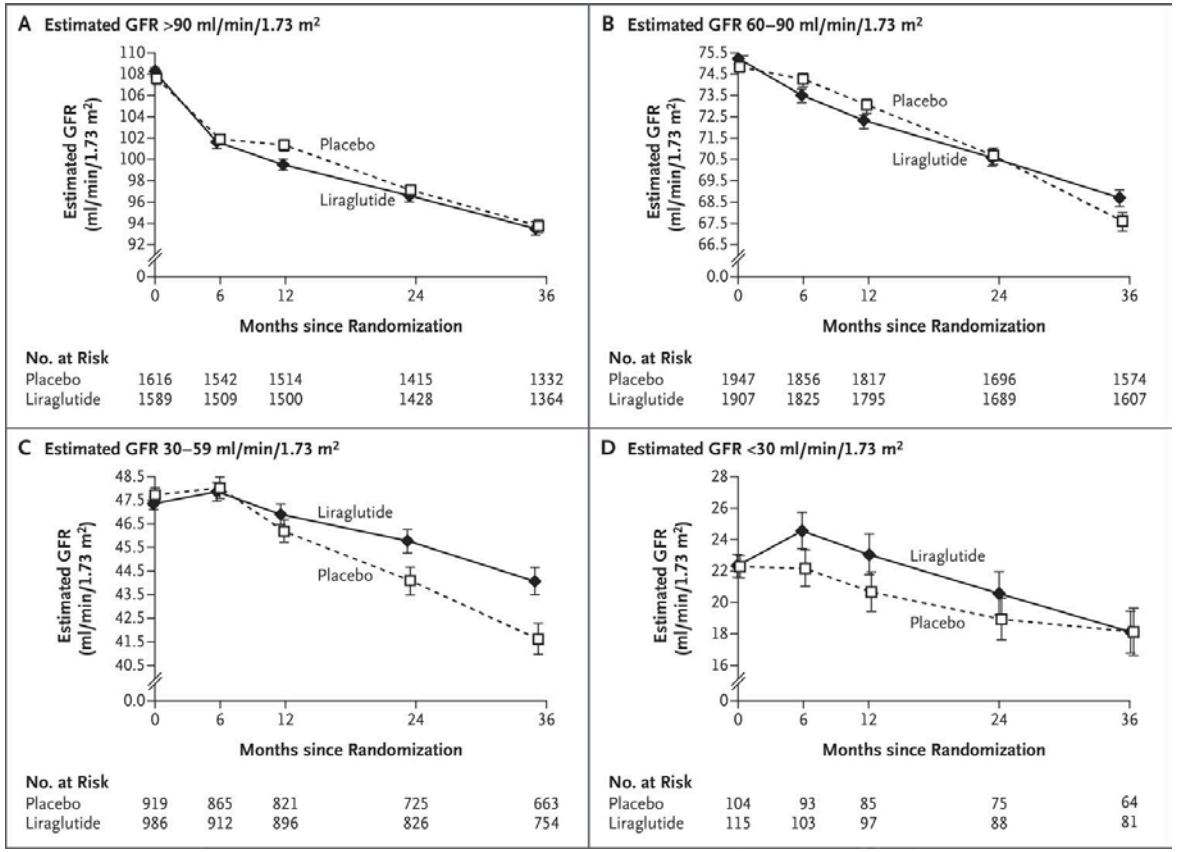
Table 1. Composite Renal Outcome and Individual Components of the Composite Outcome.*

Outcome	Liraglutide (N=4668)	Placebo (N=4672)	Total (N=9340)	Hazard Ratio (95% CI)	P Value
	no. of patients (rate per 1000 patient-yr of observation)				
Composite renal outcome	268 (15.0)	337 (19.0)	605 (17.0)	0.78 (0.67–0.92)	0.003
Components of composite renal outcome†					
New-onset persistent macroalbuminuria	161 (9.0)	215 (12.1)	376 (10.6)	0.74 (0.60–0.91)	0.004
Persistent doubling of serum creatinine level	87 (4.9)	97 (5.5)	184 (5.2)	0.89 (0.67–1.19)	0.43
Renal-replacement therapy	56 (3.1)	64 (3.6)	120 (3.4)	0.87 (0.61–1.24)	0.44
Death due to renal disease	8 (0.4)	5 (0.3)	13 (0.4)	1.59 (0.52–4.87)	0.41

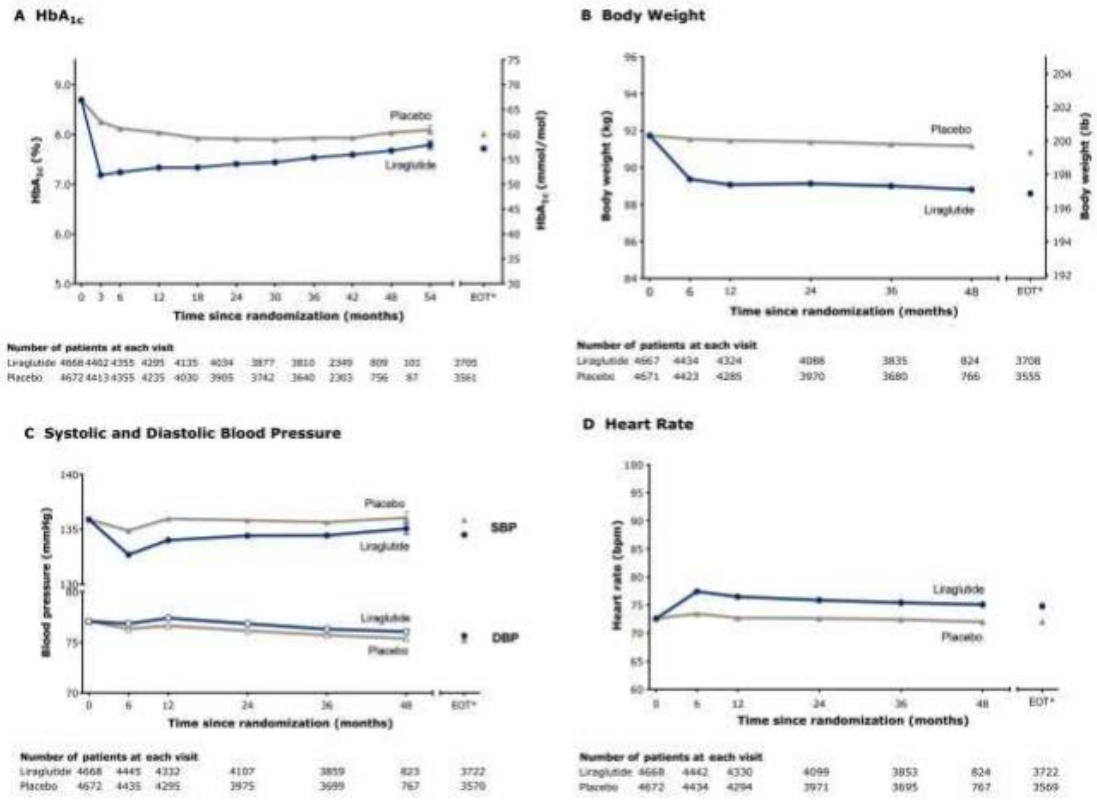
* There were 17,822 patient-years of observation in the liraglutide group and 17,741 in the placebo group. All the events were adjudicated. Hazard ratios and P values were estimated with the use of a Cox proportional-hazards model with trial group as a covariate.

† The composite renal outcome consisted of new-onset persistent macroalbuminuria, persistent doubling of the serum creatinine level and an estimated glomerular filtration rate of 45 ml or less per minute per 1.73 m^2 of body-surface area (referred to as persistent doubling of the serum creatinine level), the need for continuous renal-replacement therapy (end-stage renal disease), or death due to renal disease. One patient who had macroalbuminuria at baseline had an event of new-onset persistent macroalbuminuria that was confirmed by adjudication after the patient had regression to microalbuminuria earlier in the trial.

Liraglutide and Renal Outcomes in T2DM



Liraglutide and Renal Outcomes in T2DM



Bupropion and Naltrexone (Contrave)

Mechanism of Action

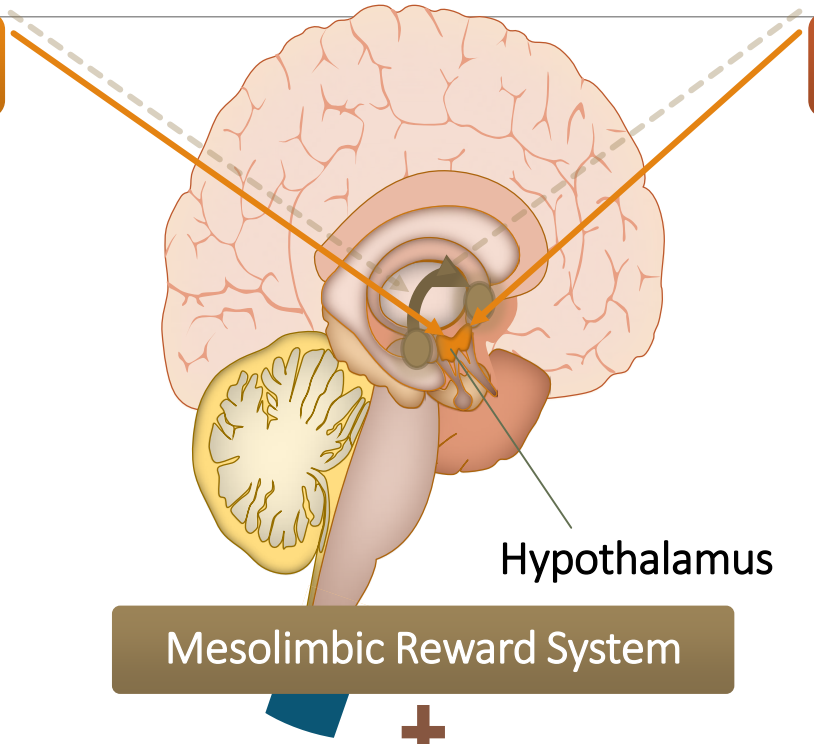
Clinical Studies

Bupropion and Naltrexone: MOA

Bupropion HCl

Indicated for the treatment of major depressive disorder and as an aid to smoking cessation

Stimulates POMC cells, which suppresses appetite



Naltrexone HCl

Indicated for the treatment of alcohol dependence and for prevention of relapse to opioid dependence

Blocks β-endorphin negative feedback loop on POMC neurons, which further contributes to appetite suppression

POMC=pro-opiomelanocortin.

Figure adapted from Billes et al,[1] © 2014, with permission from Elsevier.

1. Billes SK, et al. *Pharmacol Res*. 2014;84:1-11.

Weight loss Bupropion-Naltrexone

[a]Completer population; 303 based on 28-week endpoint, all others based on 56-week endpoint.

BMOD=behavior modification; **DM**=type 2 diabetes mellitus.

1. CONTRAVE [summary of product characteristics]. Dublin, Ireland: Orexigen Therapeutics Ireland Limited; 2017.

Bupropion and Naltrexone: Contraindications

Naltrexone-related:

- Chronic opioid use
- Abrupt discontinuation of alcohol or drug use
- Severe hepatic impairment
- Severe renal impairment

Bupropion-related:

- Uncontrolled hypertension
- Bupropion containing drugs
- Seizures, Bulimia, Anorexia
- MAOI use
- Thioridazine use
- Severe hepatic impairment
- Severe renal impairment

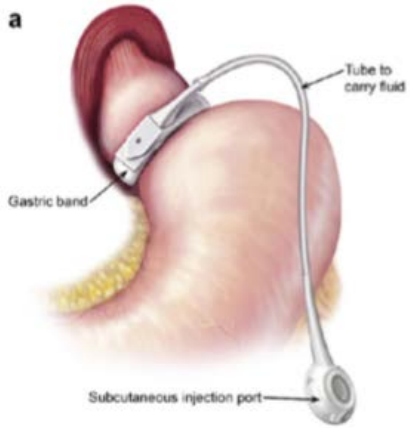
Unique to Contrave:

- Pregnancy

1. CONTRAVE [product monograph]. Laval, QC: Valeant Canada LP; 2018 CONTRAVE [product monograph]. Laval, QC: Valeant Canada LP; 2018.

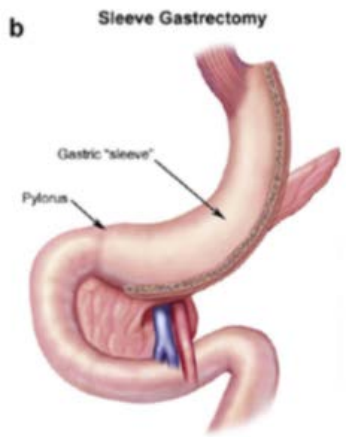
Bariatric Surgery

LAGB



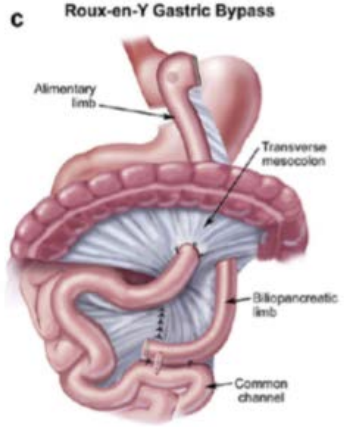
10-15%

Sleeve Gastrectomy



20-25%

Gastric Bypass



30-40%

Bariatric Surgery in CKD (Japan)

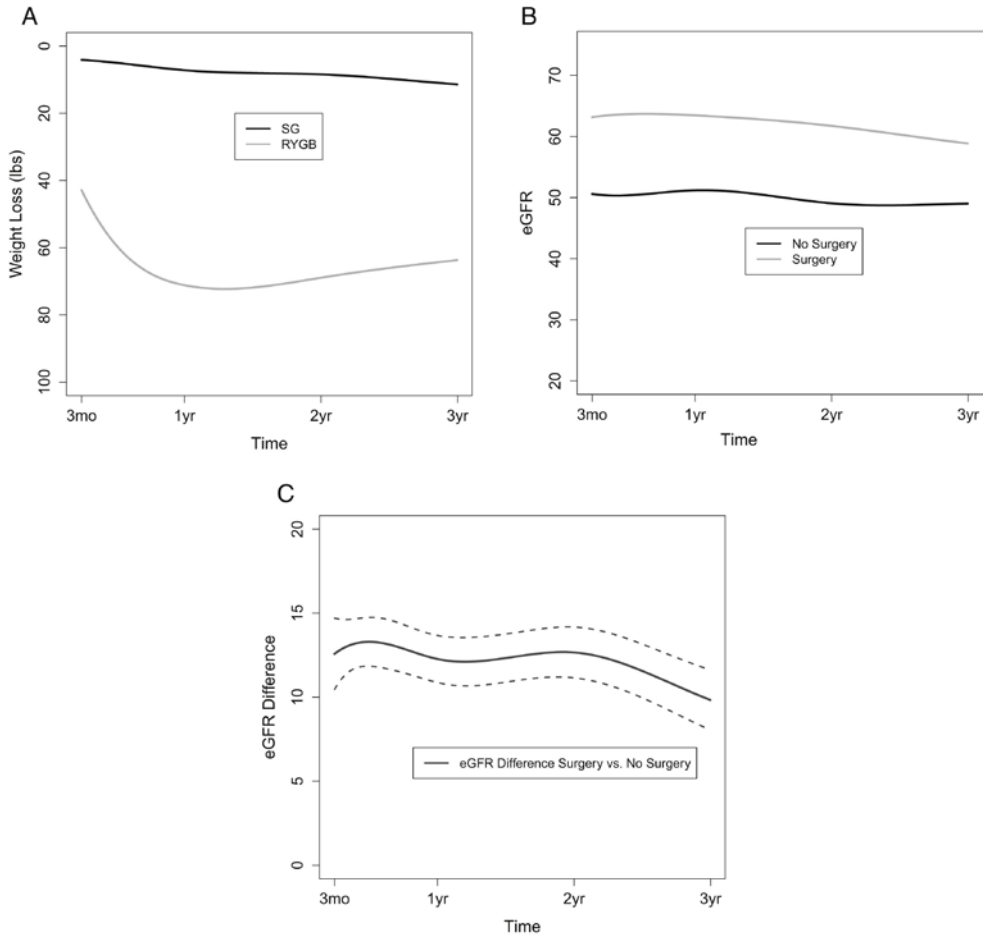
Table 3

The changes in the eGFRcys and BMI values after bariatric surgery

CKD	eGFRcys baseline	One year	*p* value	BMI baseline	One year	*p* value
1	101 (94–108)	114 (103–127)	< 0.01	38.1 ± 6.2	26.5 ± 3.4	< 0.01
2	79 (74–84)	97 (87–104)	< 0.01	38.5 ± 6.9	26.7 ± 3.6	< 0.01
3	44 (42–47)	45 (43–63)	0.08	36.1 ± 6.3	26.6 ± 3.6	< 0.01
4	27.1	33		30.9	27.1	

Wakamatsu K, Seki Y, Kasama K, Uno K, Hashimoto K, Seto Y, Kurokawa Y. Prevalence of chronic kidney disease in morbidly obese Japanese and the impact of bariatric surgery on disease progression. Obesity surgery. 2018 Feb 1;28(2):489-96.

Bariatric Surgery in CKD (US): GYGB



Imam TH, Fischer H, Jing B, Burchette R, Henry S, DeRose SF, Coleman KJ. Estimated GFR before and after bariatric surgery in CKD. American Journal of Kidney Diseases. 2017 Mar 1;69(3):380-8.

What are the surgical outcomes for patients with ESKD who undergo bariatric surgery?

CJASN
Clinical Journal of American Society of Nephrology

Retrospective cohort study

MedPAR: Medicare claims database

Bariatric surgery recipients
N=2698 ESKD
N=201,104 non-ESKD

2006-2016

Bariatric surgery in patients with ESKD increased

9X

Bariatric procedure choice in ESKD patients

	2006	2016
Lap sleeve gastrectomy	<1%	84%

Risk adjusted outcomes

	Complications	Length of stay	Readmission
ESKD	3.4% [2.5-4.2]	2.2 [2.1-2.4]	8.6% [7.3-10.0]
Non-ESKD	3.6% [3.4-3.9]	1.9 [1.8-2.1]	5.4% [5.1-5.5]

Conclusions Laparoscopic sleeve gastrectomy is the most common bariatric surgical procedure in patients with ESKD. There is a favorable complication profile in sleeve gastrectomy patients.

Kyle H. Sheetz, Kenneth J. Woodside, Vahakn B. Shahinian, Justin B. Dimick, John R. Montgomery, and Seth A. Waits. ***Trends in Bariatric Surgery Procedures Among Patients with ESKD in the United States.*** CJASN DOI: https://doi.org/10.2215/CJN.01480219. **Visual Abstract by Michelle Rheault, MD**

Barriers to Treatment

Perceptions of barriers obesity management in Canada

Clinical Obesity, Volume: 9, Issue: 5, First published: 11 July 2019, DOI: (10.1111/cob.12329)

Weight Bias In Healthcare: Perception

Physicians were rated the second most common source of obesity bias

53% of women with obesity received inappropriate comments from physicians regarding their weight

Younger women reported more stigma than older women

Participants also reported weight stigma from nurses (46%), dieticians (37%), and mental health practitioners (21%)

Weight Bias In Healthcare

40% of physicians reported a negative reaction toward a patient with obesity

>50% of primary care physicians regarded patients with obesity as "**awkward, unattractive, ugly, and non-compliant.**"

Primary care physicians spend less time with obese patients compared with thin patients

Implicit bias is similar in health professionals specializing in obesity

Stigma

Weight Stigma: stereotypes and labels we assign to patients with obesity.

$$\textbf{Treatment Access} \propto 1 / \textbf{Stigma}$$

How do we improve treatment access?

Improving Treatment Access

1. Broad recognition that stigma undermines prevention & treatment

1. Have public health policies that identify stigma as a barrier to care

1. Funding toward research and programs to reduce stigma

1. Media buy-in for more compassionate portrayals of the individual

Weight Bias Starter Kit: Language

People-First Language

Rather than: *"Mr. A is a 50 y/o obese male"*

Use: *"Mr. A is a 50 y/o male with obesity"*

Use Neutral Terms

Rather than*: fat, obese, heavy set*

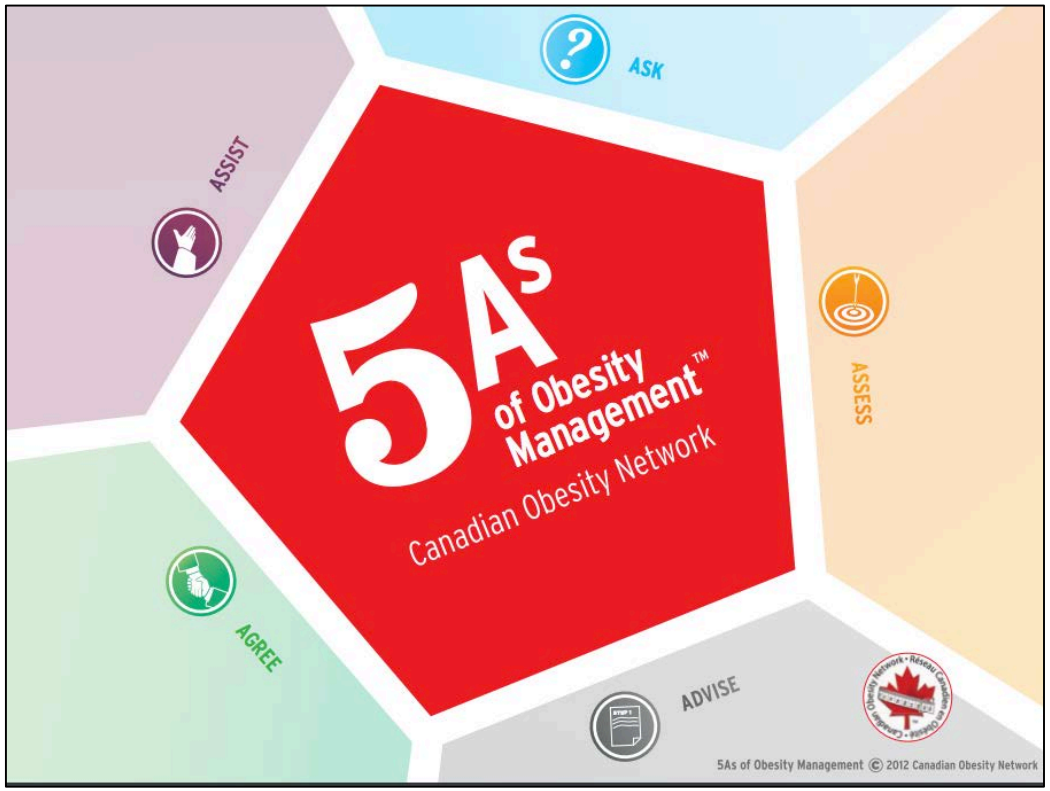
Use*: excess weight, unhealthy weight, BMI*

Avoid Blaming Language

Rather than: *"have you thought about losing weight?"*

Use: *"can we talk about your weight today?" or "how do you feel about your weight?"*

Obesity Management Starter Kit



1. Ask: for permission to discuss weight
1. Assess: obesity class, causes, complications
1. Advise: obesity risks and treatment benefits & options
1. Agree: realistic expectations & sustainable weigh loss goals
1. Assist: address drivers/barriers, resources, referrals, follow-up

5 A's of Obesity Management, Obesity Canada (2012)

Expert Obesity Toolkit

https://www.canadaobesitypeersupport.com/

Summary

1. Obesity is a chronic disease with a complex underlying physiology and a high level of morbidity

2. Effective treatments exist—though its use in kidney disease is poorly studied

3. Weight bias is a barrier to effective treatment of obesity and overall care of our patients with obesity

Questions