ΕΘΝΙΚΟ ΚΑΙ ΚΑΠΟΔΙΣΤΡΙΑΚΟ ΠΑΝΕΠΙΣΤΗΜΙΟ ΑΘΗΝΩΝ
ΙΑΤΡΙΚΗ ΣΧΟΛΗ
Γ' ΚΑΡΔΙΟΛΟΓΙΚΗ ΚΛΙΝΙΚΗ
ΓΝΝΘΑ «Η ΣΩΤΗΡΙΑ»
ΔΙΕΥΘΥΝΤΗΣ: ΚΑΘ. Ε. ΒΑΒΟΥΡΑΝΑΚΗΣ

Καρδιαγγειακά νοσήματα και παχυσαρκία

ΓΕΡΑΣΙΜΟΣ ΣΙΑΣΟΣ
Καθηγητής Καρδιολογίας

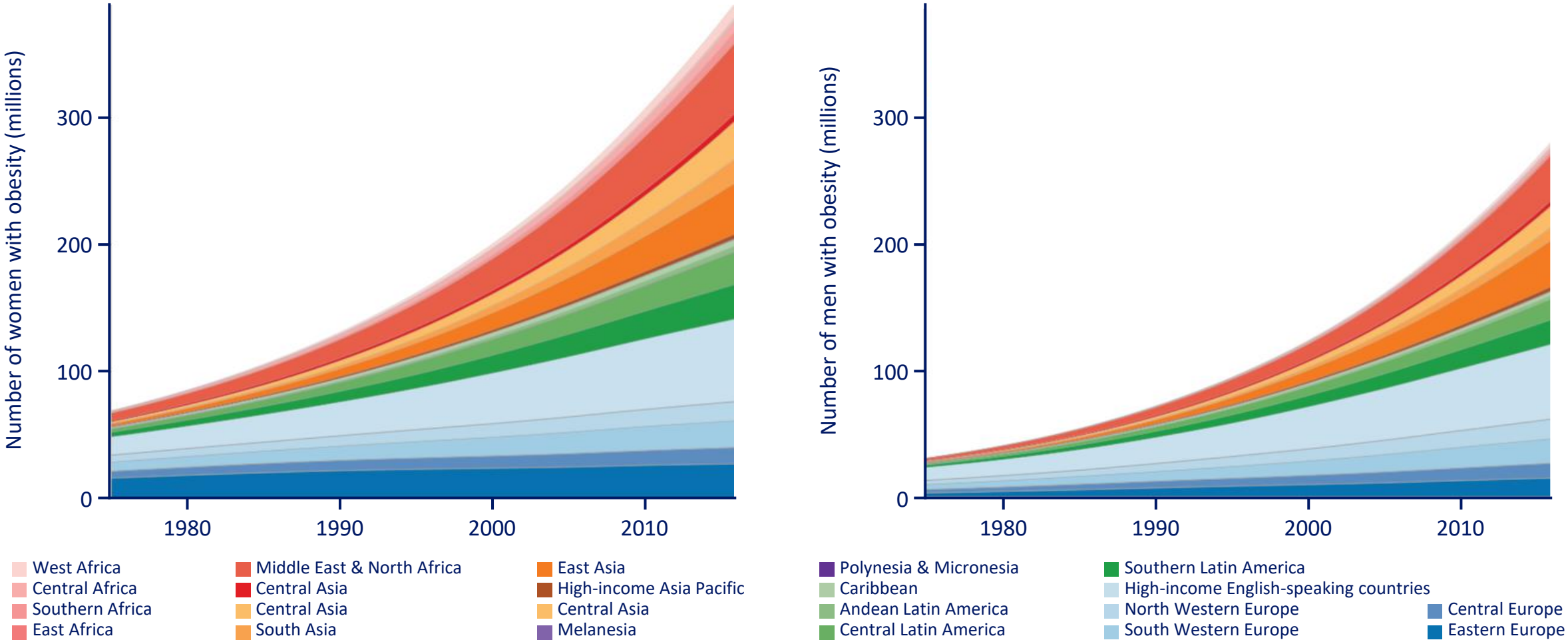
Δήλωση σύγκρουσης συμφερόντων

- Boehringer Ingelheim, NovoNordisk

Obesity

Obesity rates worldwide are increasing

Trends in the number of obese and underweight adults (20 years and older) by region

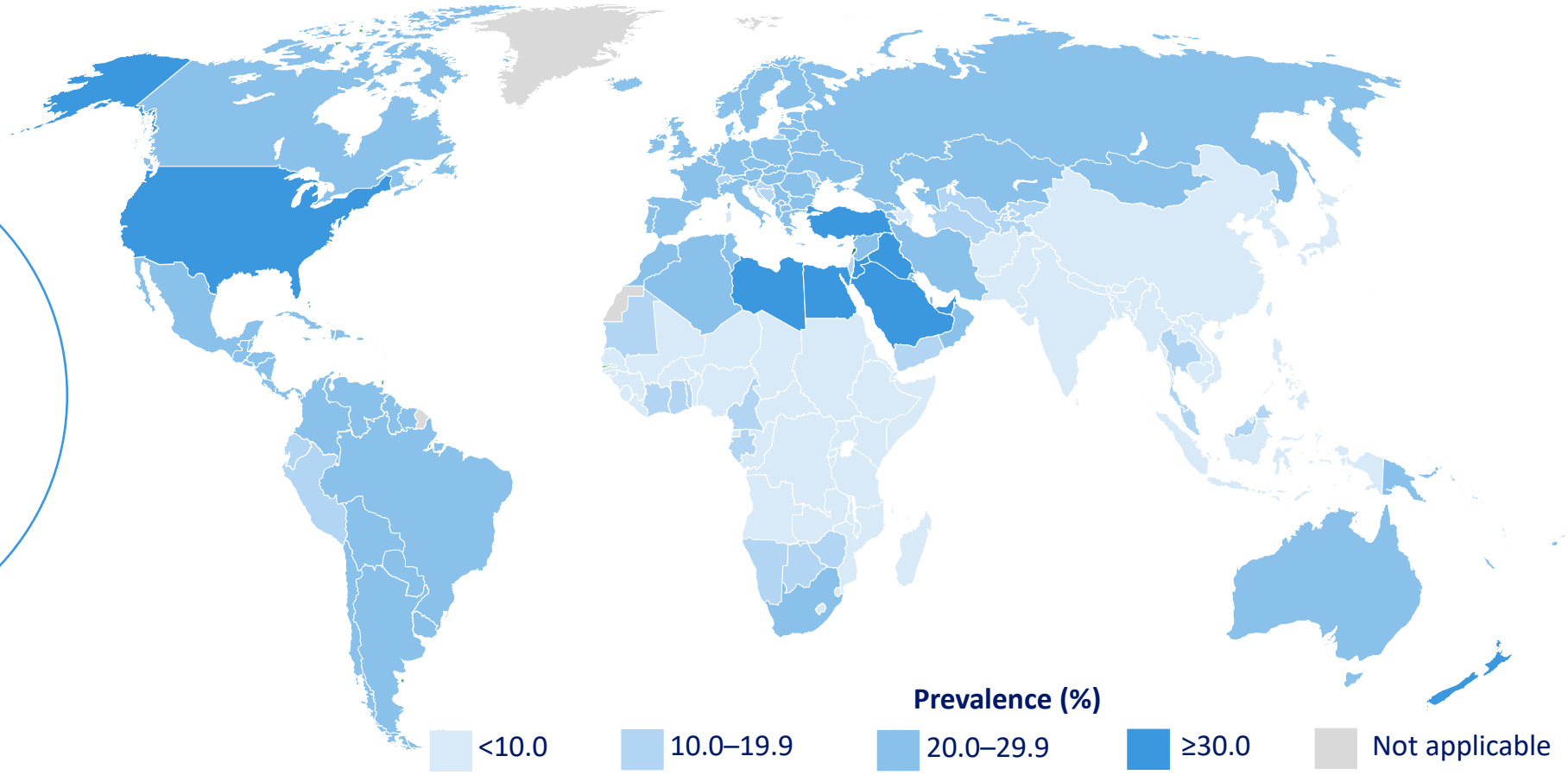


Adapted from NCD Risk Factor Collaboration (NCD-RisC). Lancet 2017:390(Supplement);2627–2642.

Global prevalence of obesity in adults

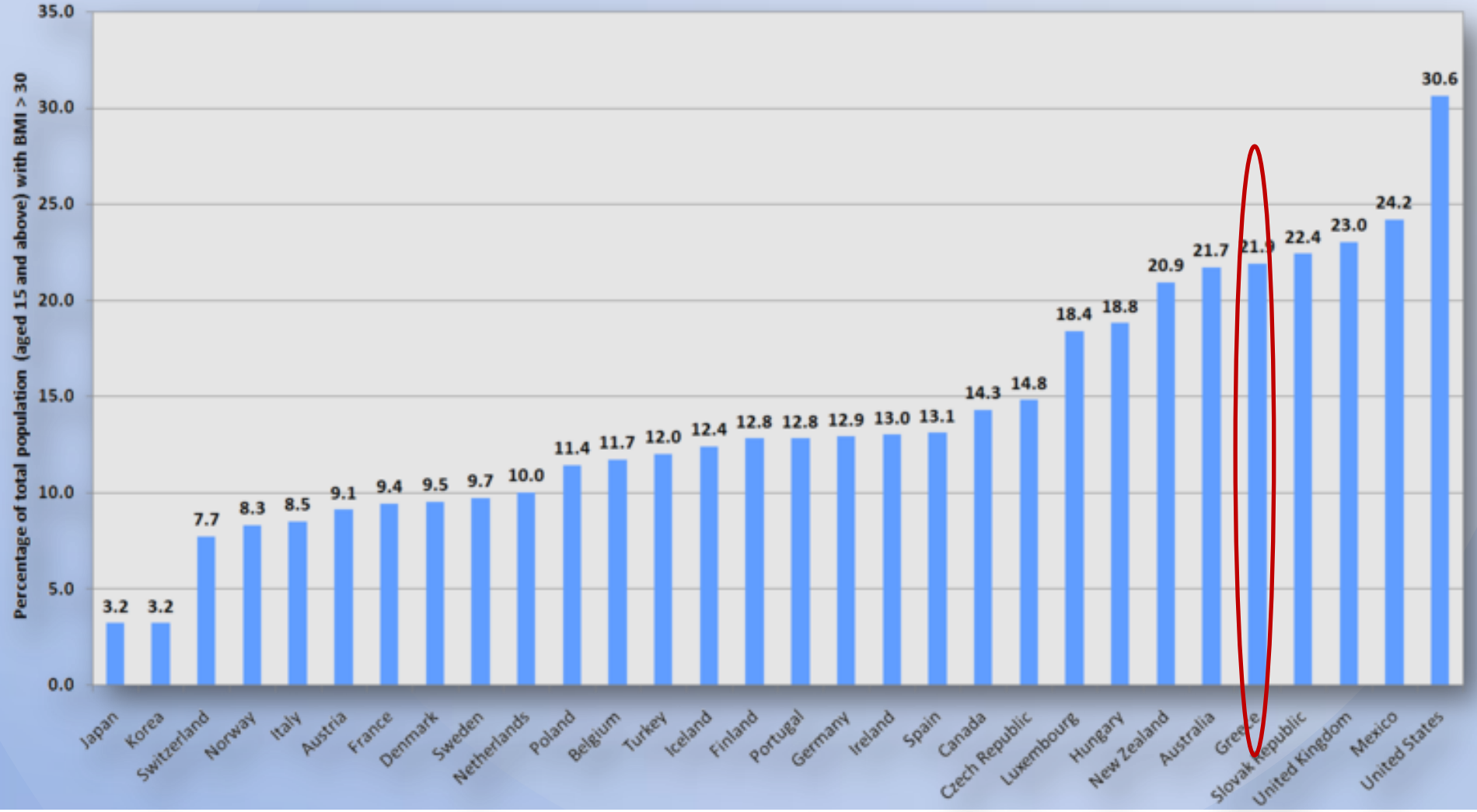
650

million people
with obesity as
of 2020



WHO, world health organization.
WHO. Global Health Observatory (GHO) data. 2017. Prevalence of obesity among adults. Available from https://www.who.int/gho/ncd/risk_factors/overweight_obesity/obesity_adults/en/. Accessed August 2020; WHO, Obesity & Overweight. 2020. Available from https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight. Accessed August 2020.

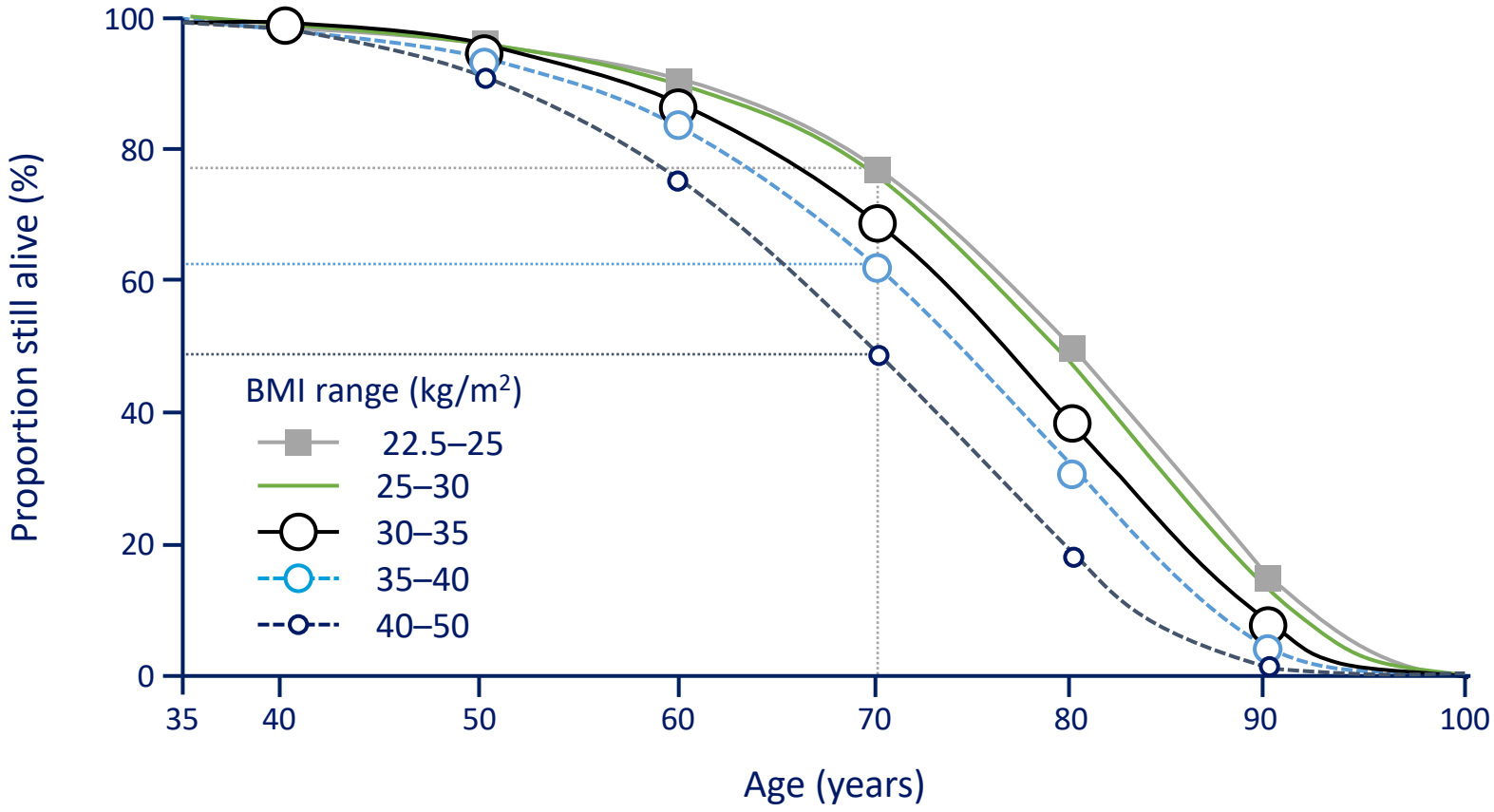
Where do nations rank in the global obesity stakes?



International Diabetes

Bariatric Surgical and Procedural Interventions in the Treatment of Obese Patients with Type 2 Diabetes

Life expectancy decreases as BMI increases



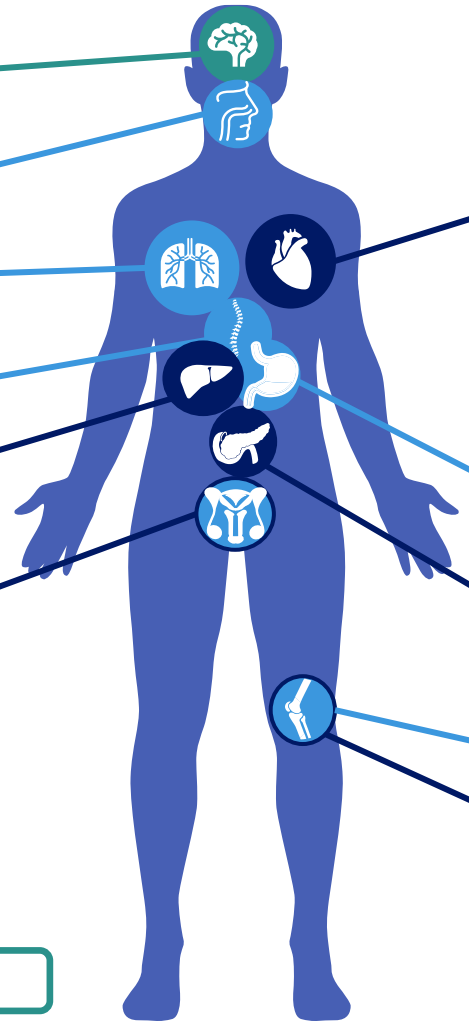
Normal BMI =
almost 80% chance of reaching age 70

BMI 35–40 kg/m² =
~60% chance of reaching age 70

BMI 40–50 kg/m² =
~50% chance of reaching age 70

Data are based on male subjects; n=541,452. BMI, body mass index.
Prospective Studies Collaboration. Lancet 2009;373:1083–1096.

Obesity is associated with multiple complications



Depression
Anxiety

Sleep Apnoea
Asthma

Chronic back pain

NALFD

Hypogonadism
PCOS
Pregnancy complications
Infertility/subfertility

Cardiovascular disease:
- Stroke
- Dyslipidaemia
- Hypertension
- Coronary artery disease
- Pulmonary embolism
- HFpEF
- Thrombosis

GERD
Incontinence

Type 2 diabetes
Prediabetes

Knee osteoarthritis

Gout

Cancers*

Metabolic | Mechanical | Mind

**Including breast, colorectal, endometrial, oesophageal, kidney, ovarian, pancreatic and prostate. GERD, gastro-oesophageal reflux disease; HFpEF, heart failure with preserved ejection fraction; NAFLD, non-alcoholic fatty liver disease.*
Adapted from Sharma AM. Obes Rev 2010;11:808–809; Guh DP et al. BMC Public Health 2009;9:88; Luppino FS et al. Arch Gen Psychiatry 2010;67:220–229; Simon GE et al. Arch Gen Psychiatry 2006;63:824–830; Church TS et al. Gastroenterology 2006;130:2023–2030; Li C et al. Prev Med 2010;51:18–23; Hosler AS. Prev Chronic Dis 2009;6:A48.

Obesity and Diabetes

Diabetes

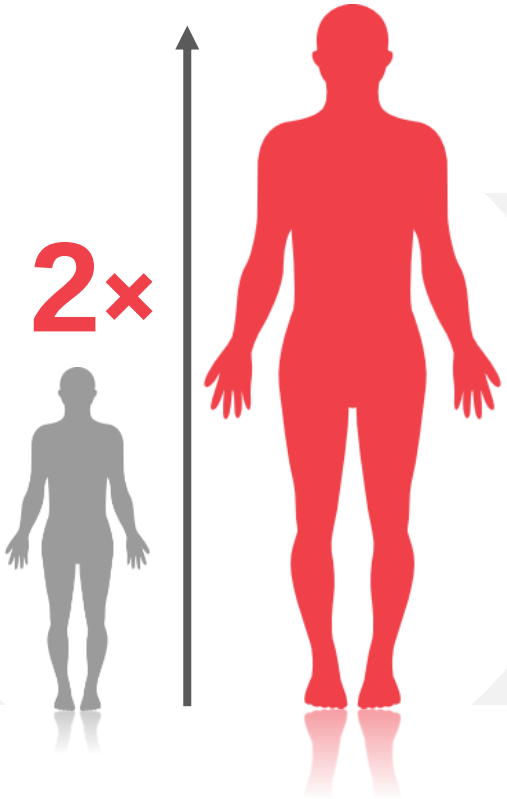
- Risk of DM increases with BMI, even at normal weight[a]
- Obesity worsens glycemic control in DM[b]

Metabolic syndrome

- Insulin resistance
- Obesity
- Hypertension
- Dyslipidemia

a. Colditz GA, et al. *Ann Intern Med*. 1995;122:481-486; b. Yki-Järvinen H, et al. *J Clin Endocrinol Metab*. 1997;82:4037-4043.

Diabetes and CVD



Οι ασθενείς με ΣΔτ2 έχουν

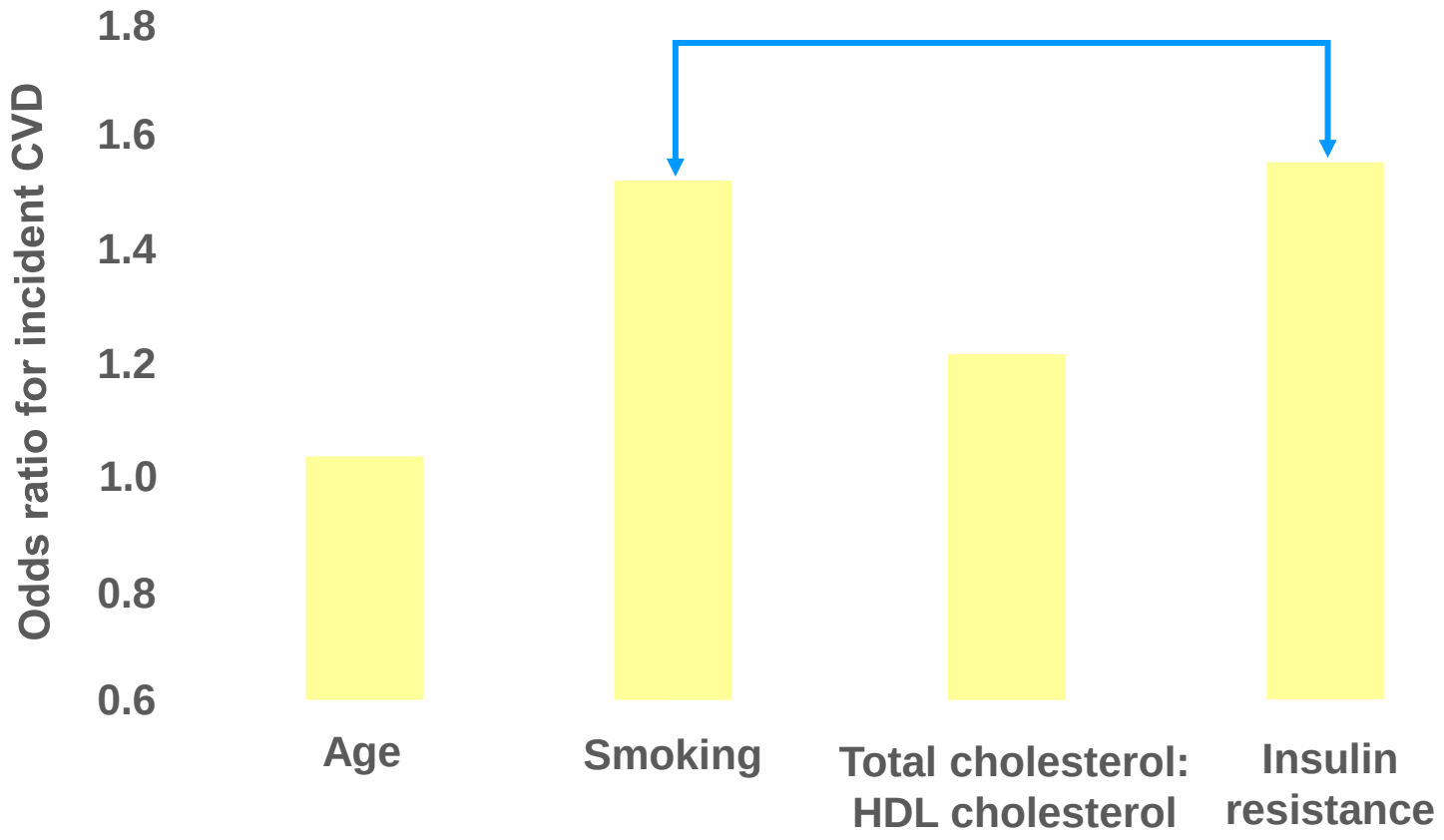
Διπλάσιο Κίνδυνο

για ΚΔ Νόσο σε σύγκριση με τον γενικό πληθυσμό

ΚΔ: Καρδιαγγειακής, ΣΔτ2 Σακχαρώδης Διαβήτης τύπου 2

Sarwar N *et al. Lancet* 2010;375:2215

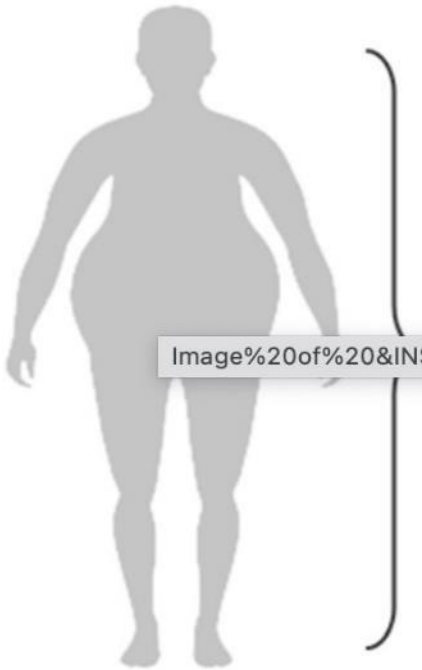
Insulin resistance is as strong a risk factor for cardiovascular disease as smoking



Bonora E, *et al. Diabetes Care* 2002; 25:1135–1141.

Obesity and CVD

Obesity and CVD



Cardiovascular and metabolic effects of obesity	
Insulin Resistance	**Cardiac Function**
Glucose intolerance	LV diastolic dysfunction
Metabolic syndrome	LV systolic dysfunction
Type 2 diabetes mellitus	RV failure
Dyslipidemia	**Neurohormonal Disturbances**
Elevated triglycerides	Sympathetic nervous system activation
Increased VLDL-triglycerides	Activation of the renin–angiotensin–aldosterone system
Elevated small, dense LDL particles	Reduced levels of atrial and brain natriuretic peptide
Elevated apolipoprotein B levels	
Decreased HDL-cholesterol levels	**Metabolic Dysregulation**
Elevated non HDL-cholesterol levels	Accumulation of toxic lipid metabolites
	Mitochondrial dysfunction
ɔdynamics	Reduced metabolic flexibility
Increased blood volume	Impaired myocardial metabolism
Increased stroke volume	
Increased cardiac output	**Ectopic Lipid Accumulation**
Decreased peripheral vascular resistance	
Increased arterial pressure	**Low-Grade Systemic Inflammation**
Increased left ventricular (LV) wall stress	
Pulmonary artery hypertension	**Increased Pro-Inflammatory Adipo(cyto)kines**
Cardiac Structure	**Endothelial Dysfunction**
LV hypertrophy	
LV enlargement	
Left atrial enlargement	
Right ventricular (RV) enlargement	

Cardiovascular outcomes
▪ Increased cardiovascular risk factor burden
▪ Increased coronary artery disease
▪ Increased heart failure
▪ Increased atrial fibrillation
▪ Increased cerebrovascular disease
▪ Increased sudden death

M.-E. Piché et al. / Progress in Cardiovascular Diseases 61 (2018) 103–113

Obesity and CVD

Hypertension	• Risk of HTN up to 5x higher in obesity[a] • 60 to 70% of HTN in adults attributable to weight[b]
Dyslipidemia	• Patients with obesity have higher triglycerides, lower HDL-C[c]
CAD	• Each unit change in BMI increases ischemic events by 9%[d]

a. Cassano PA, et al. *Ann Epidemiol.* 1990;1:33-48. b. Kotchen T. *Am J Hypertens.* 2010;23:1170-1178; c. Karaouzene N. *Nutr Metab Cardiovasc Dis.* 2011; 21:792-799. d. James WT, et al. *Overweight and obesity*. WHO;2004:497-596.

Obesity and CVD

Atrial fibrillation

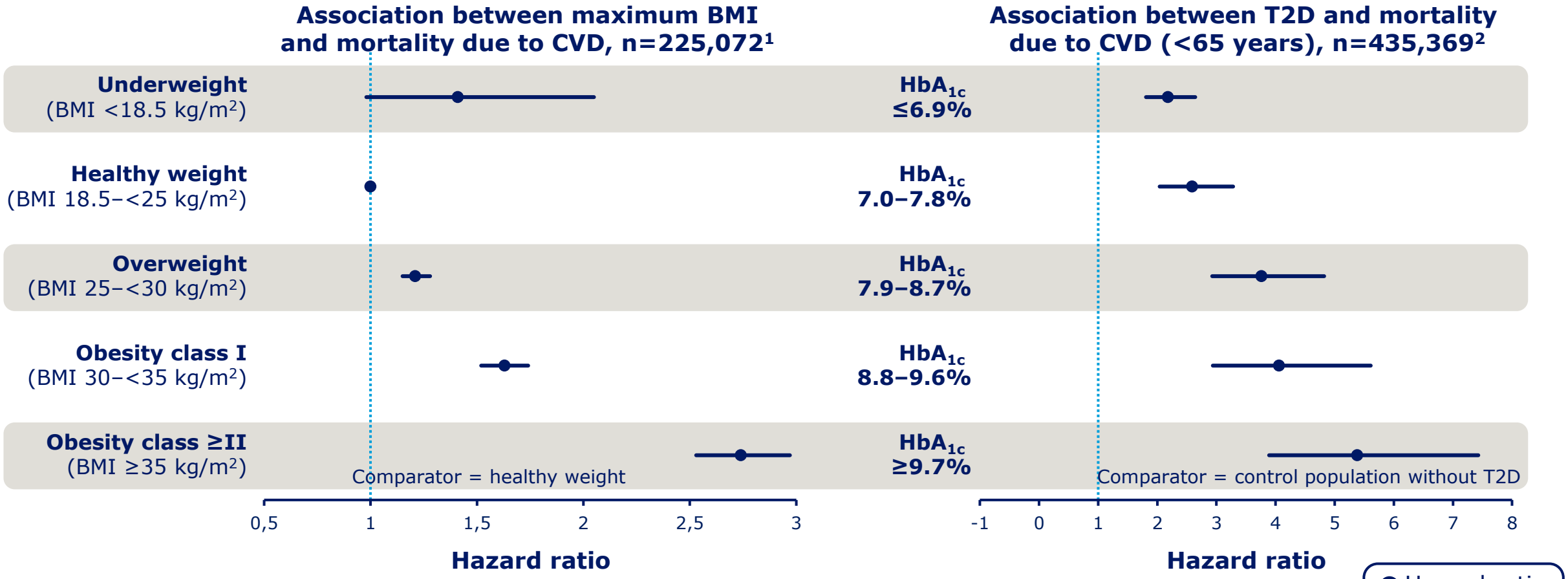
- Each unit change in BMI increases AF risk by 4.7%[a]

HFpEF

- 1-SD increase in BMI associated with 1.34-fold increased risk of future HFpEF[b]

a. Tedrow UB, et al. *J Am Coll Cardiol.* 2010;55:2319-2327; b. Savji N, et al. *JACC Heart Fail.* 2018;6:701-709.

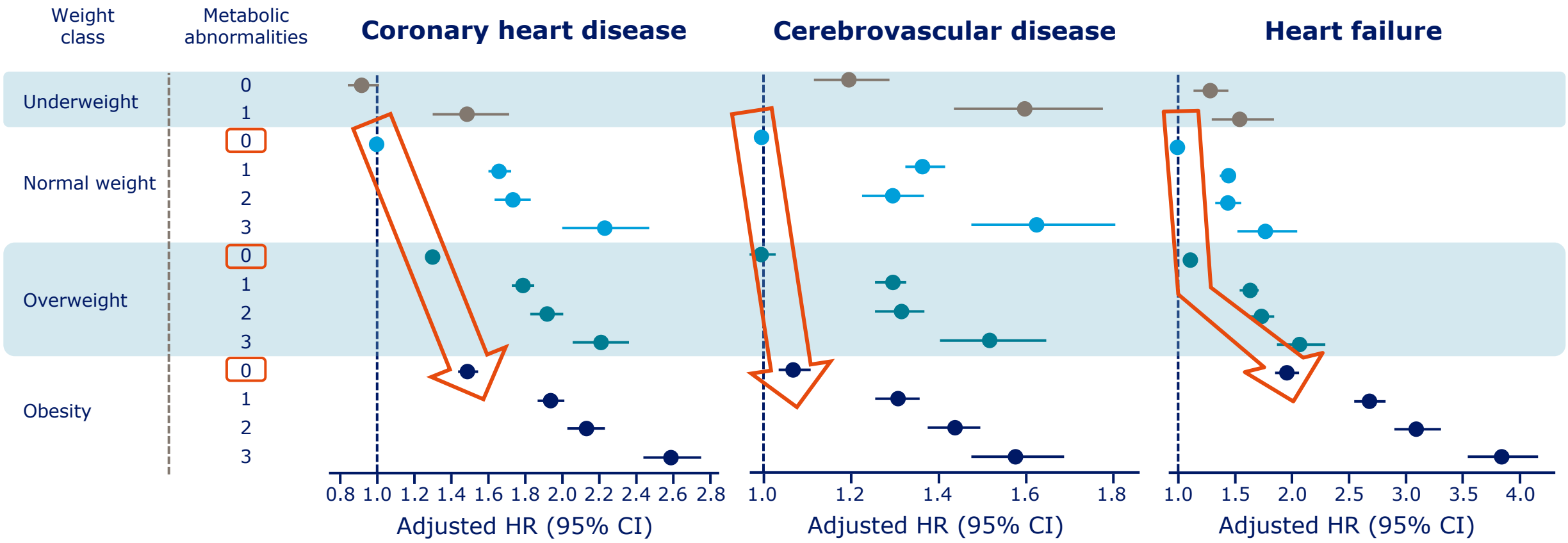
Greater risk of CVD mortality with increased BMI and HbA_{1c}



Left, pooled data from the Nurses Health Study, Nurses Health Study II and Health Professionals Follow-Up Study
Right, individuals with T2D from the Swedish National Diabetes Register and controls without T2D matched for age, sex and county
Multivariate analyses, adjusting for various CVD risk factors. CI, confidence interval; CVD, cardiovascular disease

1. Yu et al. *Ann Intern Med* 2017;166:613-620; 2. Tancredi et al. *N Engl J Med* 2015;373:1720–32

Overweight and obesity increase the risk of CVD even in the absence of metabolic abnormalities



Body size, metabolic status and CVD events in 3.5 million UK adults. Analyses adjusted for age, sex, smoking status, and social deprivation. The reference category is normal weight, no metabolic abnormalities. CVD, cardiovascular disease

Caleyachetty *et al. J Am Coll Cardiol*. 2017;70:1429–37

Obesity and Heart Failure

Heart failure poses a significant global disease burden

>60 million patients worldwide have heart failure

In **2016**, HF was the underlying cause in almost **80,000** deaths in the US

Approximately **50% of patients** diagnosed with heart failure will **die within 5 years**

1. GBD 2016 Disease and Injury Incidence and Prevalence Collaborators. *Lancet* 2017;390:1211; 2. Benjamin EL *et al. Circulation* 2019;139:e56; 3. Mozaffarian D *et al. Circulation* 2016;133:e38; 4. Mamas MA *et al. Eur J Heart Fail* 2017;19:1095

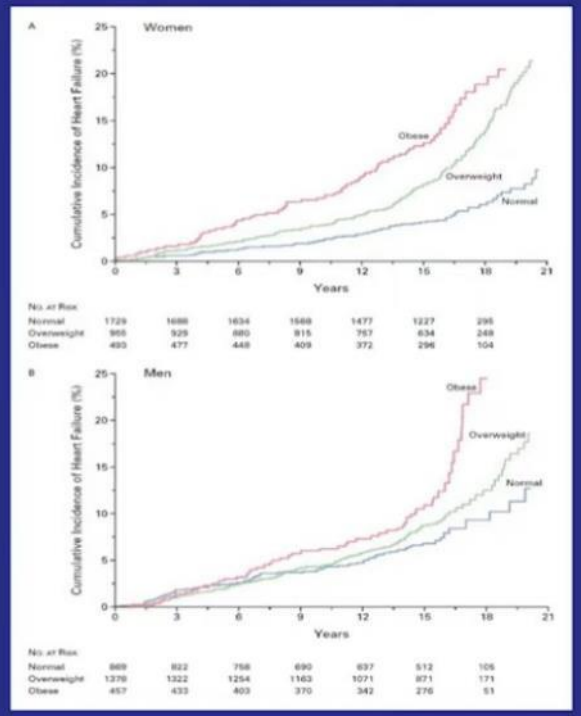
Obesity ⟷ Heart Failure

- Obesity is a risk factor for the development of HF[1-2]
- Obesity and HF often co-exist [3]
  - 15% to 35% of patients with HF are obese
  - 30% to 60% of patients with HF are overweight

Obesity and Heart Failure

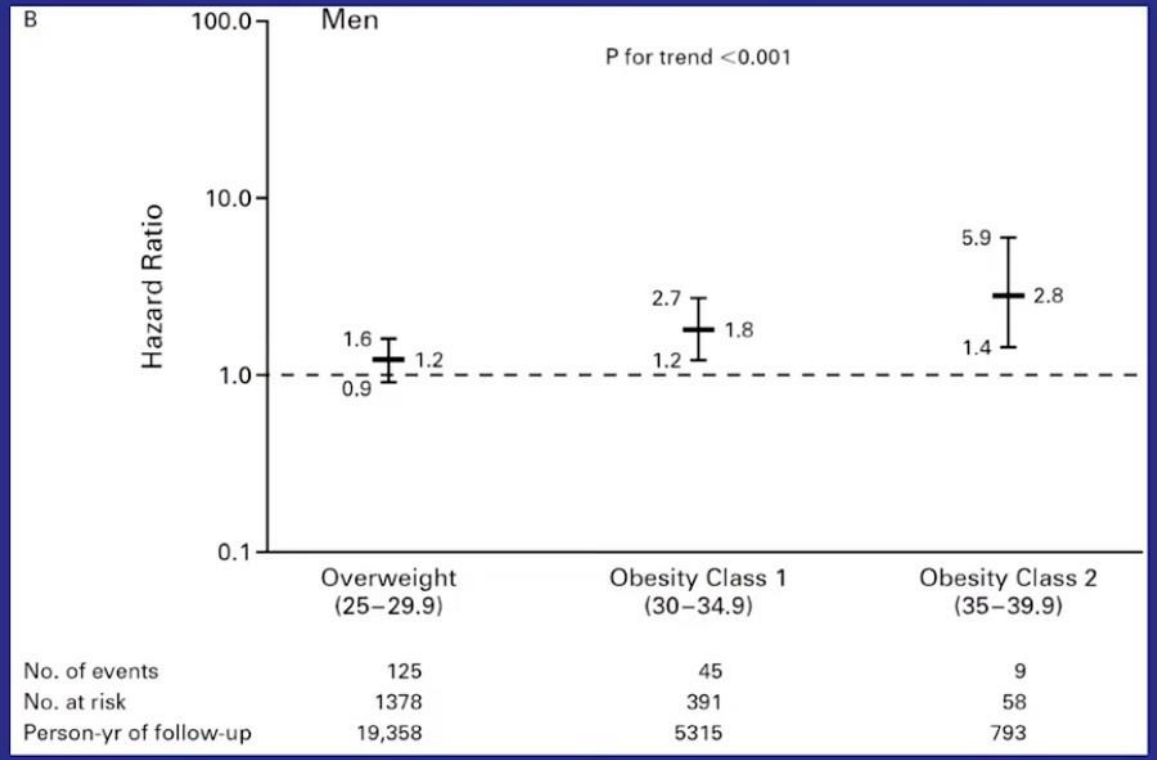
Obesity and the Risk of Heart Failure

- 5881 participants in Framingham Heart Study
- HF developed in 496 participants over 14 years of follow-up
- Used Cox models to relate baseline BMI groups to incident HF



Kenchaiah et al. N Engl J Med 2002; 347:305-13

Obesity and the Risk of Heart Failure



Kenchaiah et al. N Engl J Med 2002; 347:305-13

Obesity and Heart Failure

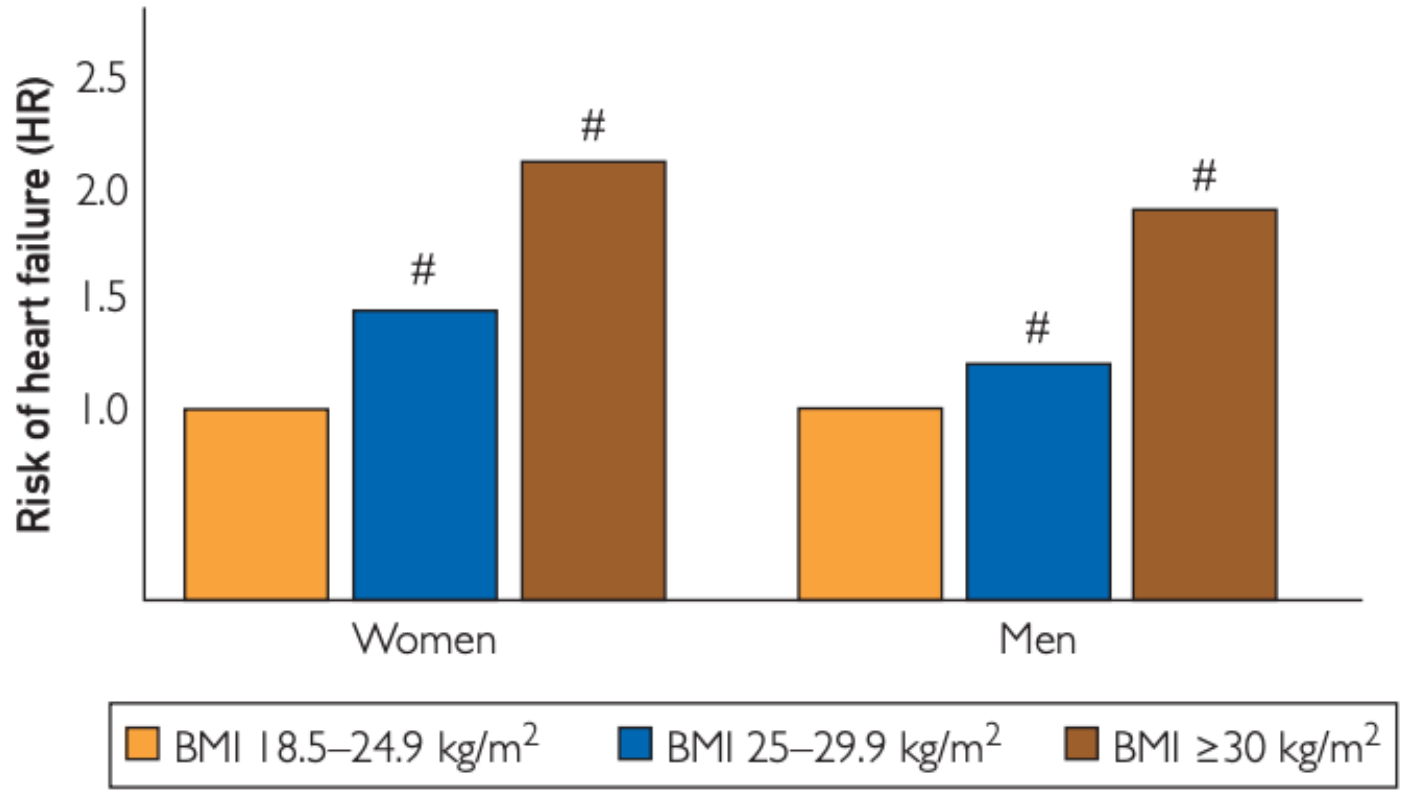


FIGURE 1. Risk of heart failure according to categories of body mass index (BMI). Considering a BMI of 18.5 to 24.9 kg/m^2 as the reference category, overweight and obese individuals had an increased risk of heart failure with reduced and preserved ejection fraction, described as hazard ratio (HR). # = $P<.01$ vs BMI of 18.5 to 24.9 kg/m^2. Data from *N Engl J Med*.[4]

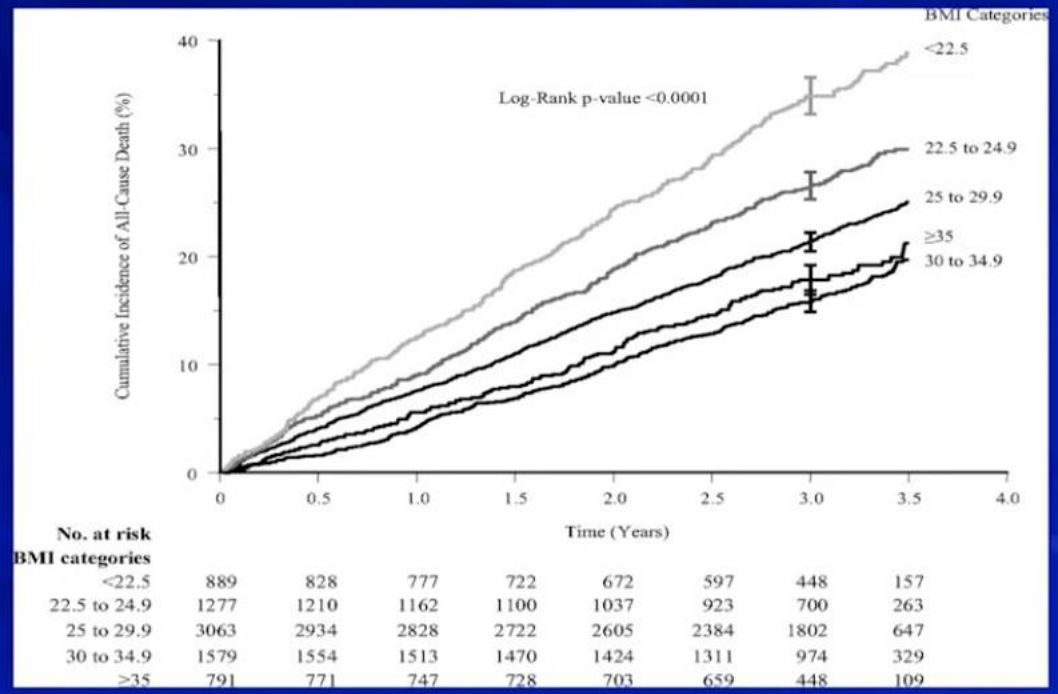
Obesity and Heart Failure

BMI and Mortality in Established HF

- 7599 participants in Candesartan in Heart failure: Assessment of Reduction in Mortality and morbidity (CHARM) Program
- NYHA Class II to IV
- Mean EF 39%

Kenchaiah et al. Circulation. 2007;116:627-636

BMI and Mortality in Established HF

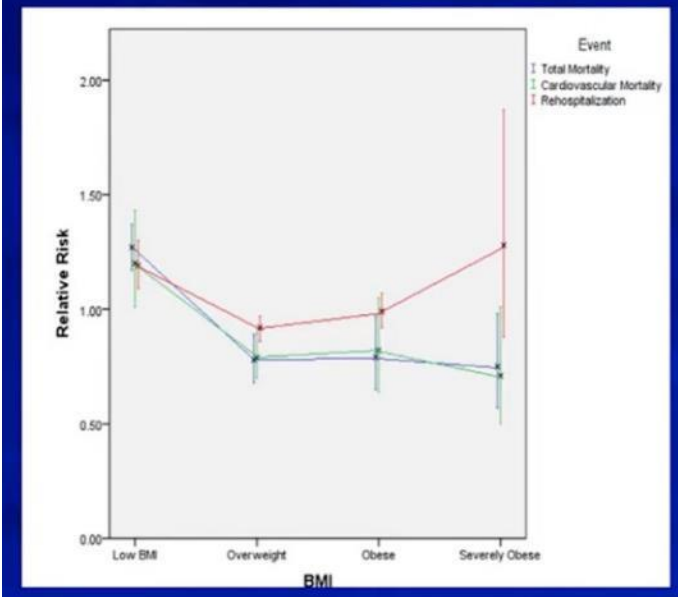


No. at risk BMI categories								
<22.5	889	828	777	722	672	597	448	157
22.5 to 24.9	1277	1210	1162	1100	1037	923	700	263
25 to 29.9	3063	2934	2828	2722	2605	2384	1802	647
30 to 34.9	1579	1554	1513	1470	1424	1311	974	329
≥35	791	771	747	728	703	659	448	109

Kenchaiah et al. Circulation. 2007;116:627-636

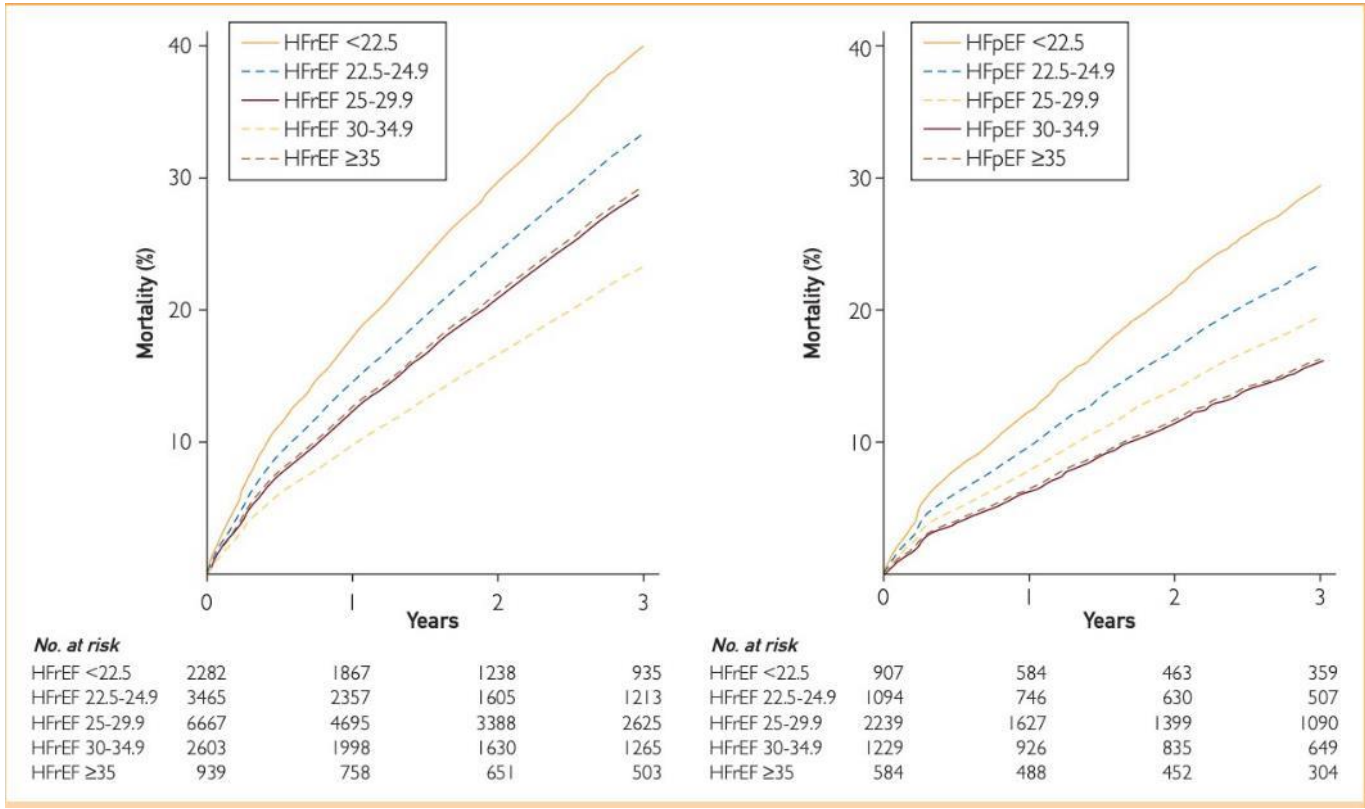
Obesity and Heart Failure

Total mortality, cardiac mortality, and hospitalization by BMI categories



Obesity associated with higher risk of HF readmission but lower cardiac and all-cause mortality

Sharma et al. Am J Cardiol 2015;115:1428-1434)



No. at risk				
HFrEF <22.5	2282	1867	1238	935
HFrEF 22.5-24.9	3465	2357	1605	1213
HFrEF 25-29.9	6667	4695	3388	2625
HFrEF 30-34.9	2603	1998	1630	1265
HFrEF ≥35	939	758	651	503

No. at risk				
HFrEF <22.5	907	584	463	359
HFrEF 22.5-24.9	1094	746	630	507
HFrEF 25-29.9	2239	1627	1399	1090
HFrEF 30-34.9	1229	926	835	649
HFrEF ≥35	584	488	452	304

Obesity, Heart Failure and Cardiorespiratory fitness

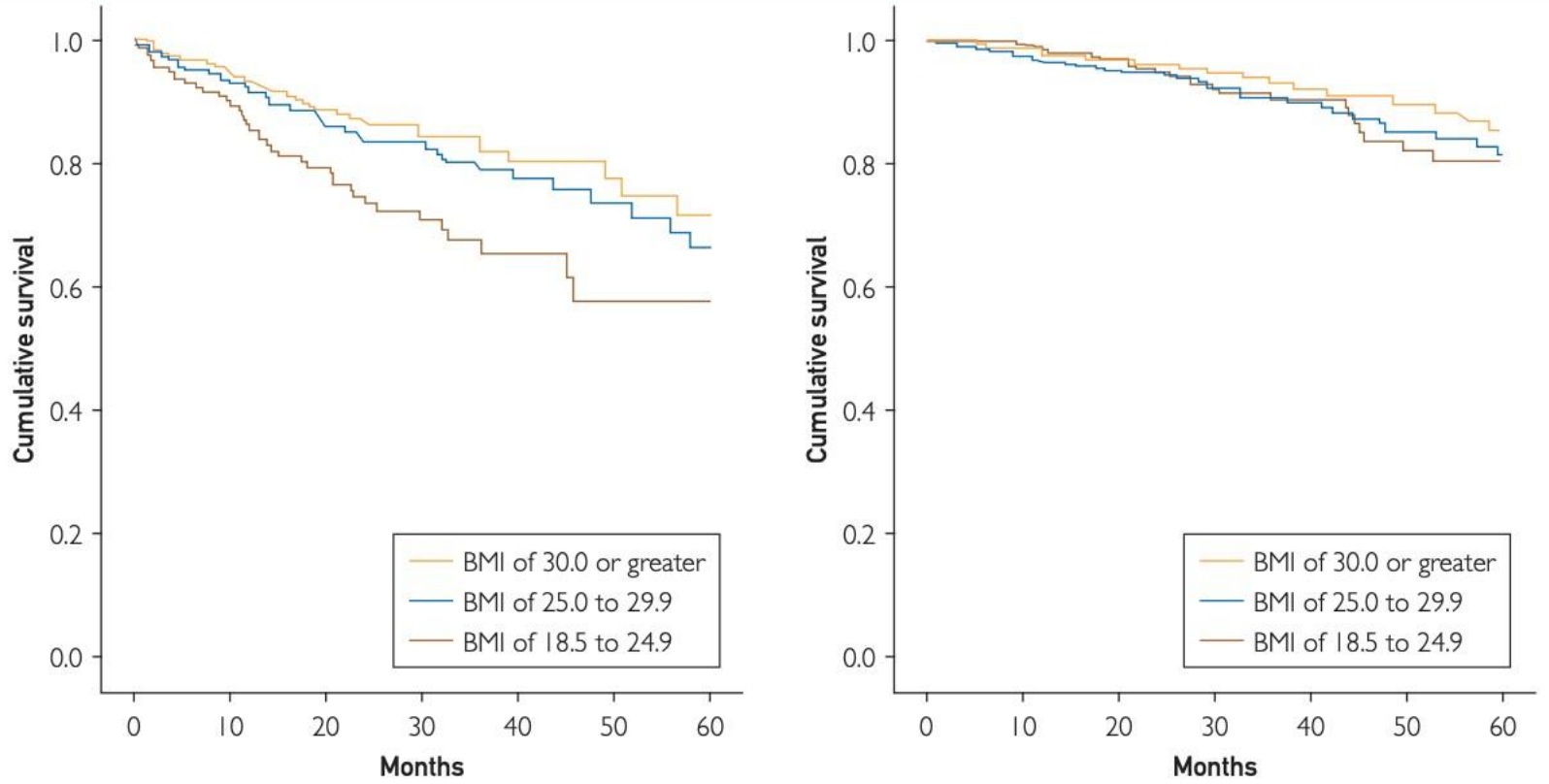


FIGURE 5. Obesity paradox and cardiorespiratory fitness (CRF). Kaplan-Meier analysis according to body mass index (BMI) in the low CRF group (peak oxygen consumption <14 mL•kg^{-1}•min^{-1}) (left) and in the high CRF group (peak oxygen consumption >14 mL•kg^{-1}•min^{-1}) (right). This figure describes the absence of the obesity paradox in patients with relatively high CRF (right) compared with those who have low CRF, in which the obesity paradox is apparent. From *Mayo Clin Proc.*[122]

JAMA. 2014;311(8):806-814.

Reasons for the obesity paradox

- Age: heavier patients are often younger
- Smoking: smokers are more likely to be lean than overweight or obese
- Better nutritional status and energy reserves
- Increased muscle mass and strength in patients who are overweight or obese
- Reverse epidemiology, frailty, and cachexia in patients who are leaner
- Implications of fitness levels for health

Heart Failure Clin (2018)

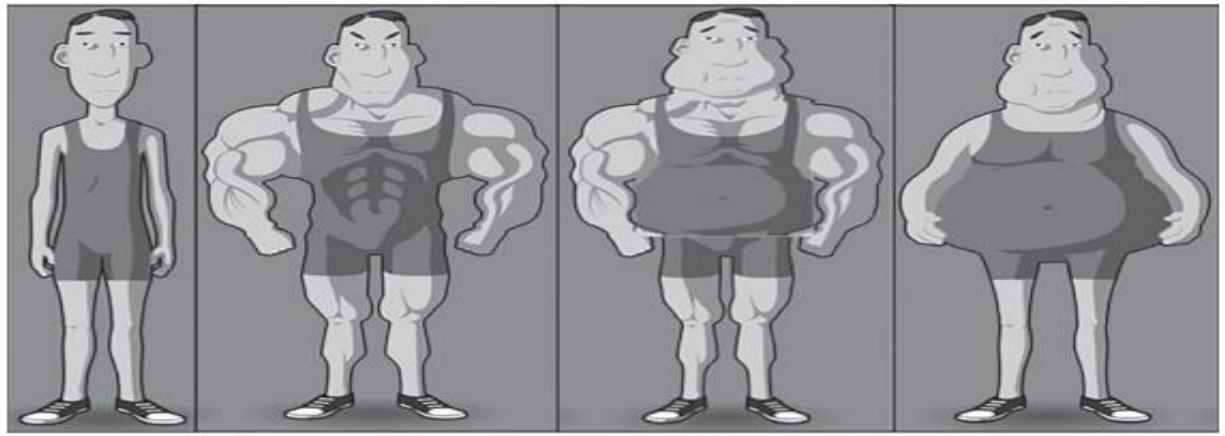
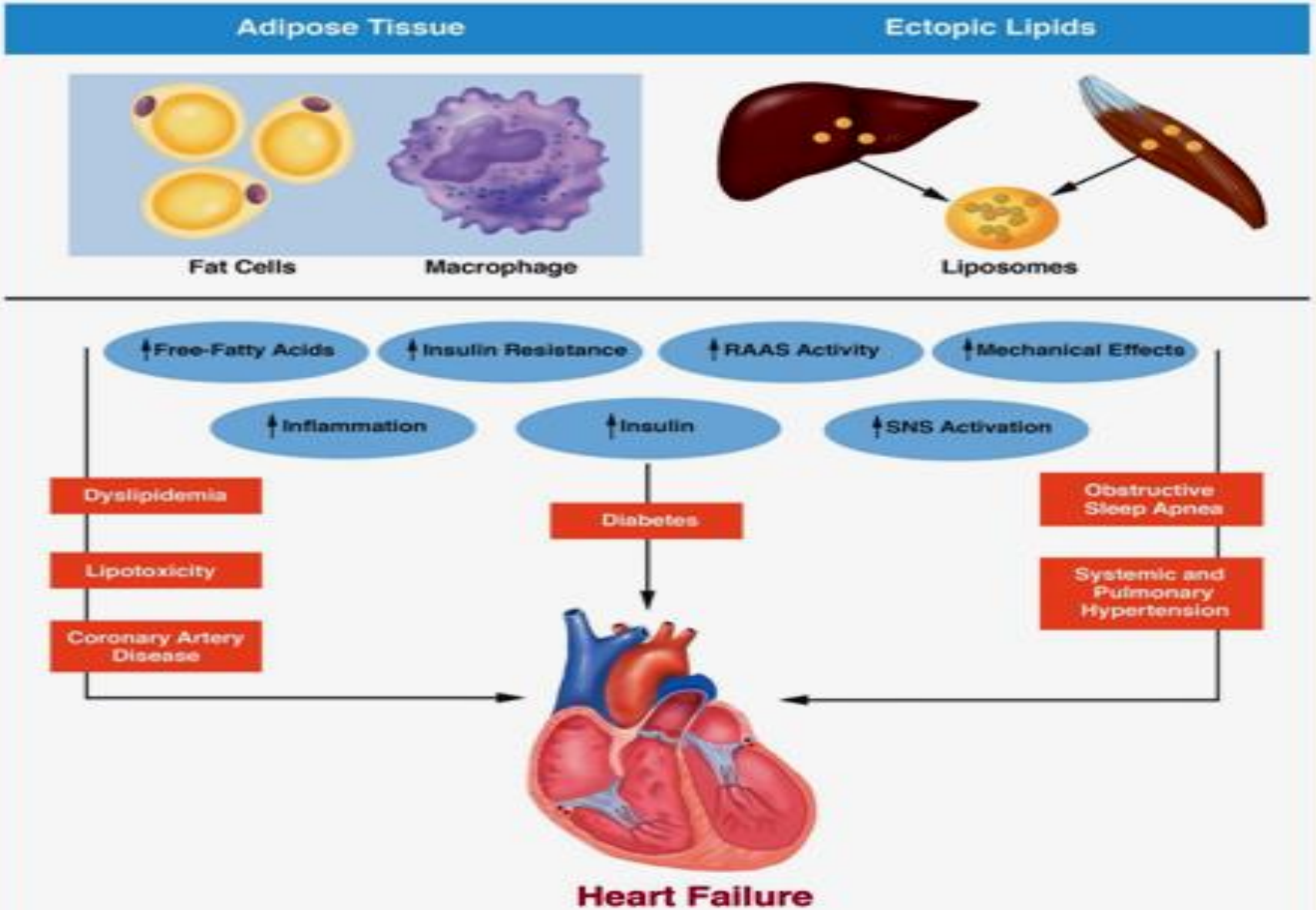
Body composition and obesity phenotypes				
	Normal weight	**Athlete**	**Nonsarcopenic obese**	**Sarcopenic obese**
BMI (kg/m^2)	18.5-25	>30	>30	>30
Fat mass	Normal	Decreased	Increased	Increased
Lean mass	Normal	Increased	Increased	Decreased
Cardiac function	Normal	Normal	***Mild dysfunction?***	***Severe dysfunction?***
Cardio-respiratory fitness	Normal	Increased	***Mild impairment?***	***Severe impairment?***

FIGURE 4. Hypothetical relationship between obesity phenotypes, cardiac function, and cardiorespiratory fitness in patients with heart failure. The figure highlights the proposed major role of body composition, obesity phenotypes, and lean mass in the development and progression of cardiac dysfunction and cardiorespiratory fitness abnormalities. BMI = body mass index. Adapted from *EC Cardiol.*[114]

Mayo Clin Proc. 2017;92(2):266-279

Mechanisms of Disease

CENTRAL ILLUSTRATION: Adiposity-Associated Major Risk Factors for Developing HF and Other Weight-Related Comorbidities



Gadde, K.M. et al. J Am Coll Cardiol. 2018;71(1):69-84.

Obesity and Cardiac dysfunction

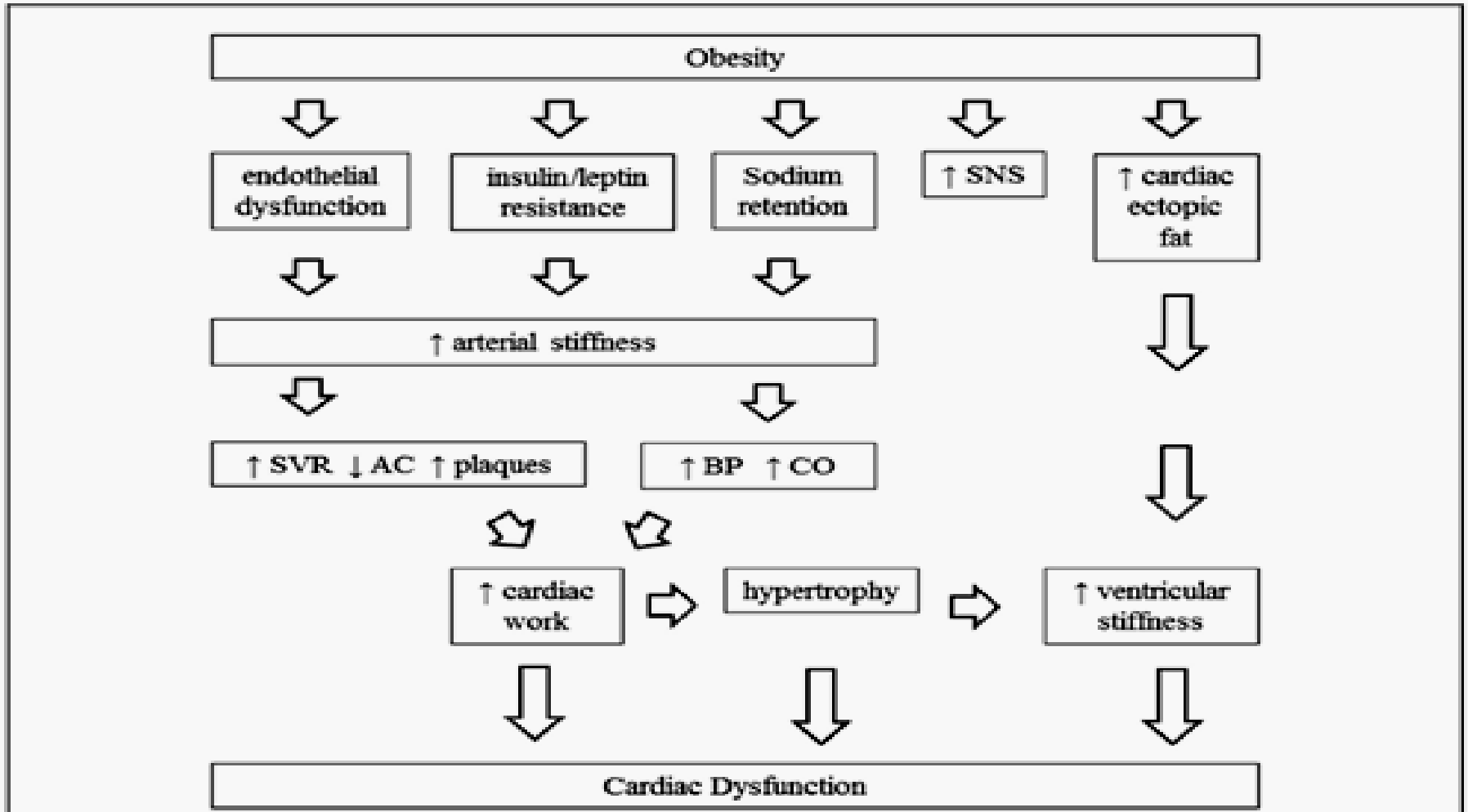
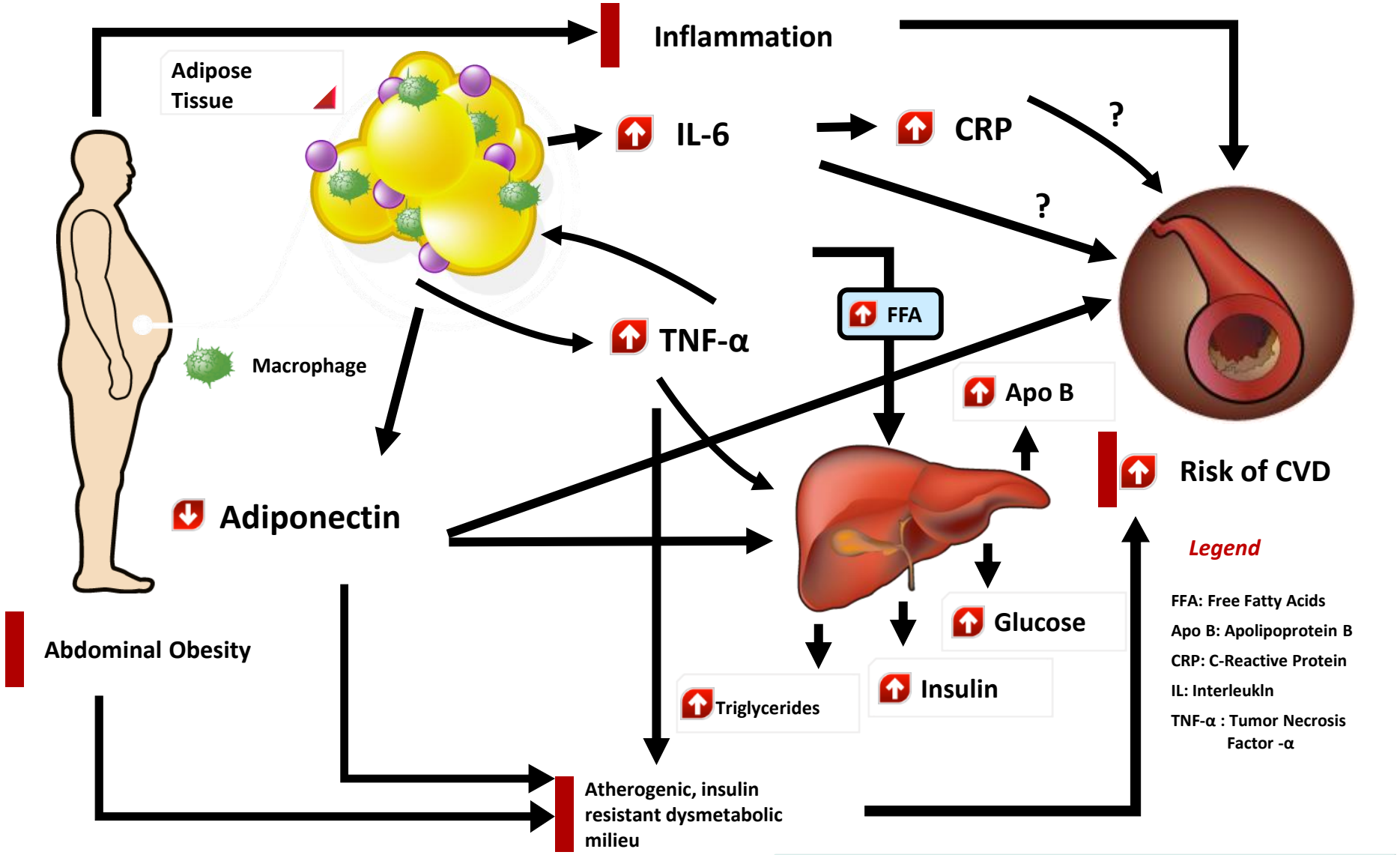


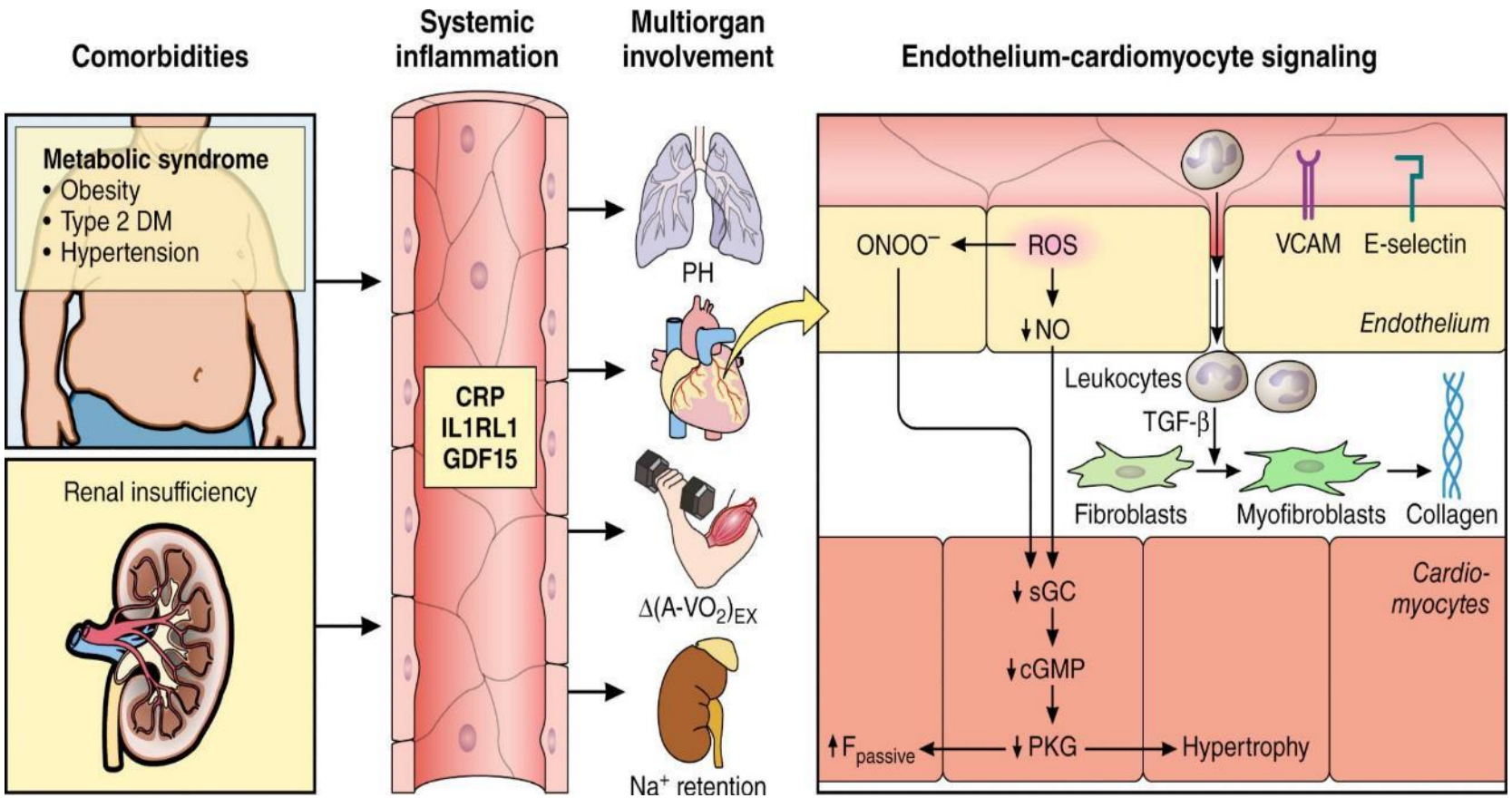
Figure 1 Obesity-Related Mechanisms of Cardiovascular Dysfunction in Children

INFLAMMATION: THE LINK BETWEEN OBESITY AND CVD

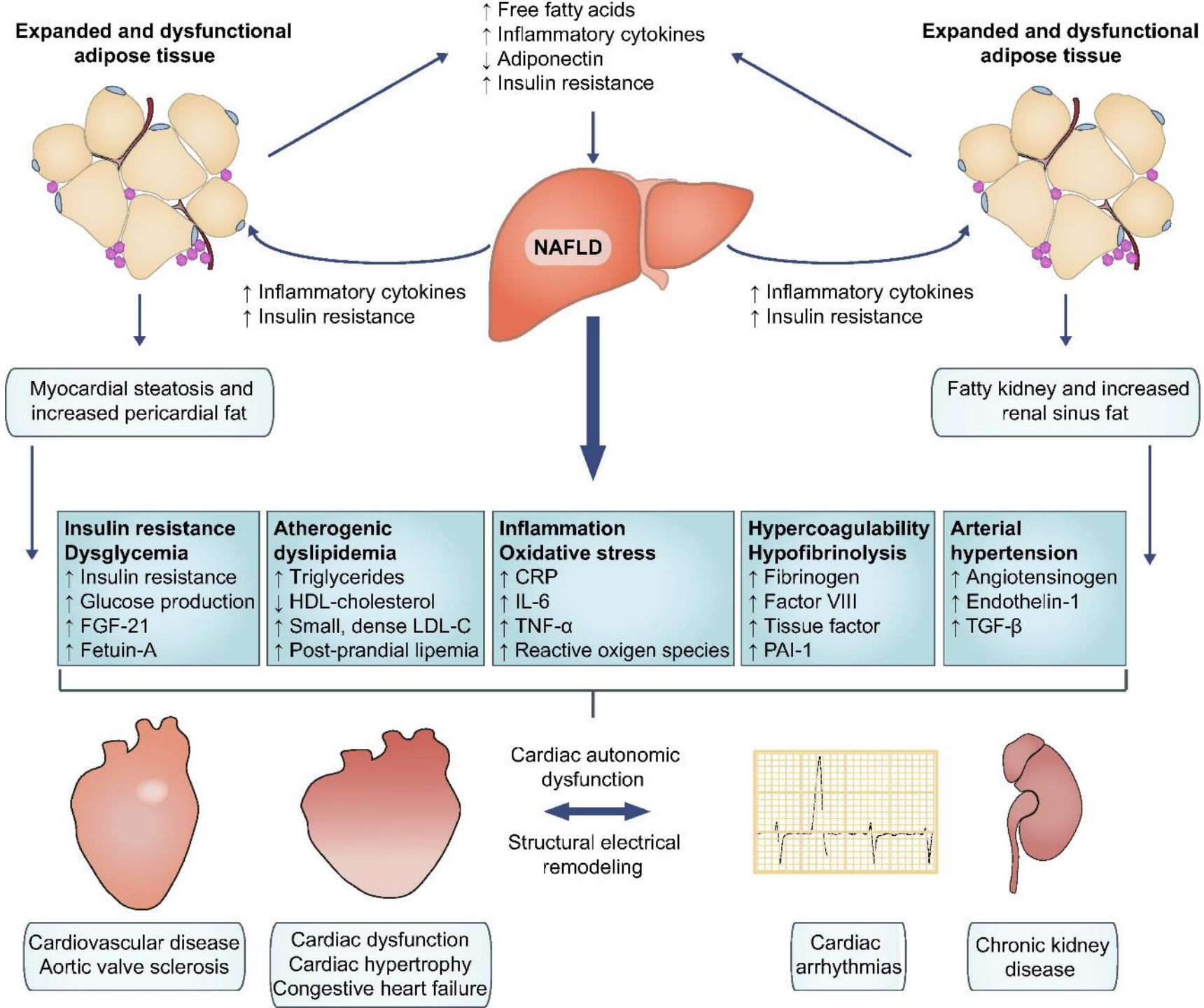


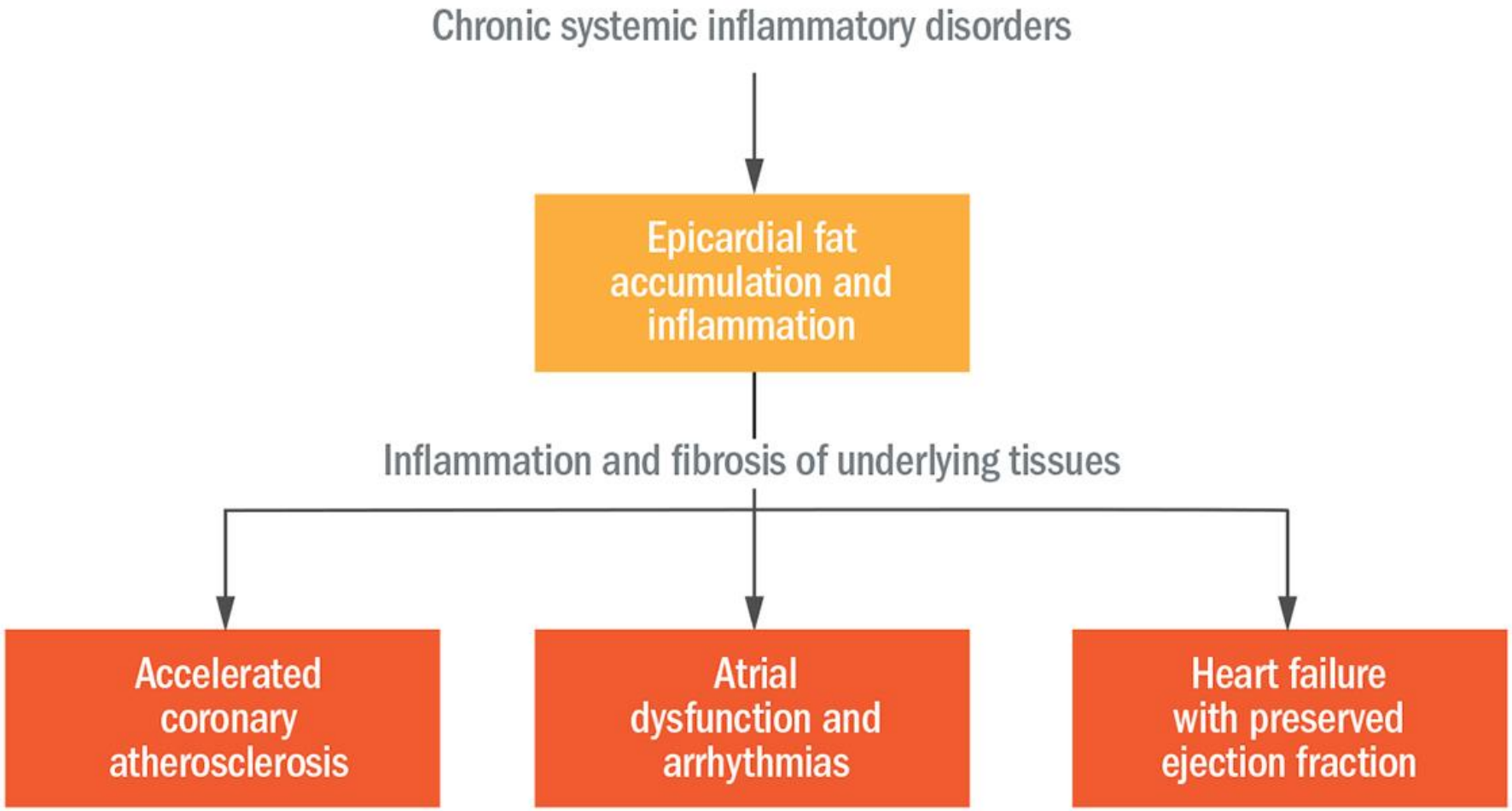
Adapled from Després JP Int J Obes Metab Disord 2003; 27: 5224

Obesity, Inflammation and myocardial signaling



Circulation. 2016 July 5; 134(1): 73–90.

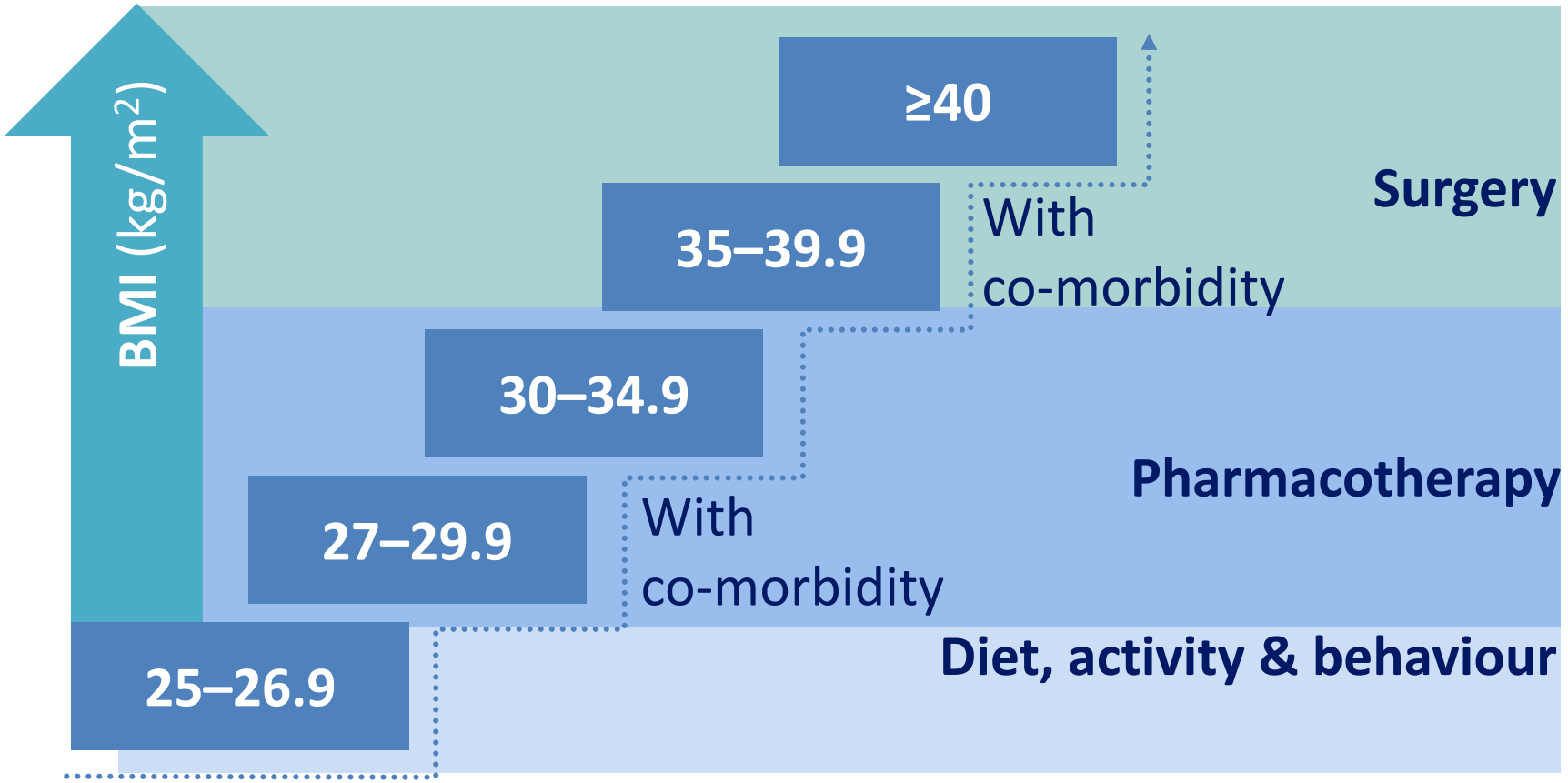

Expanded and dysfunctional adipose tissue
↑ Free fatty acids
↑ Inflammatory cytokines
↓ Adiponectin
↑ Insulin resistance
Expanded and dysfunctional adipose tissue
NAFLD
↑ Inflammatory cytokines
↑ Insulin resistance
↑ Inflammatory cytokines
↑ Insulin resistance
Myocardial steatosis and increased pericardial fat
Fatty kidney and increased renal sinus fat
Insulin resistance
Dysglycemia
↑ Insulin resistance
↑ Glucose production
↑ FGF-21
↑ Fetuin-A
Atherogenic dyslipidemia
↑ Triglycerides
↓ HDL-cholesterol
↑ Small, dense LDL-C
↑ Post-prandial lipemia
Inflammation
Oxidative stress
↑ CRP
↑ IL-6
↑ TNF-α
↑ Reactive oxigen species
Hypercoagulability
Hypofibrinolysis
↑ Fibrinogen
↑ Factor VIII
↑ Tissue factor
↑ PAI-1
Arterial hypertension
↑ Angiotensinogen
↑ Endothelin-1
↑ TGF-β
Cardiac autonomic dysfunction
Structural electrical remodeling
Cardiovascular disease
Aortic valve sclerosis
Cardiac dysfunction
Cardiac hypertrophy
Congestive heart failure
Cardiac arrhythmias
Chronic kidney disease



Inflammation of epicardial adipose tissue may cause dysfunction and inflammation in the underlying tissues, leading to atherosclerosis in coronary vessels as well as microcirculatory rarefaction and fibrosis in the underlying atria and ventricles, leading to atrial tachyarrhythmias and heart failure with preserved ejection fraction. Reprinted with permission from *J Am Coll Cardiol* 2018;71:2360-72.

Treatment

Obesity management



LIFESTYLE THERAPY

Evidence-based lifestyle therapy for treatment of obesity should include 3 components

MEAL PLAN	PHYSICAL ACTIVITY	BEHAVIOR
• Reduced-calorie healthy meal plan • ~500–750 kcal daily deficit • Individualize based on personal and cultural preferences • Meal plans can include: Mediterranean, DASH, low-carb, low-fat, volumetric, high protein, vegetarian • Meal replacements • Very low-calorie diet is an option in selected patients and requires medical supervision Team member or expertise: dietitian, health educator	• Voluntary aerobic physical activity progressing to >150 minutes/week performed on 3–5 separate days per week • Resistance exercise: single-set repetitions involving major muscle groups, 2–3 times per week • Reduce sedentary behavior • Individualize program based on preferences and take into account physical limitations Team member or expertise: exercise trainer, physical activity coach, physical/occupational therapist	An interventional package that includes any number of the following: • Self-monitoring (food intake, exercise, weight) • Goal setting • Education (face-to-face meetings, group sessions, remote technologies) • Problem-solving strategies • Stimulus control • Behavioral contracting • Stress reduction • Psychological evaluation, counseling, and treatment when needed • Cognitive restructuring • Motivational interviewing • Mobilization of social support structures

AACE/ACE ALGORITHM FOR THE MEDICAL CARE OF PATIENTS WITH OBESITY

WHEN TO INITIATE WEIGHT-LOSS MEDICATIONS IN PATIENTS WITH OVERWEIGHT / OBESITY

1. Failure on Lifestyle Therapy.
Add medication for patients who have progressive weight gain or who have not achieved clinical improvement in weight-related complications on lifestyle therapy alone.

2. Weight Regain on Lifestyle Therapy.
Add medication for patients with overweight (BMI 27–29.9 kg/m^2) or obesity who are experiencing weight regain following initial success on lifestyle therapy alone.

3. Presence of Weight-Related Complications.
Initiate medication concurrent with lifestyle therapy for patients with overweight (BMI 27–29.9 kg/m^2) or obesity who have weight-related complications, particularly if severe, in order to achieve sufficient weight loss to ameliorate the complication (tertiary prevention).

AACE/ACE ALGORITHM FOR THE MEDICAL CARE OF PATIENTS WITH OBESITY

2019 ESC Guidelines on diabetes, pre-diabetes, and cardiovascular diseases

Developed in collaboration with EASD

ESC European Society of Cardiology

Task Force Members:

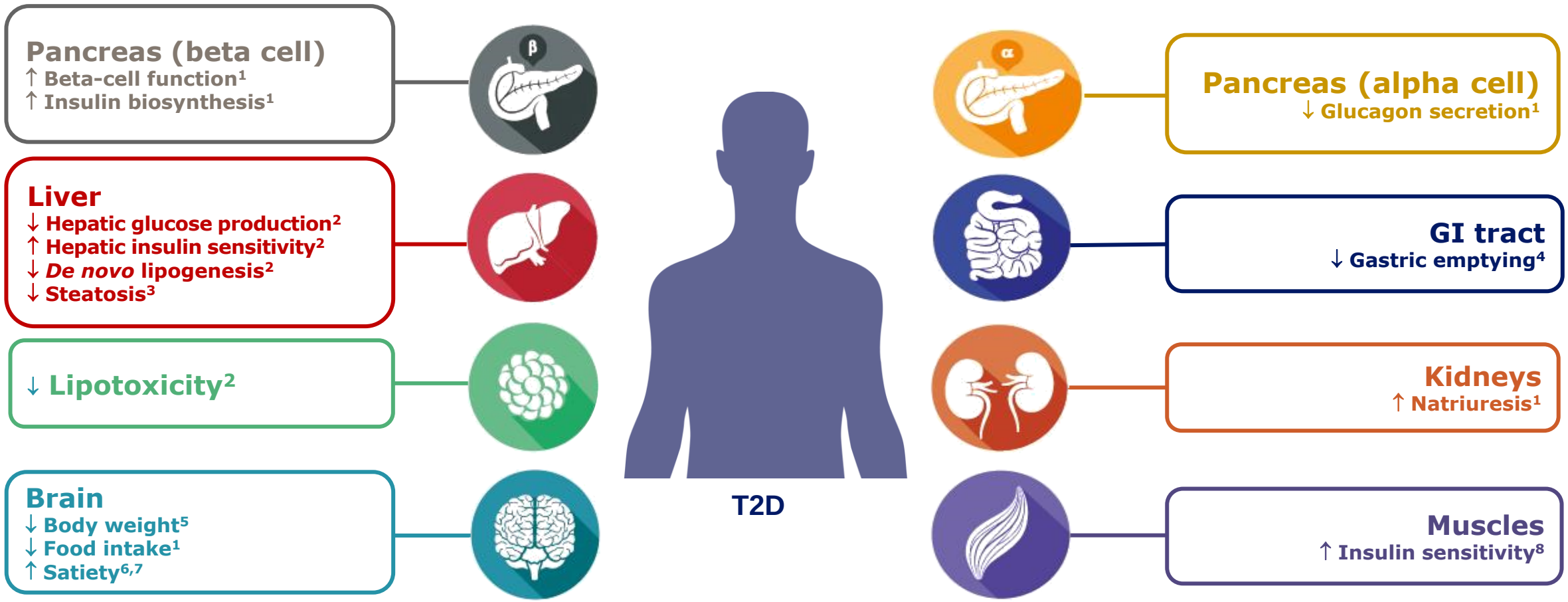
Francesco Cosentino (ESC Chairperson) (Sweden), Peter J. Grant (EASD Chairperson) (UK)

Victor Aboyans (France), Clifford J. Bailey[1] (United Kingdom), Antonio Ceriello[1] (Italy), Victoria Delgado (Netherlands), Massimo Federici[1] (Italy), Gerasimos Filippatos (Greece), Diederick E. Grobbee (Netherlands), Tina Birgitte Hansen (Denmark), Heikki V. Huikuri (Finland), Isabelle Johansson (Sweden), Peter Jüni (Canada), Maddalena Lettino (Italy), Nikolaus Marx (Germany), Linda G. Mellbin (Sweden), Carl J. Östgren (Sweden), Bianca Rocca (Italy), Marco Roffi (Switzerland), Naveed Sattar[1] (United Kingdom), Petar M. Seferović (Serbia), Miguel Sousa-Uva (Portugal), Paul Valensi (France), David C. Wheeler [1] (United Kingdom).

[1]*Representing the European Association for the Study of Diabetes (EASD)*

GLP-1RAs have multifactorial effects

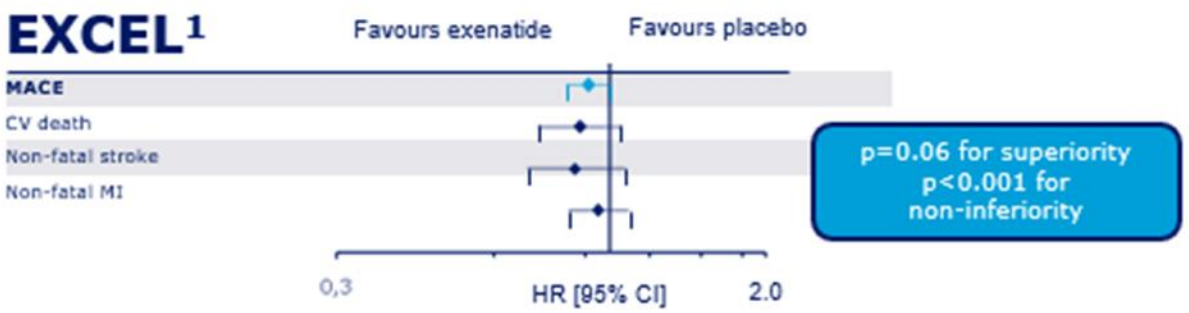
HbA1c reduction, BW reduction, CV outcomes*, Safety

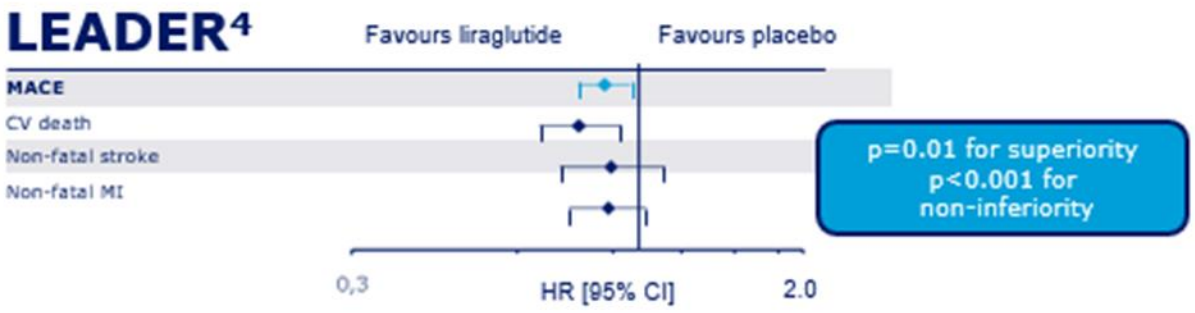


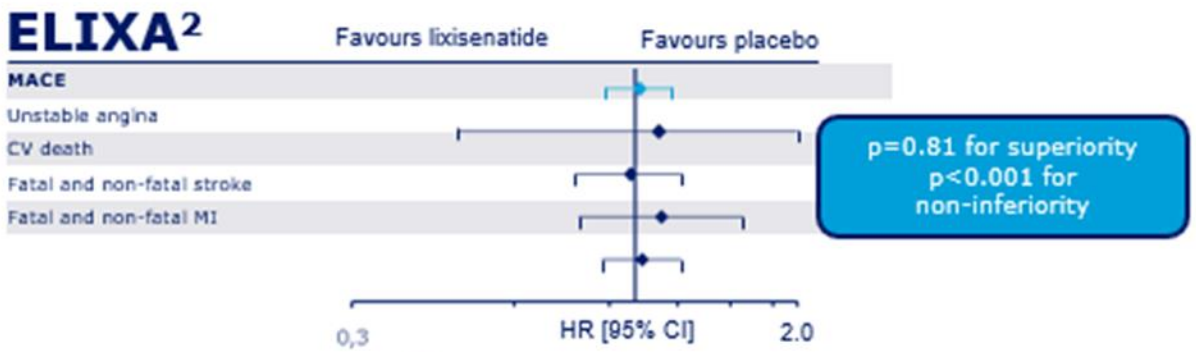
GI, gastrointestinal; GLP-1RA, glucagon-like peptide-1 receptor agonist.

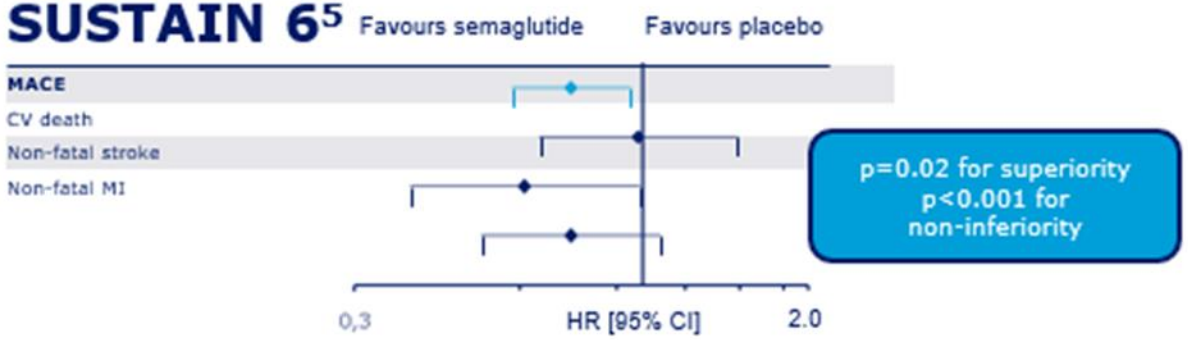
1. Campbell JE, DJ Drucker. *Cell Metab* 2013;17:819–37; 2. Armstrong MJ et al. *J Hepatol* 2016;64:399–408; 3. Armstrong MJ et al. *Lancet* 2016;387:679–90; 4. Tong J, D'Alessio D. *Diabetes* 2014;63:407–9; 5. Baggio LL, Drucker DJ. *J Clin Invest* 2014;124:4223–6; 6. Flint A et al. *J Clin Invest* 1998;101:515–20; 7. Blundell J et al. *Diabetes Obes Metab* 2017;19:1242–51; 8. MacDonald PE et al. *Diabetes* 2002;51(Suppl 3):S434–42.

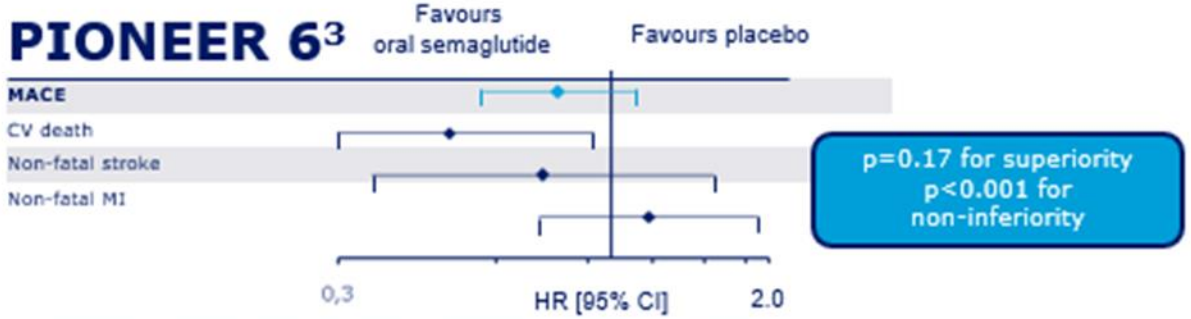
CVOTs with GLP-1RAs have varying results

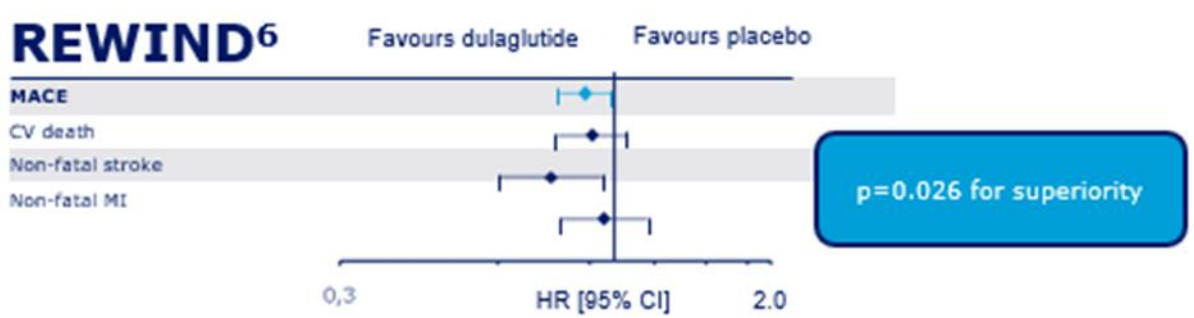












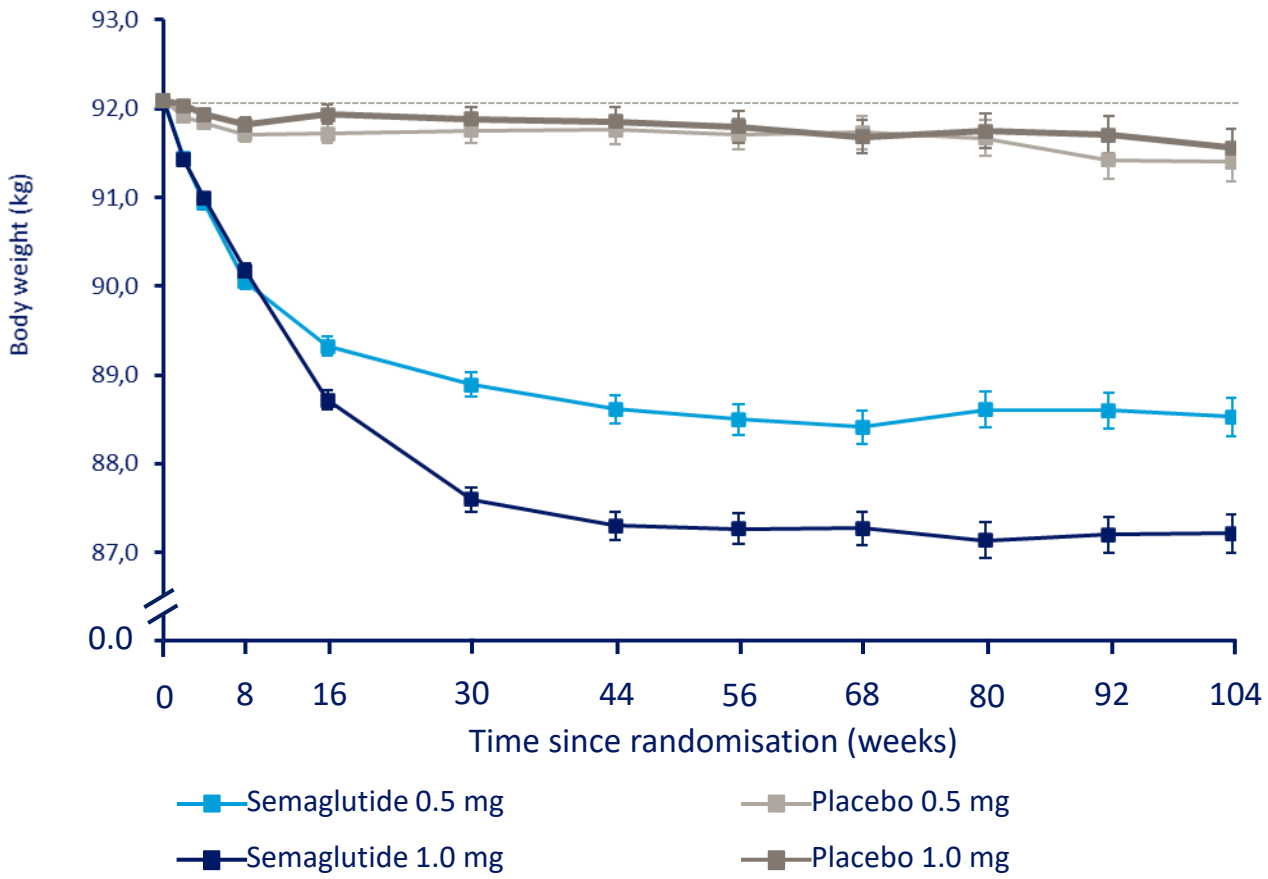
p-values for superiority/non-inferiority are for the primary endpoint.

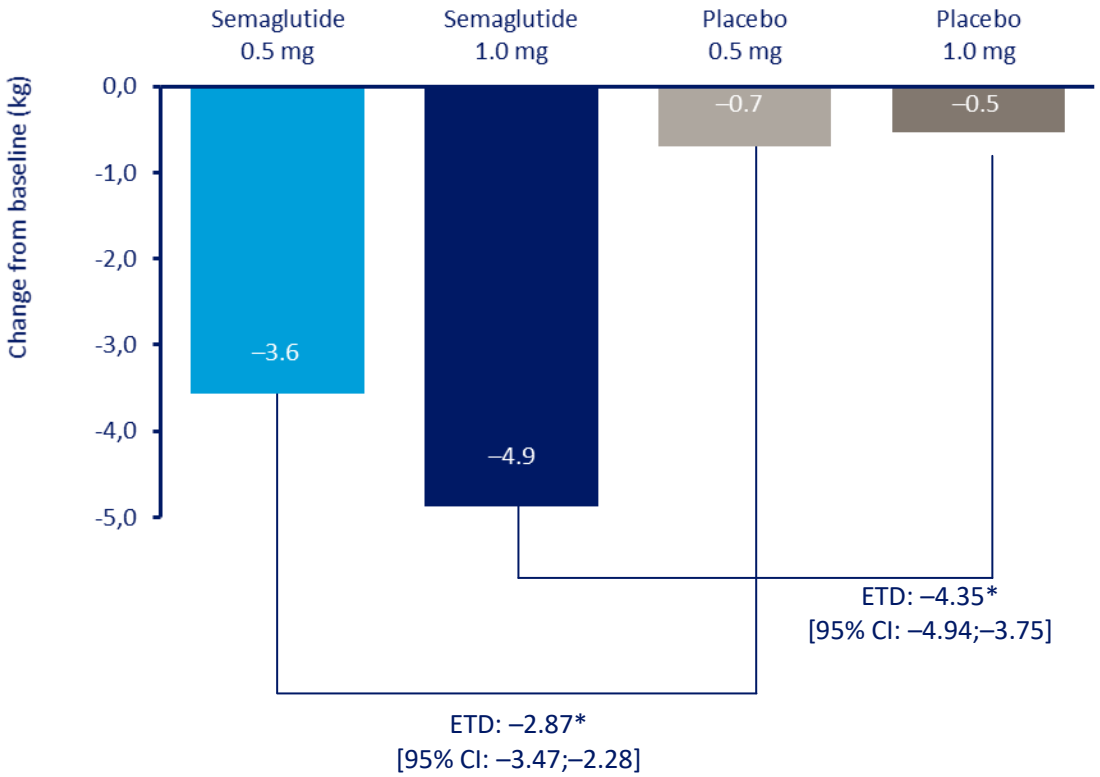
CI, confidence interval; CV, cardiovascular; CVOT, cardiovascular outcomes trial; GLP-1RA, glucagon-like peptide-1 receptor agonist; HR, hazard ratio; MACE, major adverse cardiovascular events; MI, myocardial infarction. 1. Holman RR et al. *N Engl J Med* 2017;377:1228–39; 2. Pfeffer MA et al. *N Engl J Med* 2015;373:2247–57; 3. Husain M et al. *N Engl J Med* 2019;381:841–51; 4. Marso SP et al. *N Engl J Med* 2016;375:311–22; 5. Marso SP et al. *N Engl J Med* 2016;375:1834–44; 6. Gerstein HC et al. *Lancet* 2019;394:121–30.

Change in body weight

SUSTAIN 6

Overall mean at baseline: 92.1 kg





*Data are estimated mean ± standard error of the mean based on in-trial data for scheduled visits from the full analysis set, analysed by a mixed model for repeated measures with treatment group (semaglutide 0.5 and 1.0 mg and corresponding placebo doses) and stratification (9 levels) as fixed factors and the corresponding baseline value as a covariate, all nested within visit. *p<0.0001.*

CI, confidence interval; ETD, estimated treatment difference.

Marso SP et al. N Engl J Med 2016;375:1834–44.

Long-term outcomes

SUSTAIN 6

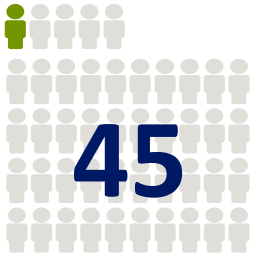
Primary outcome of CV death, non-fatal MI or non-fatal stroke

26%

risk reduction

vs placebo

Number needed to treat to prevent one CV death, non-fatal MI or non-fatal stroke



for 2 years

Semaglutide significantly reduced

HbA_{1c} and body weight

vs placebo

Risk of new or worsening nephropathy

36%

risk reduction

vs placebo

Risk of diabetic retinopathy complications

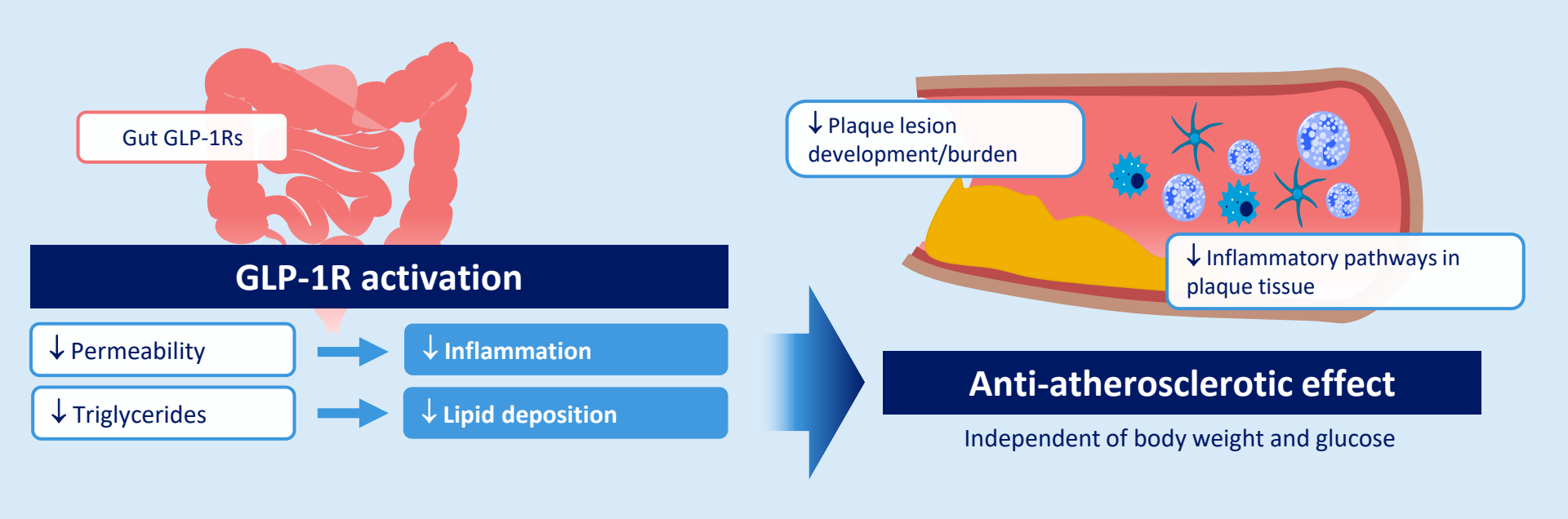
76%

increased risk

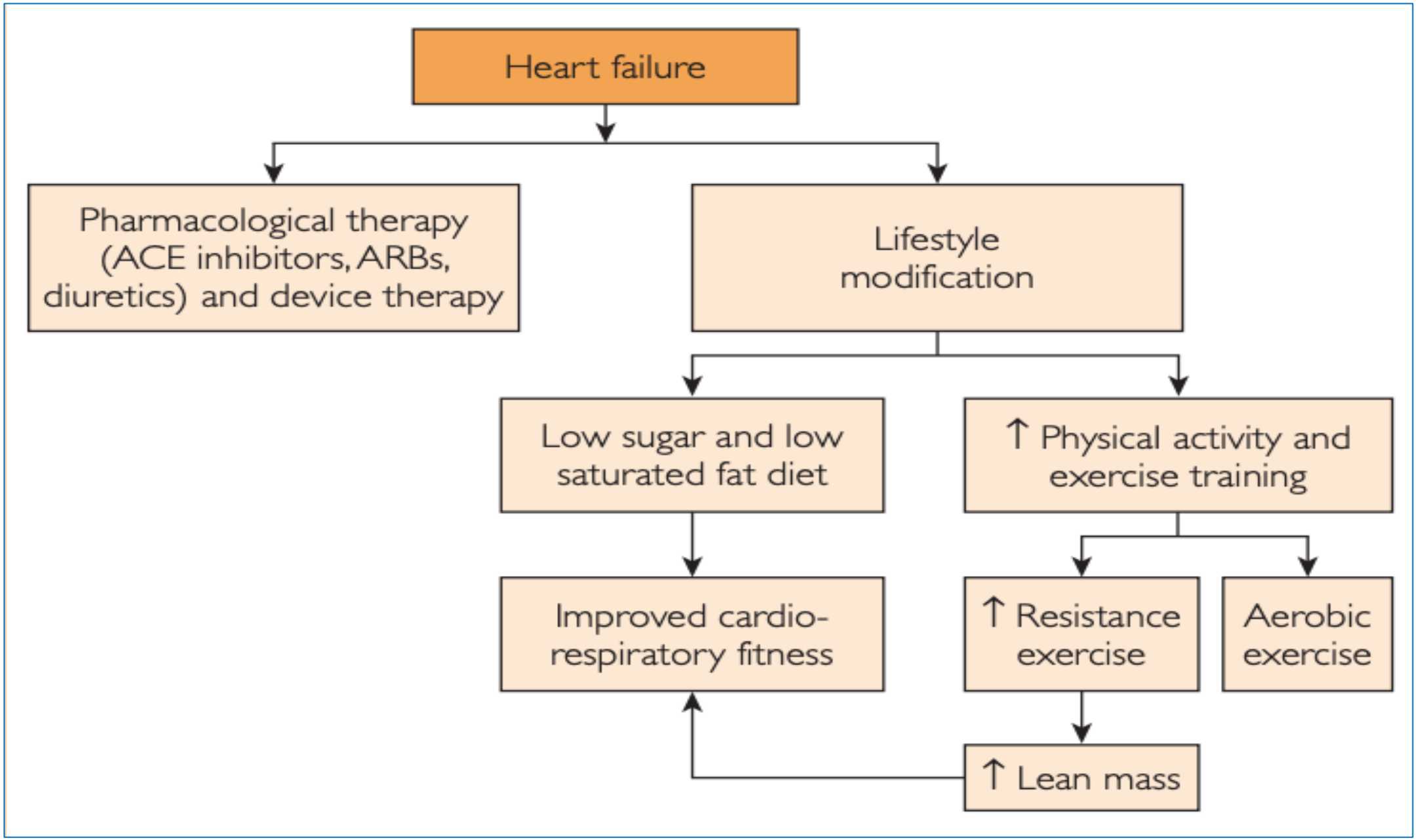
vs placebo

CV, cardiovascular; MI, myocardial infarction.
Marso SP et al. N Engl J Med 2016;375:1834–44.

Potential mechanism for long-acting GLP-1RAs to reduce atherosclerotic burden

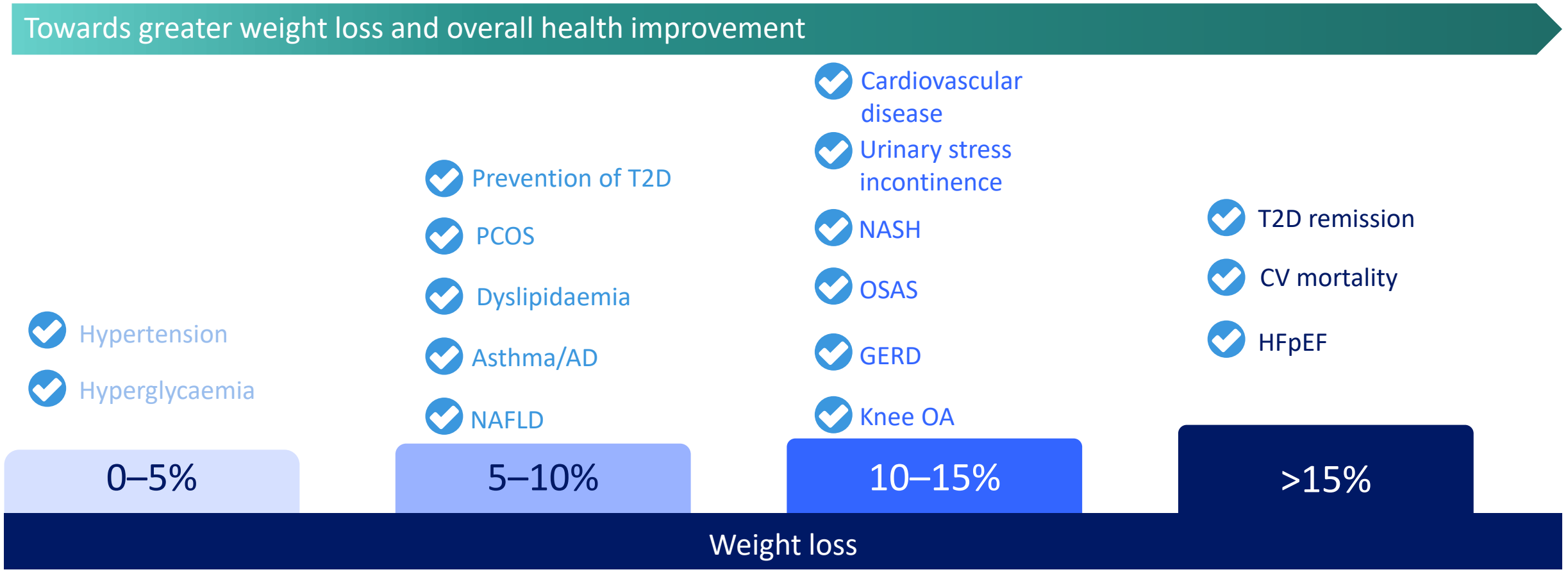


GLP-1R, glucagon-like peptide-1 receptor; GLP-1RA, glucagon-like peptide-1 receptor agonist.
Rakipovski G et al. JACC Basic Transl Sci 2018;3:844–57; Knudsen LB and Lau J. Front Endocrinol 2019;10:155.



Heart Failure Clin (2018)

Greater weight loss leads to improved health outcomes

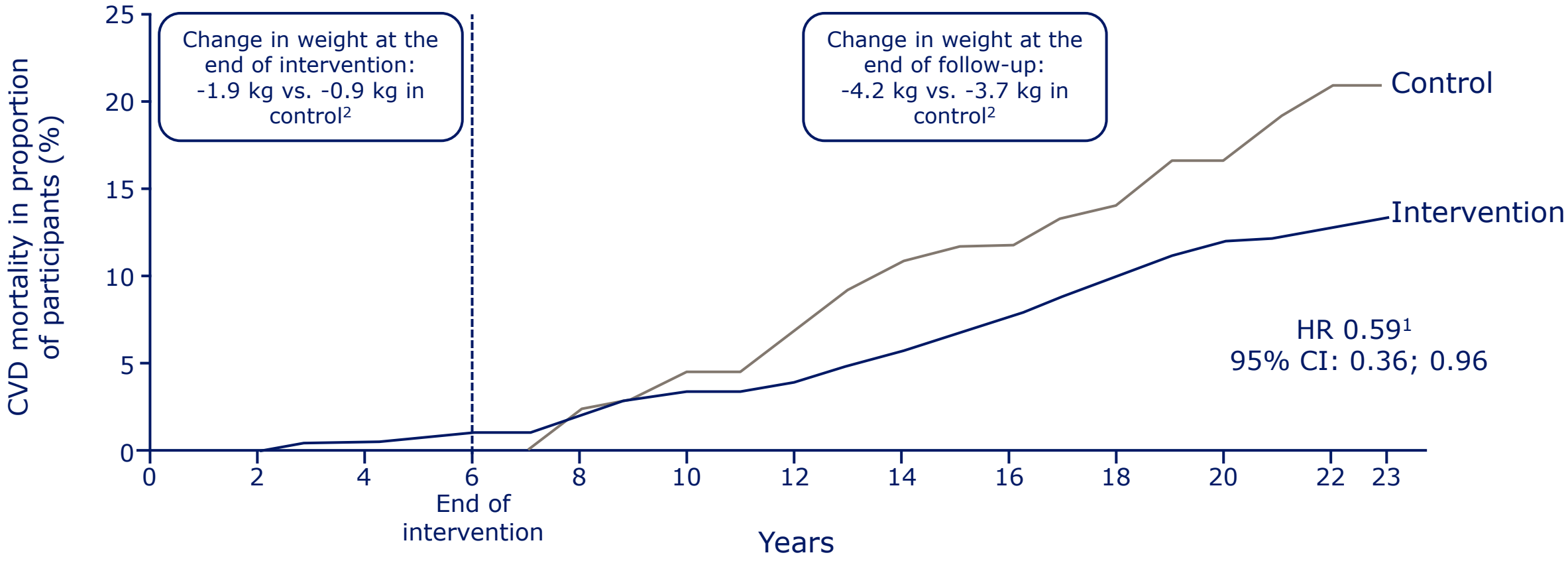


AD, airway disease; CV, cardiovascular; GERD, gastresophageal reflux disease; HFpEF, heart failure with preserved ejection fraction; NAFLD, non-alcoholic fatty liver disease; NASH, non-alcoholic steatohepatitis; OSAS, obstructive sleep apnoea syndrome; PCOS, polycystic ovary syndrome; TG, triglycerides.

Garvey WT et al. Endocr Pract 2016;22(Suppl. 3):1–203; Look AHEAD Research Group. Lancet Diabetes Endocrinol 2016;4:913–921; Lean ME et al. Lancet 2018;391:541–551; Benraoune F and Litwin SE. Curr Opin Cardiol 2011;26:555–561; Sundström J et al. Circulation 2017;135:1577–1585.

Weight loss reduces the risk of CV mortality

Da Qing Diabetes Prevention Study



1. Li *et al. Lancet* 2008;371:1783–9 2. Li *et al. Lancet Diabetes Endocrinol* 2014;2:474–80

Benefits of early intervention

Treating obesity early can **reduce the risk** of developing chronic, expensive-to-treat complications

A patient with no existing complications going from obesity class I to overweight would reduce their risk of developing type 2 diabetes or sleep apnoea by **more than 50%**

Complication	Risk of developing complication vs BMI 18.5–24.9	
	Obesity I BMI 30–34.9	Overweight BMI 25–29.9
Type 2 diabetes	5.17	2.41
Sleep apnoea	5.11	2.22
Osteoarthritis	1.68	1.35
Heart failure	1.57	1.12
Hypertension	1.99	1.44
Dyslipidaemia	1.83	1.42

Haase CL et al. Obes Sci Pract. 2020:1–11.

Conclusions

Conclusions

- Obesity is a global pandemic associated with decrease in life expectancy and severe cardiovascular complications
- Major CV complications of obesity: HFpEF, CAD, hypertension
- Main mechanisms in cardiovascular complications: inflammation, oxidative stress, insulin resistance, dyslipidemia
- Weight loss leads to improved CV outcomes
- GLP-1RA significantly reduced body weight and improved CV outcomes

ΕΘΝΙΚΟ ΚΑΙ ΚΑΠΟΔΙΣΤΡΙΑΚΟ ΠΑΝΕΠΙΣΤΗΜΙΟ ΑΘΗΝΩΝ
ΙΑΤΡΙΚΗ ΣΧΟΛΗ

ΕΙΔΙΚΟ ΙΑΤΡΕΙΟ ΚΑΡΔΙΑΣ – ΔΙΑΒΗΤΗ - ΠΑΧΥΣΑΡΚΙΑΣ

ΕΛΛΗΝΙΚΗ ΚΑΡΔΙΟΛΟΓΙΚΗ ΕΤΑΙΡΕΙΑ

HELLENIC SOCIETY OF CARDIOLOGY

ΟΜΑΔΑ ΕΡΓΑΣΙΑΣ

«ΚΑΡΔΙΑ & ΔΙΑΒΗΤΗΣ»