Medical treatments for obesity

Julian Emmanuel

Case

1. 28 year old female, strong family history of diabetes, no past cardiovascular or cerebrovascular events, nor CKD/ Liver disease, feeling lethargic and falling asleep in the afternoon, finding it difficult to concentrate at work, attends GP, and was noted to have a BMI- 42.5, and random glucose of 8 mmol, Bloods: normal renal profile/ but ALT (56) and ALP (134) were high and HBA1c was 48, Triglycerides 2.6 mmol/L, with total HDL ratio – 5.4, and low HDL at 0.90 mmol/L.

2. What next

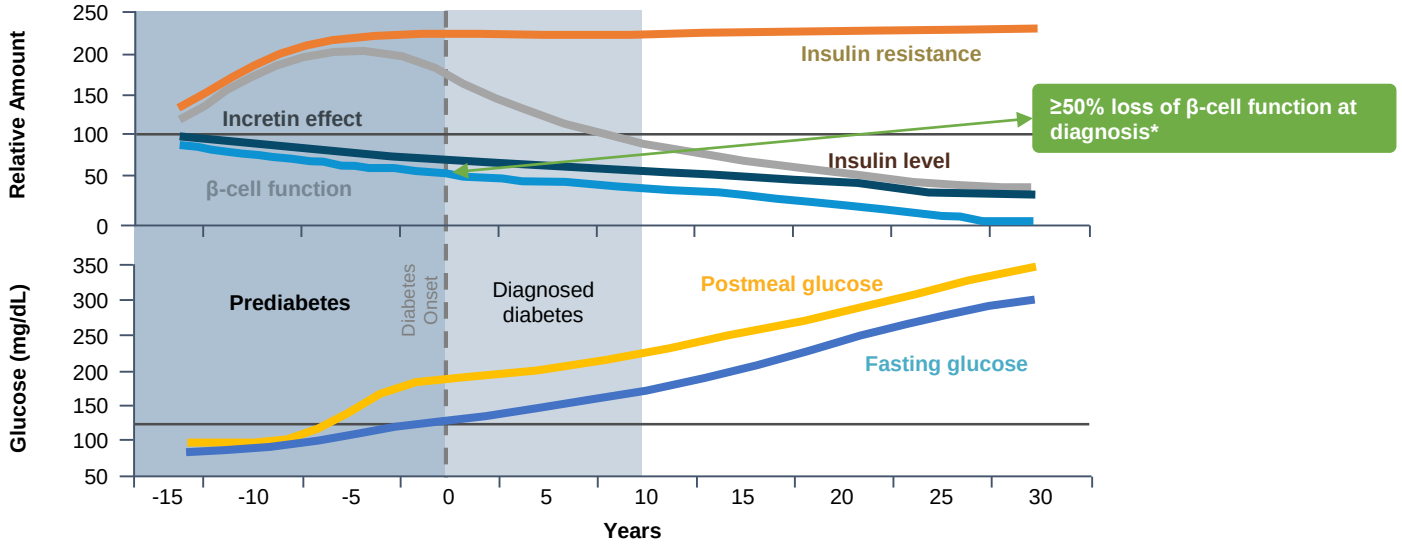
3. Anything else influence decision

4. What treatment is best first line

Metformin / GLP-1 / SGLT-2 inhibitors/ Acarbose / Insulin therapy

Diabetes (Diabesity) is a chronic and progressive disease[1–3]

1. Decline in insulin sensitivity, β-cell function, and incretin effects all occur prior to development of overt type 2 diabetes



Adapted from Kendall DM et al (2009) *Am J Med* **122**:S37–50.

*By the time diabetes is clinically diagnosed, β-cell function may be reduced by ≥50%; subjects in the upper tertile of IGT are near-maximally insulin resistant and have lost more than 80% of their β-cell function.[1–3] Representative depiction of the natural progression of type 2 diabetes (time course and function).[1]

IGT=impaired glucose tolerance.

1. Kendall DM et al (2009) *Am J Med* **122**: S37–50; 2. DeFronzo RA (2009) *Diabetes* **58**: 773–95;
3. Holman RR (1998) *Diab Res Clin Prac* **40**(suppl):S21–5

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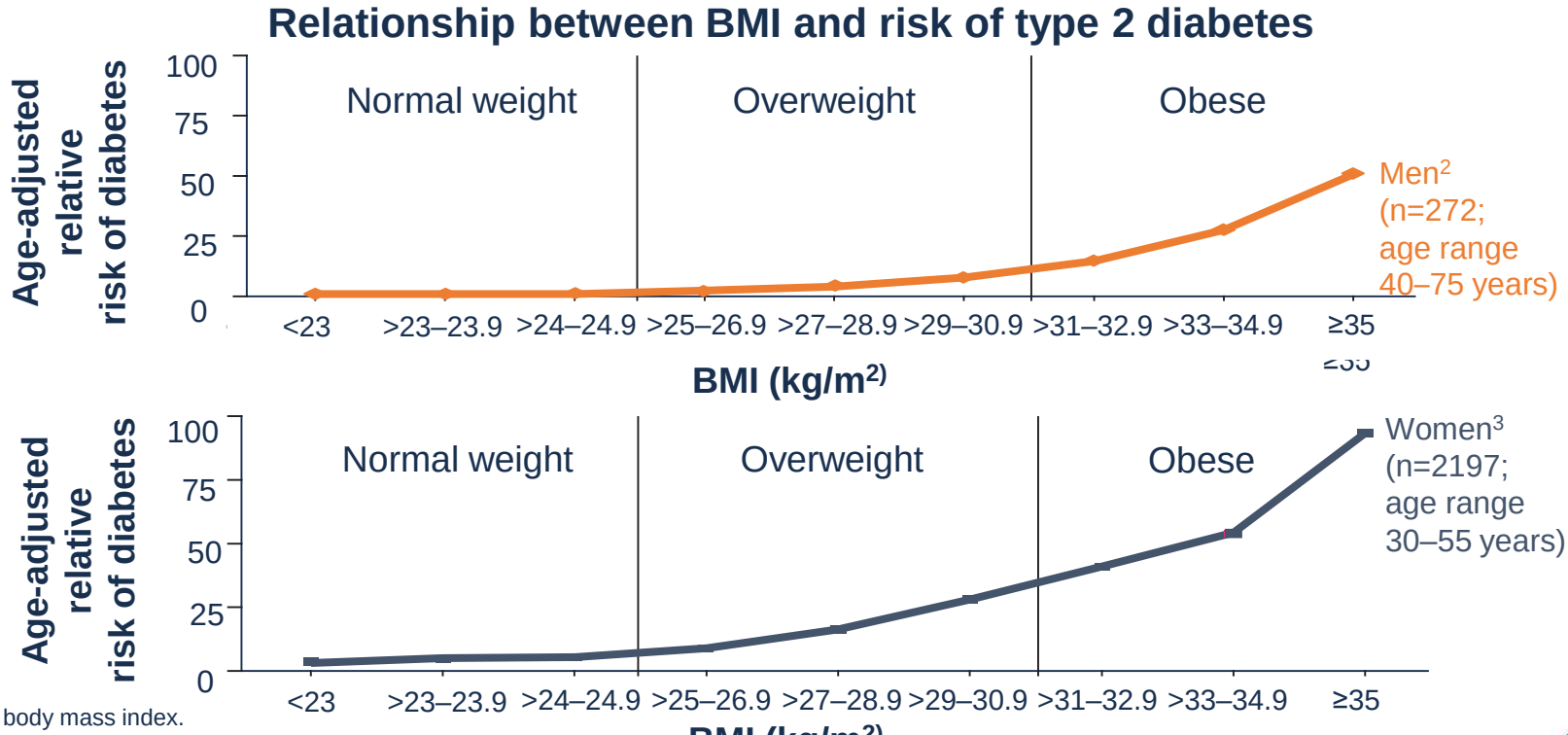
Diabetes and obesity are closely related

- 90% of individuals with type 2 diabetes are overweight or obese[1]



BMI, body mass index.

1. WHO (2003) *Obesity and overweight.* Available at: http://www.who.int/dietphysicalactivity/media/en/gsfs_obesity.pdf (accessed 29.01.2014)
1. Chan J *et al. Diabetes Care* 1994;**17**:961–9
2. Colditz GA *et al. Ann Intern Med* 1995;**122**:481–6

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Obesity prevalence

1. Obesity affects just over a quarter of adults in England (Public Health England, 2016) Retrieved from http://www.noo.org.uk/NOO_about_obesity

2. 5-10% of the population has T2DM- McCombie L et al; BMJ Sept 2017

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Guidance from Society for Endocrinology 2015 & American Society of Bariatric Physicians 2013

1. In patients with T2DM who are overweight or obese, Society for Endocrinology guidance suggest use of antidiabetic medications that have additional actions to promote weight loss

   (GLP-1/ SGLT-2i with metformin; Feb 2015 JCEM)

1. American society of Bariatric physicians, obesity algorithm 2013: Metformin, GLP-1, SGLT2i (Bays H, Obesity research 2004; Hasnain M et al, Post Grad Med 2012; Astrup A, Obesity Res 2004; Astrup A, Int J Obes Lon 2012; Bays H, Curr Med Res Opin 2009)

Emerging trials and data

- **Dapagliflozin once daily plus exenatide once weekly in obese adults without diabetes: Sustained reductions in body weight, glycaemia and blood pressure over 1 year,** Per Lundkvist MD, Maria J. Pereira PhD, Petros Katsogiannos MD, C. David Sjöström MD, PhD, Eva Johnsson MD, PhD, Jan W. Eriksson MD, PhD, jan.eriksson@medsci.uu.se orcid.org/0000-0002-2639-9481 First published: 27 March 2017https://doi.org/10.1111/dom.12954

- **A Randomized Controlled Trial of Dapagliflozin Plus Once-Weekly Exenatide Versus Placebo in Individuals with Obesity and Without Diabetes: Metabolic Effects and Markers Associated with Bodyweight Loss** Maria J. Pereira, Per Lundkvist, Prasad G. Kamble, Joey Lau, Julian G. Martins, C. David Sjöström, Volker Schnecke, Anna Walentinsson, Eva Johnsson & Jan W. Eriksson ORCID: orcid.org/0000-0002-2639-9481

- Sodium-glucose cotransporter (SGLT) 2 inhibitors for prevention or delay of type 2 diabetes mellitus and its associated complications in people at risk for the development of type 2 diabetes mellitus; Bianca Hemmingsen , Jesper Krogh, Maria-Inti Metzendorf, Bernd Richter, Department of Internal Medicine, Herlev University Hospital, Herlev, Denmark. Department of Endocrinology, Herlev University Hospital, Herlev, Denmark. Cochrane Metabolic and Endocrine Disorders Group, Institute of General Practice, Medical Faculty of the Heinrich-Heine-University Düsseldorf, Düsseldorf, Germany

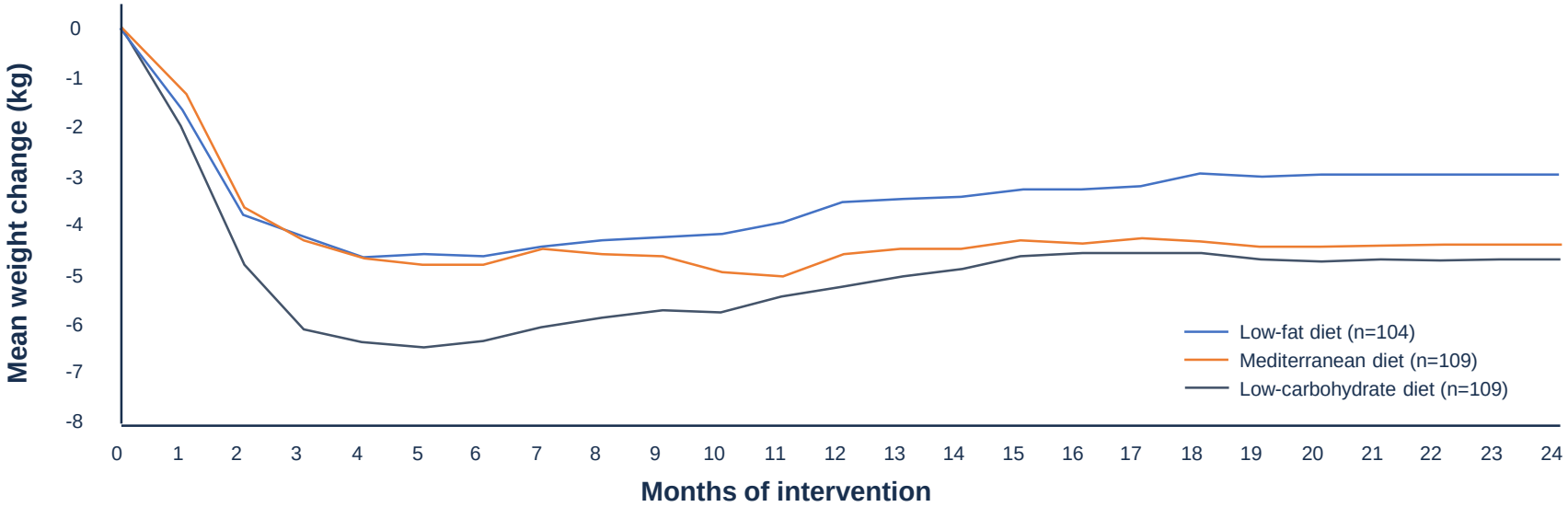
Pharmacotherapy- Recent evidence

1. Recent RCT show GLP1 agonist liraglutide to be efficacious in a dose dependent manner for weight loss in both diabetics and non-diabetics.(Pi-Sunyer, Astrup, & Fujioka, 2015, Vilsbøll, Christensen, Junker, & Knop, 2012, Astrup, Rössner, Gaal, & Rissanen, 2009)

2. In diabetic patients Sodium-glucose co-transporter 2 inhibitors (SGLT2i) are associated with weight loss which is significantly increased when used in conjunction with a GLP1 agonist (Pinto, Rados, & Remonti, 2015, Lundkvist, Sjöström, & Amini, 2017, Guja, Hardy, Ahmed, & Dong, 2016)

3. Emerging Role of SGLT-2 Inhibitors for the Treatment of Obesity, Drugs. 2019; 79(3): 219–230. Published online 2019 Jan 30. doi: 10.1007/s40265-019-1057-0

Different dietary compositions and their effect on weight loss



Baseline mean weight (all, n=322) = 91.4 kg

P<0.001 for both comparisons with the low-fat diet

In this 2-year trial, 322 moderately obese subjects (mean age 52 years, mean BMI 31 kg/m^2, mean baseline weight 91.4 kg, male 86%) were randomly assigned to one of three diets: Low fat, restricted calorie; Mediterranean, restricted calorie; or low carbohydrate, non-calorie restricted.
BMI: body mass index.
Shai I, *et al*. *N Engl J Med* 2008;**359**:229–41

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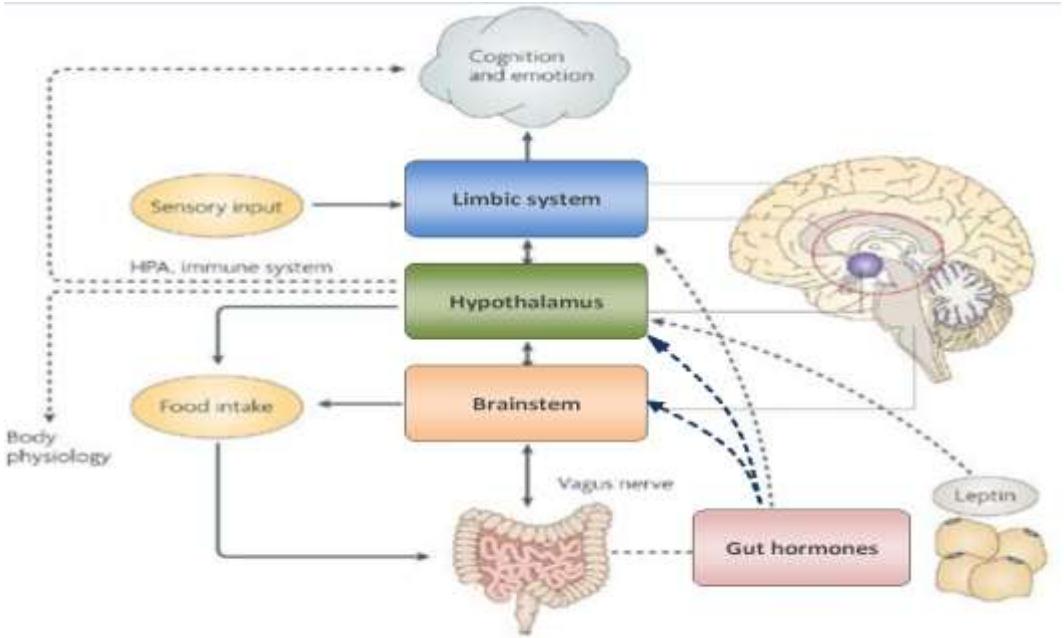
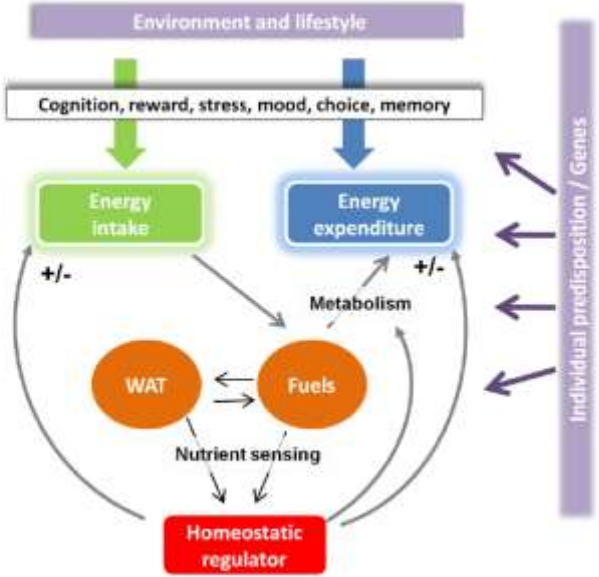
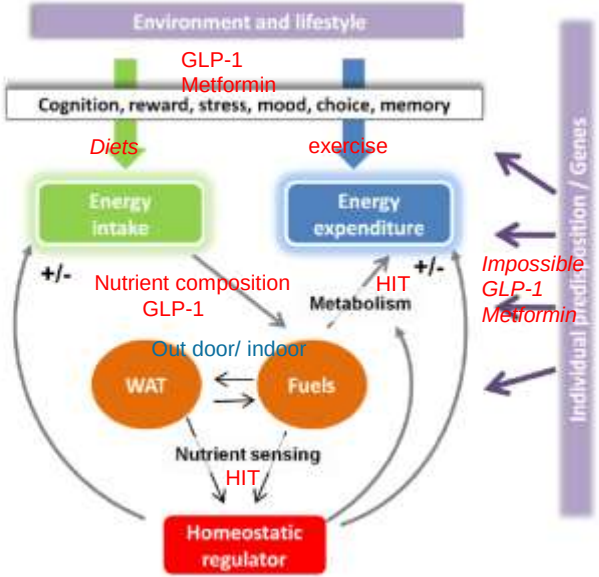


Complex neural circuitry governs many aspects of energy balance. Information from the periphery is conveyed by circulating hormones and vagal afferents to the caudal brainstem, hypothalamus and cortico-limbic brain regions. Cortico-limbic regions integrate meal-related sensory input and nutrient information with internal emotional factors. Together, these circuits regulate ingestive behaviour and bodyweight. (Adapted from Gomez-Pinilla, 2008)



The control of energy balance is a complex process requiring bidirectional integration of information regarding nutrient status and body fat stores with homeostatic circuits in the central nervous system. A homeostatic regulator is thought to modulate energy intake and expenditure. Environment and genetics further influence the control of homeostatic processes. (WAT = white adipose tissue) (Adapted from Lenard and Berthoud, 2008)



The control of energy balance is a complex process requiring bidirectional integration of information regarding nutrient status and body fat stores with homeostatic circuits in the central nervous system. A homeostatic regulator is thought to modulate energy intake and expenditure. Environment and genetics further influence the control of homeostatic processes. (WAT = white adipose tissue) (Adapted from Lenard and Berthoud, 2008)

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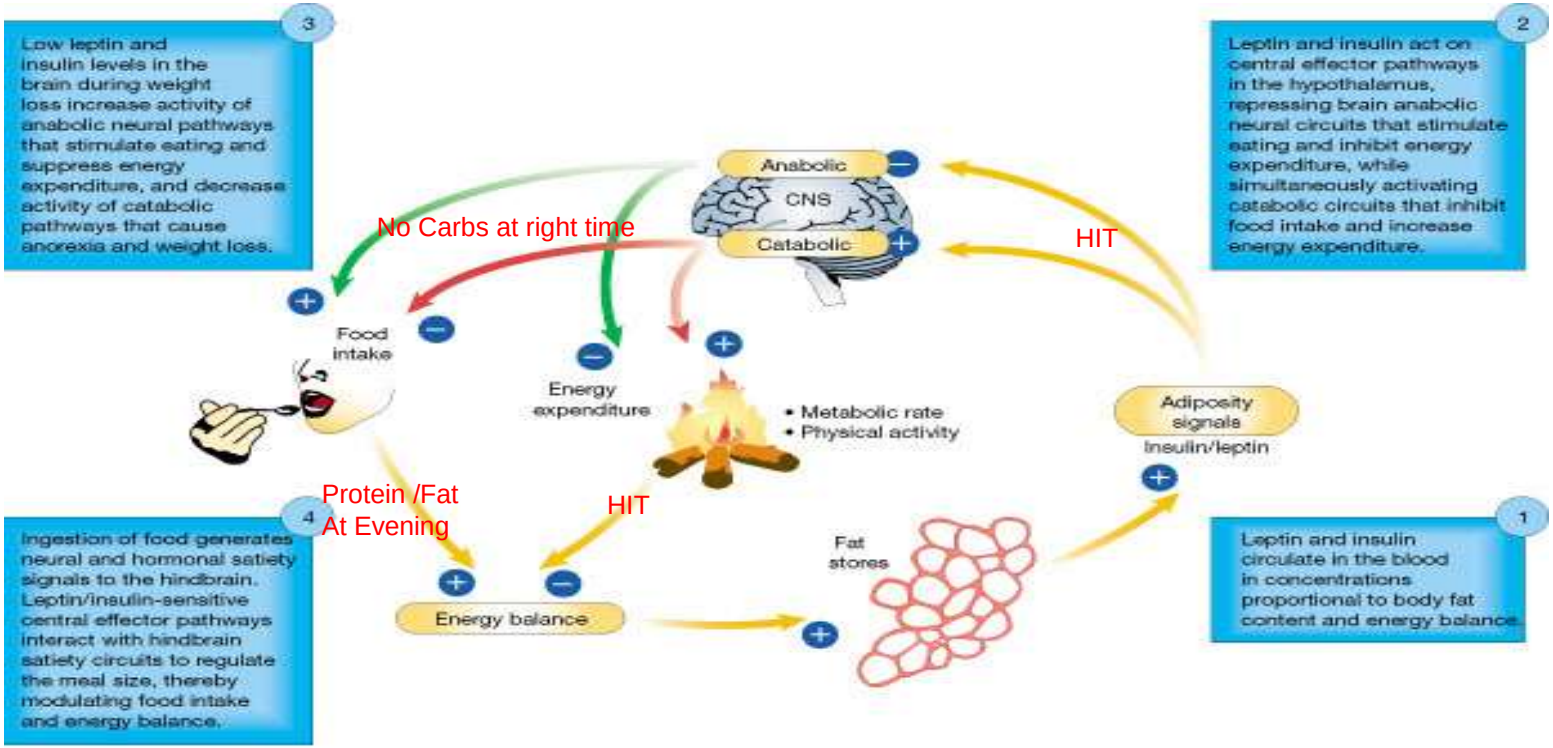
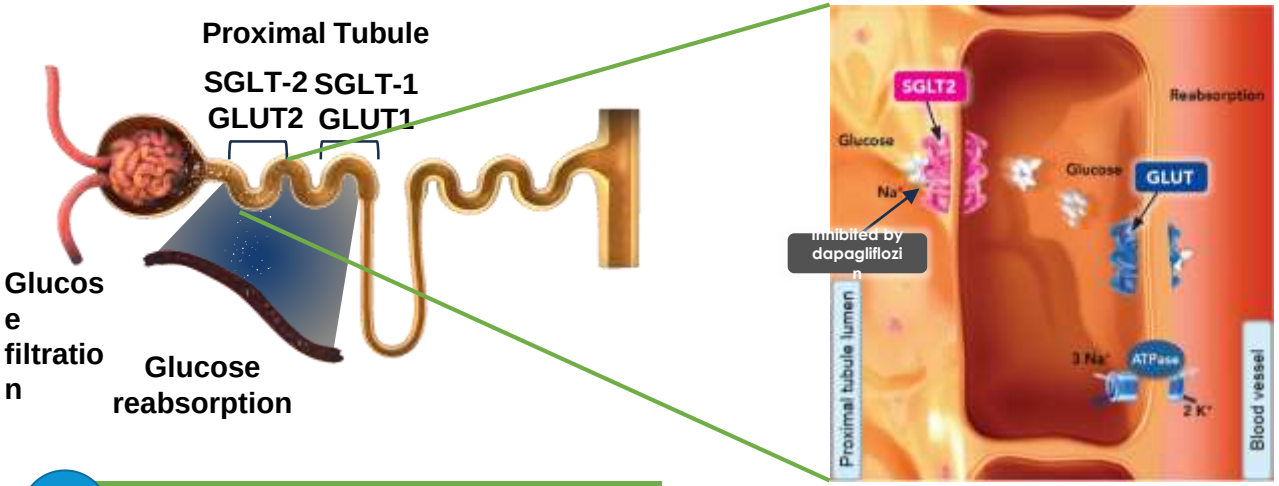


The central role of leptin and insulin in energy balance is displayed in this schematic diagram. Leptin and insulin are secreted in proportion to body fat stores. They act on the homeostatic (hypothalamus and brainstem) and reward pathways to modulate energy balance. A reduction in adiposity leads to a compensatory changes in anabolic and catabolic pathways. This in turn maintains adiposity. This diagram was reproduced from a recent publication by Schwartz and colleagues (Schwartz MW et al 2000).

Physiologic Actions and Effects of SGLT-2 Inhibitors



Effect of SGLT-2i

- ↑ Urinary glucose excretion
- ↓ HbA_{1C} levels
- ↓ FPG and PPG
- ↓ Body weight
- ↓ Systolic blood pressure
- ↓ Vascular stiffness

* For illustrative purposes only.

1. Bays H. *Curr Med Res Opin*. 2009;25;671-681. 2. Abdul-Ghani MA et al. *Endocr Pract*. 2008;14:782-790. 3. Marsenic O. *Am J Kidney Dis*. 2009;53:875-883. 4. Mather A et al. *Kidney Int*. 2011;79(suppl 120):S1-S6. 5. Forxiga SmPC available at www.medicines.co.uk. Last Accessed Sep 20th 2016. 6. Inzucchi SE. *Diab Vasc Dis Res*. 2015;12(2):90-100. 7. Asano T et al. *Curr Med Chem.* 2004;11:2717-2724.

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Phase 2 Trials in SGLT2

1. Dapagliflozin- **DB06292,** BMS-512148: Increased the amount of glucose excreted in the urine in healthy subjects and in patients with T2DM. ClinicalTrials.gov Identifier: NCT00162305

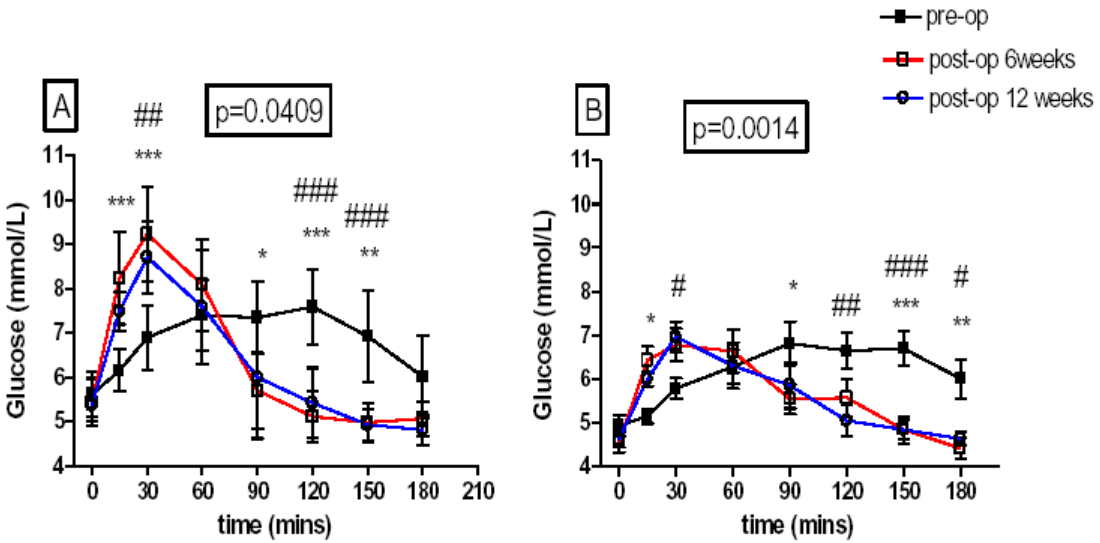
Canagliflozin	Dapagliflozin	Empagliflozin
SGLT 2 inhibitor least selective	Intermediate	SGLT2 inhibitor most selective
110- 120 g/ day glucose loss	70 g/ day loss	40 g/day loss

Phase 2 Trials in SGLT2

1. Dapagliflozin- **DB06292,** BMS-512148: Increased the amount of glucose excreted in the urine in healthy subjects and in patients with T2DM. ClinicalTrials.gov Identifier: NCT00162305

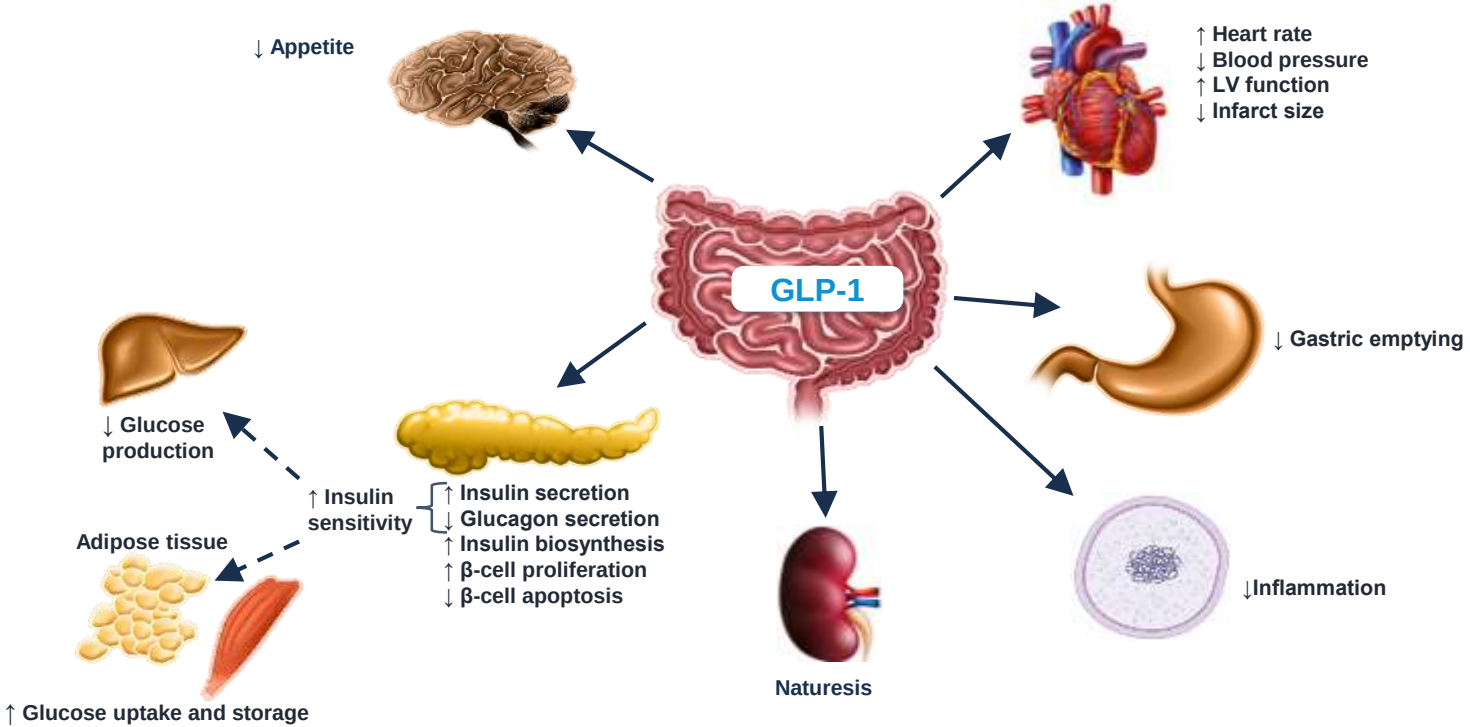
2. A Dapagliflozin10 mg day in patients with T2DM, for 12 weeks, resulted in excretion of approximately 70 grams of glucose in the urine per day at week 12.

Why SGLT2i
RYGBP (A) SG (B)



1. *Comparison of plasma glucose following the standard liquid meal in the RYGBP (A) and SG (B) groups are shown. There was a significant (two way matched ANOVA, p=0.0409) decline in the temporal profile of glucose, comparing pre-operative time point to post-operative time points after RYGBP. Bonferroni post test analysis did confirm significant decline at t=15 (p<0.001), t=30 (p<0.001), t=90 (p<0.05), t=120 (p<0.001), t=150 (p<0.01) at 6 weeks after surgery, and at t=30 (p<0.01), t=120 (p<0.001), t=150 (p<0.001) at 12 weeks after surgery. There was also a significant (two way matched ANOVA, p=0.0014) decline in the temporal profile of glucose after SG, comparing pre-operative time point to post-operative time points. Bonferroni post test analysis confirm a significant decline in glucose at t=15 (p<0.05), t=90 (p<0.05), t=150 (p<0.001), t=180 (p<0.01) at 6 weeks after surgery, and at t=30 (p<0.05), t=120 (p<0.01), t=150 (p<0.001), t=180 (p<0.05) at 12 weeks after surgery. Over the three visits: *p<0.05, ** p<0.01, ***p<0.001 at 6 weeks, # p<0.05, ## p<0.01, ### p<0.001 at 12 weeks, reproduced from JE thesis 2015- UCL*

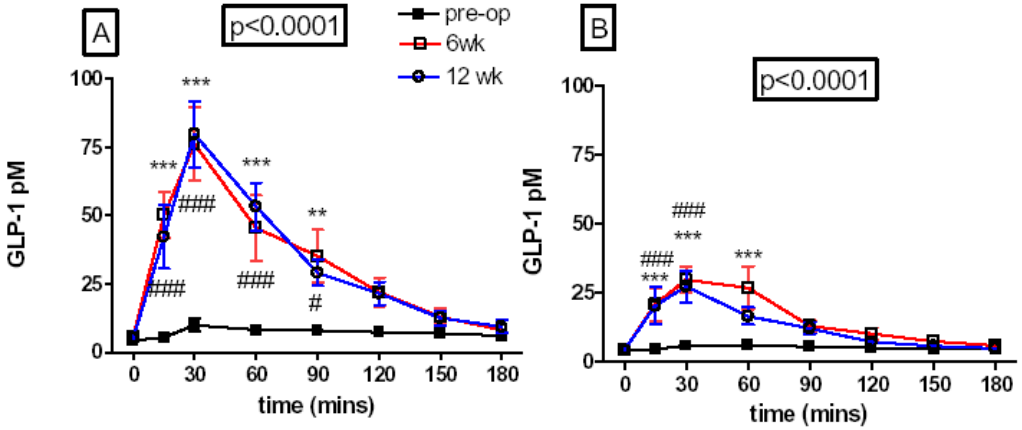
Physiologic Actions and Effects of GLP-1 Receptor Agonists



1. Drucker DJ, et al. *Diabetes*. 2015;64:317-326. 2. Campbell JE, et al. *Cell Metab.* 2013;17:819-837.

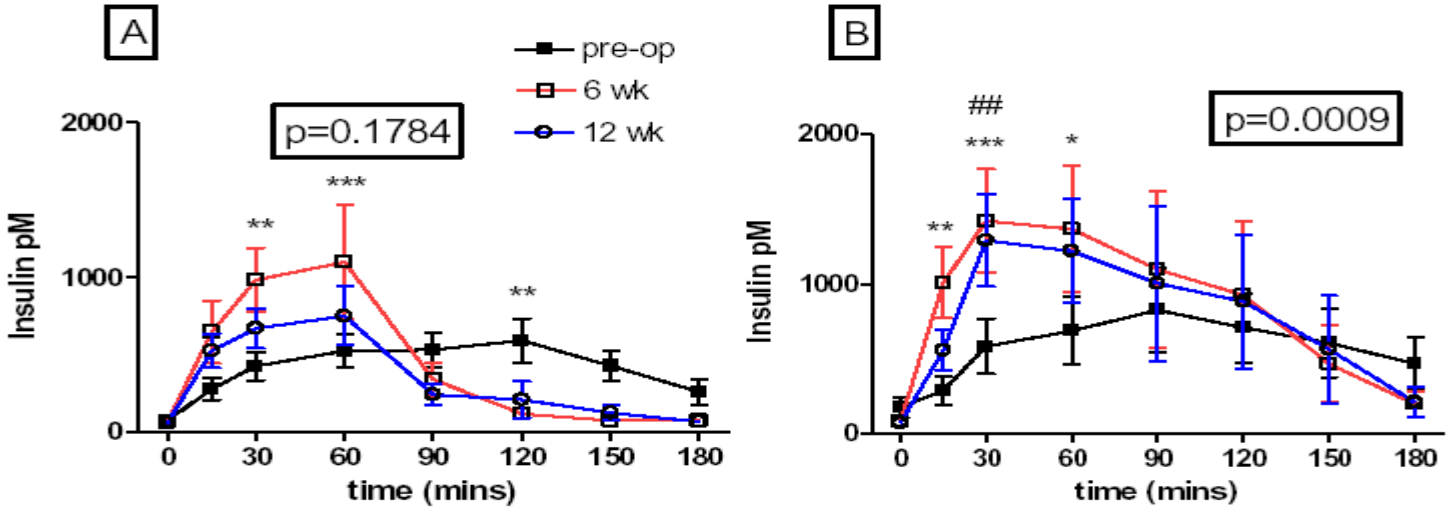
3. Baggio LL et al. *Gastroenterology*. 2007;132:2131-2157. 4. Ussher JR, et al. *Circ Res.* 2014;114:1788-1803.

Why GLP-1 analogues RYGBP (A) SG (B)



1. *Analysis of the plasma temporal profile of GLP-1 after a mixed meal test utilising a matched two-way ANOVA, comparing pre-operative time point to post-operative time points did show a significant increase after both RYGBP (p<0.0001) and SG (p<0.0001). Bonferroni post-hoc analysis did show significant increase at t=15 (p<0.001), 30 (p<0.001), 60 (p<0.001), and t=90 (p<0.01) at 6 weeks, and at t=15 (p<0.001), 30 (p<0.001), 60 (p<0.001) and at t=90 (p<0.05) at 12 weeks after RYGBP. This analysis in the SG group did also show significant increase at t=15 (p<0.001), 30 (p<0.001), 60 (p<0.001) at 6 weeks, and at t=15 (p<0.001), t=30 (p<0.001) at 12 weeks (figure-40). Over the three visits: *p<0.05, ** p<0.01, ***p<0.001 at 6 weeks, # p<0.05, ## p<0.01, ### p<0.001 at 12 weeks- Metabolic hormones in bariatric surgery and reward behaviour, JE Thesis UCL, 2015*

Insulin profile before and after weight loss surgery: RYGBP (A) and SG (B)



1. *Comparison of plasma insulin concentrations after the liquid meal following RYGBP (A) and SG (B) groups are shown. There is no change in the (two way matched ANOVA, p=0.1784) temporal profile of glucose, comparing pre-operative time point to post-operative time points after RYGBP. Bonferroni post test analysis did confirm a significant increase at t=30 (p<0.01), t=60 (p<0.001), and a significant decline at t=120 (p<0.01) at 6 weeks after surgery. The analysis at 12 weeks after surgery did not identify any significant increase or decline at any time points. There is a significant (two way matched ANOVA, p=0.0009) increase in the temporal profile of glucose after SG, comparing pre-operative time point to post-operative time points. Bonferroni post-test analysis confirms an increase at t=15 (p<0.01), t=30 (p<0.001), t=60 (p<0.05) at 6 weeks after surgery and at t=30 (p<0.01) at 12 weeks after surgery. Over the three visits: *p<0.05, ** p<0.01, ***p<0.001 at 6 weeks, # p<0.05, ## p<0.01, ### p<0.001 at 12 weeks reproduced from JE thesis 2015 UCL*

GLP-1 glucoregulatory effects in humans

Enhances satiety[1]

Suppresses postprandial glucagon secretion, which decreases hepatic glucose production[1,2]

Slows gastric emptying[1]

Stimulates glucose-dependent insulin secretion[1]

GLP-1 glucagon-like peptide-1.

1. Drucker DJ, Nauck MA. *Lancet* 2006;**368**:1696–705; 2. Larsson H, *et al. Acta Physiol Scand* 1997;**160**:413–22.

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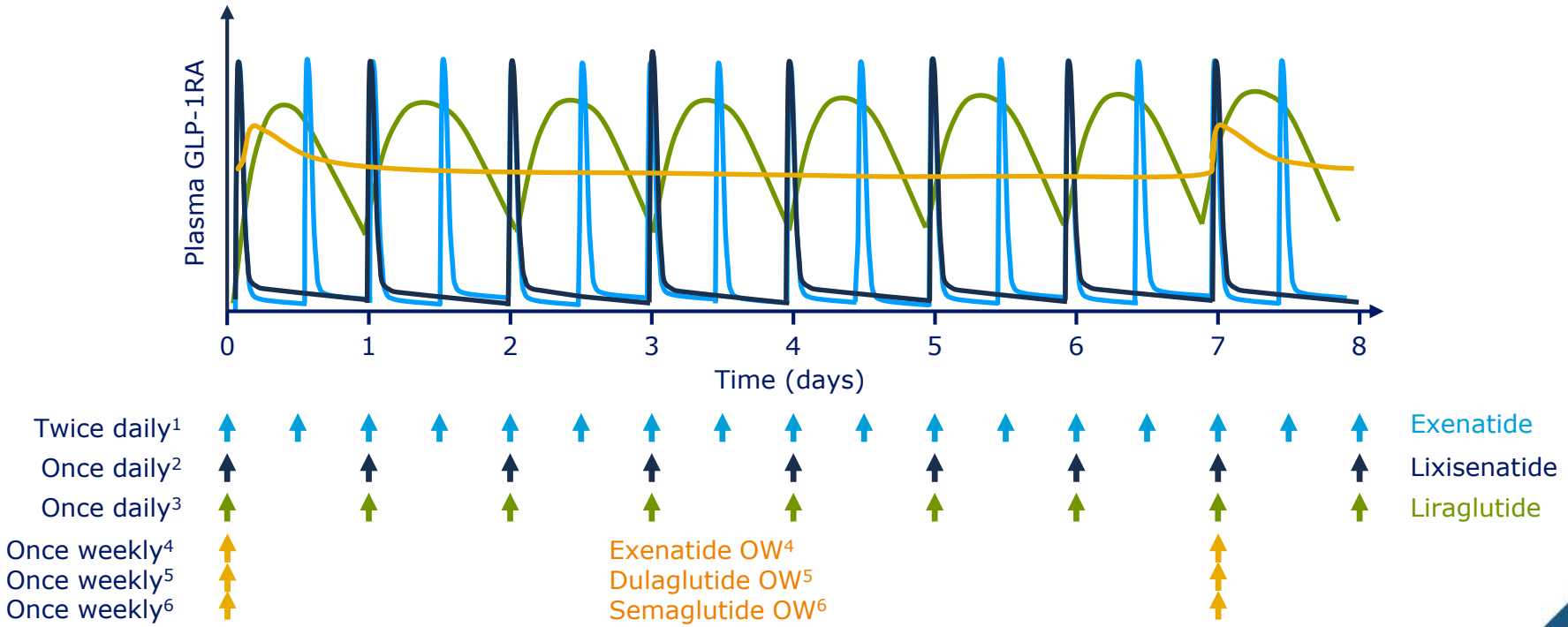
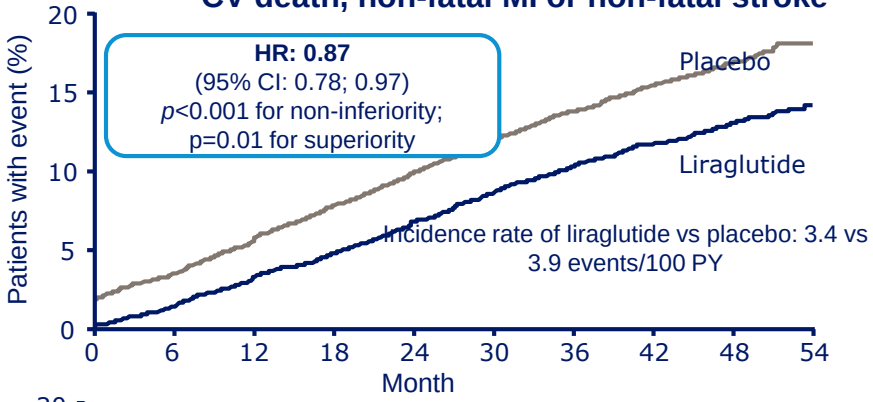
Broad illustration of different pharmacokinetics of GLP-1RAs at steady state



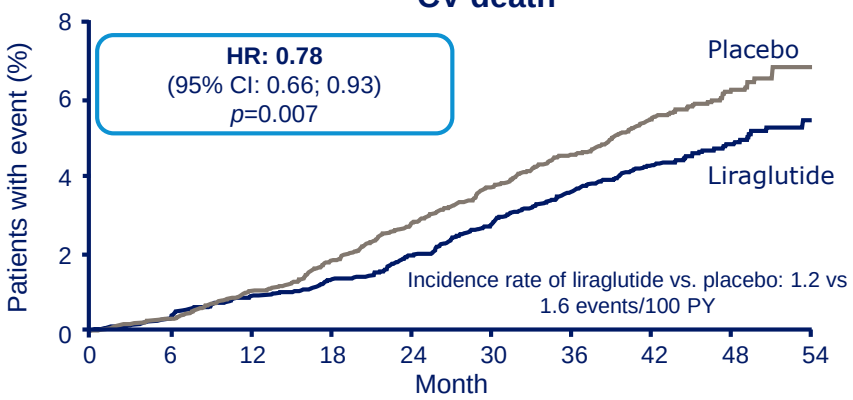
LEADER outcomes

job bag no: SAGB.TJO.19.10.1725
DOP: OCT 2019

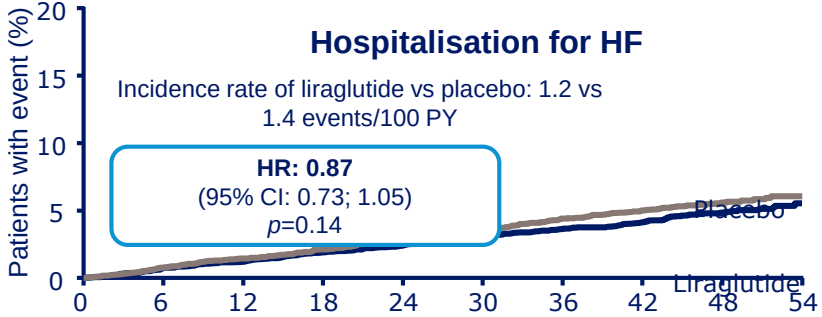
CV death, non-fatal MI or non-fatal stroke



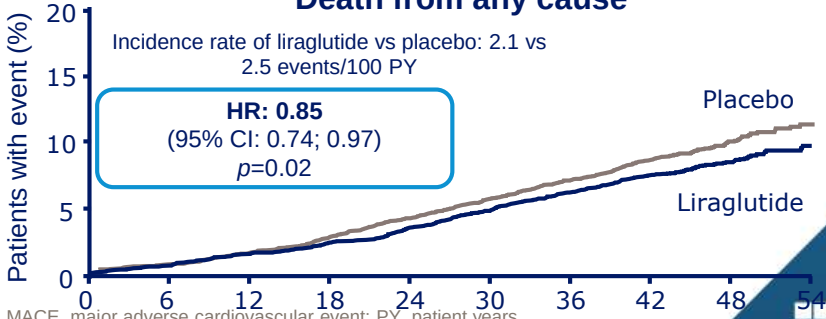
CV death



Hospitalisation for HF



Death from any cause



CI, confidence interval; CV, cardiovascular; EAC, event adjudication committee; HF, heart failure; HR, hazard ratio; MACE, major adverse cardiovascular event; PY, patient years

Adapted from Marso *et al. N Engl J Med* 2016;375:311–22

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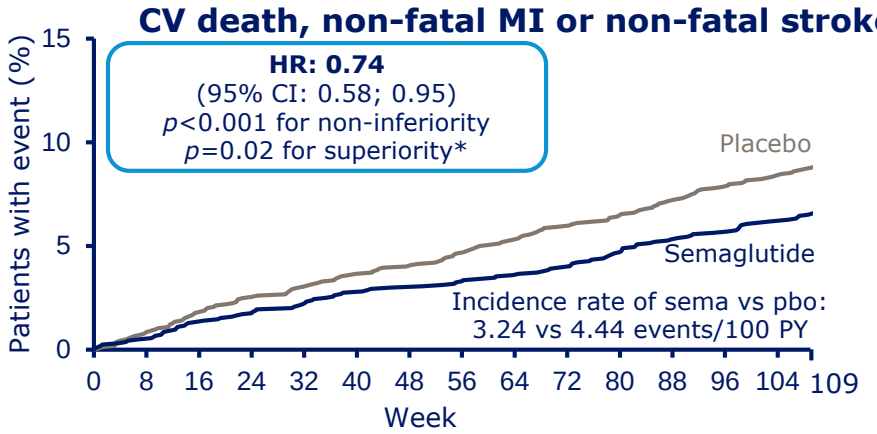
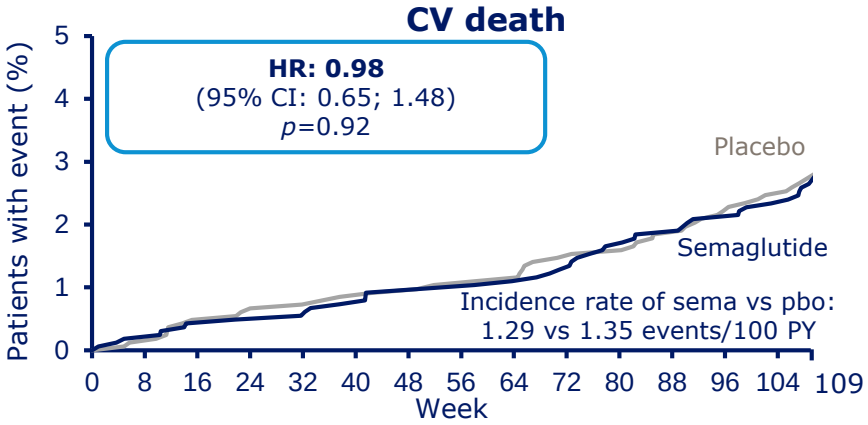
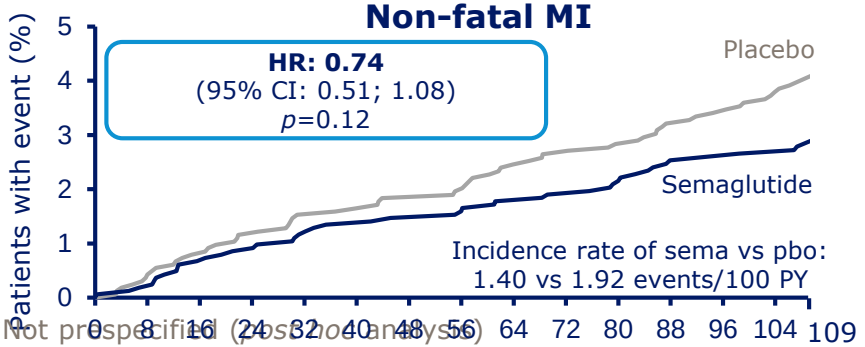
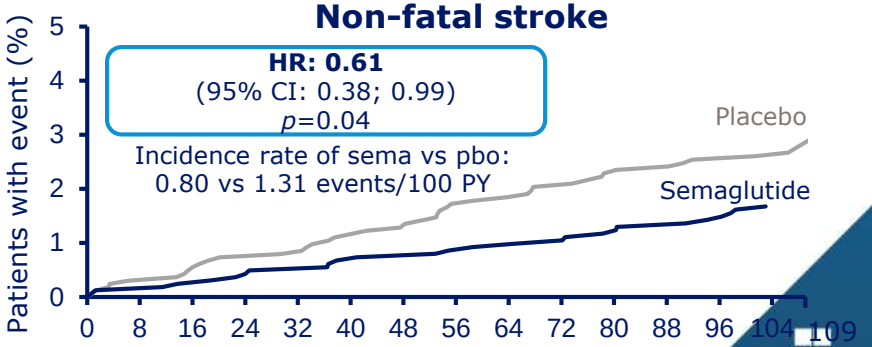
SUSTAIN outcomes









*Not prespecified (*post hoc* analysis)

CI, confidence interval; CV, cardiovascular; HR, hazard ratio; MI, myocardial infarction; Pbo, placebo; PY, patient-year; Sema, semaglutide Adapted from Marso *et al. N Engl J Med* 2016;375:1834–44

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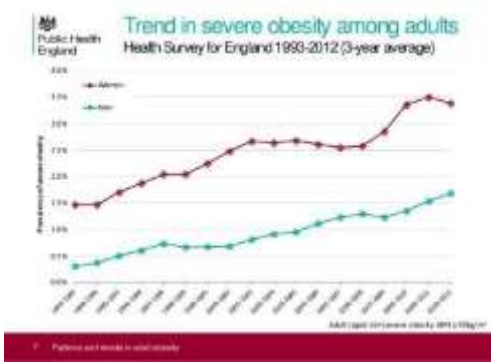
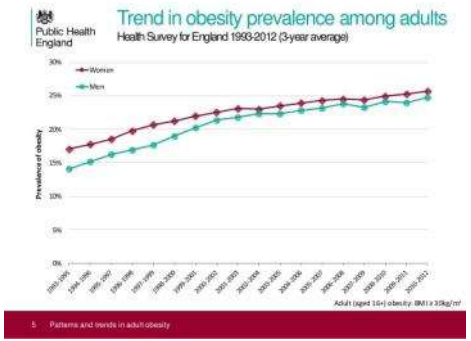
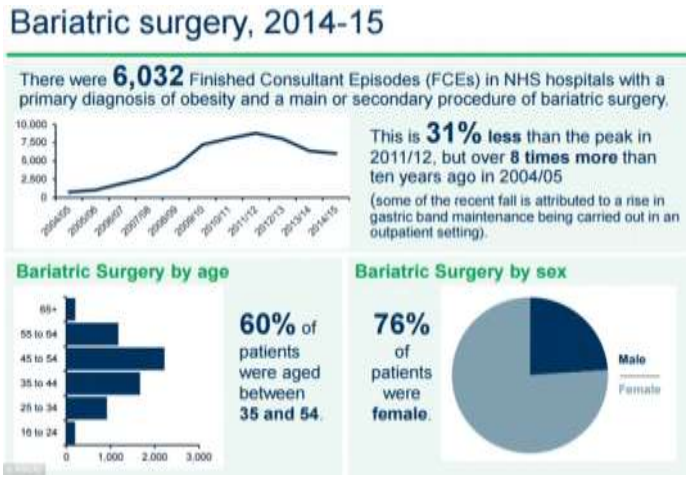
Mild and moderate obesity



The obesity epidemic has reached the UK, with over one million hospital admissions for obesity last year- 2020
If BMI>50 kg/m2, surgical management can be considered as first-line treatment option.
Eligible- 33/1000- 1,980,000 (house of commons library January 2021)





Daily Mail 28 April 2016

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Methods

1. 506 patient records examined between January 2014 and September 2016.
2. Of these 204 met the inclusion criteria of being in one of the treatment groups and having at least two BMI readings available.
3. Data treated nonparametrically, corrections for multiple comparisons- Bonferonni method.
4. Median time adjusted ratio of BMI change calculated for: Metformin alone, metformin + GLP, metformin + SGLT2i, or metformin + GLP + SGLT2i (triple).

Baseline characteristics

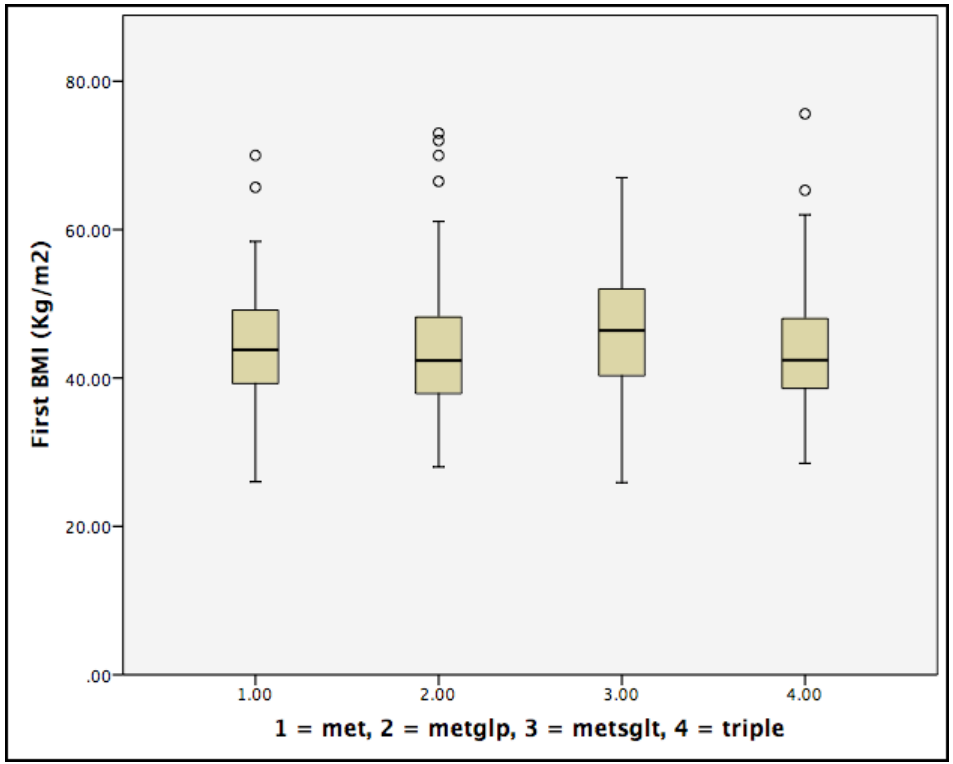
Group	N	Gender F/M	Median BMI loss per year	Mean Age (SD)	Time in clinic	First BMI mean (SD)
met	92	55/37	-0.005	46.1 (12.2)	467 (749)	46.1 (19.7)
metglp	88	65/23	0.0191	46.8 (14.1)	959 (4143)	43.9 (8.9)
metsglt2	45	32/13	0.0137	46.6 (8.8)	601 (874)	46.6 (8.8)
triple	54	37/17	0.0194	43.9 (9.1)	646 (826)	43.9 (9.1)

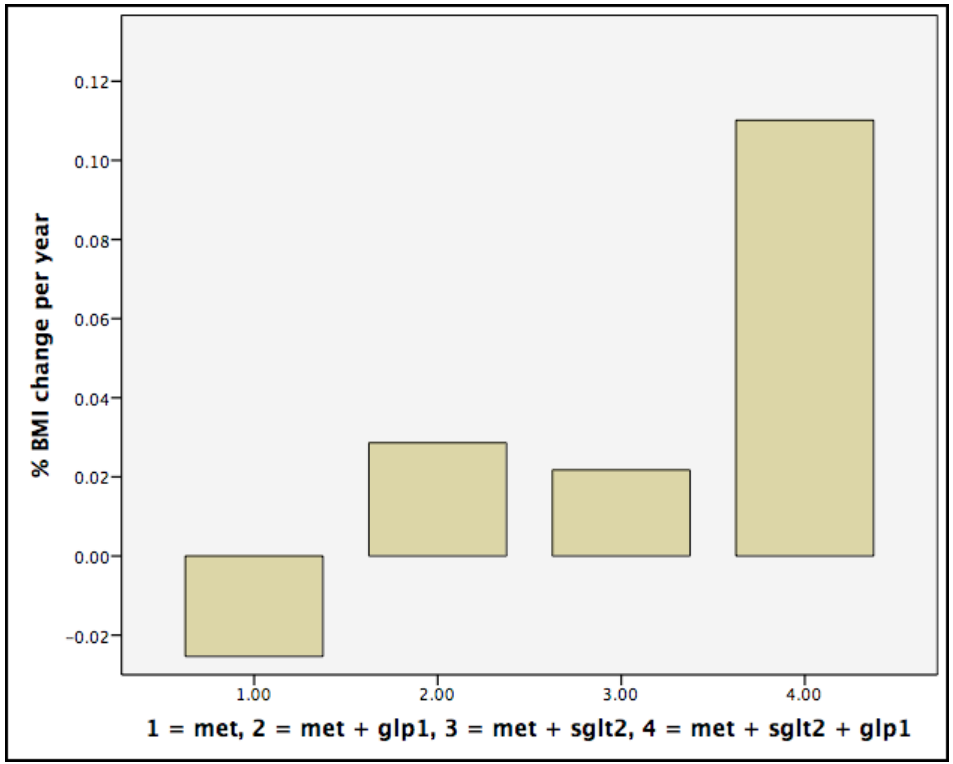
Analysis

1. Non-parametric ANNOVA- Kruskal-wallis test, based on rank value

2. The data is not normally distributed on a histogram

3. The data also failed the Shapiro-wilks test.


80.00
60.00
40.00
20.00
.00
First BMI (Kg/m2)
1.00
2.00
3.00
4.00
1 = met, 2 = metglp, 3 = metsglt, 4 = triple


% BMI change per year
0.12
0.10
0.08
0.06
0.04
0.02
0.00
-0.02
1.00
2.00
3.00
4.00
1 = met, 2 = met + glp1, 3 = met + sglt2, 4 = met + sglt2 + glp1

Analysis- non parametric ANNOVA

BMI						
Test used: independent samples kruskal-wallis			(rank based non-parametric annova)			
Null hypothesis: The distribution of ratio BMI change per year is the same across metformin, met + GLP, met + sglt2 and triple						
p = 0.016	reject the null hypothesis					
Pairwise comparisons (with bonferoni correction for multiple comparisons)						
	p value					
met vs metglp	p = 0.254					
met vs metsglt2	p = 0.339					
met vs triple	p = 0.017					

Results

1. There was a significant difference between distributions of % change in BMI per year between the treatment groups (p = 0.016).
2. An inspection of pairwise tests revealed a significant comparison between triple therapy and metformin (median % BMI change per year 1.94 vs -0.495, p = 0.017 (0.017 after adjustment for multiple comparisons)
3. None of the other pairwise comparisons were significant (median % BMI change 1.91, 1.37 for Metformin + GLP/Metformin + SGLT respectively).
4. Patients on metformin as single therapy gained weight (median increase 0.5% in BMI per year).

Discussion

1. Not an RCT, a retrospective audit

2. Under-estimate the rate of weight loss per year- start time of oral hypo-glycaemic agent not known

3. Not a standardized approach to escalation in therapy, rather patient preference and clinician choice- Real World Evidence

Conclusions

1. Notable because our cohort suffers from more severe obesity than those from recent RCTs.

1. Likely to underestimate the rate of BMI loss per year, as we over-estimated the time on therapy, as exact time started on therapy not known, and thus HBA1c at start not available.

2. Despite this the result remains significant.

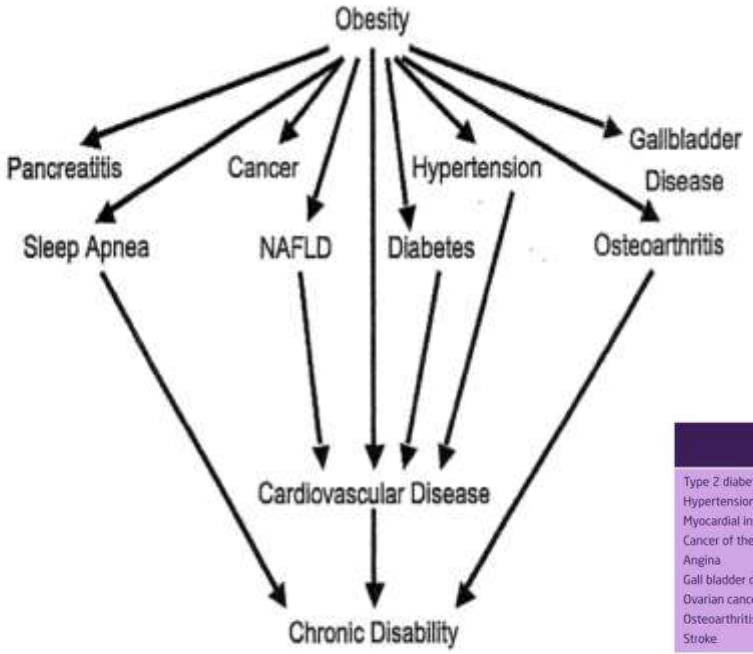
Conclusion: Combination therapy in T2DM and pre-diabetes with obesity may become the norm

1. SGLT2i/ GLP-1 dual therapy reduced body weight, others have shown reduced frequency of prediabetes and SBP over 24 weeks and was well tolerated in obese adults without diabetes (Lundkvist P et al 2016- Diabetes obesity and metabolism, Lundkvist et al 2017).

2. Greater weight loss in the combination treatment group compared to either therapy alone, greater proportion of patients with weight loss of 5% or more (Frias J P et al 2016, Lancet diabetes)

Obesity related co-morbidity

Julian Emmanuel and Simon Coppack, *Obesity, Bariatric and Metabolic Surgery: A Practical Guide,* published by Springer.



Abbreviation: NAFLD, nonalcoholic fatty liver disease.

	Numbers	
	Men	Women
Type 2 diabetes	5.2	12.7
Hypertension	2.6	4.2
Myocardial infarction	1.5	3.2
Cancer of the colon	3.0	2.7
Angina	1.8	1.8
Gall bladder diseases	1.8	1.8
Ovarian cancer	–	1.7
Osteoarthritis	1.9	1.4
Stroke	1.3	1.3

Obesity and Cancer risk;

Julian Emmanuel and Simon W Coppack, **Obesity, Bariatric and Metabolic Surgery**

A Practical Guide

Editors Sanjay Agrawal

Springer publishing, 2018

Cancer type	Relative risk Men	Relative risk women	Suggested causal mechanism
Endometrium		1.59	Estrogen excess
Adenocarcinoma esophagus	1.52	1.51	GERD, barrett's esophagus
Thyroid	1.33	1.14	
Adenocarcinoma colon	1.24	1.09	Hyperinsulinemia and/or IGF-1
Renal	1.24	1.34	Hypertension partly
Hepatoma	1.24	1.07	NAFLD, cirrhosis
Breast, estrogen receptor positive		1.18	Estrogen excess
Malignant melanoma	1.17	0.96	
Multiple myeloma	1.11	1.11	Inflammatory cytokines, e.G. Il-6
Rectum	1.09	1.02	
Gall bladder	1.09	1.59	Gall stones
Leukemia	1.08	1.17	Inflammatory cytokines, e.G. Il-6
Pancreas	1.07	1.12	
Non-hodgkin's	1.06	1.07	Inflammatory cytokines, e.G. Il-6
Breast, estrogen receptor negative		1.03	Inflammatory cytokines, e.G. Il-6
Ovary		1.03	
Prostate	1.03		
Stomach	0.97	1.04	
Lung	0.76	0.80	Negative association with smoking
Squamous esophageal	0.71	0.57	Negative association with smoking

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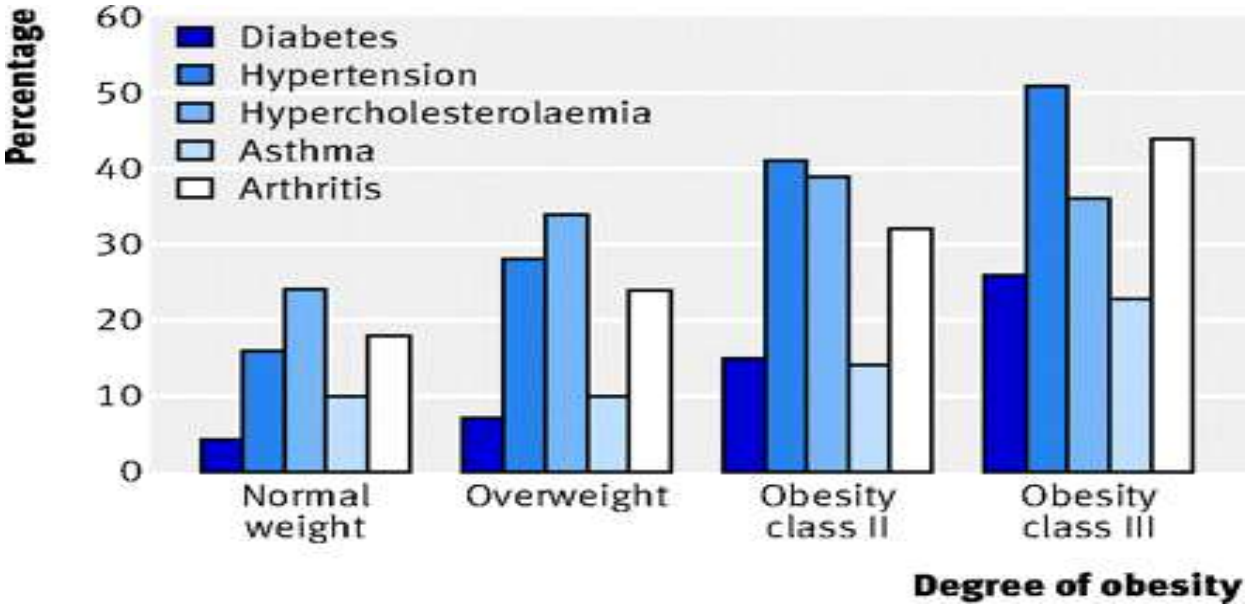

Proportion of people with a major co-morbidity, by degree of obesity (Leff and Heath 2009)

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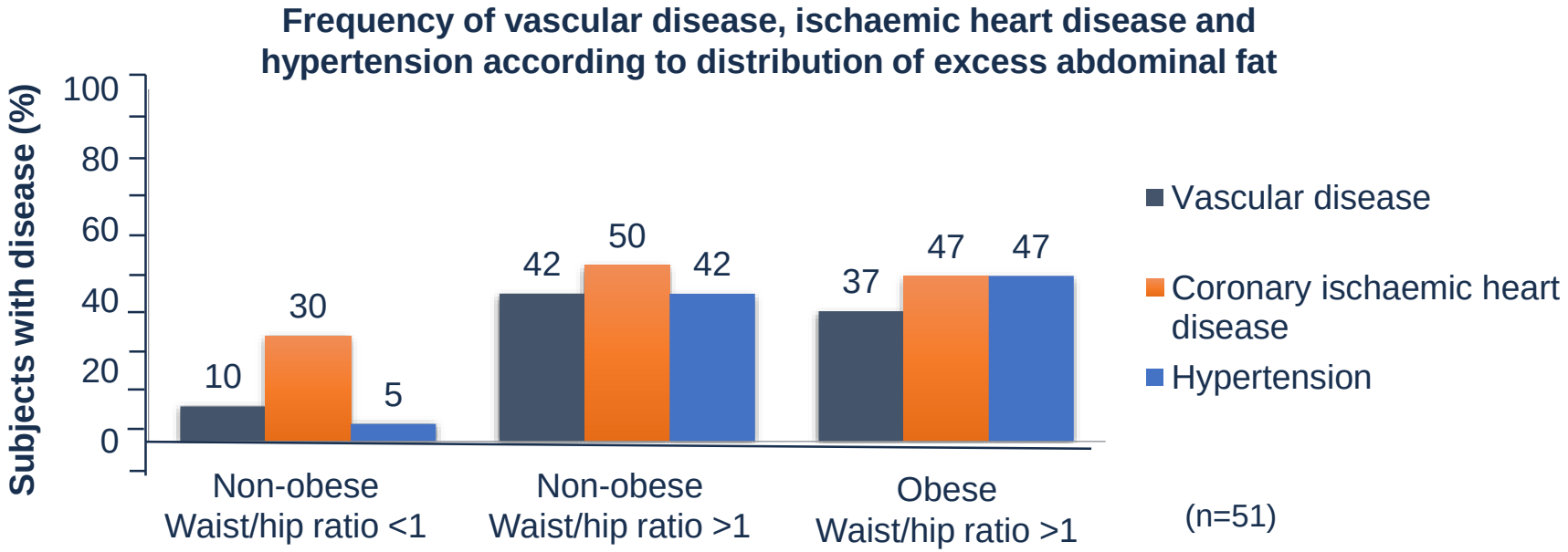
Obesity related co-morbidities:

Health Consequences–Obesity Associated Comorbidities: Julian J. Emmanuel and Simon W. Coppack

Relative risk	Diseases associated with metabolic consequences (indirect association)	Diseases associated with excess weight (direct association)
Greatly increased risk (>3)	Type 2 diabetes, gallbladder disease, hypertension, dyslipidaemia, insulin resistance, non-alcoholic fatty liver	Sleep apnoea, breathlessness, asthma, social isolation, depression, daytime sleepiness/fatigue
Moderately increased risk (2-3)	Coronary heart disease, stroke, gout	Osteoarthritis, respiratory disease, hernia, psychological problems
Slight increased risk (1-2)	Cancer, impaired fertility, polycystic ovaries, skin complications, cataract	Varicose veins, musculoskeletal problems, backache, stress incontinence, oedema/cellulitis

Obesity is associated with increased cardiovascular risk



- The frequency of peripheral vascular disease, coronary ischaemic heart disease and hypertension was most prominent in patients with type 2 diabetes and an abdominal fat mass distribution

Van Gaal LF, *et al. Diabetes Care* 1988;**11**:103–6

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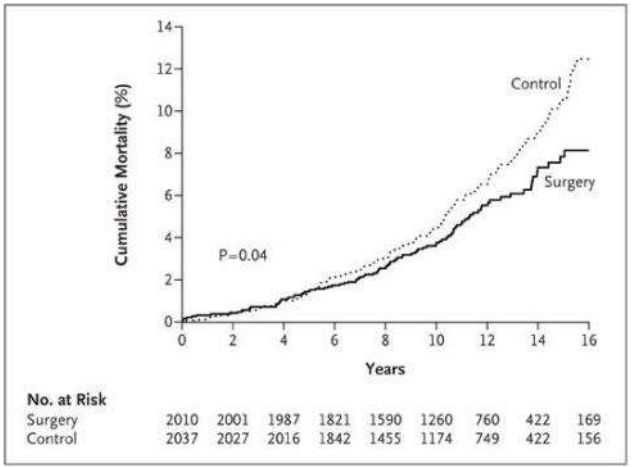
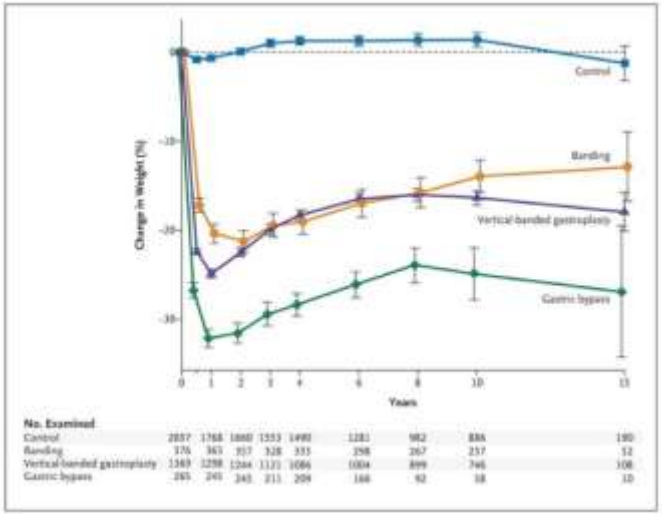
SOS

Lars Sjöström, M.D., Ph.D
N Engl J Med 2007;
357:741-752





NICE 2020

A body mass index (BMI) of at least 35 kg/m^2 (or at least 32.5 kg/m^2 for members of minority ethnic groups known to be at equivalent risk of the consequences of obesity at a lower BMI than the white population) and they have non-diabetic hyperglycaemia or a fasting plasma glucose level of 5.5 mmol/litre to 6.9 mmol/litre) and
they have a high risk of cardiovascular disease based on risk factors such as hypertension and dyslipidaemia and it is prescribed in secondary care by a specialist multidisciplinary tier 3 weight management service

Table 2. Cause of Death.*

Variable	Surgery Group (N=2010)	Control Group (N=2037)
	no. of subjects	
Cardiovascular condition		
Any event	43	53
Cardiac	35	44
Myocardial infarction	13	25
Heart failure	2	5
Sudden death	20	14
Stroke	6	6
Intracerebral hemorrhage	2	4
Infarction	1	2
Subarachnoid bleeding	3	0
Other	2	3
Aortic aneurysm	1	2
Aortic thrombosis	0	1
Diabetic gangrene	1	0
Noncardiovascular condition		
Any event	58	76
Tumor	29	48
Cancer	29	47
Meningioma	0	1
Infection	12	3
Thromboembolic disease	5	7
Pulmonary embolism	4	7
Vena caval thrombosis	1	0
Other	12	18
Total no. of deaths	101	129

* During the first 90 days after study initiation, there were five deaths in the surgery group (four from peritonitis with organ failure and one sudden death) and two deaths in the control group (one from cancer of the pancreas and one from alcohol-related causes).

Liraglutide for managing overweight and obesity
Technology appraisal guidance [TA664] Published: 09 December 2020

Ramsay Health Care

SASH 2021 - Obesity Surgery Patient Pathway
Ms Kirstin Carswell

Before

- Surgical OPC - 30min
- Dietetics:
  Tier 3
  Peri-op dietetic support
- Bloods
  T2DM/metabolic testing
  OSA testing +/- CPAP loan
  Cardiac Ix

Around

- Pre-operative assessment inc high-risk anaesthetic review
- Liver shrinkage diet (if appropriate)
- On the day admission
- pLMWH training
  Diet regimen
  Pedometer
- POD1 - Mx protocol and discharge

After

- 6w Surgical OPC
- 6m and 1y dietetic follow-up
- 2y Surgical OPC and discharge

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