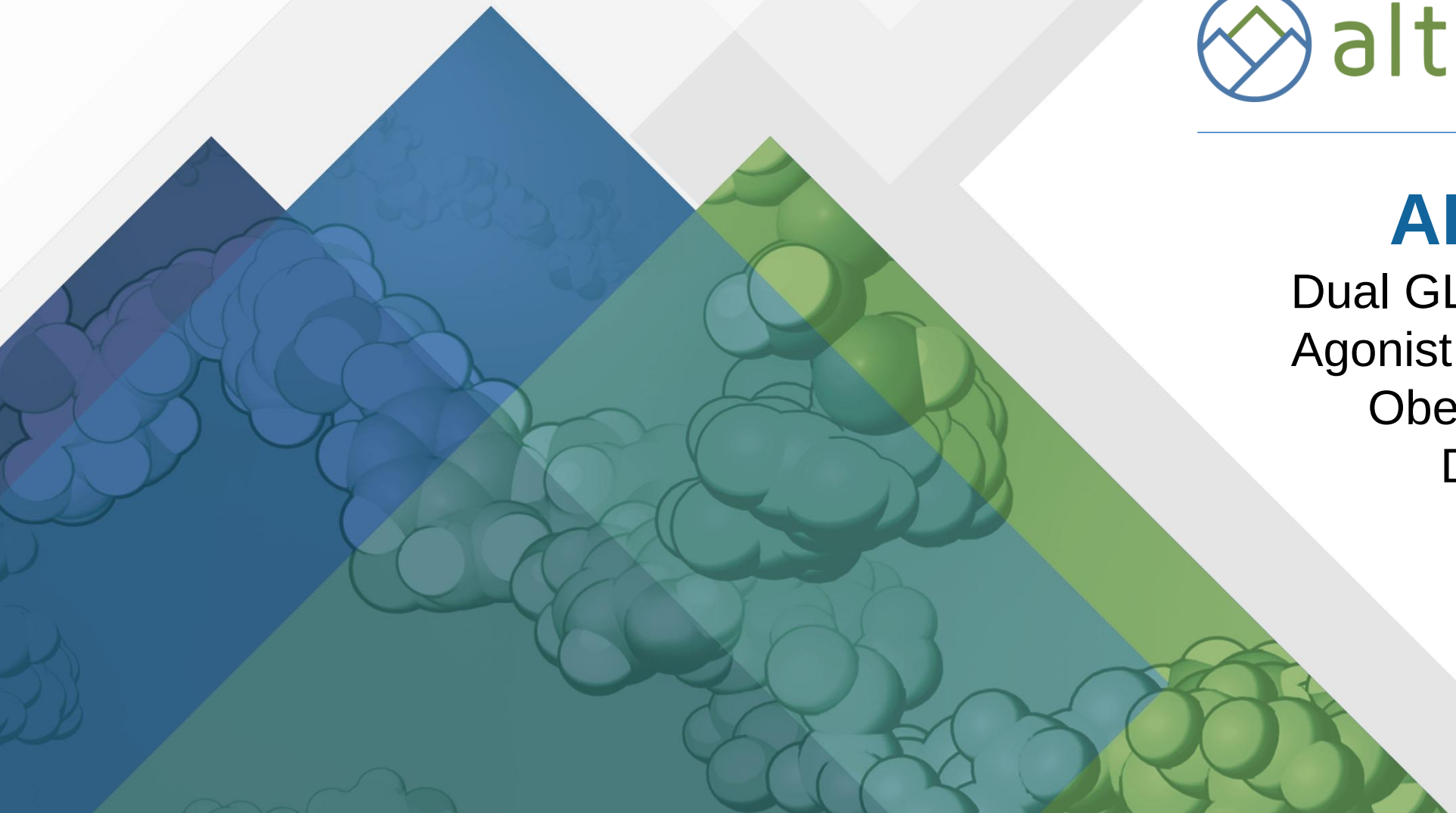

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ALT-801

Dual GLP-1/Glucagon Agonist for NASH and Obesity-Driven Disease

FORWARD-LOOKING STATEMENTS

Safe-Harbor Statement

Any statements made in this presentation relating to future financial or business performance, conditions, plans, prospects, trends, or strategies and other financial and business matters, including without limitation, the closing of the Spitfire Pharma acquisition, the timing of key milestones for ALT-801, the filing of the IND for ALT-801 in 2020, the initiation of a Phase 1 clinical study in 2020, cash on hand to fund the development of ALT-801, and the prospects for regulatory approval or commercializing ALT-801, are forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. In addition, when or if used in this press release, the words “may,” “could,” “should,” “anticipate,” “believe,” “estimate,” “expect,” “intend,” “plan,” “predict” and similar expressions and their variants, as they relate to Altimmune, Inc. (the “Company”) may identify forward-looking statements. The Company cautions that these forward-looking statements are subject to numerous assumptions, risks, and uncertainties, which change over time. Important factors that may cause actual results to differ materially from the results discussed in the forward looking statements or historical experience include risks and uncertainties, including risks relating to: the Company’s ability to close the Spitfire Pharma acquisition on the timelines anticipated, or at all, the reliability of the results of the studies relating to human safety and possible adverse effects resulting from the administration of ALT-801; the Company may encounter substantial delays in its clinical trials, or its clinical trials may fail to demonstrate the safety and efficacy of our product candidates to the satisfaction of applicable regulatory authorities; the Company’s ability to predict the time and cost of product development; competition from other pharmaceutical and biotechnology companies, which may result in others discovering, developing or commercializing NASH products before, or more successfully, than the Company the Company’s ability to obtain potential regulatory approvals on the timelines anticipated, or at all; the Company’s ability to obtain additional patents or extend existing patents on the timelines anticipated, or at all; the Company’s ability to expand its pipeline of products and the success of future product advancements, including the success of future clinical trials, and the Company’s ability to commercialize its products; third-party claims of intellectual property infringement or misappropriation may prevent or delay the Company’s development and commercialization efforts the Company’s anticipated financial or operational results; the Company’s ability to obtain additional capital resources; unforeseen safety and efficacy issues; the Company’s ability to receive stockholder approval to issue shares of its common stock in satisfaction of milestone payments; and the Company’s ability to continue to satisfy the listing requirements of the NASDAQ Global Market. Further information on the factors and risks that could affect the Company's business, financial conditions and results of operations are contained in the Company’s filings with the U.S. Securities and Exchange Commission, including under the heading “Risk Factors” in the Company’s annual reports on Form 10-K and quarterly reports on Form 10-Q filed with the SEC, which are available at www.sec.gov.

NASH
LARGELY A DISEASE OF OBESITY AND ECTOPIC BODY FAT

NAFLD is present in up to **90% of obese patients**

Liver fat mainly represents the breakdown of **peripheral fat**, not *de novo* hepatic synthesis

40% of NASH patients develop NAFLD recurrence one year after liver transplant—i.e., the underlying disease is still present

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NASH

7-10% BODY WEIGHT LOSS REVERSES NASH PROGRESSION[1]

The **treatment of obesity** remains the cornerstone of NASH and NAFLD therapy

Meaningful weight loss is rarely achieved without medical intervention

Drugs have failed to deliver the weight loss achieved by bariatric surgery

[1] *Pomrat et al Hepatology 2010; Glass et al Dig Dis Sci 2015; Vilar-Gomez et al Gastroenterology 2015; Marchesini et al Hepatology 2016; Koutowkidis et al JAMA Intern Med 2019*

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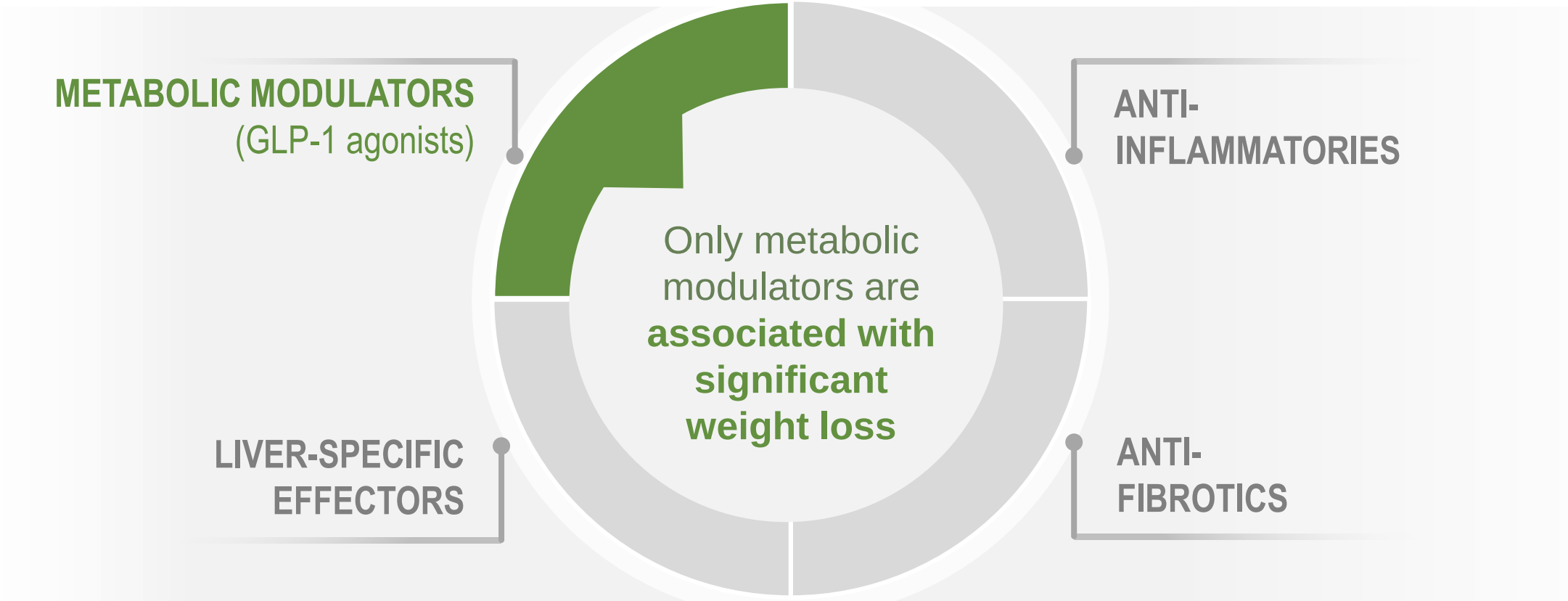
NASH

DUAL AGONISTS SIGNIFICANTLY IMPROVE UPON GLP-1 AGONIST-INDUCED WEIGHT LOSS



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OPTIMIZED FOR NASH AND WEIGHT LOSS

GLP-1

blood glucose

appetite

inflammation

Indirect effects on liver

GLUCAGON

energy expenditure

adipose browning

lipolysis/ gluconeogenesis

mobilization of liver fat

Direct effects on liver

SUBSTANTIAL REDUCTIONS IN

body weight

liver fat, inflammation and resulting fibrosis

blood glucose

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ALT-801
ACTS AT AN EARLY STAGE TO REVERSE NASH PROGRESSION

[1]https://aasldpubs.onlinelibrary.wiley.com/doi/pdf/10.1002/hep.28392; Marchesini et al Hepatology, Vol. 63, No.6, 2016

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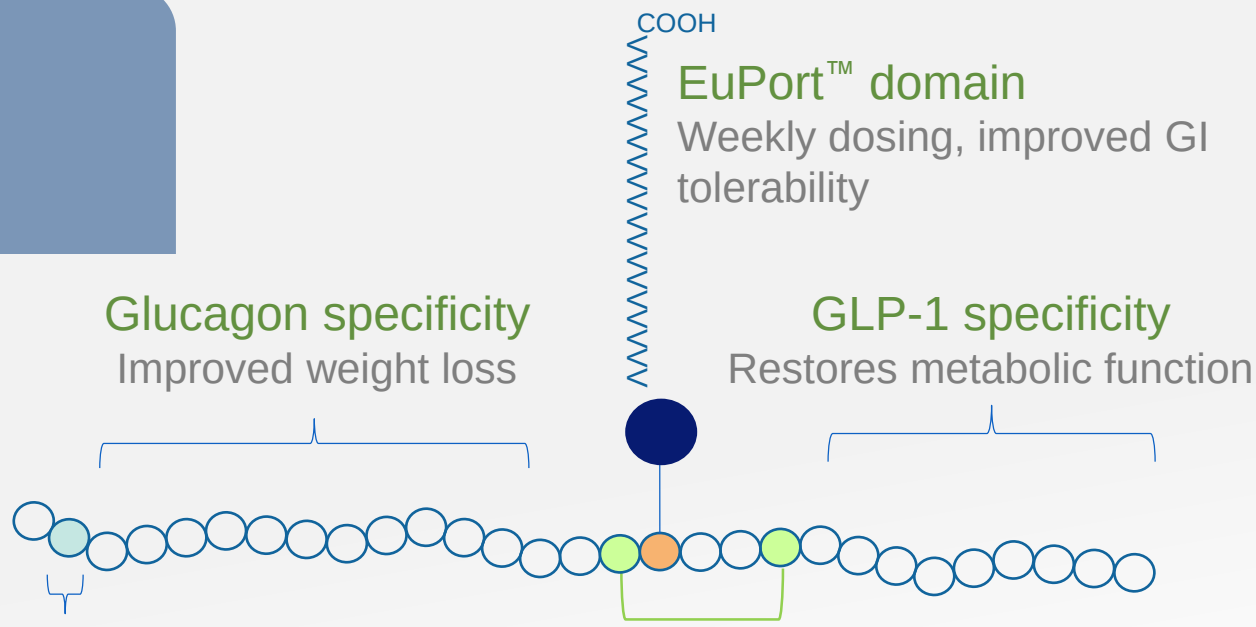
ALT-801

STRUCTURE IS KEY TO DIFFERENTIATION

Proprietary EuPort™ domain provides improved PK

Balanced GLP-1:Glucagon Agonism



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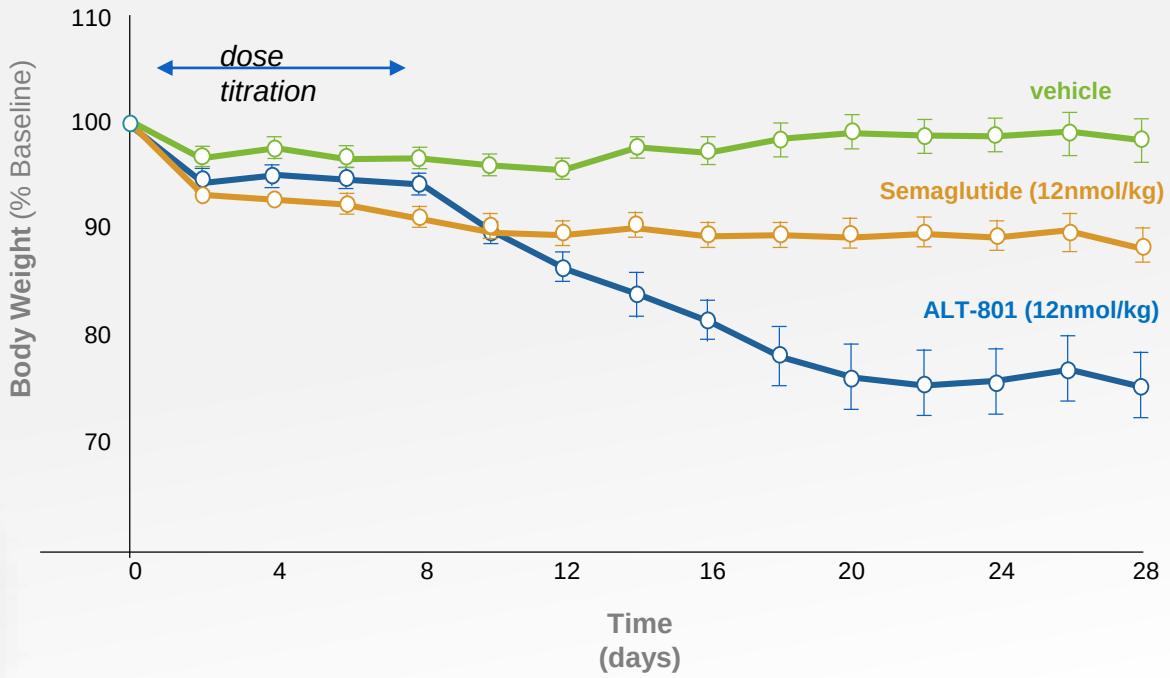
ALT-801
25% WEIGHT LOSS OVER ONE MONTH

Mouse DIO Model After 4 Weeks of Treatment



More than **2x** the weight loss of **semaglutide**

Body weight decreased to **lean normal**

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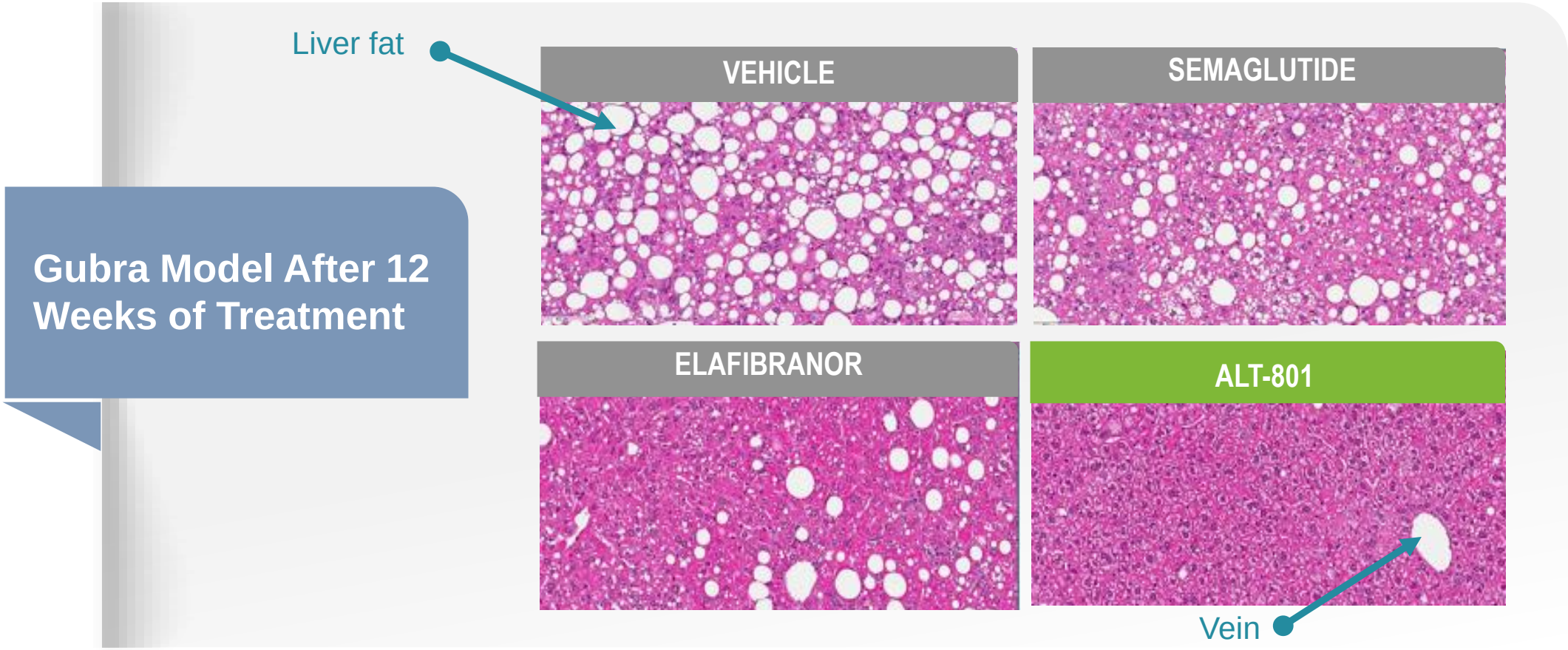
ALT-801
REDUCTION IN LIVER FAT TO LEAN NORMAL



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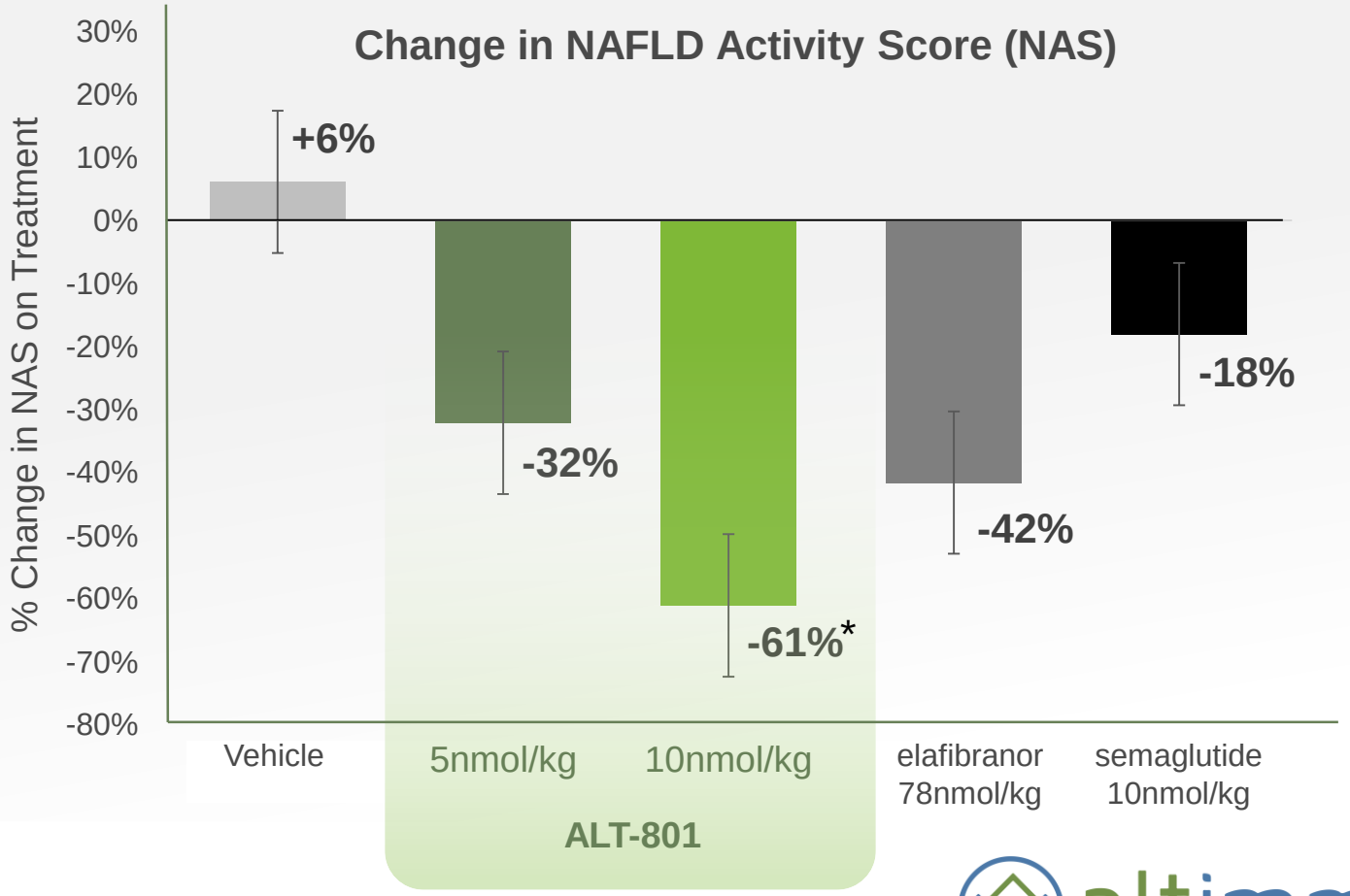
ALT-801
GREATER REDUCTION IN FAT-DRIVEN LIVER INFLAMMATION

Gubra Model After 12 Weeks of Treatment



*All animals receiving ALT-801 10nmol/kg achieved NAS ≤ 3

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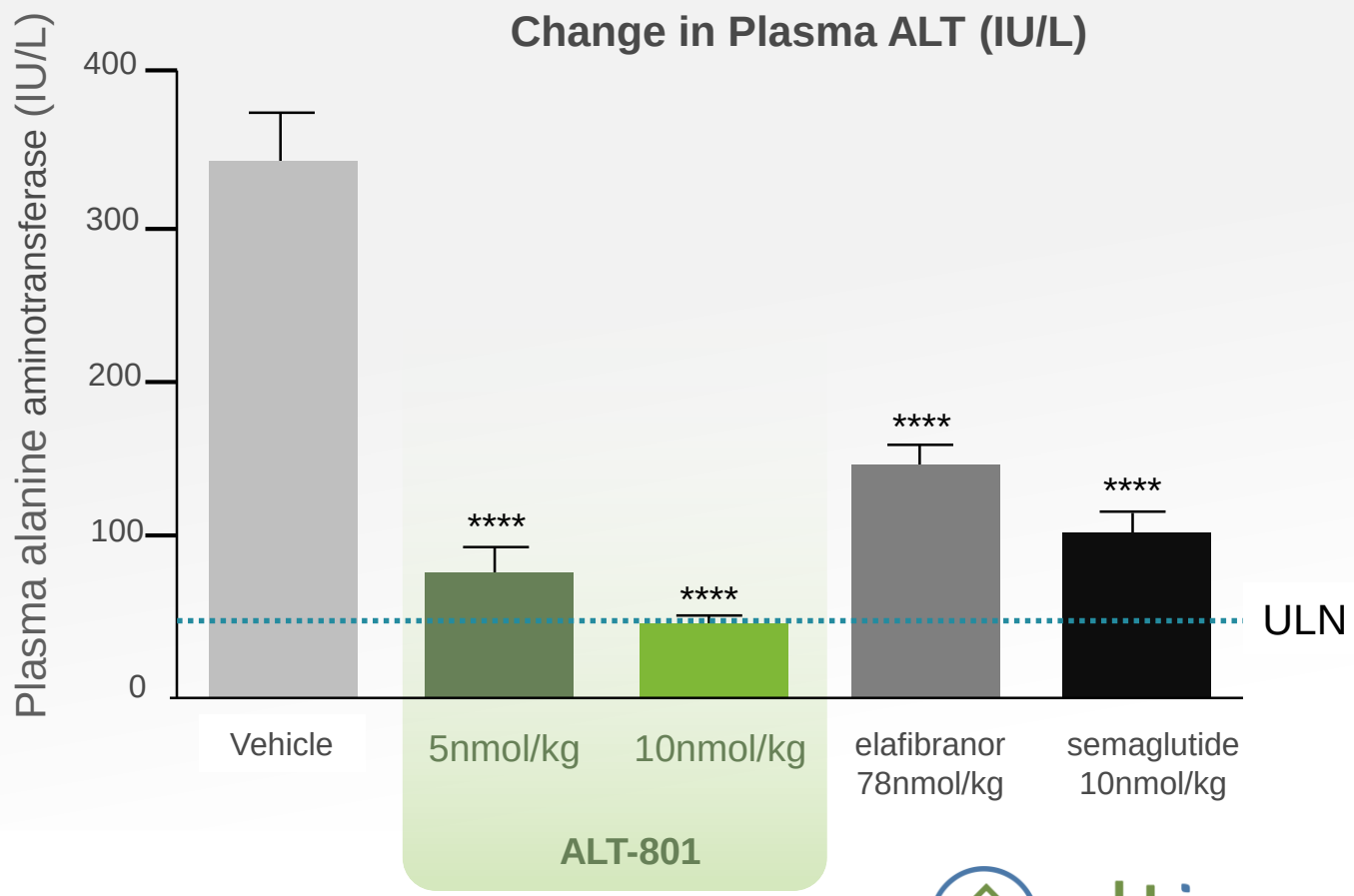
ALT-801
NORMALIZATION OF PLASMA ALT

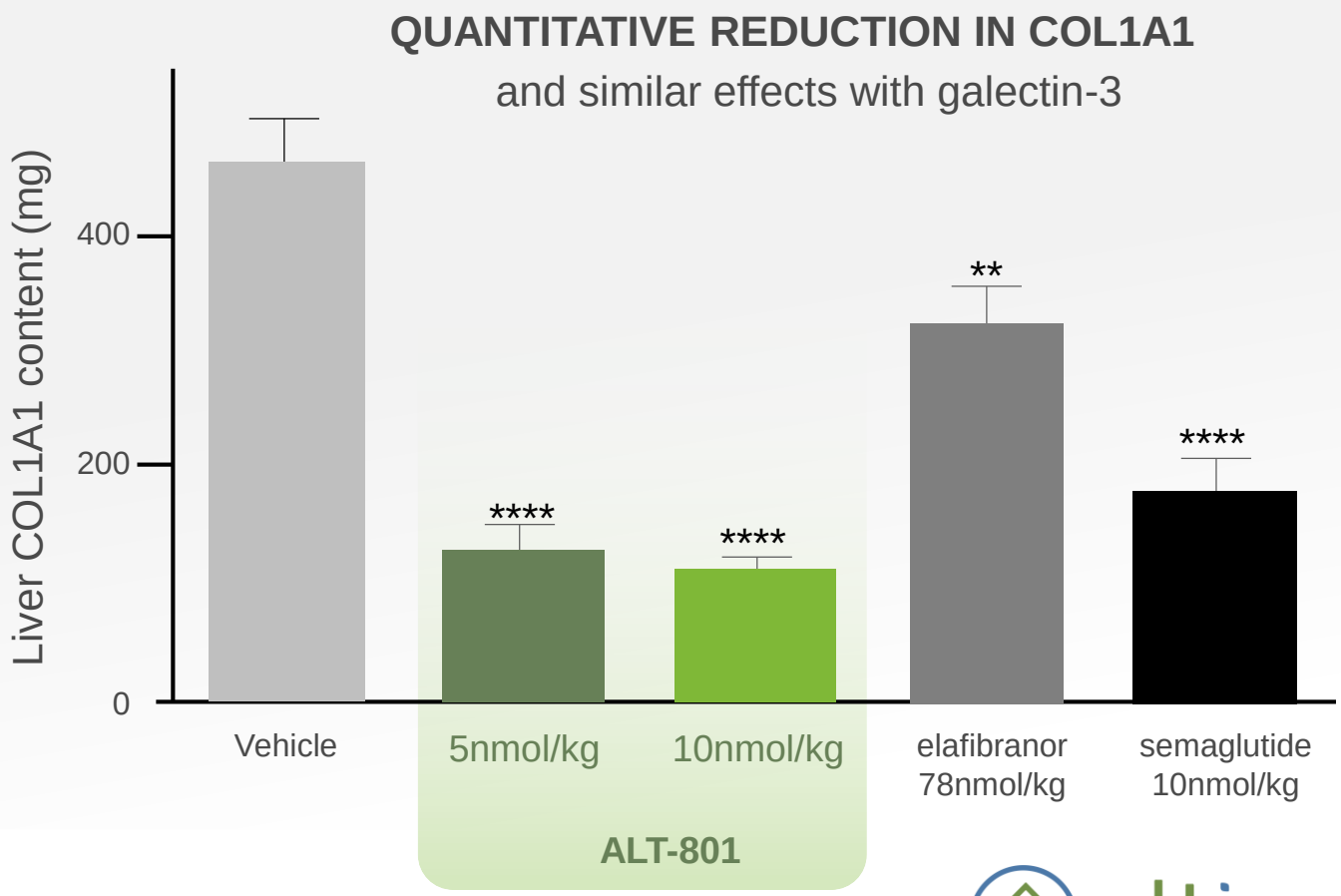
Gubra Model After 12 Weeks of Treatment



**** p < .0001 vs. vehicle; ULN: upper limit of normal

ALT-801
GREATER EFFECTS ON FIBROSIS

Gubra Model After 12 Weeks of Treatment



** p < .0001, **** p < .0001 vs. vehicle

altimmune

DIFFERENTIATED

Balanced and potent dual GLP-1 and glucagon agonist

Superior therapeutic activity in accepted preclinical models

Novel peptide stabilization mechanisms

Molecular classes with **known safety profiles**

Weekly dosing

ALT-801

GLP-1/Glucagon Dual Agonist for NASH

DEVELOPMENT PLAN

File **IND in 2H** 2020

Phase 1 study with mechanistic readout on liver fat and body weight in 1H 2021

Prosecute **6 global supporting patent families**

Evaluate aligned disease indications including obesity and type 2 diabetes

Overview of NASH and the Potential Role of ALT-801

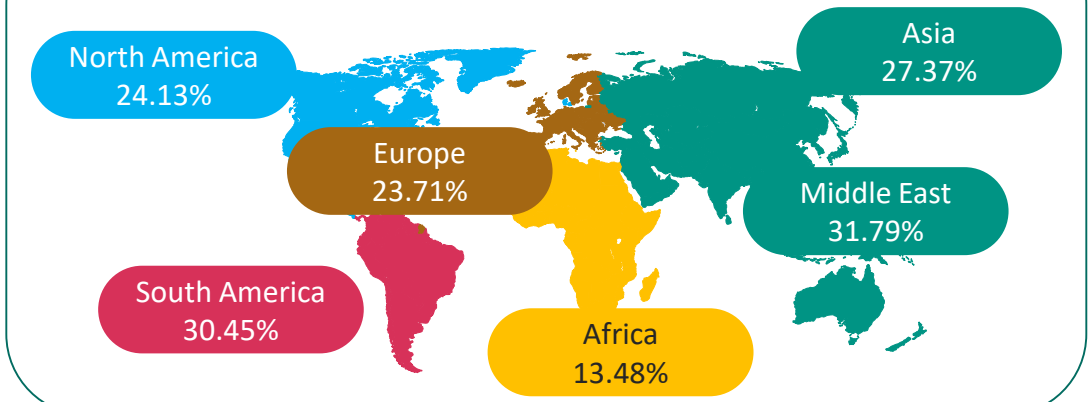
Stephen A. Harrison, MD, FACP, FAASLD
COL (ret.), USA, MC
Visiting Professor of Hepatology
Radcliffe Department of Medicine, University of Oxford

Prevalence of NAFLD around the world…

…is increasing in line with **obesity**, **type 2 diabetes** and **age**

…is about **25%** in the general population

…varies across the **globe**

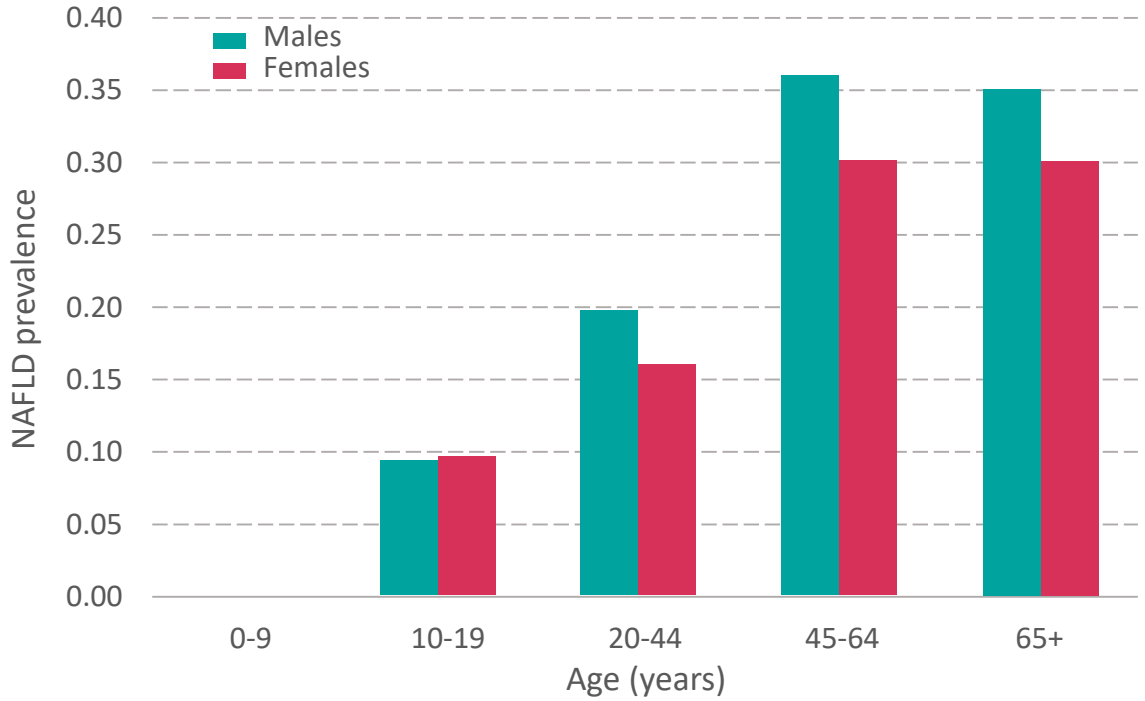


…varies across **ethnicities**

(Hispanics > non-Hispanic white > African Americans)

is higher in **urban** than rural population

NAFLD prevalence by age and sex (US, Southwest China and Spain)

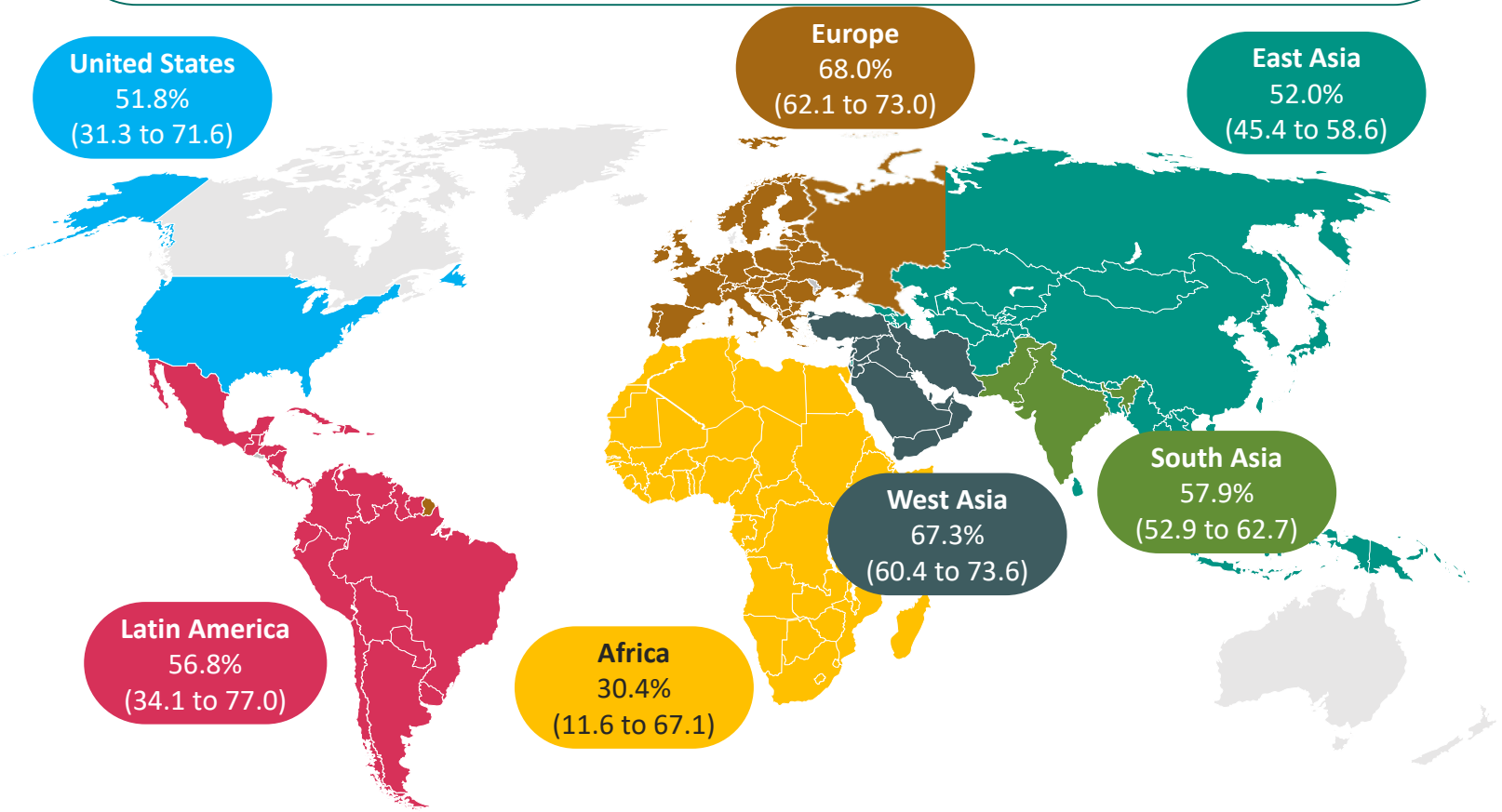


Note: *Other studies demonstrated a higher incidence of NAFLD in females than males*

Younossi Z, et al, Hepatology 2019;69(6):2672-2682; Younossi ZM et al, J Hepatol 2019;70(3):531-544.

Prevalence of NAFLD among patients with T2D

Global prevalence of NAFLD among patients with T2D:
55.5% (47.3 to 63.7)



Prevalence of **NAFLD** in patients with **T2D** is higher than in the **general population** **>2X**

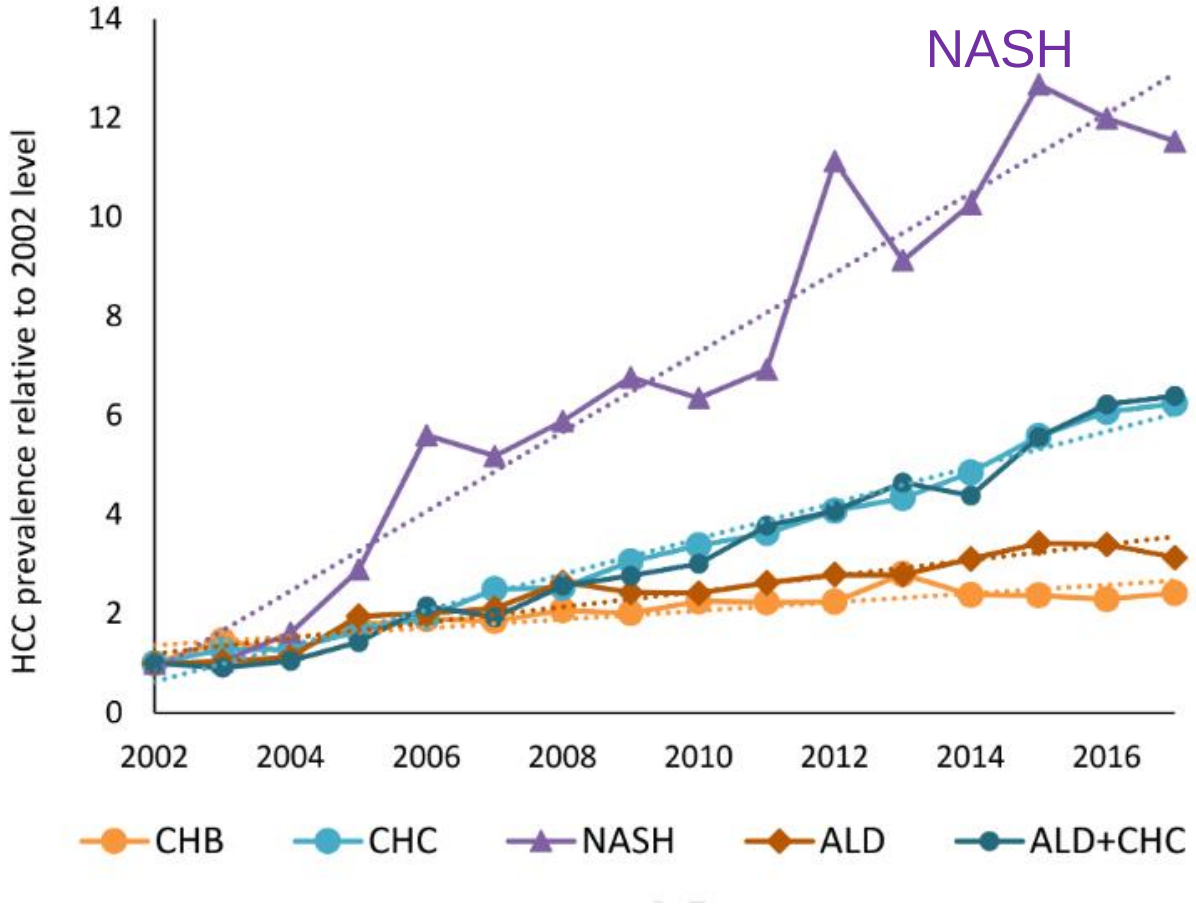
55.5% of patients with **T2D** globally have **NAFLD**

37.3% of patients with **T2D** globally have **NASH**

17% of patients with **T2D** and **NAFLD** who undergo liver biopsy, have **advanced fibrosis**

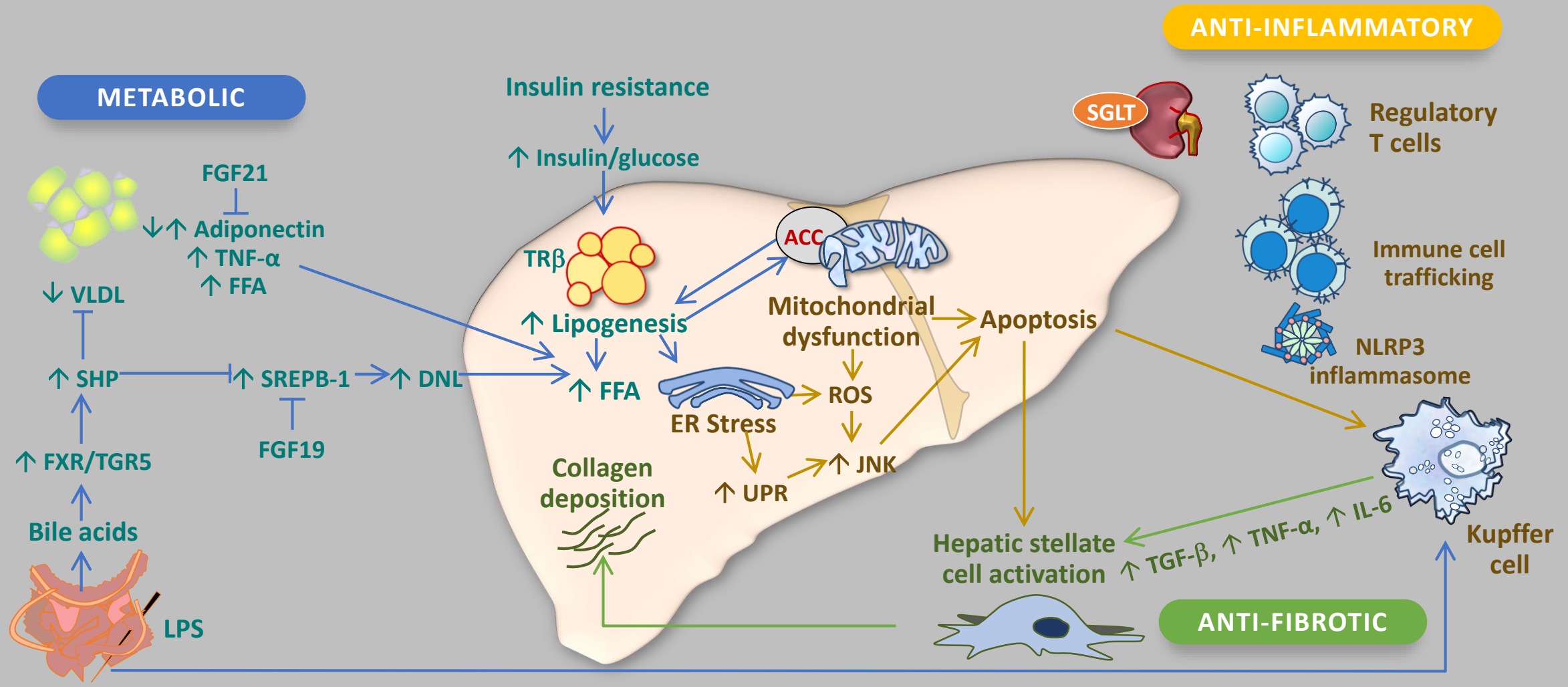
NAFLD diagnosed by ultrasound or H-MRS. Data displayed as prevalence (95% CI). NAFLD, non-alcoholic fatty disease; NASH, non-alcoholic steatohepatitis; T2D, type 2 diabetes.
Younossi ZM, et al. J Hepatol. 2019; 71(4):793-801. doi: 10.1016/j.jhep.2019.06.021. Epub 2019 Jul 4.

NASH is the fastest growing cause of HCC among liver diseases



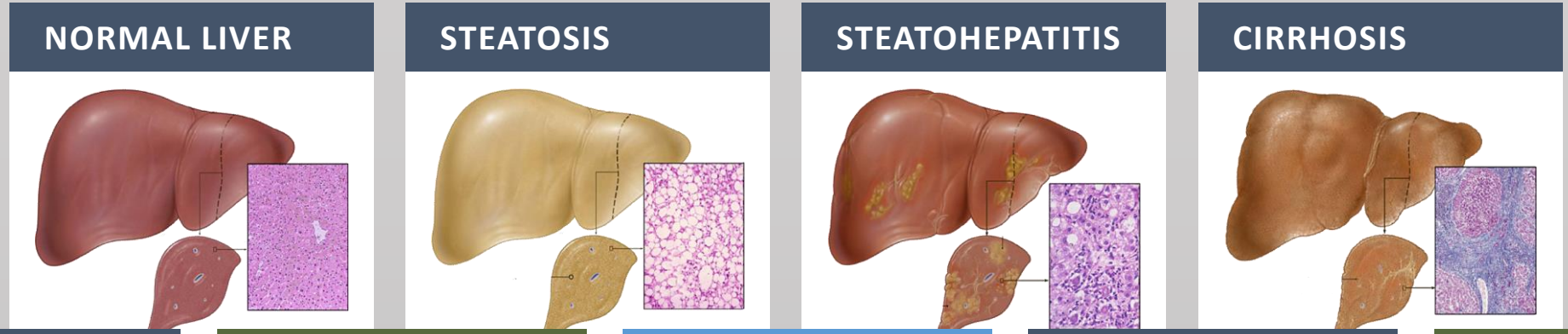
Younossi et al, *J Clin Gastro Hep*, 2018

NASH: Potential therapeutic targets



ACC, acetyl-CoA carboxylase; AOC, amine oxidase, copper containing; ASK, apoptosis signal-regulating kinase; CCR, CC chemokine receptor; DNL, de novo lipogenesis; ER, endoplasmic reticulum; FFA, free fatty acids; FGF, fibroblast growth factor; FXR, farnesoid X receptor; IL, interleukin; JNK, Jun N-terminal kinases; LPS, lipopolysaccharide; NLRP3, nucleotide-binding oligomerization domain and leucine rich repeat and pyrin domain containing protein 3; PPAR, peroxisome proliferator-activated receptor; ROS, reactive oxygen species; SCD, stearoyl CoA desaturase; SGLT, sodium-glucose linked transporter; SHP, small heterodimer partner; SREBP, sterol regulatory element binding proteins; TGF, transforming growth factor; TGR5, G protein-coupled bile acid receptor 1; TLR, toll like receptor; TNF, tumor necrosis factor; TR, thyroid receptor; UPR, unfolded protein response VLDL, very low density lipoprotein. Adapted from Konerman MA et al. J Hepatol. 2018;68:362–375.

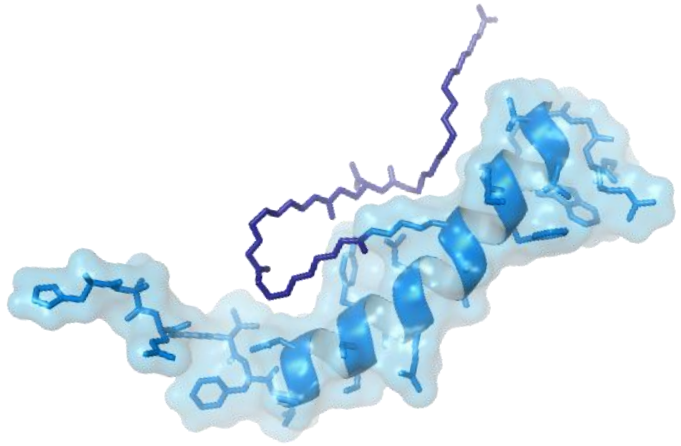
Targeting pathophysiological processes



Targets related to insulin resistance and/or lipid metabolism		Targets related to lipotoxicity & oxidative stress		Targets related to inflammation and immune activation		Targets related to cell death (apoptosis and necrosis)		Targets related to fibrogenesis & collagen turnover	
PPARγ	Pioglitazone	**PPARα/∂**	Elafibranor	**CCR2/5**	Cenicriviroc	**ASK1**	Selonsertib	**LOXL2**	Simtuzumab
GLP-1	Liraglutide, Semaglutide	**PPARα/∂/γ**	Lanifibranor	**AOC3**	BI 1467335	**Caspases**	Emricasan	**Galectin**	GR-MD-02
GLP-1/GR	MEDI0382	**PPARα/γ**	Saroglitazar	**TLR4**	JKB-121				
ACC	GS-0976, PF-05221304	**mTOT**	MSDC-0602K	**Anti-LPS**	IMM-124E				
SCD1	Aramchol	**FXR**	OCA, GS-9674, tropifexor, LMB-763						
SGLT1/2	LIK066	**TGR5**	INT-767, INT-777						
FGF21	BMS-986036, AKR-001	**ASBT:**	Volixibat						
THR-β	MGL-3196, VK2809	**FGF19**	NGM282						
GLP-1/Gluc	ALT-801	**Vitamin E**							
FGFR1/KLB	BFKB8488A								

GLP-1 analogs have multifactorial effects

NASH
- Inflammation[3] ↓
- Body weight[8] ↓
- Lipids[*4,5] ↓
- Glucose[1] ↓



CV disease
- Inflammation[3] ↓
- Lipids[*4,5] ↓
- Systolic blood pressure ↓
- Heart rate[6] ↑

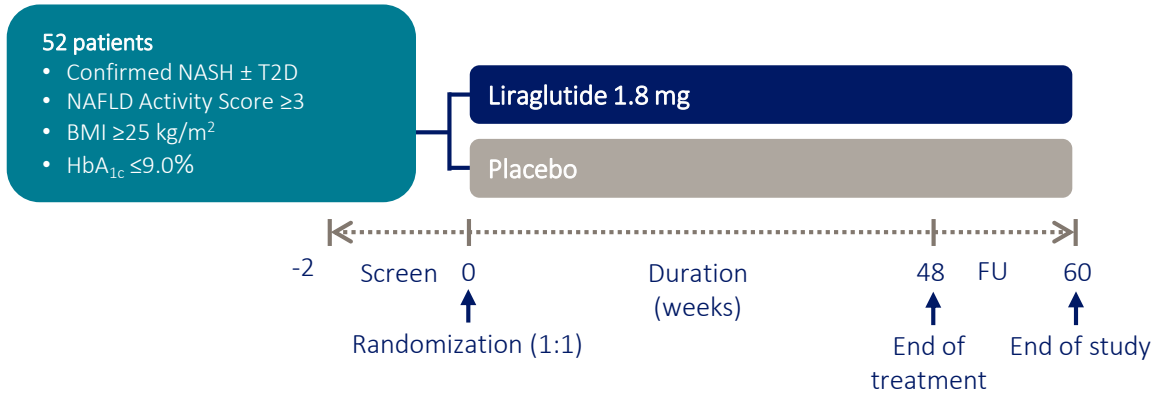
Obesity
- Energy intake[7] ↓
- Appetite[8] ↓
- Body weight[8] ↓

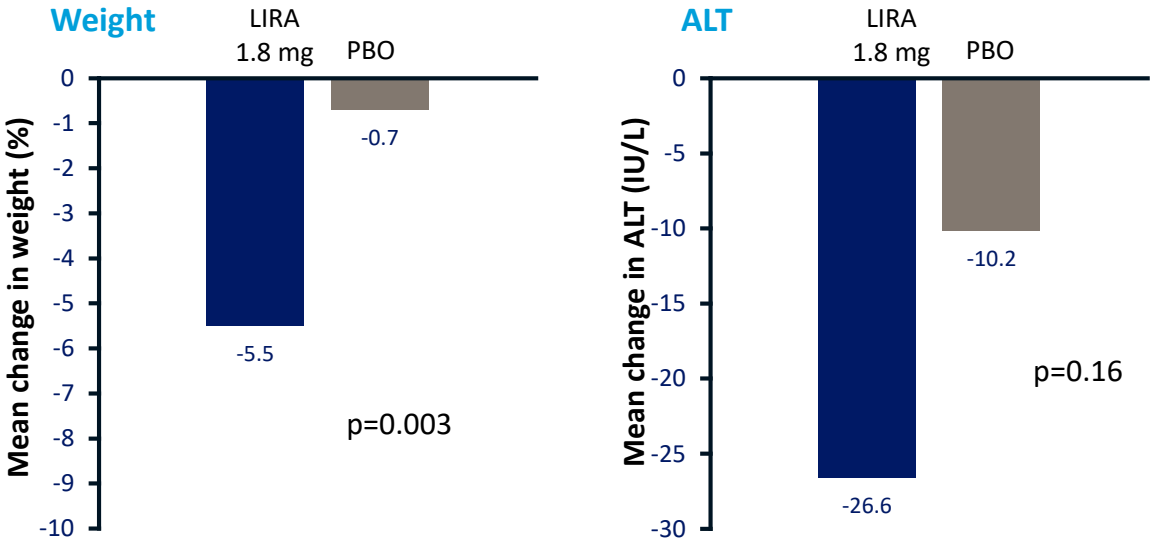
Kidney disease
- Inflammation[3] ↓
- Systolic blood pressure[6]

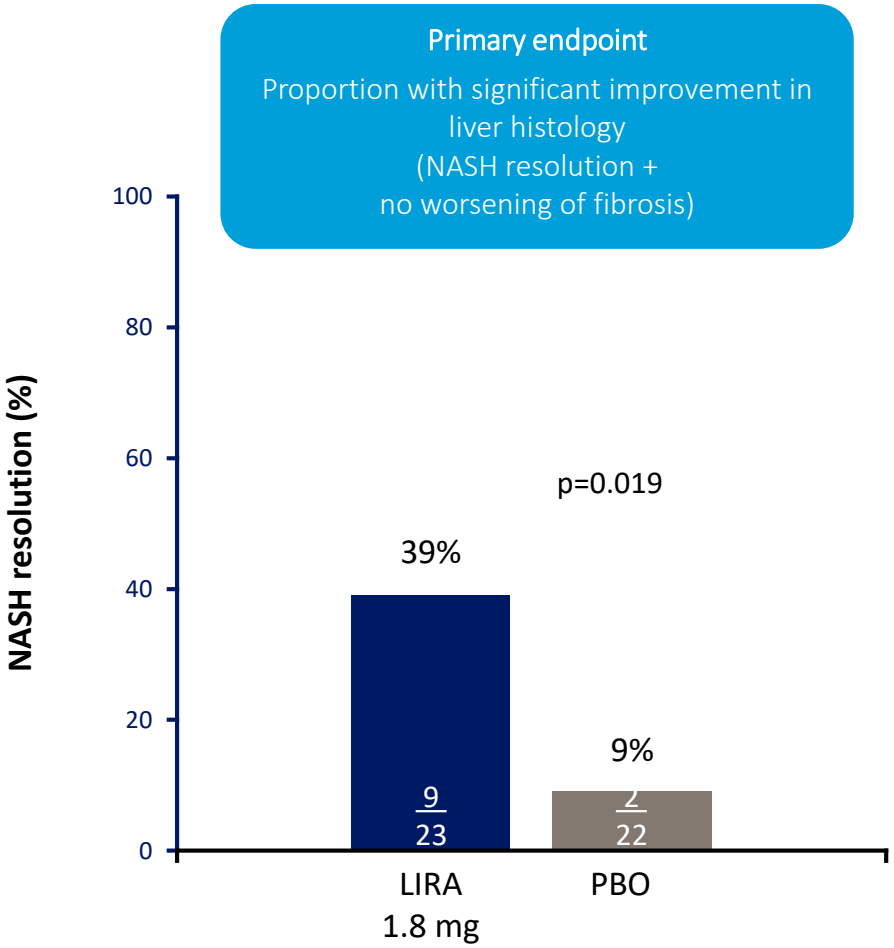
Diabetes
- Insulin[1] ↑
- β-cell function[1] ↑
- Glucagon[1] ↓
- Gastric emptying[2] ↓

[*]Fasting and post-prandial lipids.
CV, cardiovascular; GLP-1, glucagon-like peptide-1; NASH, non-alcoholic steatohepatitis.
1. Campbell JE and Drucker DJ. *Cell Metab* 2013;17:819–37; 2. Tong J and D'Alessio D. *Diabetes* 2014;63:407–9; 3. Hogan AE, *et al. Diabetologia* 2014;57:781–4;
4. Hermansen K, *et al. Diabetes Obes Metab* 2013;15:1040–8; 5. Ahrén B, *et al. Lancet Diabetes Endocrinol* 2017;5:341–54;
6. Ryan D and Acosta A. *Obesity* 2015;23:1119–29; 7. Bagger JI, *et al. J Clin Endocrinol Metab* 2015;100:4541–52; 8. Flint A, *et al. J Clin Invest* 1998;101:515–20.

Liraglutide in NASH trial (LEAN)





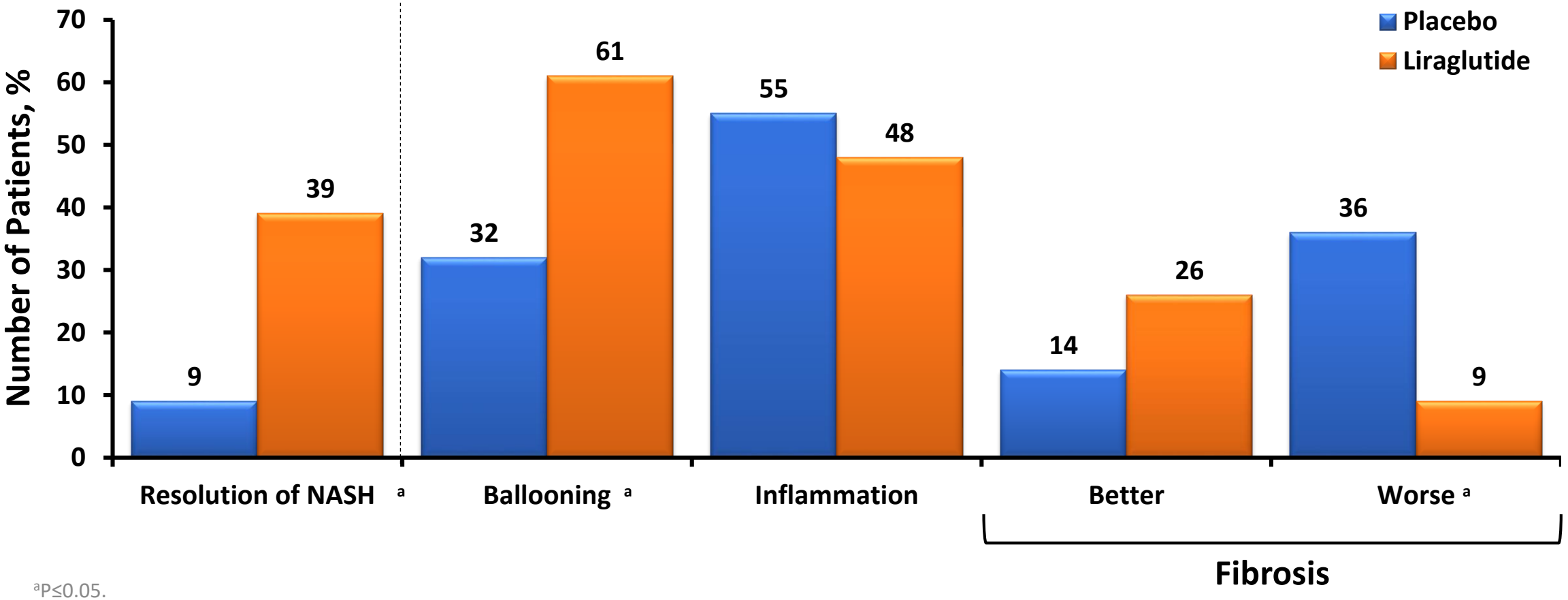


ALT, alanine aminotransferase; BMI, body mass index; FU, follow-up; LIRA, liraglutide; NAFLD, non-alcoholic fatty liver disease; NASH, non-alcoholic steatohepatitis; PBO, placebo; T2D, type 2 diabetes.

Armstrong MJ, *et al. Lancet* 2016;13:679–90; Armstrong MJ, *et al. BMJ Open* 2013;3:e003995.

LEAN with Liraglutide

Histologic Improvement With A GLP-1 RA



[a]P≤0.05.

HbA1c, hemoglobin A1c.

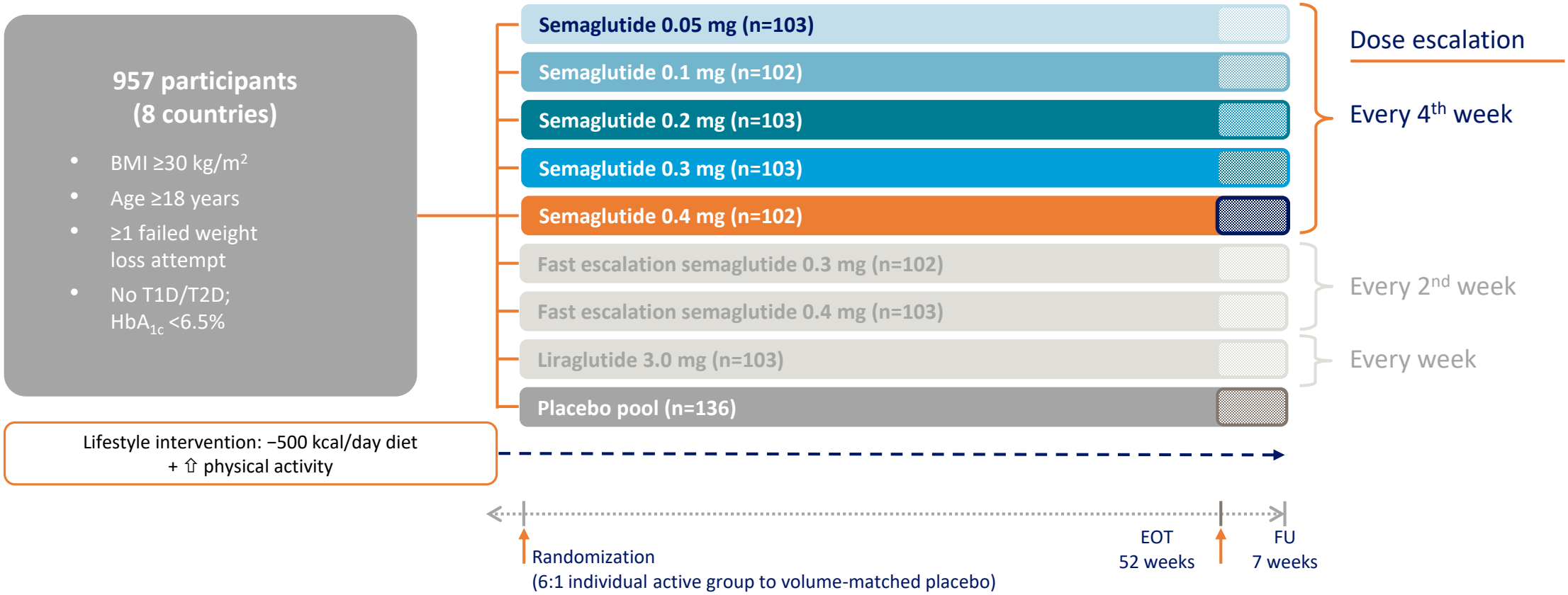
N=52 patients age 18- 70 years with biopsy-confirmed NASH and, if diabetic, HbA1c <9.0% and managed by either diet or stable dose of metformin or sulfonylurea, were randomized to 1.8 mg liraglutide or placebo daily for 48 weeks.

Armstrong MJ, et al. *Lancet*. 2016;387(10019):679-690.

Semaglutide obesity study: Trial design

Multinational, double-blind, placebo- and active-controlled, phase 2 dose-ranging RCT (NCT02453711)[1]

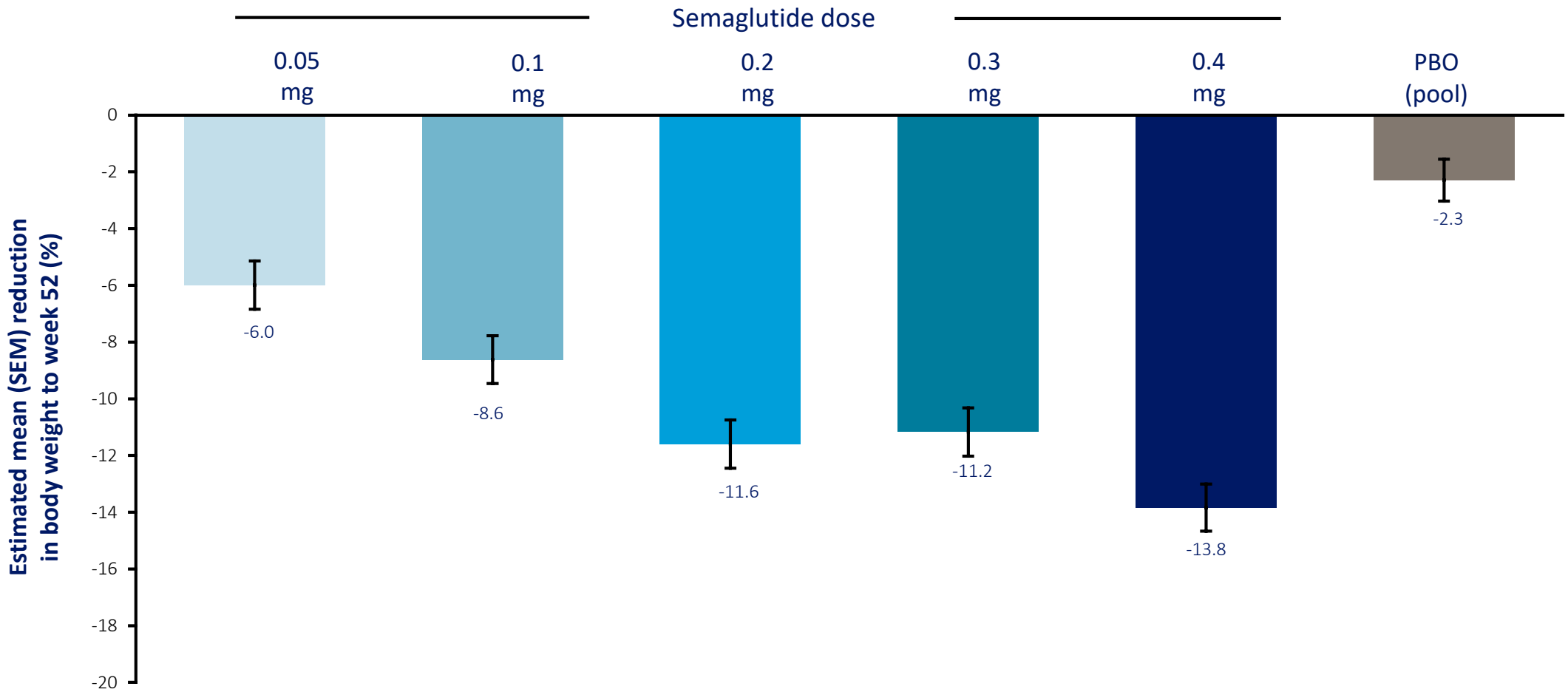
One-year safety, tolerability, and weight loss associated with once-daily semaglutide



BMI, body mass index; EOT, end of treatment; FU, follow-up; RCT, randomized controlled trial; T1D/T2D, type 1 or type 2 diabetes.

1. O'Neil PM, *et al. Lancet* 2018;392:637–49.

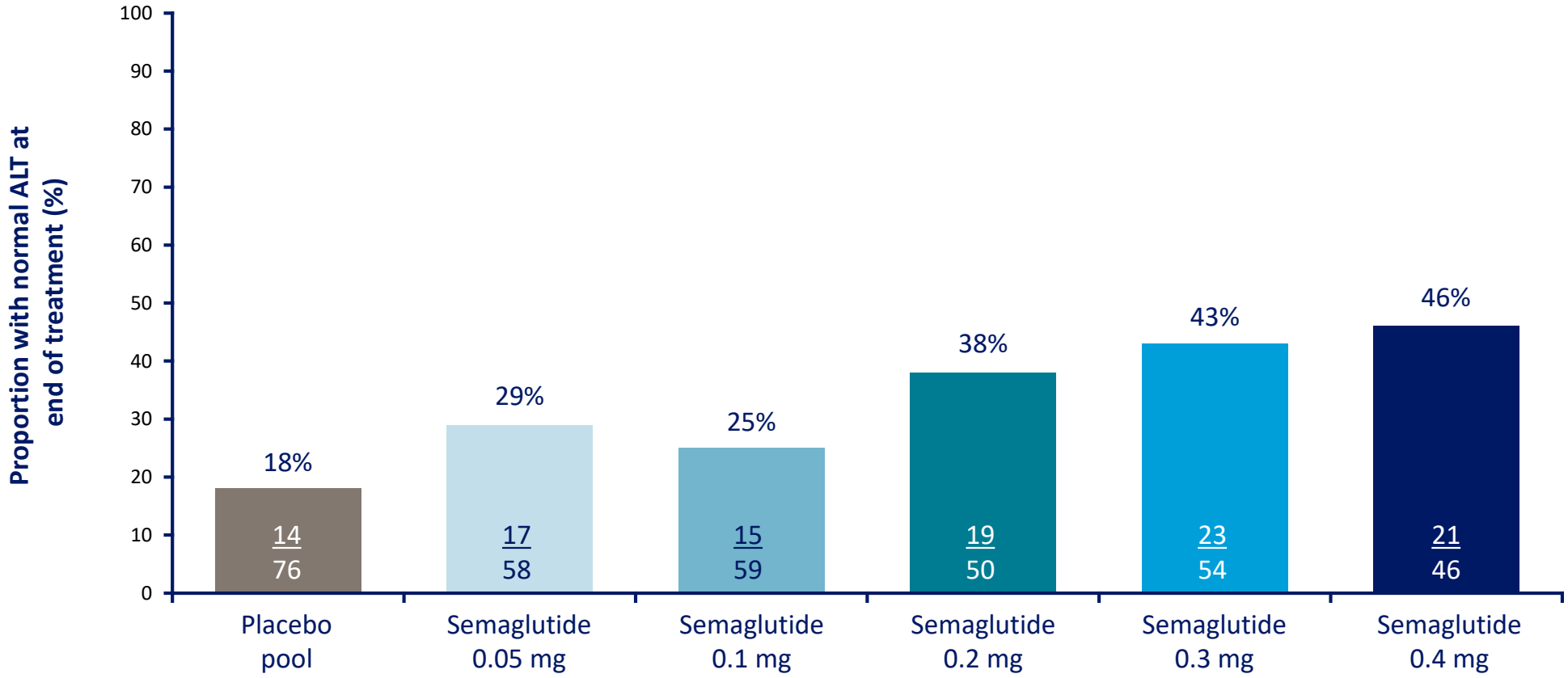
Change in body weight



Estimated changes are ANCOVA-modelled with J2R-MI of missing data.
ANCOVA, analysis of covariance; J2R-MI, jump-to-reference multiple imputation; PBO, placebo; SEM, standard error of the mean.
O'Neil PM, *et al. Lancet* 2018;392:637–49.

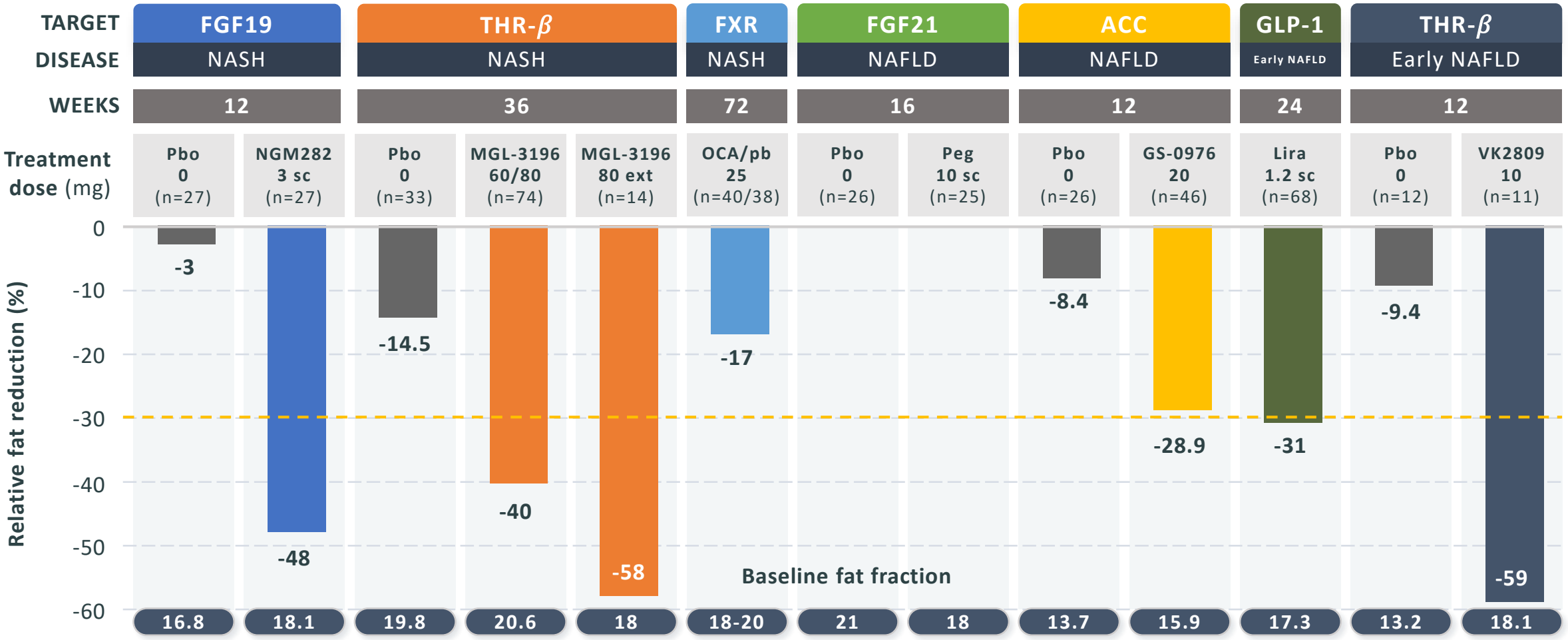
ALT normalization at week 52

Subjects with elevated baseline ALT



ALT, alanine aminotransferase.

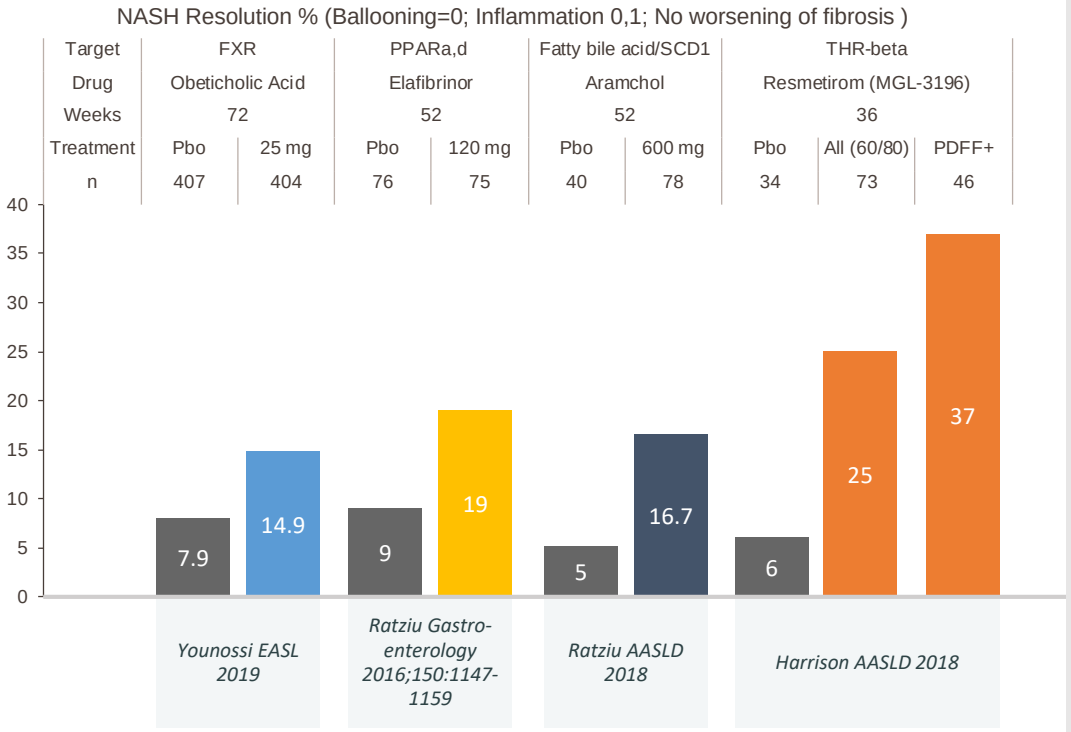
MRI-PDFF relative fat reduction (%)



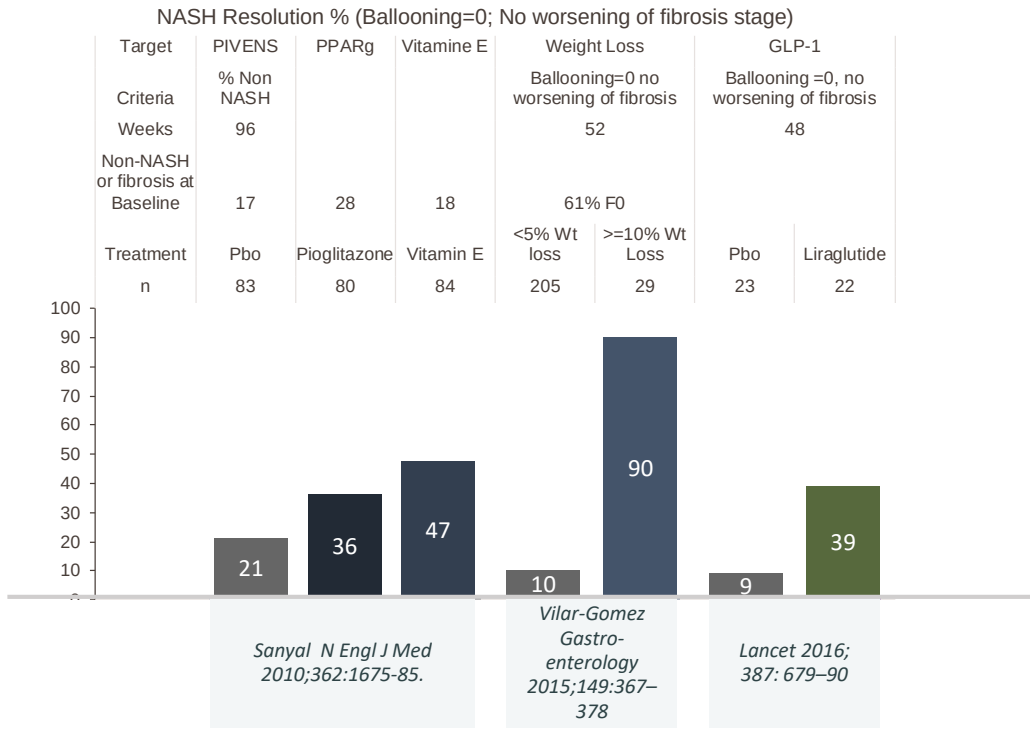
ACC, acetyl-CoA carboxylase; FGF, fibroblast growth factor; FXR, farnesoid X receptor; GLP-1, glucagon-like peptide-1; Lira, liraglutide; MRI-PDFF, magnetic resonance imaging – proton density fat fraction; NAFLD, non-alcoholic fatty liver disease; NASH, non-alcoholic steatohepatitis; OCA, obeticholic acid; Pbo, placebo; Peg, pegbelfermin; sc, subcutaneous; THR, thyroid hormone receptor. Harrison SA. Lancet. 2018;391:1174–85; Harrison AASLD 2018; Loomba AASLD 2017; Sanyal AJ. Lancet. 2018;392:2705-717; Loomba R. Gastroenterology. 2018;155:1463–1473; Petit JM. J Clin Endocrinol Metab. 2017;102:407–15; Loomba AASLD 2018.

NASH resolution (varying definitions)

% NASH Resolution (Ballooning=0; Infl =0,1) No worsening of fibrosis stage Baseline NASH Fibrosis Stage 1-3



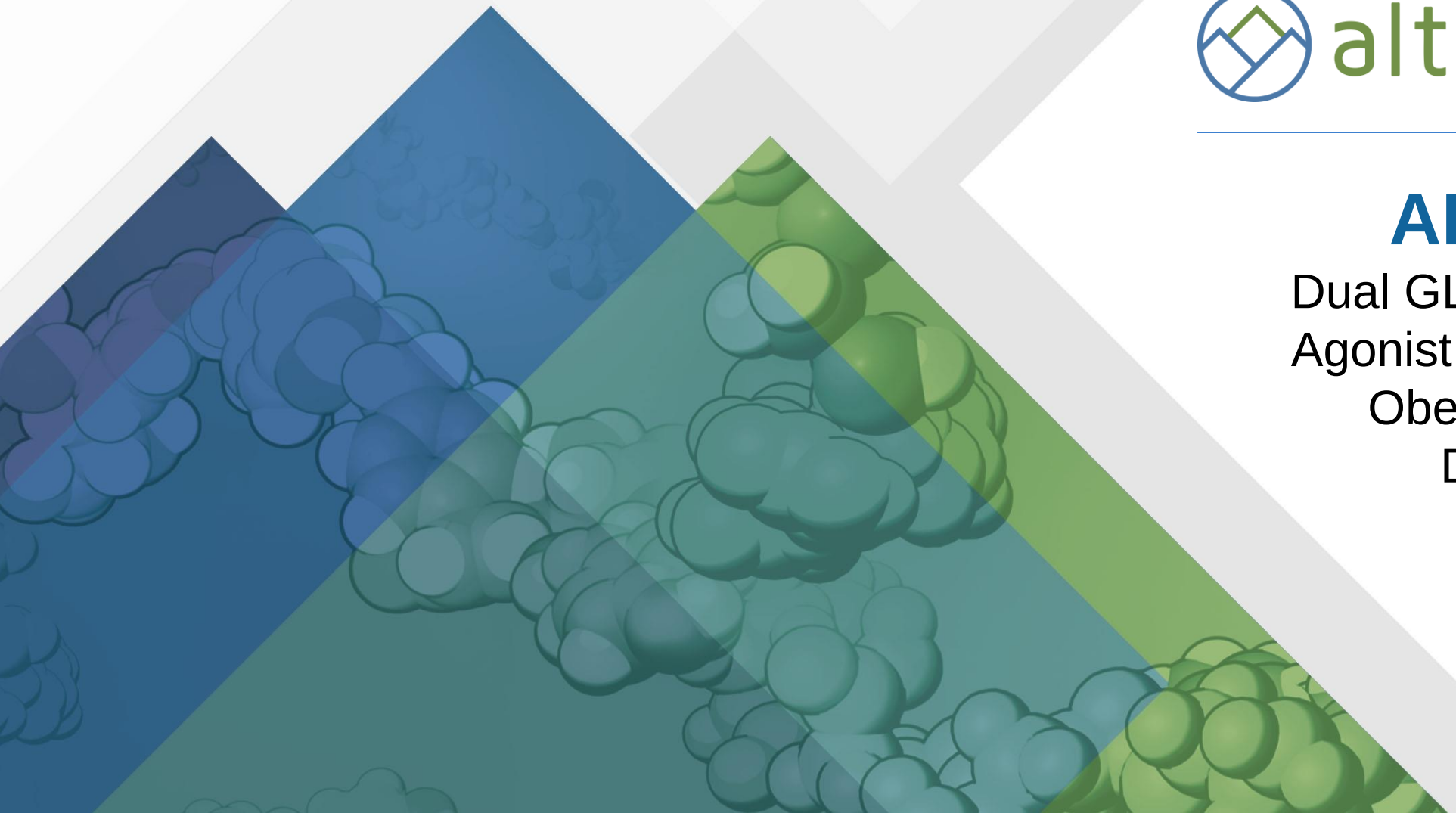
% NASH Resolution (non-NASH, or ballooning=0)) Baseline NASH Fibrosis Stage 0-3



MGL-3196 required NASH resolution (ballooning =0; infl =0, 1) with at least 2 point decrease in NAS; Elafibranor data included enrolled patients with NAS>3; other targets, selonsertib, cenicriviroc did not demonstrate NASH resolution relative to placebo (not shown); obeticholic acid, F1-F3 shown

Summary

- NASH prevalence is increasing; is particularly prominent in diabetic patients; and linked to the rising prevalence of HCC
- Multiple targets for therapeutics. Ideal candidate would be pleiotropic in mechanism and include metabolic pathways
- Data with GLP-1 agonists look promising for NASH
- Significant opportunity exists to improve on current late stage product candidates both for NASH resolution and fibrosis improvement



altimmune

ALT-801

Dual GLP-1/Glucagon Agonist for NASH and Obesity-Driven Disease