Child and Adolescent Obesity

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ACKNOWLEDGEMENT OF COUNTRY

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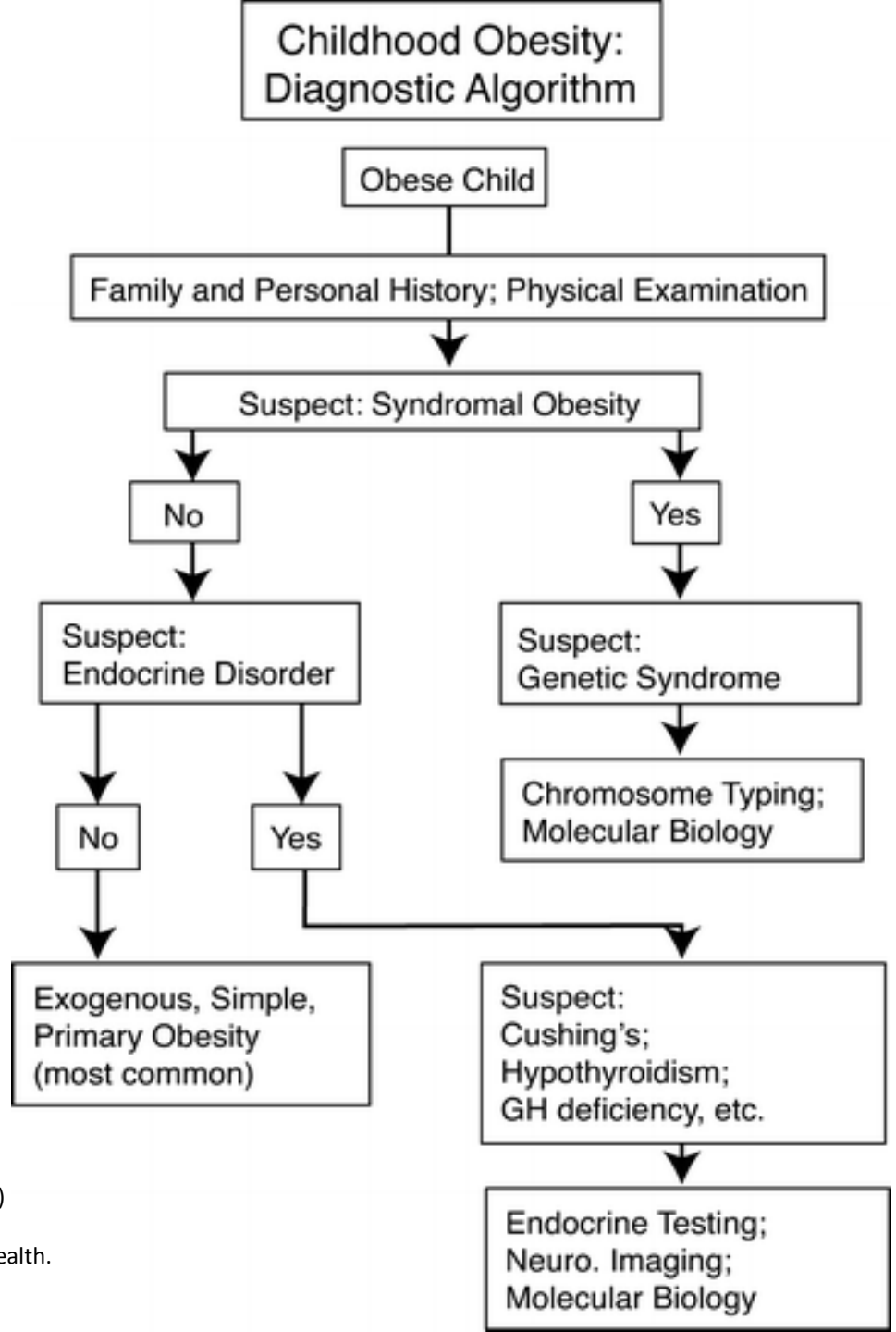
1. Endocrine and Genetic causes
2. Medical implications
3. Treatment

Endocrine and Genetic causes

Most obesity is lifestyle related



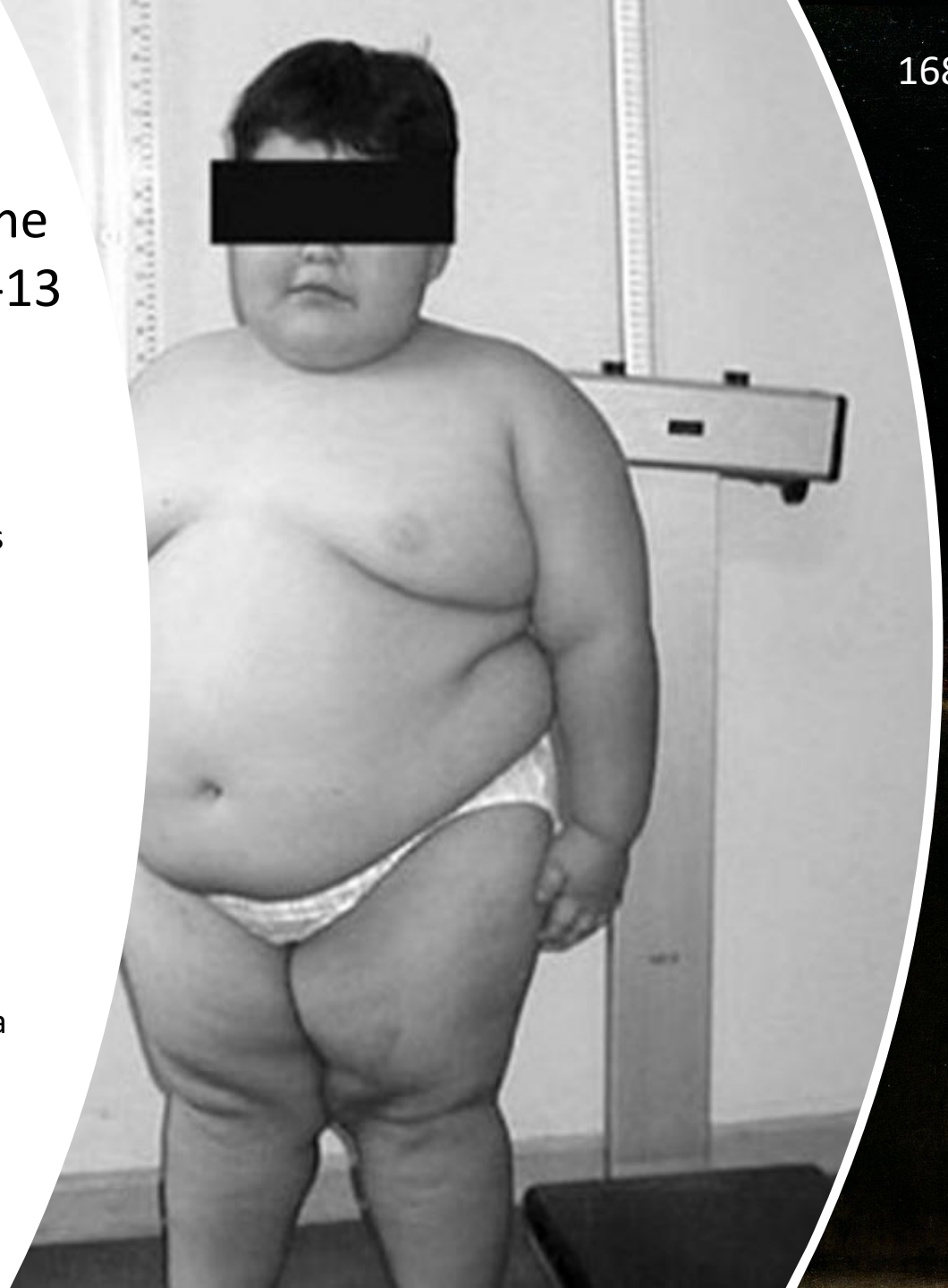
Hale P.M., Cushman T.T., Kimball E.S., Nair A., Shaffer R.G. (2011)
Secondary Causes of Obesity in Childhood. In: Ferry, Jr. R. (eds)
Management of Pediatric Obesity and Diabetes. Nutrition and Health.
Humana Press. https://doi.org/10.1007/978-1-60327-256-8_16

- Prospective cohort study followed 1,405 children and adolescents four to 16 years of age who were obese, and who were referred to an endocrinology clinic for evaluation
- A syndromal or endocrinologic disorder was diagnosed as the cause of obesity in 13 patients (0.9 percent; number needed to refer = 108)
- All 13 patients had clinical findings, and none were identified solely based on laboratory test results

Reinehr T, Hinney A, de Sousa G, Austrup F, Hebebrand J, Andler W. Definable somatic disorders in overweight children and adolescents. J Pediatr. 2007;150(6):618-622

Prader Willi Syndrome
Chromosome 15q11-13

- 1/10,000 to 1/25,000 live births
- Decreased foetal movements
- Hypotonia – failure to thrive
- Altered temperature sensation
- Hypogonadism, cryptorchidism
- Growth hormone deficient/short stature
- Global developmental delay
- Decreased Caloric requirement (60-80% RDA)
- Absent satiety & hyperphagia
- Temper tantrums
- Inability to vomit



1680 painting of Eugenia Martínez Vallejo

https://en.wikipedia.org/wiki/Prader–Willi_syndrome

Other Genetic Causes

- Prader Willi
- Bardet-Biedl
- Down Syndrome
- Alstrom syndrome
- Carpenter syndrome
- Cohen syndrome
- Albright hereditary osteodystrophy
- Monogenic obesity

Consider:

Non familial facial features

Developmental delay

Deafness

Hypotonia

Craniosynostosis

Syndactyly

Endocrine Causes for Obesity

- Hypothyroidism
- Cushing Syndrome
- Growth hormone deficiency
- Pseudo-hypoparathyroidism
- CNS disorders/hypothalamic obesity
    - Tumour, surgery, trauma

Consider:

Short stature

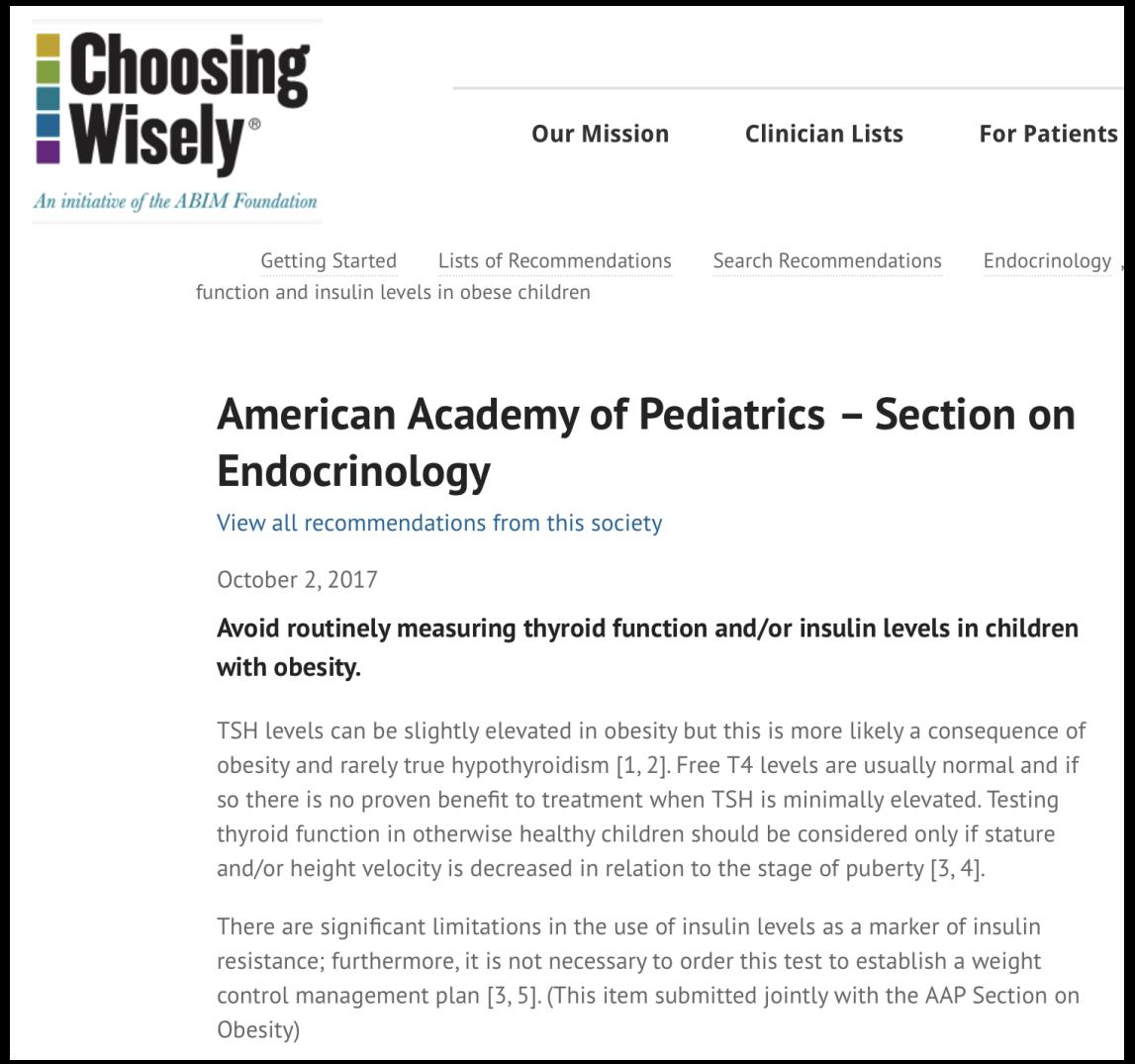
Cold intolerance

Short 5th metacarpal

Peripheral field defect

Hypothyroidism

- Thyroid hormone role in the regulation of metabolism, thermogenesis, food intake, and fat oxidation
- With obesity most frequent hormonal abnormalities are slight hyperthyrotropinaemia and moderate increases in fT3
- Consequence vs Cause



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function and insulin levels in obese children

American Academy of Pediatrics – Section on Endocrinology

View all recommendations from this society

October 2, 2017

Avoid routinely measuring thyroid function and/or insulin levels in children with obesity.

TSH levels can be slightly elevated in obesity but this is more likely a consequence of obesity and rarely true hypothyroidism [1, 2]. Free T4 levels are usually normal and if so there is no proven benefit to treatment when TSH is minimally elevated. Testing thyroid function in otherwise healthy children should be considered only if stature and/or height velocity is decreased in relation to the stage of puberty [3, 4].

There are significant limitations in the use of insulin levels as a marker of insulin resistance; furthermore, it is not necessary to order this test to establish a weight control management plan [3, 5]. (This item submitted jointly with the AAP Section on Obesity)

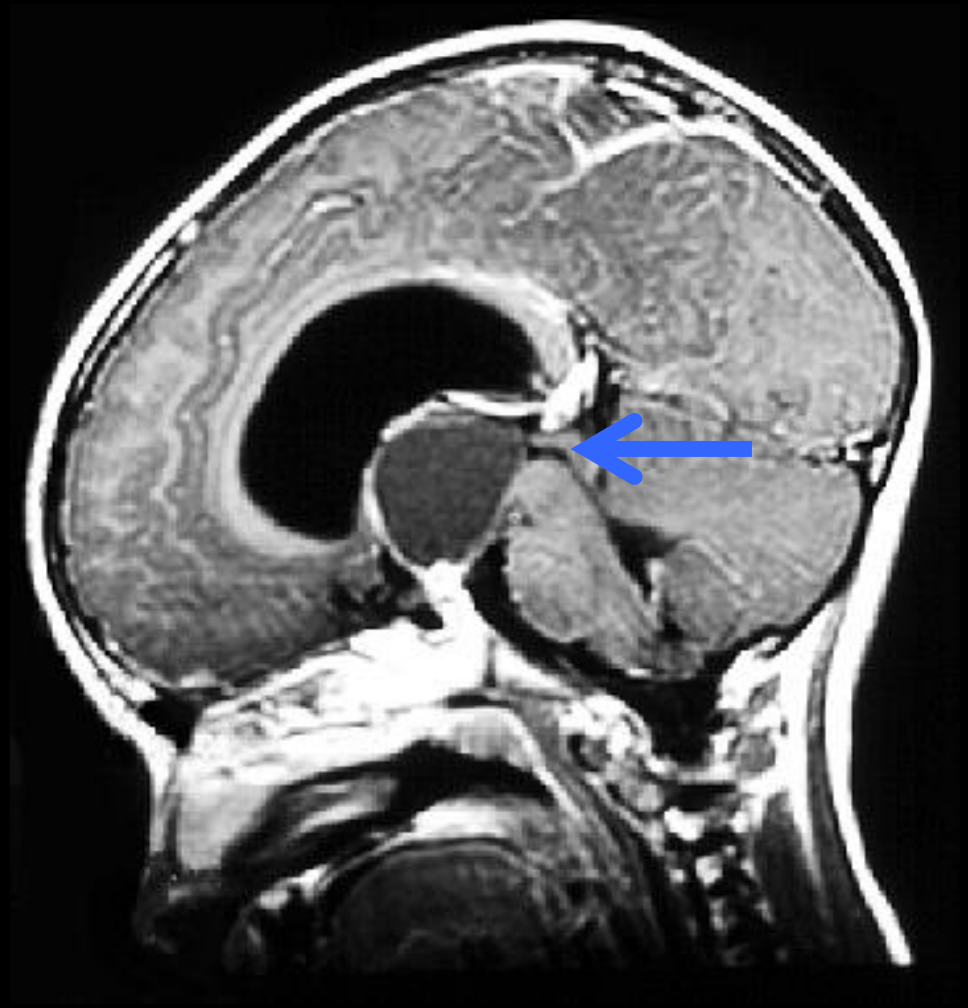
Hypothalamic dysfunction

- Features include:
  - Behavioural changes
  - Disturbed circadian rhythm and sleep
  - Daytime sleepiness
  - Imbalances of regulation of temperature, thirst, heart rate, BP
  - Disturbances of appetite regulation & satiety ➔ hyperphagia
  - Reduced energy expenditure

➔ Rapid weight gain:

Unresponsive to conventional lifestyle modification

Physical causes of hypothalamic obesity or hypothalamic injury associated obesity

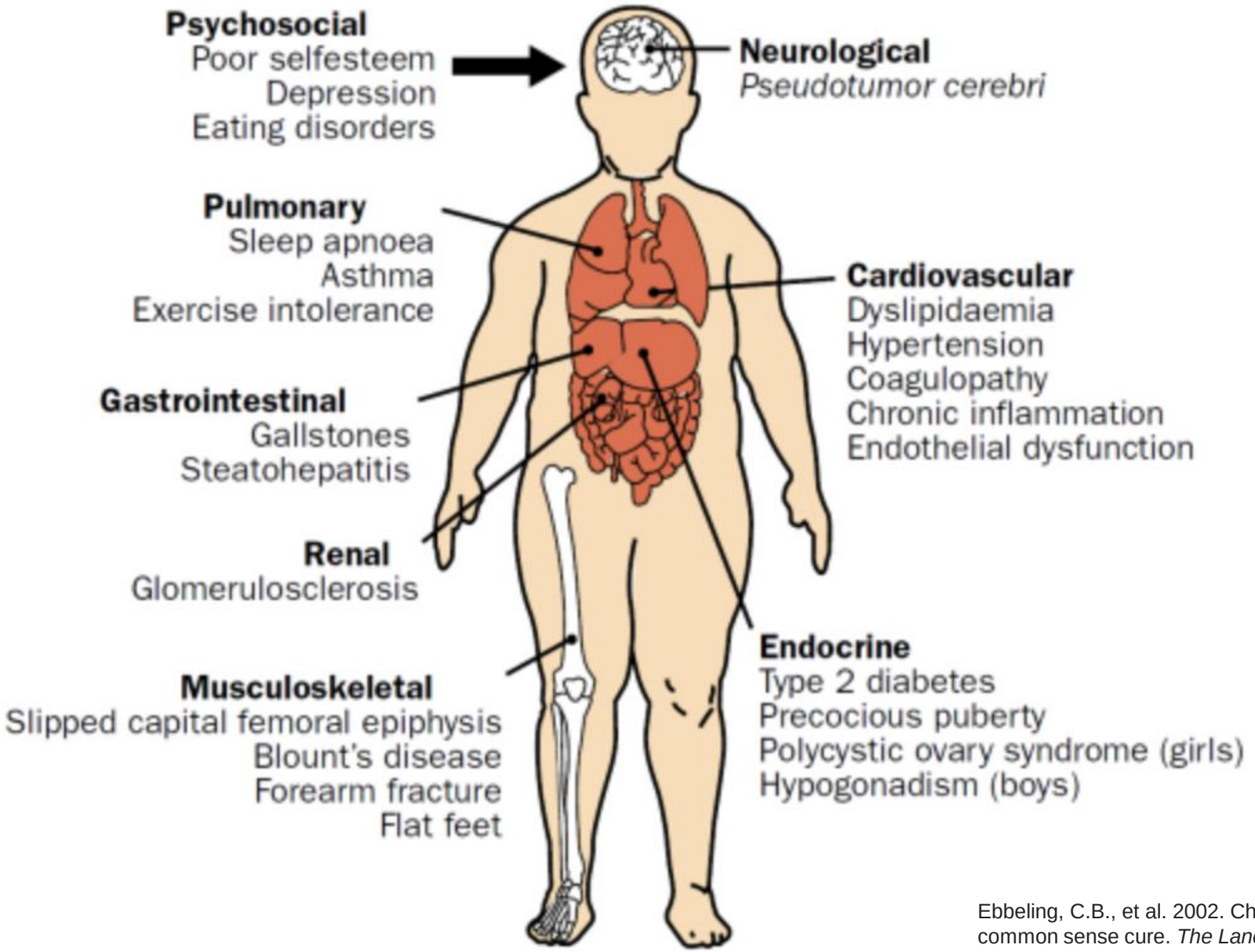


- Craniopharyngioma
- Neurosurgery, cranial irradiation
- Glioma, meningioma
- Astrocytoma
- Teratoma, germ cell tumour
- Metastasis
- Aneurysm

➔lack of satiety, hyperphagia, reduced physical activity, sleep disturbances

Medical implications

COMPLICATIONS OF CHILDHOOD OBESITY



Ebbeling, C.B., et al. 2002. Childhood obesity: public-health crisis, common sense cure. *The Lancet*, *360*(9331), pp.473-482.

Endocrine Complications of Obesity

- Insulin Resistance
- Hyperlipidemia
- Type 2 Diabetes
- Polycystic Ovarian Syndrome
- Accelerated linear growth
- Early Puberty

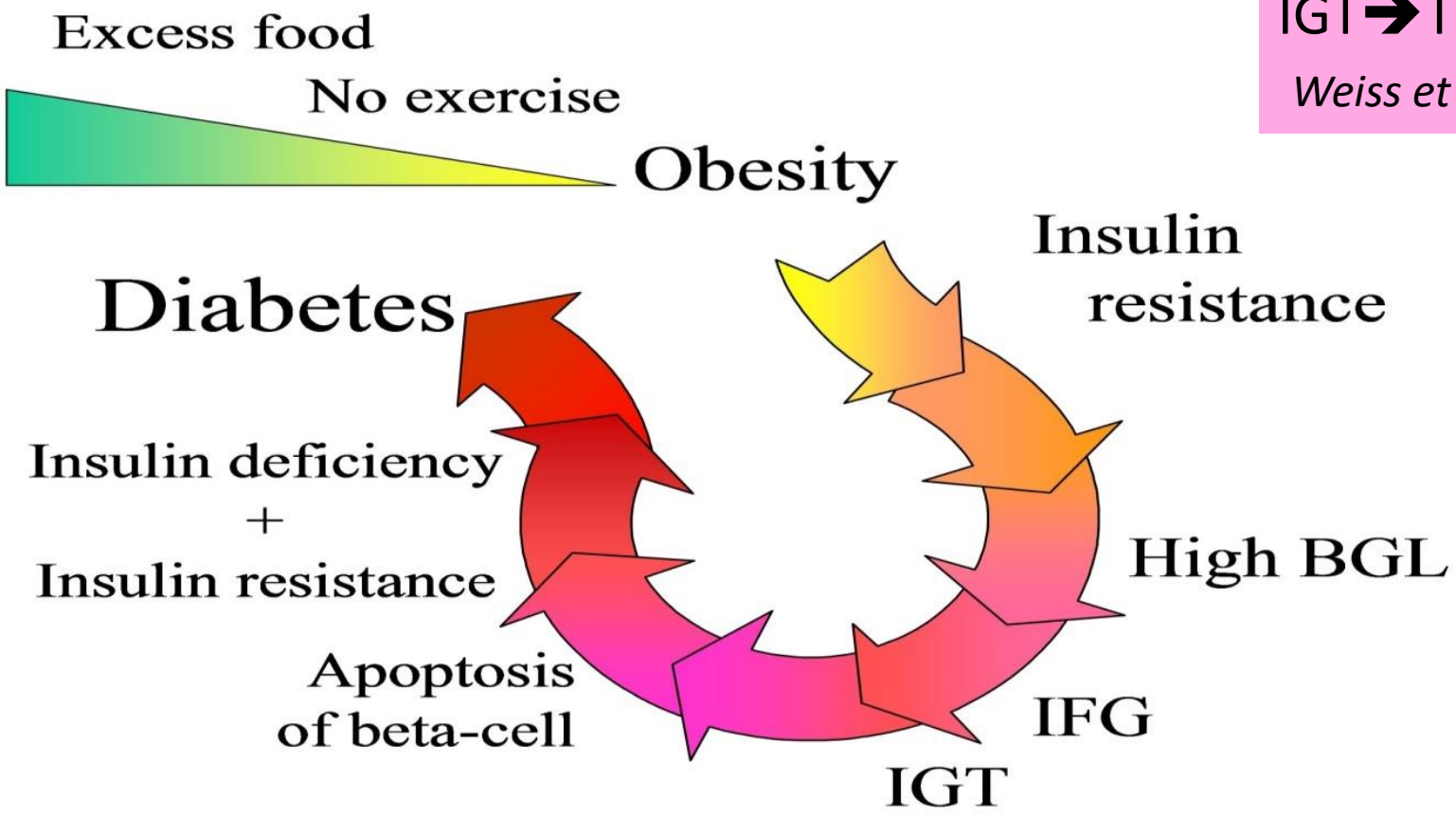
What is insulin resistance?

- The impaired ability of plasma insulin at usual concentrations to
  - adequately promote peripheral glucose disposal
  - suppress hepatic glucose production
  - inhibit VLDL output
- Inferred on clinical evidence
- Screened by fasting insulin/glucose values
- Confirmed by OGTT

The progression from insulin resistance to diabetes

24% of adolescents
IGT➔T2D in <2 years

Weiss et al Diabetes Care 2005



OGTT for obese children- how to do it?

- Unrestricted diet, including carbohydrates, for at least 3 days before test
- Normal physical activity, no intercurrent illness
- Test performed in the morning after 10-16 hours fast e.g. from 9pm
- 1.75g/kg glucose (75g max) in 25% solution (Glucaid) in less than 5 minutes
- Bloods (insulin/glucose) taken immediately before and 2 hours after the glucose load +/- other bloods

Cut-off values

- Prediabetes is diagnosed according to ADA definitions:
  - IFG: FPG ≥5.6 to 6.9 mmol/L
  - IGT: Post-challenge plasma glucose is ≥7.8 to 11.1 mmol/L
  - Hemoglobin A1c 5.7% to 6.4%
  - Laboratory-based, DCCT aligned, NGSP certified methodology

- In obese adolescents, pre-diabetes often transient
- ~60% revert to normal OGTT within 2 years as the insulin resistance of puberty wanes

Diagnosing diabetes

TABLE 1 Criteria for the diagnosis of diabetes mellitus

1. Classic symptoms of diabetes or hyperglycemic crisis, with plasma glucose concentration ≥11.1 mmol/L (200 mg/dL).
or
2. Fasting plasma glucose ≥7.0 mmol/L (≥126 mg/dL). Fasting is defined as no caloric intake for at least 8 h.[a]
or
3. Two-hour postload glucose ≥11.1 mmol/L (≥200 mg/dL) during an OGTT.[a]
The test should be performed using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water or 1.75 g/kg of body weight to a maximum of 75 g.
or
4. HbA1c ≥6.5%[b]
The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay.

[a] In the absence of unequivocal hyperglycemia, the diagnosis of diabetes based on these criteria should be confirmed by repeat testing.
[b] A value of less than 6.5% does not exclude diabetes diagnosed using glucose tests. The role of HbA1c alone in diagnosing type 1 diabetes in children is unclear.

ISPAD Guidelines 2018.
Chapter 1: Definition, epidemiology, diagnosis and classification of diabetes in children and adolescents

Metabolic Syndrome

- Abdominal obesity & two or more of the other components
- FPG 5.6-6.9 mmol/L --> OGTT
- Prevalence:
  - Children 3%
  - Overweight 11.9%
  - Obese 29.2%

Friend et al Metab Syndr Relat Disord 2013

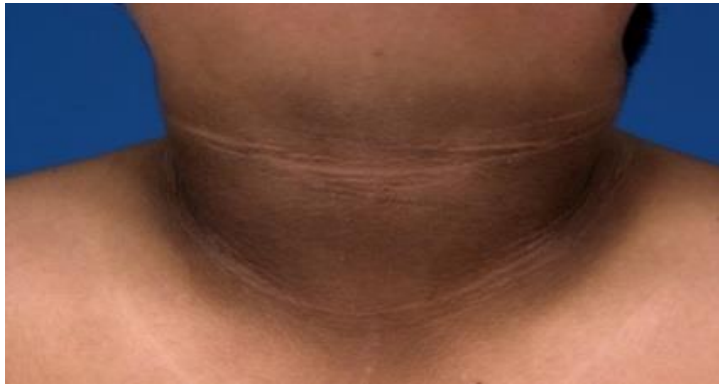
Age group (years)	Obesity (WC)	Triglycerides	HDL-C	Blood pressure	Glucose
6–<10†	≥90th percentile				
10–<16	≥90th percentile or adult cut-off if lower	≥1.7 mmol/L (≥150 mg/dL)	<1.03 mmol/L (<40 mg/dL)	Systolic BP ≥130 or diastolic BP ≥85 mm Hg	FPG ≥5.6 mmol/L (100 mg/dL)** or known T2DM
16+ (Adult criteria)	WC ≥ 94cm for Europid males and ≥ 80cm for Europid females, with ethnic-specific values for other groups*)	≥1.7 mmol/L (≥150 mg/dL) or specific treatment for high triglycerides	<1.03mmol/L (<40 mg/dL) in males and <1.29mmol/L (<50 mg/dL) in females, or specific treatment for low HDL	Systolic BP ≥130 or diastolic BP ≥85 mm Hg or treatment of previously diagnosed hypertension	FPG ≥5.6 mmol/L (100 mg/dL)** or known T2DM

Zimmet P, Alberti G, Kaufman F, Tajima N, Silink M, Arslanian S, Wong G, Bennett P, Shaw J, Caprio S; International Diabetes Federation Task Force on Epidemiology and Prevention of Diabetes. The metabolic syndrome in children and adolescents. Lancet. 2007 Jun 23;369(9579):2059-61. doi: 10.1016/S0140-6736(07)60958-1. PMID: 17586288.

8.5 year old girl

- Presents to dermatology clinic
- BMI 24kg/m^2 (98^{th} percentile)
- Severe acanthosis nigricans
- Strong family history of metabolic syndrome and early cardiac events on both maternal and paternal sides
- Past 6 months dancing or sport almost every day, healthier food choices but still gaining weight

O/E
Pre-pubertal
BP 110/68
Severe AN



Waist circumference 84cm
Waist:height ratio 0.6 (<0.5)

OGTT

	Baseline	30min	60min	90min	120min
Glucose mmol/L	4.4	6.9	7.8	6.8	7.9
Insulin pmol/L	185 (31mU/L)	1428	1377	983	2770

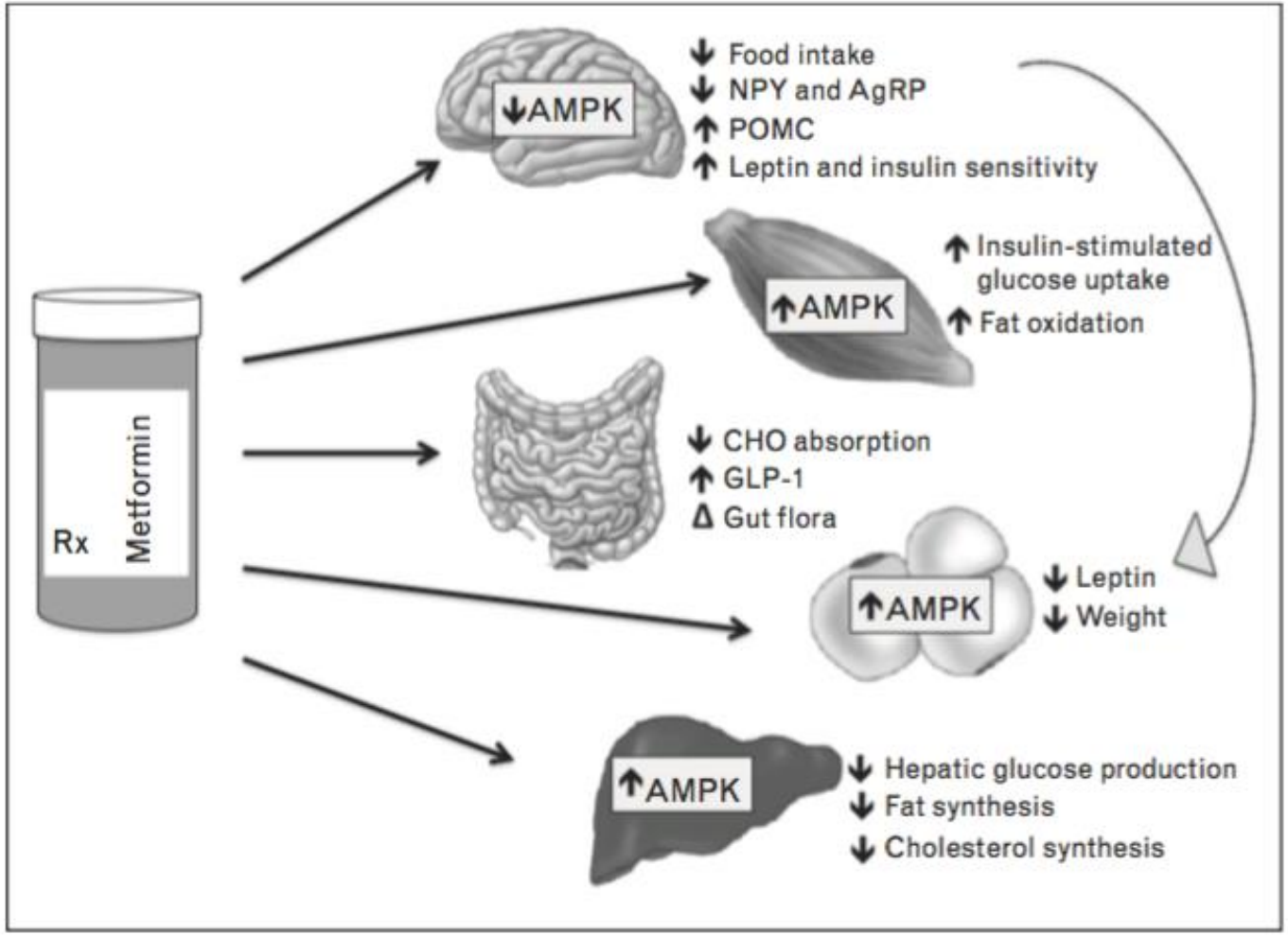
Lipid profile: Cholesterol 4.5mmol/L
LDL-cholesterol 1.5mmol/L
Triglycerides 4.2mmol/L

Treatment

Metformin

Biguanide:

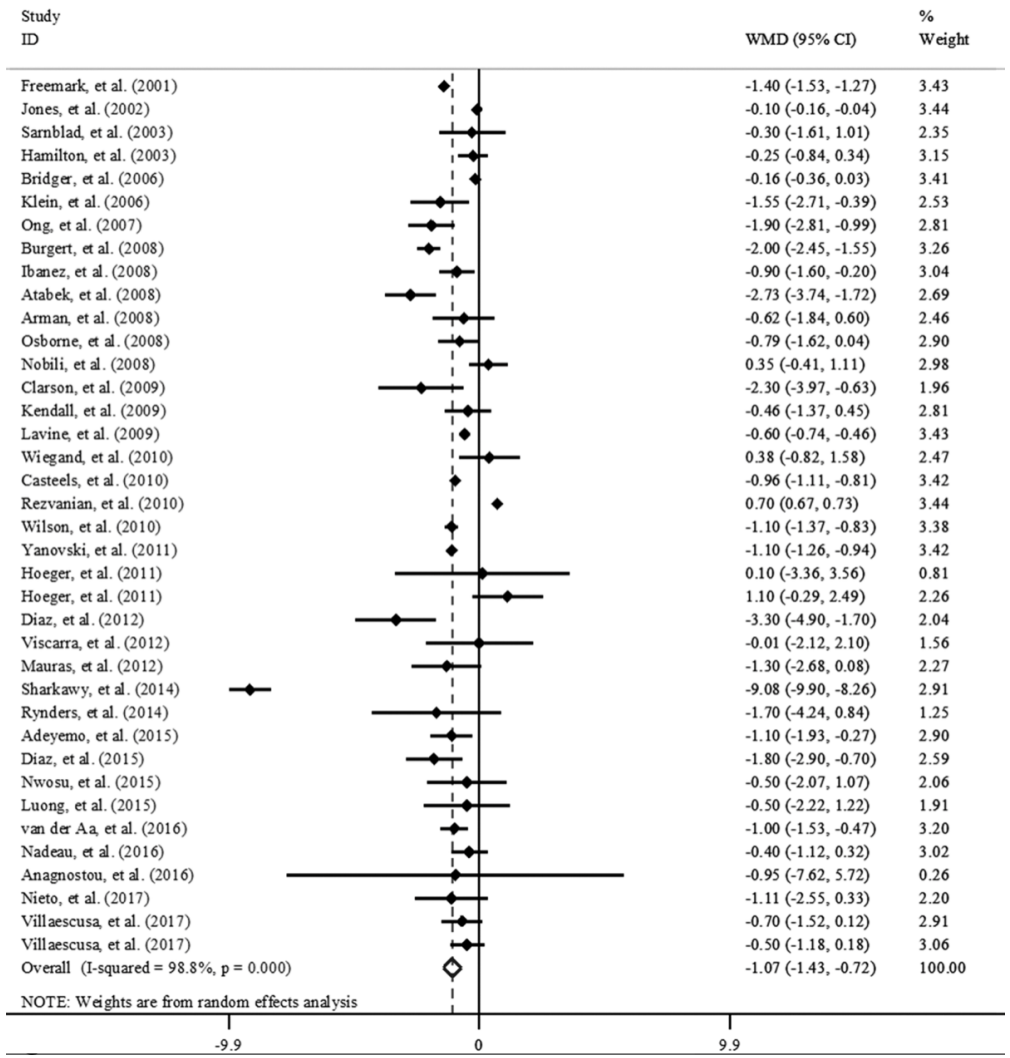
- Increase hepatic and muscle insulin sensitivity
- Decreased appetite, mild weight loss
- No effect on energy expenditure



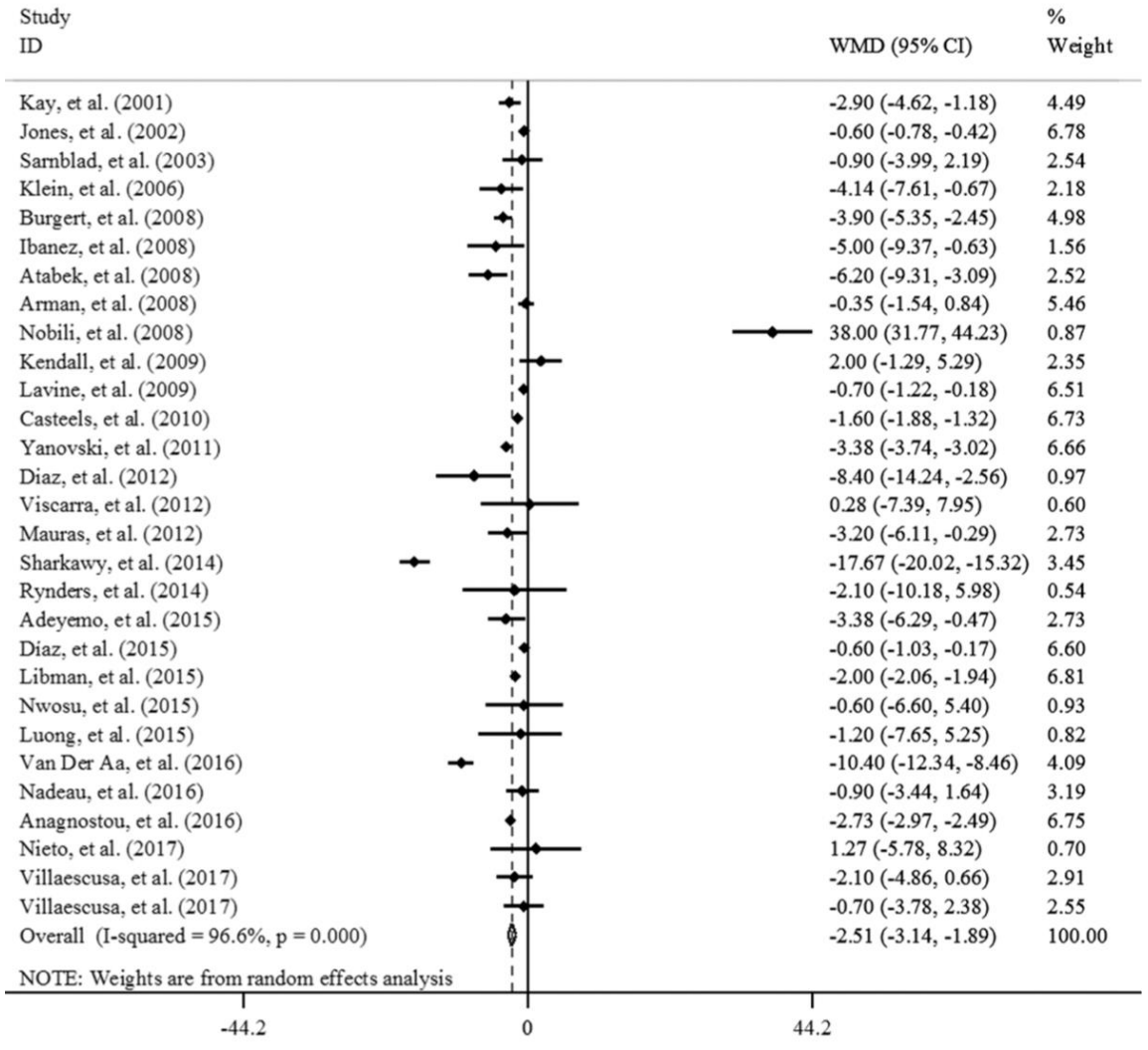
Malin et al Current Opinion Endocrinol Diabetes Obes 2014; 21

Systematic review metformin

BMI



Weight



Alireza Sadeghi, Seyed Mohammad Mousavi, Tahereh Mokhtari, Mohammad Parohan, and Alireza Milajerdi.Childhood Obesity.Apr 2020.174-191.

Metformin safety profile

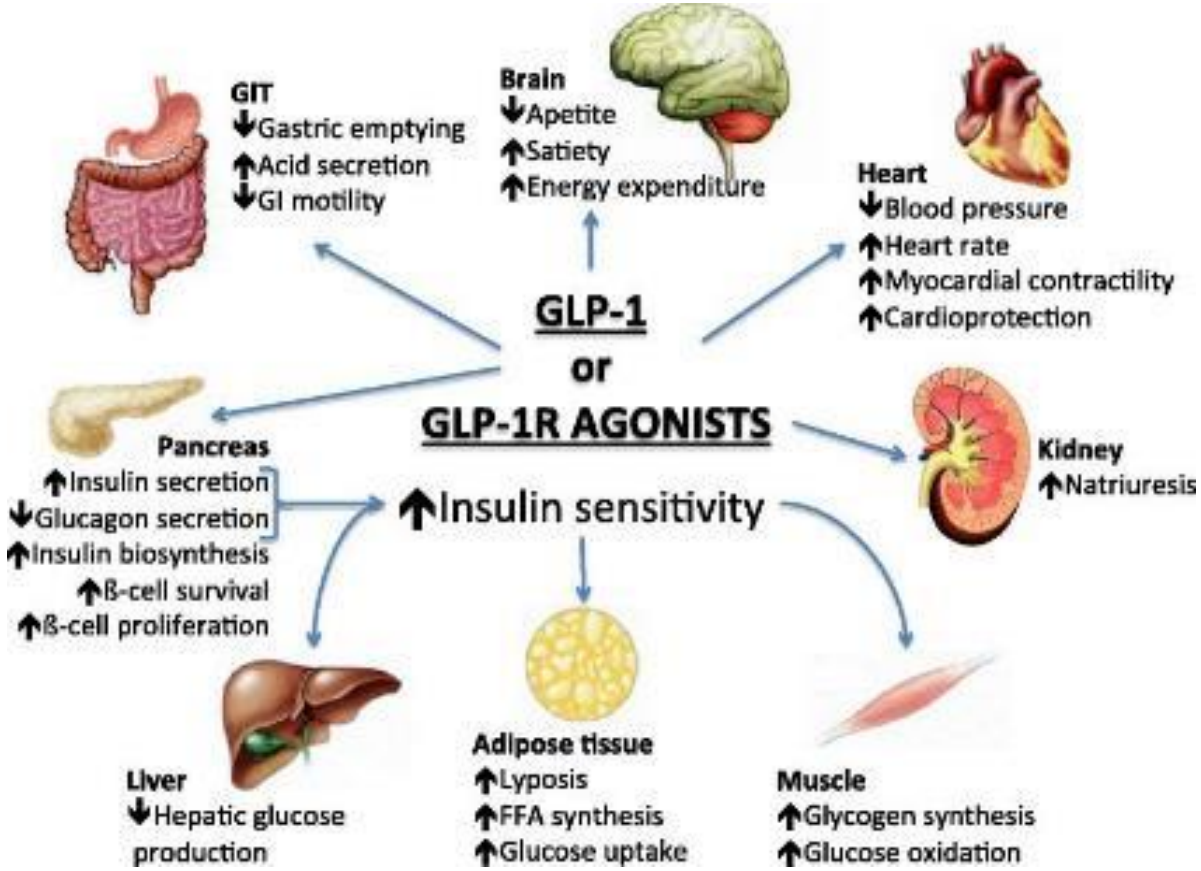
Side effects:

- Gastrointestinal (metallic taste, abdominal discomfort, diarrhoea in 5-20%, or RR~2
- Low vitamin B12 levels with chronic use due to malabsorption (usually not anaemia)
- Lactic acidosis (rare) contraindicated in impaired renal function, CHF, other forms of acidosis and clinically significant liver disease

- Guidance:
  - Obese adolescents with evidence of clinical insulin resistance (acanthosis nigricans, PCOS, hyperinsulinaemia etc)
  - Start low and go slow, building up to 2g/day over 2 months
  - XR formulation
  - Generally >10 years old

Glucagon-like peptide-1 (GLP1) receptor agonists

- Increase postprandial insulin, reduce glucagon secretion, delay gastric emptying, decrease appetite, induce weight loss
- Used as a second-line treatment for type 2 diabetes, and for reducing the risk of CVD events in people with T2DM and CVD
- Liraglutide (*Saxenda*) – daily subcutaneous
- Semaglutide (*Ozempic*) – weekly subcutaneous
- Side-effects include nausea, vomiting, diarrhoea



With thanks to Prof Baur

ORIGINAL ARTICLE

A Randomized, Controlled Trial of Liraglutide for Adolescents with Obesity

Aaron S. Kelly, Ph.D., Pernille Auerbach, M.D., Ph.D., Margarita Barrientos-Perez, M.D., Inge Gies, M.D., Ph.D., Paula M. Hale, M.D., Claude Marcus, M.D., Ph.D., Lucy D. Mastrandrea, M.D., Ph.D., Nandana Prabhu, M.Sc., and Silva Arslanian, M.D., for the NN8022-4180 Trial Investigators*

ABSTRACT

NEJM 2020; 382:2117-2128

With thanks to Prof Baur

Methods

- **Randomised, double-blind, placebo-controlled phase 3 trial**
- **Designed & overseen by the trial sponsor, Novo Nordisk**
- **Run Sept 2016 through to Aug 2019 at 32 sites in 5 countries (US, Mexico, Belgium, Russia, Sweden)**
- **12 week run-in period: All received lifestyle therapy. Randomised at the end of this (1:1 ratio). Randomisation stratified based on pubertal stage, glycaemic status**
- **56 week treatment period: Incl. dose escalation (from 0.6 mg daily up to 3mg daily)**
- **26 week follow-up period with no treatment**
- **Participants:**
  - 12 to <18 years
  - BMI >30 (or age-equivalent), with stable body weight
  - Those with T2DM were eligible

With thanks to Prof Baur

Methods - 2

- **Endpoints:**
  - Primary: Change from baseline in BMI standard deviation score at week 56
  - Secondary efficacy: Included change in BMI SDS from wks 56 to 86, % of participants BMI reduction >5% and >10% at wk 56, and change in waist, glucose metabolism, BP, quality of life at wk 56
  - Safety end-points: adverse events from 0-wk 56, hypoglycaemic episodes; changes in bone age, height SD score, hormone levels and HR. mental health

Sample size estimation – 228 participants

With thanks to Prof Baur

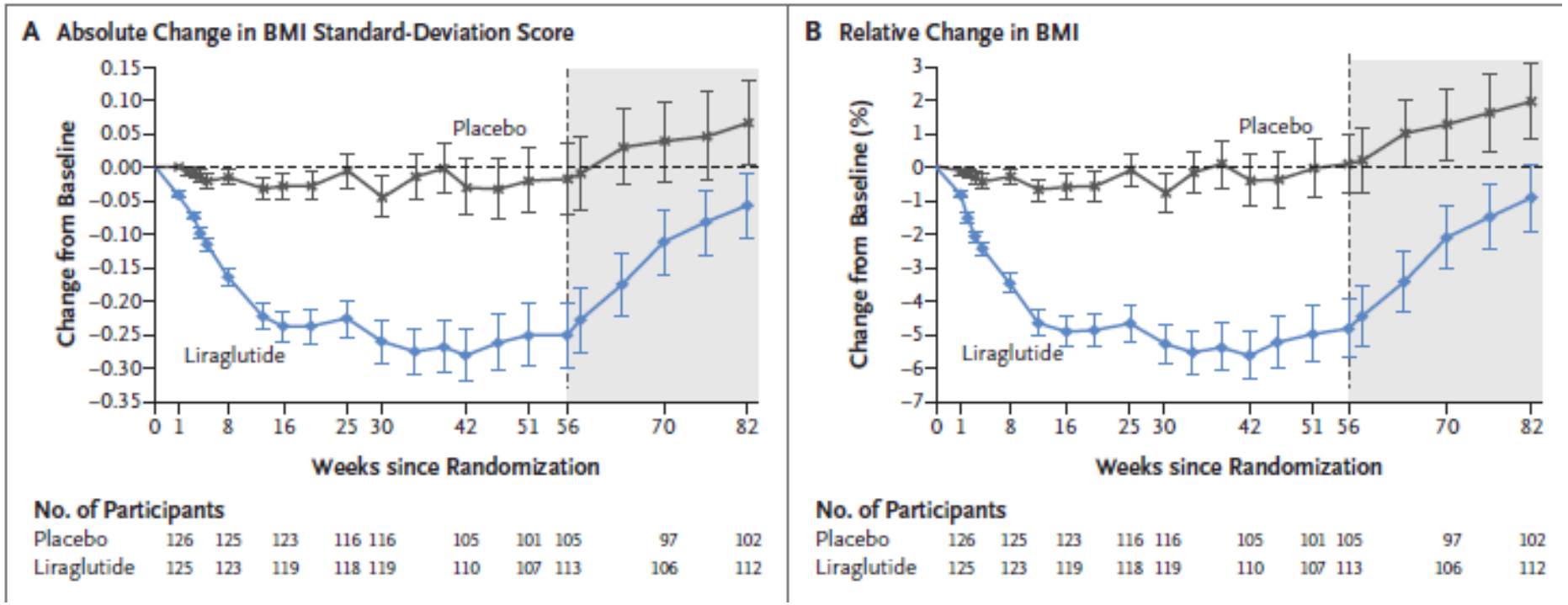
Table 1. Characteristics of the Participants at Baseline.*

Characteristic	Liraglutide (N= 125)	Placebo (N= 126)
Female sex — no. (%)	71 (56.8)	78 (61.9)
Age — yr	14.6±1.6	14.5±1.6
Race or ethnic group — no. (%)†		
White	105 (84.0)	115 (91.3)
Black	14 (11.2)	6 (4.8)
Asian	2 (1.6)	0
American Indian or Alaska Native	0	1 (0.8)
Other	4 (3.2)	4 (3.2)
Hispanic ethnic group — no. (%)†	32 (25.6)	24 (19.0)
Tanner stage — no. (%)‡		
2	6 (4.8)	8 (6.3)
3	16 (12.8)	13 (10.3)
4	38 (30.4)	40 (31.7)
5	65 (52.0)	65 (51.6)
Height — m	1.7±0.1	1.7±0.1
Body weight — kg	99.3±19.7	102.2±21.6
BMI§	35.3±5.1	35.8±5.7
BMI standard-deviation score¶	3.14±0.65	3.20±0.77
BMI as percentage of the 95th percentile — %	137.7±18.0	139.6±21.4
Waist circumference — cm	104.87±12.67	106.99±13.57
Waist:hip ratio	0.908±0.079	0.915±0.080
Glycated hemoglobin — %	5.3±0.4	5.3±0.4
Fasting plasma glucose — mg/dl	94.1±7.6	94.5±11.1
Dysglycemia — no. (%)‖	32 (25.6)	33 (26.2)
Blood pressure — mm Hg		
Systolic	116±10	117±12
Diastolic	72±8	73±8
Cholesterol — mg/dl		
Total	156.4±27.0	154.9±29.6
High-density lipoprotein	43.8±10.0	43.8±10.3
Low-density lipoprotein	88.6±24.0	86.6±25.2
Triglycerides — mg/dl	121.0±59.4	124.5±62.5
Free fatty acids — mg/dl	[illegible]	[illegible]

- 299 participants screened
- 251 randomised – 125 liraglutide & 126 placebo
- At week 56 – 101 (81%) completed treatment
- Baseline characteristics similar

- Escalation to 3.0mg dose achieved by 82% in liraglutide group & 98% in the control group

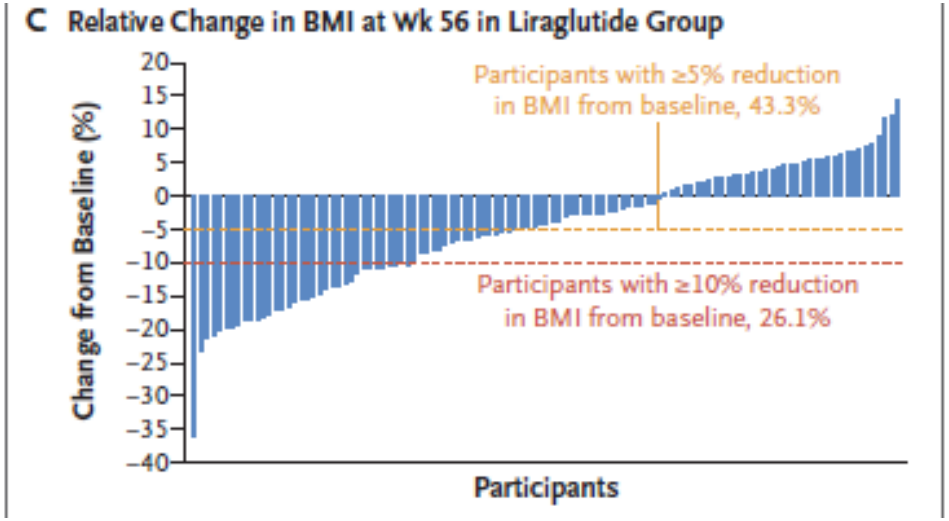
With thanks to Prof Baur

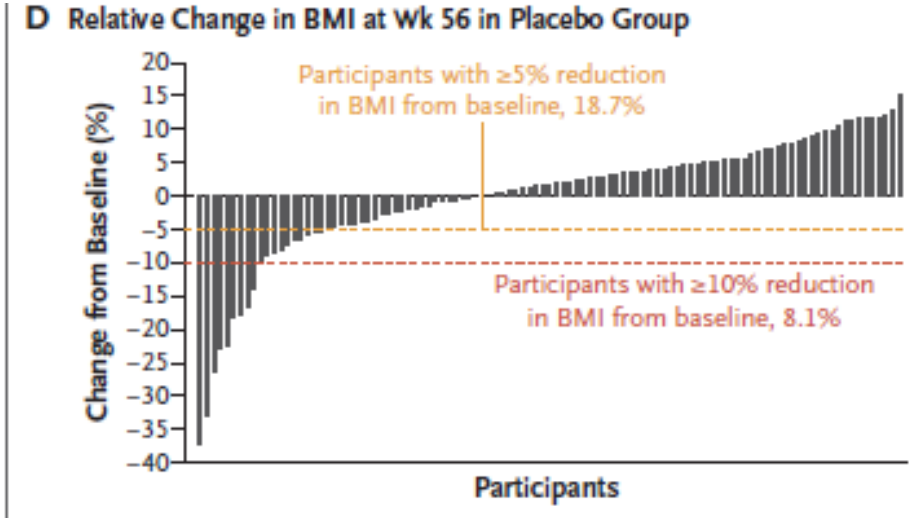


Liraglutide superior to placebo at wk 56:

- Change in BMI SDS from baseline (-0.26; 95% CI -7.14 to -2.14)
- Relative change in BMI (-4.64% points; 95% CI-7.14 to -2.14)

With thanks to Prof Baur

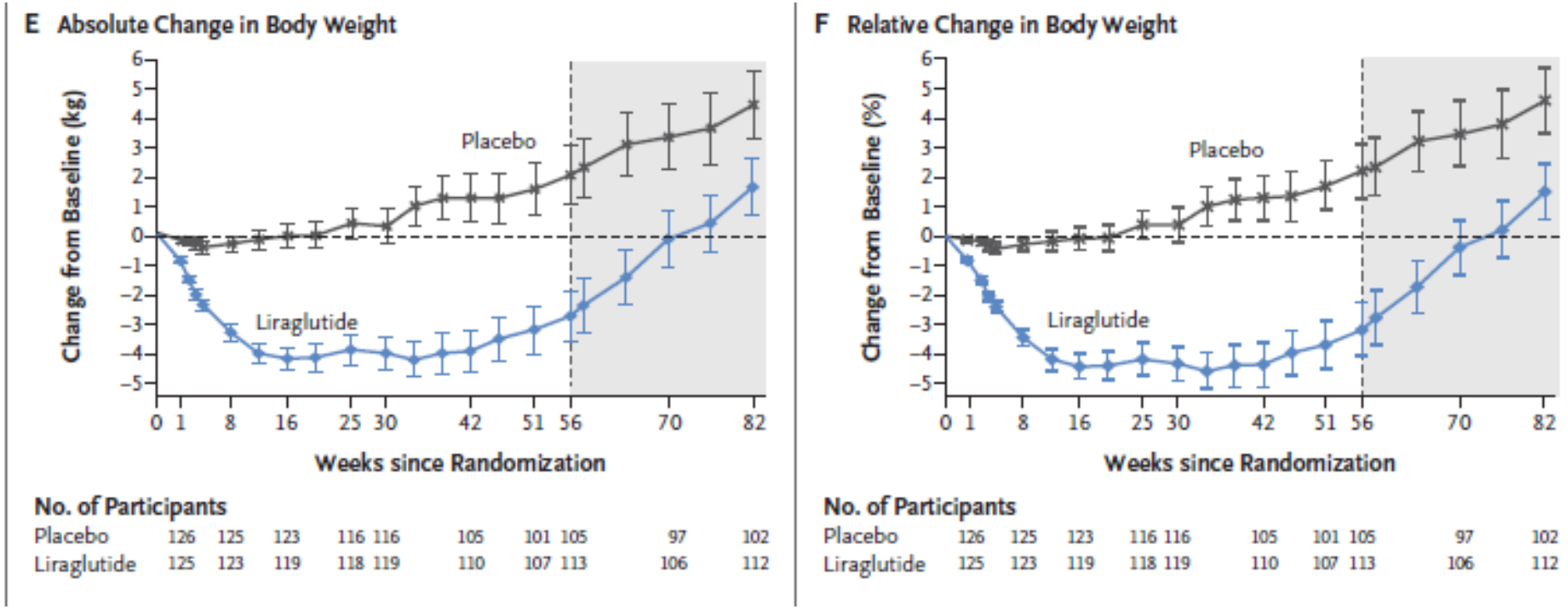




Liraglutide superior to placebo at wk 56:

- Reduction in BMI of at least **5%** observed in 43% liraglutide group vs 19% placebo group
- Reduction in BMI of at least **10%** observed in 26% vs 8%

With thanks to Prof Baur



Liraglutide superior to placebo at wk 56:

- Reduction in absolute weight: -2.79±9.1 kg vs +2.1±10.2 kg
- Reduction in relative weight change: -3.2±9.4% vs +2.2±9.5%

With thanks to Prof Baur

Table 3. Adverse Events during the Treatment Period.*

Event	Liraglutide (N=125)			Placebo (N=126)			P Value
	no. of participants (%)	*no. of events*	*events/1000 exposure-yr*	*no. of participants (%)*	*no. of events*	*events/1000 exposure-yr*	
Any adverse events	111 (88.8)	777	6187.8	107 (84.9)	627	5018.5	0.07†
Gastrointestinal adverse events	81 (64.8)	319	2540.4	46 (36.5)	121	968.5	0.001†
Serious adverse events‡	3 (2.4)	3	23.9	5 (4.0)	6	48.0	0.72§
Adverse events that led to treatment discontinuation	13 (10.4)	19	151.3	0	0	0	<0.001§
Adverse events that occurred in ≥5% of participants							
Nasopharyngitis	34 (27.2)	68	541.5	38 (30.2)	80	640.3	0.60¶
Nausea	53 (42.4)	101	804.3	18 (14.3)	25	200.1	<0.001¶
Headache	29 (23.2)	43	342.4	35 (27.8)	53	424.2	0.41¶
Vomiting	43 (34.4)	85	676.9	5 (4.0)	8	64.0	<0.001¶
Diarrhea	28 (22.4)	44	350.4	18 (14.3)	29	232.1	0.10¶
Upper abdominal pain	17 (13.6)	25	199.1	17 (13.5)	23	184.1	0.98¶
Oropharyngeal pain	11 (8.8)	11	87.6	15 (11.9)	18	144.1	0.42¶
Influenza	11 (8.8)	11	87.6	12 (9.5)	12	96.0	0.84¶
Gastroenteritis	16 (12.8)	22	175.2	6 (4.8)	9	72.0	0.02¶
Upper respiratory tract infection	11 (8.8)	14	111.5	11 (8.7)	16	128.1	0.98¶
Abdominal pain	10 (8.0)	15	119.5	11 (8.7)	15	120.1	0.83¶
Pyrexia	10 (8.0)	11	87.6	9 (7.1)	11	88.0	0.80¶
Dizziness	13 (10.4)	15	119.5	4 (3.2)	5	40.0	0.02¶
Dysmenorrhea	4 (3.2)	5	39.8	8 (6.3)	16	128.1	0.38§
Arthralgia	3 (2.4)	3	23.9	8 (6.3)	8	64.0	0.22§
Pharyngitis	4 (3.2)	5	39.8	7 (5.6)	7	56.0	0.54§

*Adverse events and serious adverse events that occurred from week 0 through week 56 among adolescents in the safety population are included in the table and presented with their preferred terms. Events were included if the date of onset was between the first day the trial drug was administered and 14 days after the last day the trial drug was administered, at the follow-up visit, or at the last trial visit.

†The P value was calculated with a negative binomial model. The number of events was analyzed with a negative binomial model with log-link function and the logarithm of the exposure time (1000 years) for which an adverse event is considered to be reported during the treatment period as an offset. The model included treatment, sex, region, baseline glycemic category, stratification factor for Tanner stage, and interaction between baseline glycemic category and stratification factor for Tanner stage as fixed effects.

‡The following serious adverse events were reported in one participant each: postprocedural hemorrhage, myositis, and completed suicide in the liraglutide group; and appendicitis, pneumonia, acute cholecystitis, cholelithiasis, and thrombophlebitis in the placebo group.

§The P value was calculated by means of Fisher's exact test on the basis of the number of participants.

¶The P value was calculated by means of Pearson's chi-square test on the basis of the number of participants.

With thanks to Prof Baur

- Overall, similar % of participants who reported adverse events during treatment period
- Most were mild to moderate and deemed to be unrelated to trial treatment
- Liraglutide – GIT events
- Adverse events that led to trial discontinuation – 13 in liraglutide group (10 were GIT) and 0 in placebo group
- 1 suicide in the liraglutide group – not deemed to be related to treatment

Further reflections

CONCLUSIONS

In adolescents with obesity, the use of liraglutide (3.0 mg) plus lifestyle therapy led to a significantly greater reduction in the BMI standard-deviation score than placebo plus lifestyle therapy. (Funded by Novo Nordisk; NN8022-4180 ClinicalTrials.gov number, NCT02918279.)

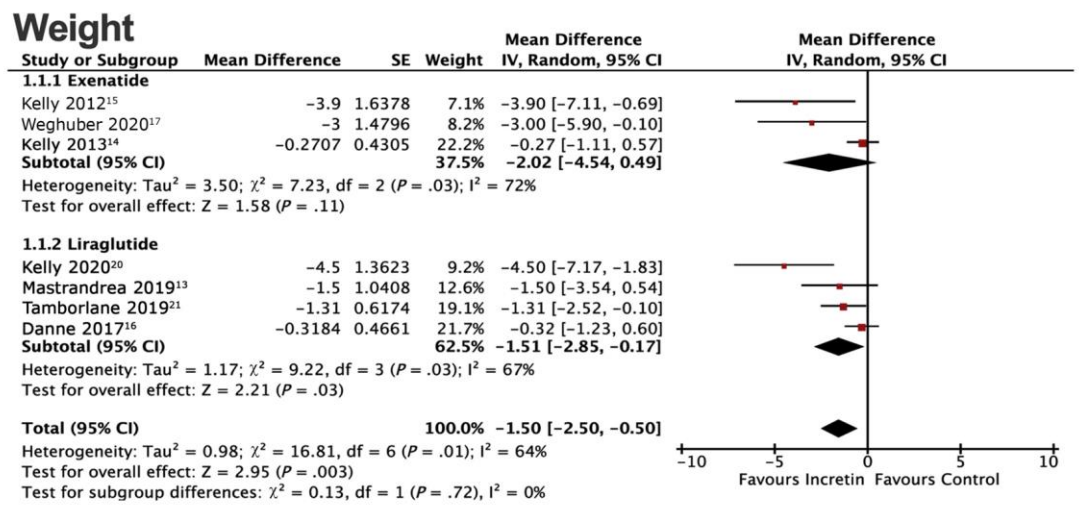
- 5.01% points in weight – "compared favourably" to 5.4% in adult trials of liraglutide
- Weight regain seen in 26 week follow-up period → reinforces need for continued treatment
- No diffs in cardiometabolic outcomes - possibly because most participants had normal values at baseline?

- → liraglutide now FDA approved for adolescents with obesity

With thanks to Prof Baur

Safety and Efficacy of Glucagon-Like Peptide-1 Receptor Agonists in Children and Adolescents with Obesity: A Meta-Analysis

A



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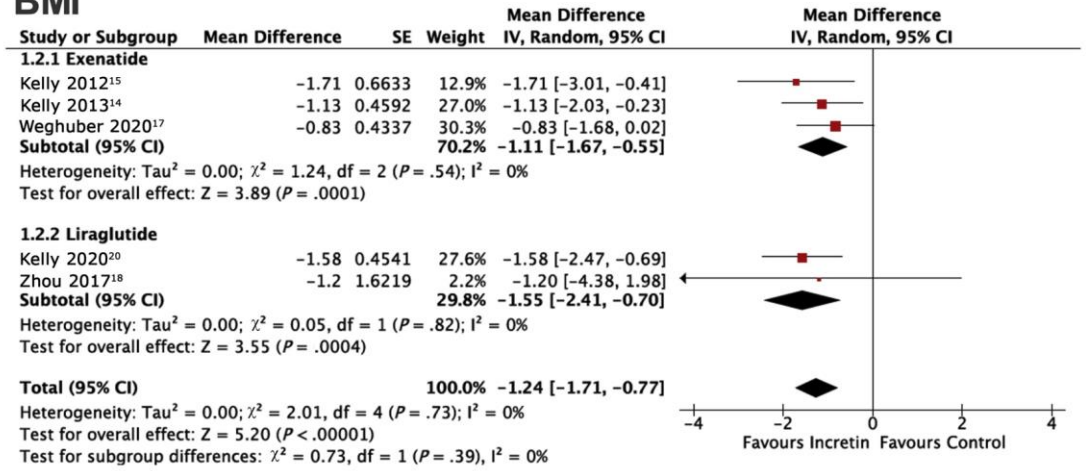
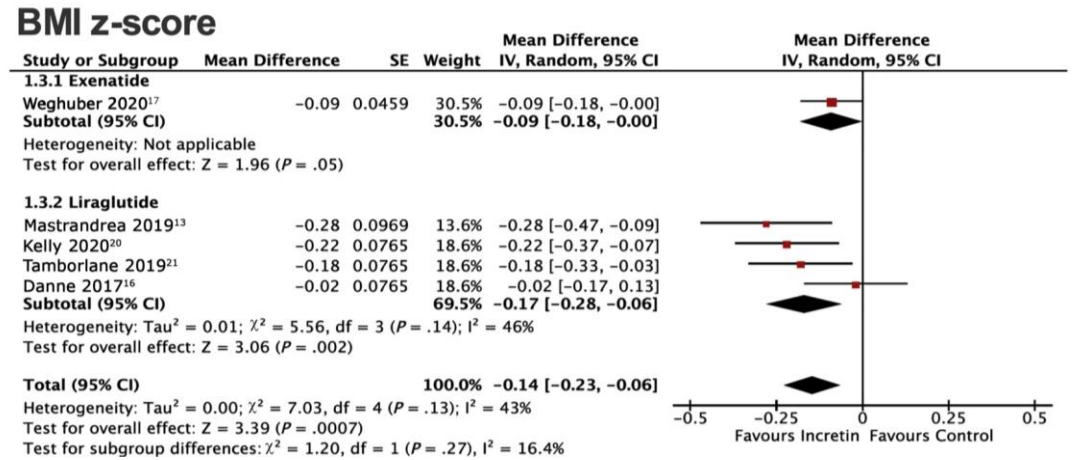


C



- Body weight (mean difference -1.50 [-2.50,-0.50] kg)
- BMI (MD -1.24 [-1.71,-0.77] kg/m^2)
- BMI z score (MD -0.14 [-0.23,-0.06])
- Hemoglobin A1c MD -1.05 [-1.93,-0.18] %)
- No lipid profile improvements noted
- Increased risk of nausea (risk ratio 2.11 [1.44, 3.09])

Ryan PM, Seltzer S, Hayward NE, Rodriguez DA, Sless RT, Hawkes CP. Safety and Efficacy of Glucagon-Like Peptide-1 Receptor Agonists in Children and Adolescents with Obesity: A Meta-Analysis. J Pediatr. 2021 Sep;236:137-147.e13. doi: 10.1016/j.jpeds.2021.05.009. Epub 2021 May 11. PMID: 33984333.

Surgery – consideration in those:

- Fully pubertal and near final height
- Extreme obesity and comorbidities which persist despite compliance with a formal program of lifestyle modification, +/-pharmacotherapy
- No underlying untreated psychiatric illness
- Stability and competence of the family unit
- Access to an experienced surgeon in a paediatric bariatric surgery centre with the necessary infrastructure for multidisciplinary patient care

RECOMMENDATIONS FOR BARIATRIC SURGERY IN ADOLESCENTS IN AUSTRALIA AND NEW ZEALAND
A position paper from the Australian and New Zealand Association of Paediatric Surgeons, the Obesity Surgery Society of Australia and New Zealand and the Paediatrics & Child Health Division of The Royal Australasian College of Physicians

With thanks to Prof Baur

Surgery – 1x RCT in adolescents

After 2 years:

↓weight vs lifestyle

34.6 kg (95% CI 30.2 to 39.0) vs 3.0 kg (95% CI 2.1 to 8.1)

↓BMI vs lifestyle

12.7kg/m^2 (95% CI 11.3 to 14.2) vs 1.3kg/m^2 (95% CI 0.4 to 2.9)

O'Brien PE et al JAMA. 2010;303(6)

With thanks to Prof Baur

Bariatric surgery in adolescents

- Several consensus guidelines for bariatric surgery, including:
  - European Society of Endocrinology *and the* Pediatric Endocrine Society
  - Surgery of Obesity and Metabolic Disorders—European Chapter *and* European Association for the Study of Obesity
  - Australia & New Zealand: Royal Australas Coll Physicians, Royal Australas. Coll Surg, Obesity Surg Soc ANZ
  - Int Diabetes Federation *and* American Diabetes Association
  - American Society for Metabolic & Bariatric Surgery Pediatric Committee
  - American Academy of Pediatrics ….

Styne DM et al. J Clin Endocrinol Metab 2017; 102:709-757; Pratte JSA et al.Surg Obes Rel Dis 2018; 14:882-901;Fried M et al. Obes Surg 2014; 24:42-55; Dixon JB et al. Diabet Med 2011; 28:628–642; Baur LA et al. J Paediatr Ch Health 2010; 46:704-7; Michalsky M et al. Surg Obes Rel Dis 2012; 8:1-7; Barlow SE and the Expert Committee of the AAP. Pediatr 2007; 120:S164-S192

With thanks to Prof Baur

Relatively common elements of consensus guidelines

- Patients
  - Severe obesity: BMI >40 kg/m^2, or >35kg/m^2 with severe co-morbidities (e.g. T2DM, mod-severe OSA, severe NASH …)
  - Late puberty, near-final adult height
  - Developmental maturity
  - (Followed 6 months of medical weight management in a specialist centre)
  - Willing to participate in pre-op and long-term post-operative treatment program
  - Informed consent
  - Family support should be optimised

With thanks to Prof Baur


Fast Track Trial
www.fasttracktrial.com

Fast Track dietary trial

Do you think your adolescent might be above a healthy weight? Are they between the age of 13 – 17 years?

The Children's Hospital at Westmead is conducting a study for young people who are above a healthy weight.

The Fast Track to Health study is a 12-month study looking at the effect of two different eating plans on health and wellbeing in young people. We want to find out which plan is the most successful at reducing weight and improving health. We may find that both work well.

We also want to find out which eating plans are acceptable to young people, so we can offer more choice and help young people with weight concerns in the future.

Contact details

How the trial works

There are three phases to the Fast Track study:

Phase 1

Everyone will follow a Very Low-Energy Diet to jump-start weight loss. This involves having meal replacements each day for 4 weeks. Meal replacements are provided free of charge.

Phase 2

Participants will be randomised to either an Intermittent Energy Restricted dietary pattern (also called the 4:3 plan) or a Reduced Calorie dietary pattern. It is not possible for participants to choose the plan they follow.

Phase 3

Everyone will continue to follow the plan they've been given with support from the dietitian.


Register
Go4FunOnline
Health Professionals
GO4FUN
HEALTHY ACTIVE HAPPY KIDS
About Go4Fun
Weight Status Calculator
Why Join?
FAQs
Find a Program
https://go4fun.com.au
Fun and fitness for kids above a healthy weight
Go4Fun is a free program for NSW children aged 7 to 13 who are above a healthy weight, and their families. Run by trained health and community professionals, it's a fun way to build self-esteem and learn about eating well, staying active and living a healthy life.
REGISTER NOW!


1300 806 258
GET STARTED
MENU
NSW GOVERNMENT
get healthy
Information & Coaching Service
For parents and family members aged 16+ years
FREE TELEPHONE-BASED HEALTH COACHING
Your **free** *NSW Health service* can help provide you with the support and motivation you need to reach your own healthy lifestyle goals.
YOUR GET HEALTHY JOURNEY
GET STARTED NOW

THANKS FOR YOUR INTEREST

SLIDES AVAILABLE HERE