Clinical Impact of New Data From the 2017 Washington, DC, Hepatology Meeting

CCO Independent Conference Coverage*

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Faculty Disclosures

Stephen A. Harrison, MD, COL (Ret.), FAASLD, has disclosed that he has received consulting fees from Axcella, Capulus, the Chronic Liver Disease Foundation, Cirius, Genfit, Echosens, Histoindex, Intercept, Madrigal, NGM Bio, Novartis, Novo Nordisk, Perspectum, and Prometheus; has received fees for non-CME/CE services received directly from a commercial interest or their agents (eg, speaker bureaus) from AbbVie, Alexion, and Gilead Sciences; and has an ownership interest in Cirius.

Paul Y. Kwo, MD, has disclosed that he has received consulting fees from AbbVie, Bristol-Myers Squibb, Gilead Sciences, and Merck and funds for research support from AbbVie and Gilead Sciences.

Norah Terrault, MD, MPH, FAASLD has disclosed that she has received consulting fees from Gilead Sciences and Merck and funds for research support from AbbVie, Gilead Sciences, and Merck.

Current HCV Therapy

AASLD/IDSA Recommendations for First-line HCV Treatment

HCV GT	Regimen	Duration, Wks	
		No Cirrhosis	Compensated Cirrhosis
1	GLE/PIB	8	12
	GZR/EBR*	12	12
	SOF/LDV	8 or 12†	12
	SOF/VEL	12	12
2 or 3	GLE/PIB	8	12
	SOF/VEL	12	12‡
4	GLE/PIB	8	12
	SOF/VEL	12	12
	GZR/EBR	12	12
	SOF/LDV	12	12
5 or 6	GLE/PIB	8	12
	SOF/LDV	12	12
	SOF/VEL	12	12

*If GT1a, use only if no baseline NS5A elbasvir RASs detected.

†If nonblack, no HIV, and HCV RNA < 6 million IU/mL, 8-wk duration recommended.

‡For GT3, if Y93H RAS detected, add RBV or consider SOF/VEL/VOX.

AASLD/IDSA. HCV guidelines. September 2017.

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AASLD/IDSA Recommendations for DAA-Experienced Pts

HCV GT	Duration, Wks	Previous Experience		
		NS3/4A	NS5B	NS5A
1	12	GLE/PIB SOF/LDV (no cirrhosis) SOF/VEL	GLE/PIB SOF/VEL (1b) SOF/VEL/VOX (1a)	SOF/VEL/VOX
2*	12	NA	GLE/PIB SOF/VEL	NA
3	12	SOF/VEL/VOX	SOF/VEL/VOX	SOF/VEL/VOX ± RBV†
4, 5, 6	12	SOF/VEL/VOX	SOF/VEL/VOX	SOF/VEL/VOX

*Recommendations for any SOF + RBV experienced pt. †RBV if NS5A inhibitor failure and cirrhosis.

AASLD/IDSA. HCV guidance. September 2017.

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STREAGER: 8-Wk GZR/EBR for Treatment-Naive, Noncirrhotic Pts With GT1b HCV

- Interim analysis of single-arm study

- n = 2 relapses
  - Pt 1: HCV RNA 14 million IU/mL, F0-F1 fibrosis, Y93H mutation
  - Pt 2: HCV RNA 453,899 IU/mL; F2 fibrosis; **GT1e HCV**; L28M, R30Q, A92T, Y93H mutations

*Interim analysis; planned N = 120.

Abergel A, et al. AASLD 2017. Abstract LB-5.

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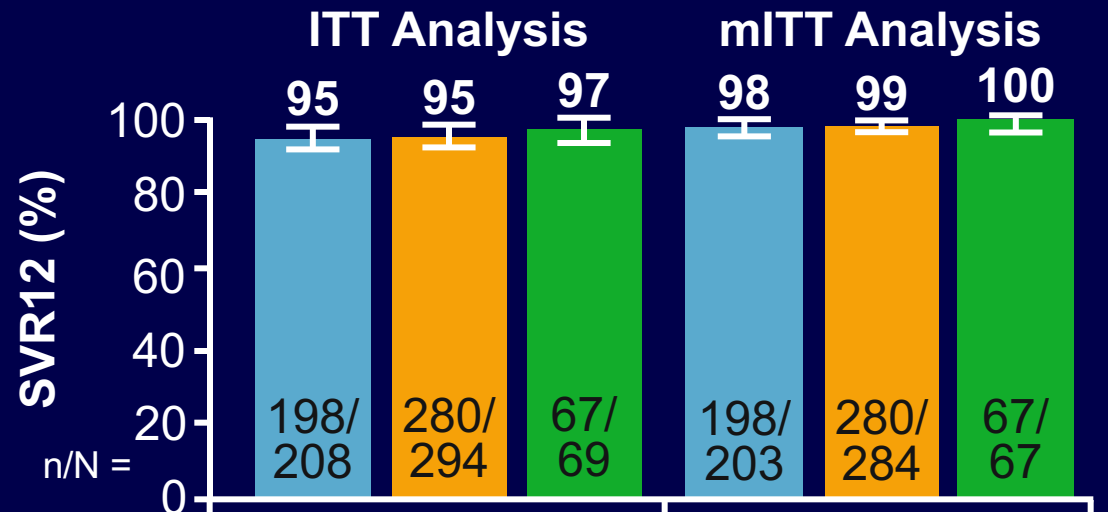
Efficacy and Safety of GLE/PIB for Treatment-Naive Pts With GT3 HCV

- Integrated analysis of phase II/III trials of **GLE/PIB** for 8 wks (noncirrhotic pts, n = 208) or 12 wks (noncirrhotic pts, n = 294; cirrhotic pts, n = 69) in treatment-naive pts with GT3 HCV



Tx Wks	8	12	12	8	12	12
Cirrhosis	No	No	Yes	No	No	Yes
OTVF	1*	1	1*			
Relapse	5	4*	0			
Non-VF	4	9	1			

*1 pt in each group failed for noncompliance.

- No statistically significant differences in SVR12 for 8-wk vs 12-wk treatment across subgroups, including:
  - Black race (n = 10)
  - OST (n = 79)
  - Recent or history of drug use (n = 35)
  - Baseline RAS (n = 181)
- Only n = 3 discontinued for AE

Flamm S, et al. AASLD 2017. Abstract 62. Reproduced with permission.

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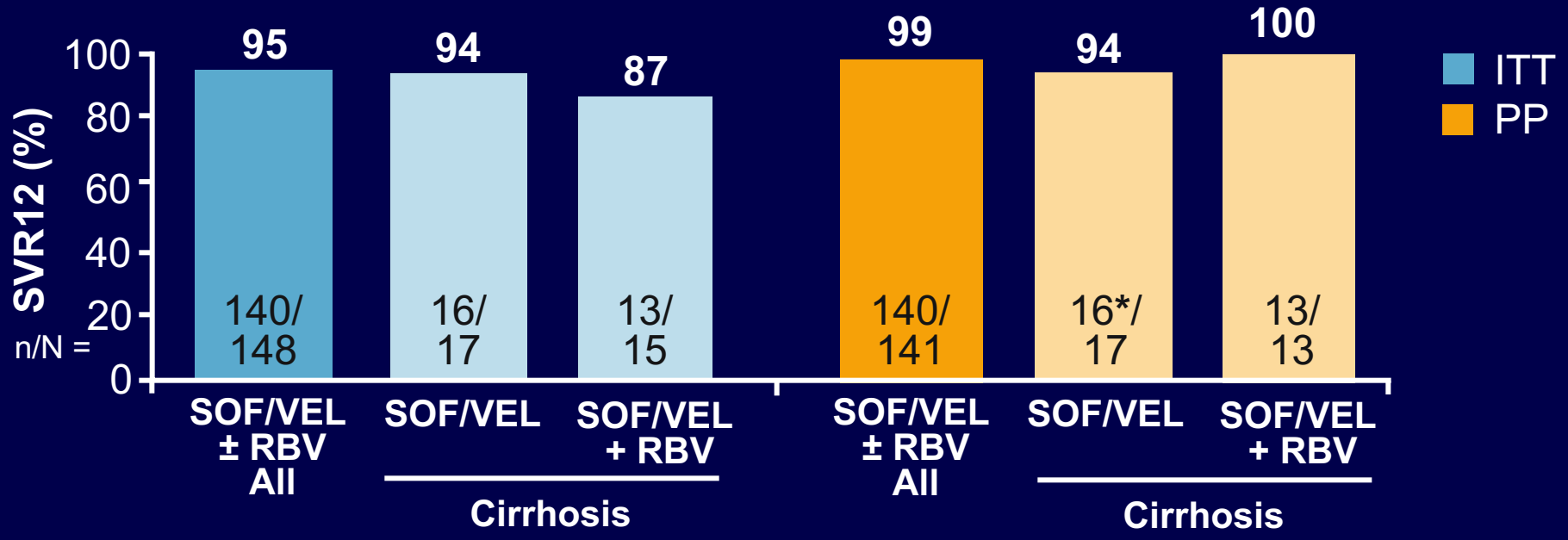
GECCO: Real-World Efficacy of SOF/VEL in Pts With GT3 HCV

- Prospective German cohort study assessing efficacy of **SOF/VEL ± RBV** for 12 wks in pts with GT3 HCV (N = 232 [n = 148 analyzed]; 14% treated with RBV)
  - 26% of pts previously treated with pegIFN + RBV or SOF-based regimen



- *n = 1 relapse (prior SOF + RBV for 6 mos, no BL RAS, emergent Y93H)
- Baseline RAS did not impact ability to achieve SVR12

Christensen S, et al. AASLD 2017. Abstract 63. Reproduced with permission.

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SOF/LDV for Pts With Severe Renal Impairment

- Single-arm, open-label phase II study

- Pharmacokinetics
  - Mean SOF, GS-331007, LDV exposures 103%, 501%, 57% higher in study population vs those in the phase II/III SOF/LDV trial populations
- Safety
  - No treatment-related serious AEs or cardiac AEs, discontinuations for AEs, clinically meaningful eGFR change

Lawitz E, et al. AASLD 2017. Abstract 1587.

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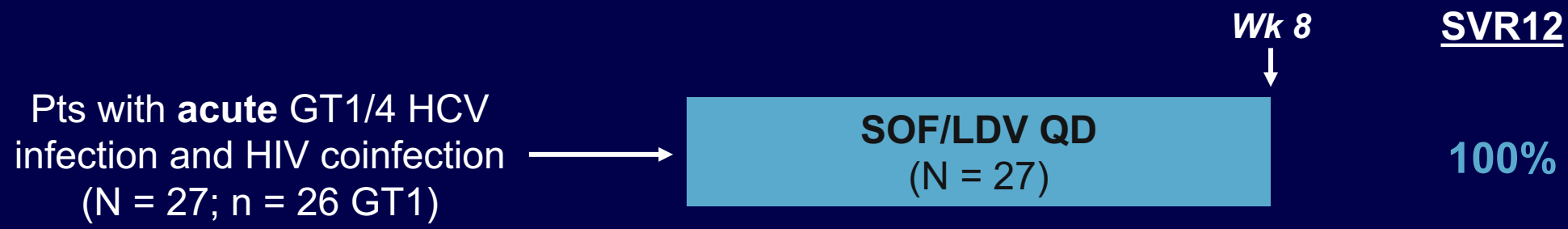
SWIFT-C: 8-Wk SOF/LDV for Pts With HIV and Acute GT1/4 HCV Coinfection

- Optimal duration of SOF/LDV for acute HCV infection in HIV-coinfected pts not known; previous study showed SVR12 rate of 77% with 6 wks of therapy[1]
- SWIFT-C: single-arm study[2]
  - Baseline HIV regimens: boosted PI, 26%; NNRTI, 30%; INSTI, 52%; TDF/FTC, 85%; ABC/3TC, 15%



1. Rockstroh JK, et al. Lancet Gastroenterol Hepatol. 2017;2:347-353.
2. Naggie S, et al. AASLD 2017. Abstract 196.

Slide credit: clinicaloptions.com

Co-STAR Part B: Assessment of Reinfection Risk in Pts on OAT Who Received GZR/EBR

- Observational study in which pts on OAT* with GT1/4/6 HCV who received GZR/EBR in Co-STAR Part A were assessed for HCV reinfection and drug use for 3 yrs (N = 199)[2]
  - Part A: SVR12 rate of 91% with 12-wk GZR/EBR; 97% of pts had > 95% adherence
- **10 reinfections occurred during 24 mos following end of HCV treatment**

Parameter at Post-Tx Mo 24	Reported IDU*	No Reported IDU*
IDU, % (n)	37 (74)	63 (125)
Reinfection rate/100 PY (95% CI)	4.2 (1.5-9.2)	0.4 (0-2.3)

*In the previous 6 mos.

Dore G, et al. AASLD 2017. Abstract 195.

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HCV Therapy and Risk of Mortality and HCC

ANRS HEPATHER: HCV DAA Therapy and Risk of Mortality, HCC, and Decompensated Cirrhosis

- Multicenter observational cohort study assessing short-term effects of DAAs on mortality, HCC, and DC risk in pts with HCV (N = 9295)
  - Median follow-up: 24 mos
- **DAA treatment associated with decreased risk of death vs no DAA treatment**

Event, n (%)	DAA Treatment (n = 6460)*	No DAA Treatment (n = 2835)†	aHR (95% CI)
All-cause death	90 (1.4)	78 (2.8)	**0.65 (0.45-0.95); *P* = .0258**
▪ Liver related	NR	NR	0.68 (0.36-1.29)
▪ Nonliver related	NR	NR	0.75 (0.43-1.31)
HCC	164 (2.5)	57 (2.0)	1.19 (0.85-1.66); *P* = .3178
DC	77 (1.2)	35 (1.2)	0.90 (0.58-1.41); *P* = .6533

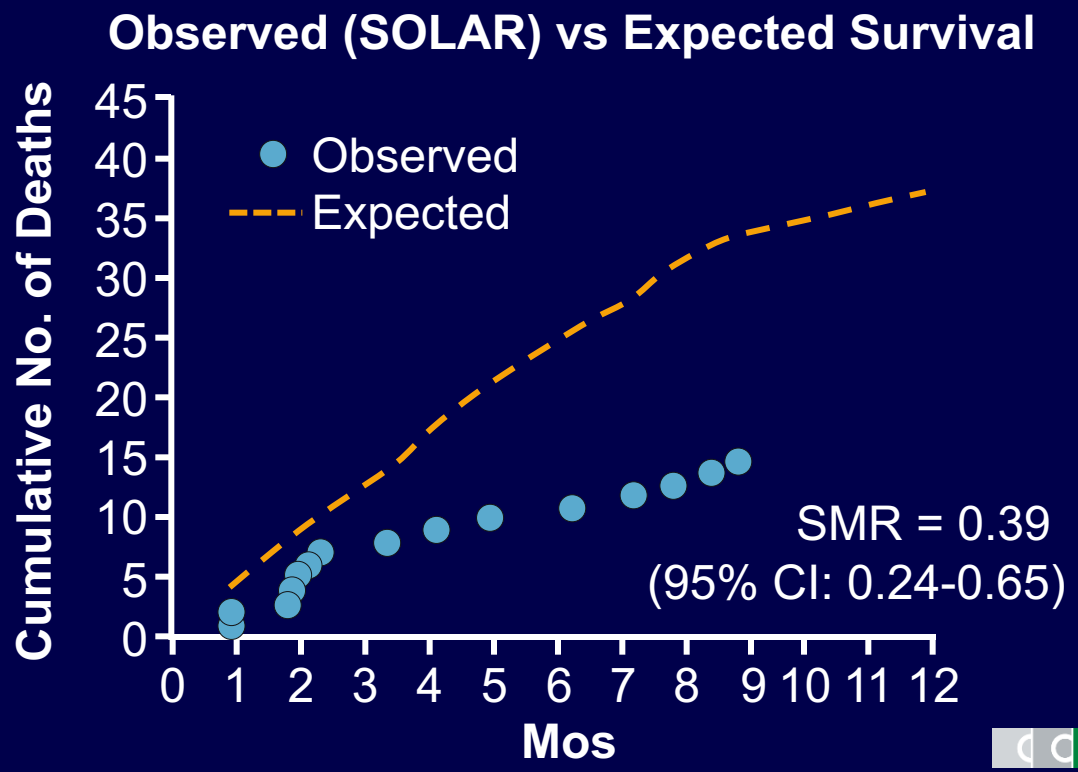
Effect of DAAs quantified with weighted Cox proportional hazards models adjusted for confounding via IPTW. IPTW scores derived from covariates associated with DAA, including age, sex, cirrhosis, and treatment experience.
*8482 person-yrs. †10040 person-yrs.

Carrat F, et al. AASLD 2017. Abstract LB-28.

HCV DAA Therapy and Survival in Pts With Decompensated Cirrhosis

- Comparison of incidence of death in pts with hepatic decompensation in the SOLAR studies of SOF/LDV + RBV (n = 212) and pre-DAA era pts in OPTN meeting SOLAR criteria (model creation set, n = 2071; validation set, n = 899)
  - Survival prediction model created using OPTN data to determine expected mortality for 1 yr in pts not receiving DAA therapy; model included age, albumin, HE grades 1/2 and 3/4, MELD, sodium

- 15 deaths in 1 yr in SOLAR study
- **DAA therapy lowered risk of death by 60%**



Kim WR, et al. AASLD 2017. Abstract LB-27. Reproduced with permission.

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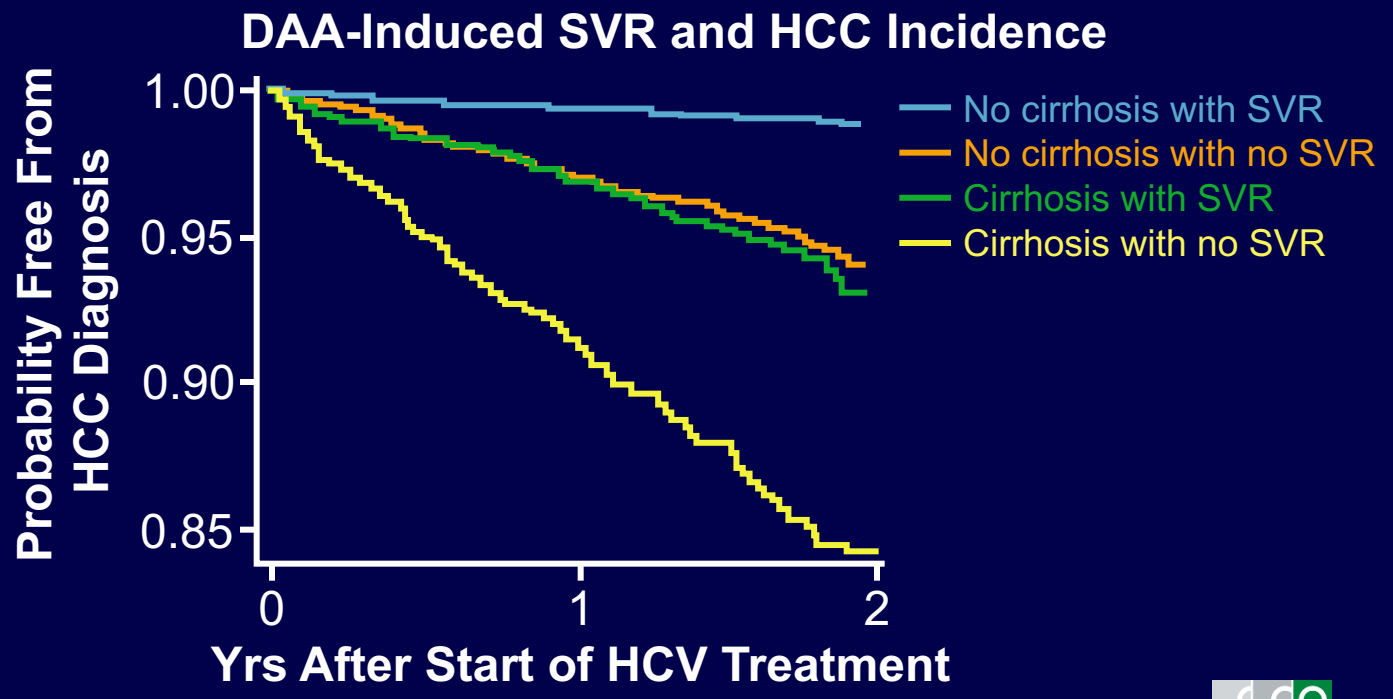
DAA Therapy and HCC Risk in Large VA Cohort

- Retrospective cohort study assessing the relationship between SVR and HCC risk in pts with HCV in the VA healthcare system receiving antiviral therapy 1999-2015 (N = 62,354)
  - 58% received IFN-only therapy, 35% received DAA-only therapy
- **SVR with DAA regimen associated with 71% decrease in HCC risk**

Regimen	HCC/100 PY	aHR
IFN only		
▪ SVR	0.28	0.32
▪ No SVR	1.07	
DAA + IFN		
▪ SVR	0.6	0.48
▪ No SVR	1.73	
DAA only		
▪ SVR	0.92	0.29
▪ No SVR	5.2	



Ioannou G, et al. AASLD 2017. Abstract 142. Reproduced with permission.

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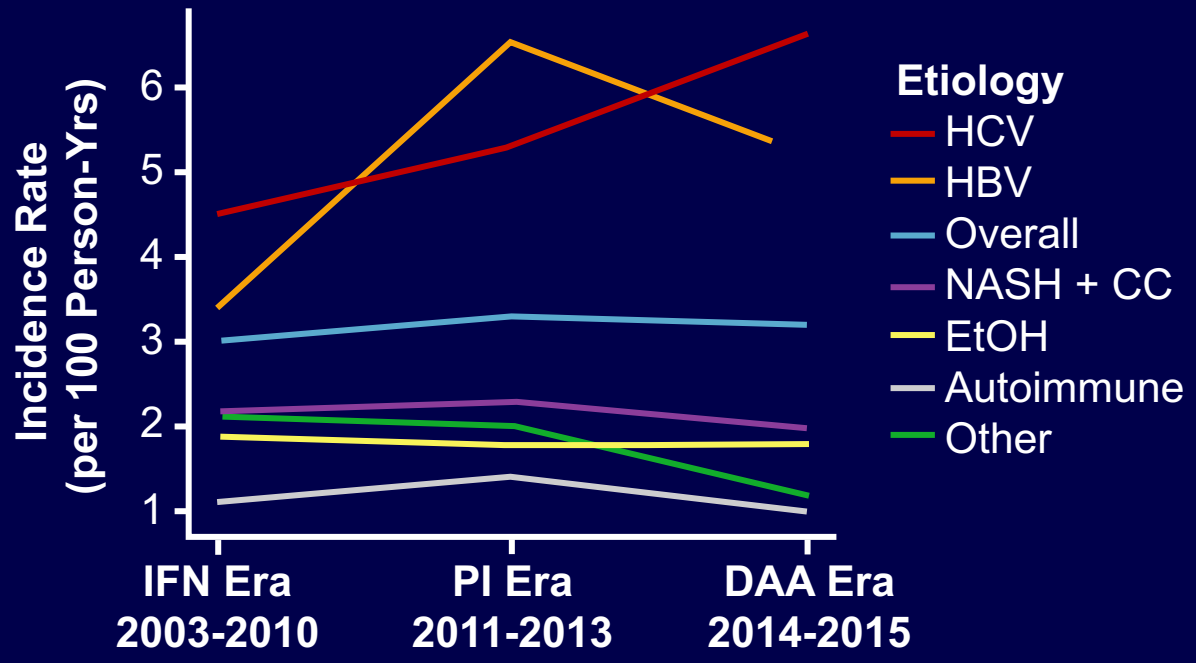
Incidence of HCC in Liver Transplant Registrants With HCV

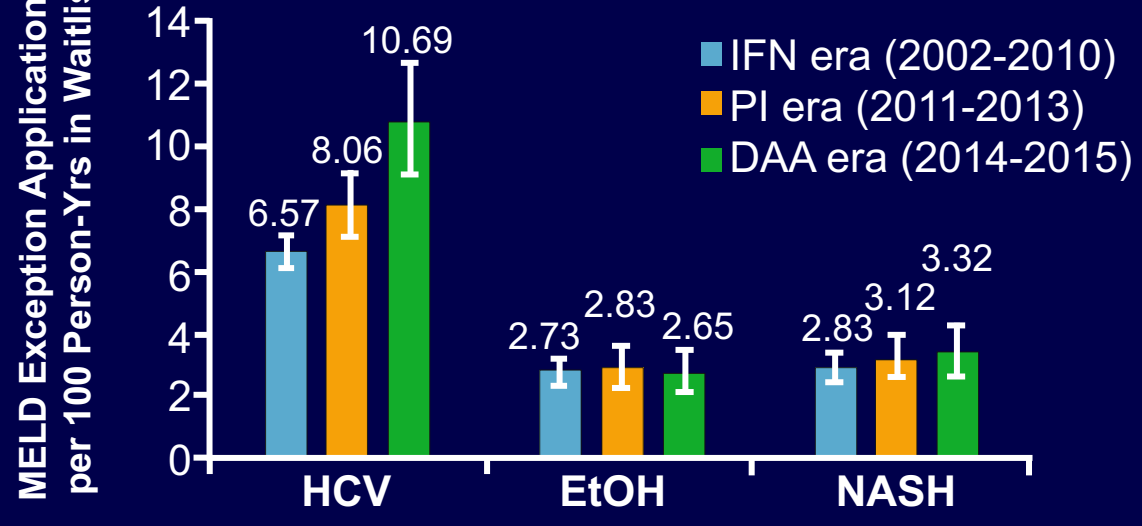
- Analysis of HCC incidence in pts awaiting liver transplantation in SRTR database (N = 48,158; n = 20,039 with HCV)[1]

Incidence of HCC by Etiology and HCV Treatment Era



- Analysis of HCC-related MELD exceptions in pts awaiting liver transplantation in OPTN database (N = 55,416; n = 23,020 with HCV)[2]

HCC-Related MELD Exceptions By Etiology and HCV Treatment Era



1. Kwong AJ, et al. AASLD 2017. Abstract 121. 2. Esteban JP, et al. AASLD 2017. Abstract 1040.

Managing HBV Infection

HBV Treatment Guidelines: Starting Treatment

When to Start

Guideline	HBeAg Positive			HBeAg Negative		
	HBV DNA, IU/mL	ALT	Liver Disease	HBV DNA, IU/mL	ALT	Liver Disease
AASLD[1]	> 20,000	≥ 2 x ULN	N/A	≥ 2000	≥ 2 x ULN	N/A
	N/A	N/A	Cirrhosis	N/A	N/A	Cirrhosis
EASL[2]	> 2000	> ULN*	Moderate inflammation or fibrosis*	> 2000	> ULN*	Moderate inflammation or fibrosis*
	> 20,000	> 2 x ULN	N/A	> 20,000	> 2 x ULN	N/A
	N/A	N/A	Cirrhosis	N/A	N/A	Cirrhosis

*In pts with HBV DNA > 2000 IU/mL, treatment indicated if ALT > ULN and/or at least moderate fibrosis.

What to Start

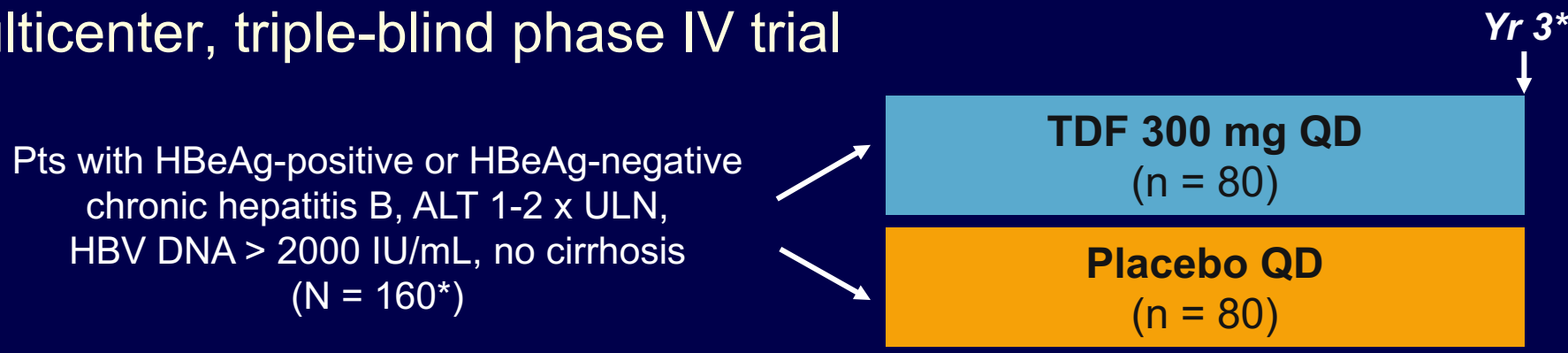
Preferred: entecavir, tenofovir alafenamide,† tenofovir disoproxil fumarate, peginterferon‡[1,2]

†AASLD guidelines not yet updated since approval of tenofovir alafenamide. ‡May be considered for select pts (eg, pts with mild/moderate CHB)

1. Terrault NA, et al. Hepatology. 2016;63:261-283. 2. EASL. J Hepatol. 2017;67:370-398.

TDF vs Placebo for HBsAg-Positive CHB With Mild ALT Elevation

- Multicenter, triple-blind phase IV trial



*Preliminary results for 114 pts completing treatment with paired biopsy.

Baseline Fibrosis Stage, %	TDF (n = 57)	Placebo (n = 57)
0/1	51	42
2	39	32
3	7	12
4	4	14

Selected Baseline Characteristic	TDF (n = 57)	Placebo (n = 57)
HBeAg positive, %	16	26
Median HBsAg, log IU/mL (IQR)	3.07 (2.39-3.63)	3.12 (2.61-3.84)

Hsu YC, et al. AASLD 2017. Abstract 25. ClinicalTrials.gov. NCT01522625.

Slide credit: clinicaloptions.com

TDF vs Placebo for HBsAg-Positive CHB With Mild ALT Elevation: Key Findings

3-Yr Outcome, n (%)	TDF (n = 57)	PBO (n = 57)
Progression		
▪ **In fibrosis stage**	**14 (24.6)***	**25 (43.9)***
▪ To cirrhosis	2 (3.5)	8 (14.0)
Inflammation score		
▪ Median (IQR)	2 (1-3)†	3 (2-4)†
▪ Decrease	31 (54.4)	24 (42.1)

*P = .03 for any progression in fibrosis stage. †P = .002.

- OR for fibrosis progression with TDF vs PBO: 0.42 (95% CI: 0.19-0.93)

- Significantly higher 3-yr rates with TDF vs PBO for:
  - Undetectable HBV DNA: 85.5% vs 10.9%
  - ALT normalization: 76.8% vs 51.8%
- Entecavir only needed for clinical flare in PBO-treated pts (n = 8)

Hsu YC, et al. AASLD 2017. Abstract 25. ClinicalTrials.gov. NCT01522625.

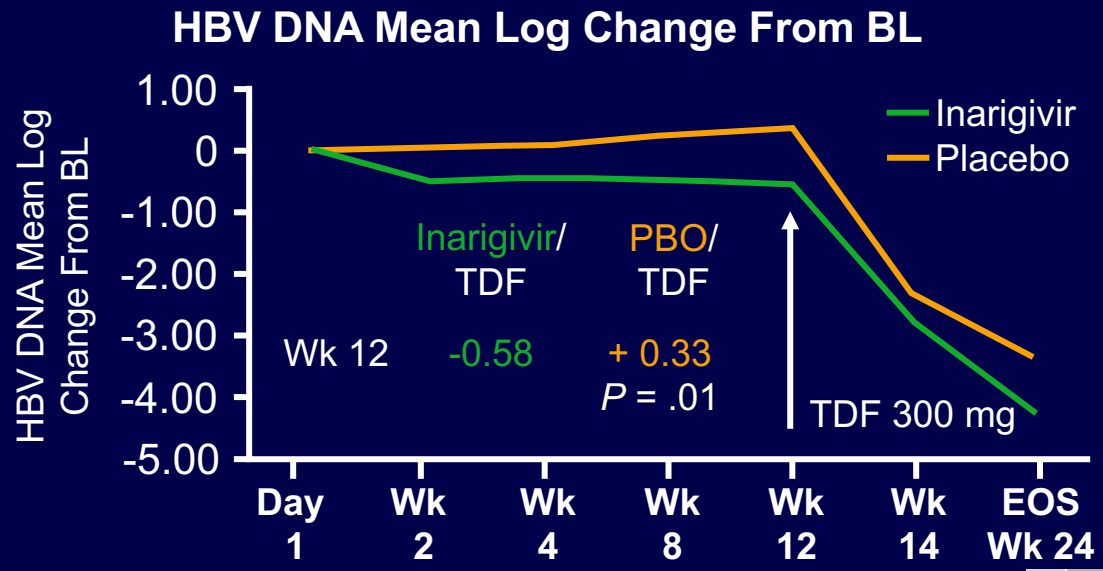
Slide credit: clinicaloptions.com

Selected Studies on Emerging Investigational Agents for Treating HBV

- Study 002*: randomized, single-blind phase IIa study of 8 wks of ARB-1467 IV Q4W (2 doses) vs PBO or open-label ARB-1467 IV Q2W in HBeAg-negative, noncirrhotic pts with chronic HBV on ETV or TDF (N = 30)[1]
  - ARB-1467: siRNAs targeting HBV RNA

	ARB-1467		
	0.2 mg/kg Q4W	0.4 mg/kg Q4W	0.4 mg/kg Q2W
Mean change in HBsAg (log IU/mL), Wk 12‡	-0.6	-0.9	-1.1

- ACHIEVE†: randomized, double-blind phase II study of 4 doses of inarigivir PO QD or PBO for 12 wks for tx-naive, noncirrhotic pts with HBV (N = 20)[2]
  - Inarigivir (formerly SB 9200): activates RIG-I, NOD-2, subsequent IFN responses

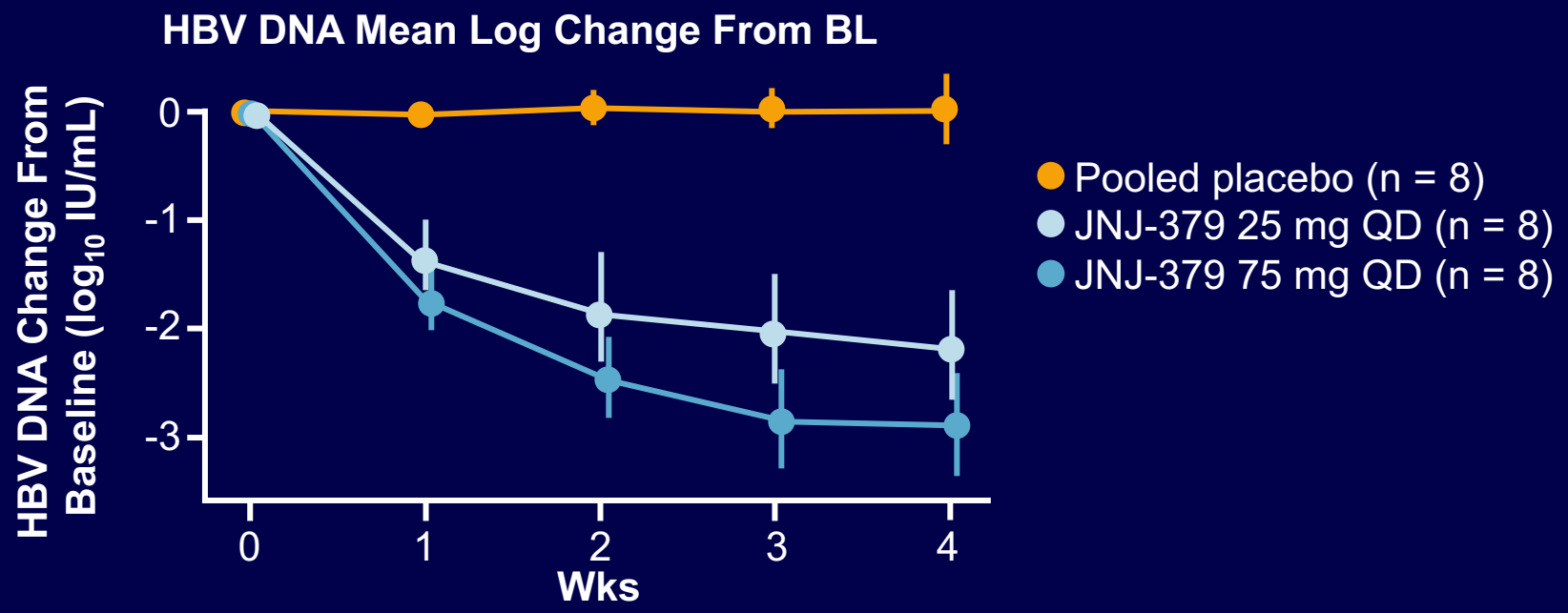


*Cohorts 1, 2, 4. †Part A, Cohort 1. ‡Estimated from graph.

1. Agarwal K, et al. AASLD 2017. Abstract 40. 2. Yuen M, et al. AASLD 2017. Abstract 39.

Selected Studies on Emerging Investigational Agents for Treating HBV

- HPB1001, Part 2: randomized, double-blind, phase I study of 2 doses of JNJ-379 QD for 28 days or placebo for pts with chronic HBV infection (N = 24)[1]
  - JNJ-379: capsid assembly modulator



Zoulim F, et al. AASLD 2017. Abstract LB-15. Reproduced with permission.

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Emerging Therapeutics for HDV Infection

Selected Studies on Emerging Investigational Agents for Treating HDV

- Open-label, randomized phase IIb study of 3 doses of Myrcludex B SC QD + TDF or TDF for 24 wks in pts with chronic HBV/HDV (N = 120)[1]
  - Myrcludex B: HBV/HDV entry inhibitor

Outcome*	MyrB + TDF			TDF
	2 mg	5 mg	10 mg	
HDV RNA reduction at Wk 24, log_{10} IU/mL	NR	-1.63 (n = 7)	-2.42 (n = 10)	-0.015 (n = 9)
Complete ALT normalization at Wk 12, n/N (%)	7/21 (33)	5/21 (24)	9/22 (41)	NR

*Interim results.

- LIMT: open-label, randomized phase II study of 2 doses of pegIFN lambda for 48 wks in pts with chronic HDV infection (N = 33)[2]
  - pegIFN λ: type III IFN with unique receptor binding; reduced cytopenias and other AEs vs IFN alfa[3]
- At Wk 24*:
  - 5/10 (50%) analyzable pts achieved ≥ 2 log HDV RNA decline
  - 4/10 (40%) analyzable pts HDV PCR negative

1. Wedemeyer H, et al. AASLD 2017. Abstract 37. Data from abstract.
2. Hamid S, et al. AASLD 2017. Abstract 927. 3. Chan HLY, et al. J Hepatol. 2016;64:1011-1019.

NAFLD/NASH:
Disease Outcomes and Novel Therapeutics

NAFLD Prevalence and Outcomes Among Racial and Ethnic Subgroups in the United States

- Meta-analysis of 34 studies of NAFLD prevalence, severity, or prognosis (N = 368,569)

Parameter	Finding
NAFLD prevalence, %*	11.2
NAFLD relative risk (95% CI)*	
▪ Hispanic vs white pts	1.36 (1.08-1.73)
▪ Black vs white pts	0.68 (0.54-0.84)
NASH prevalence in NAFLD pts, %†	31.4
NASH relative risk in NAFLD pts (95% CI)†	
▪ Hispanic vs white pts	1.24 (1.02-1.52)
▪ Black vs white pts	0.72 (0.60-0.87)

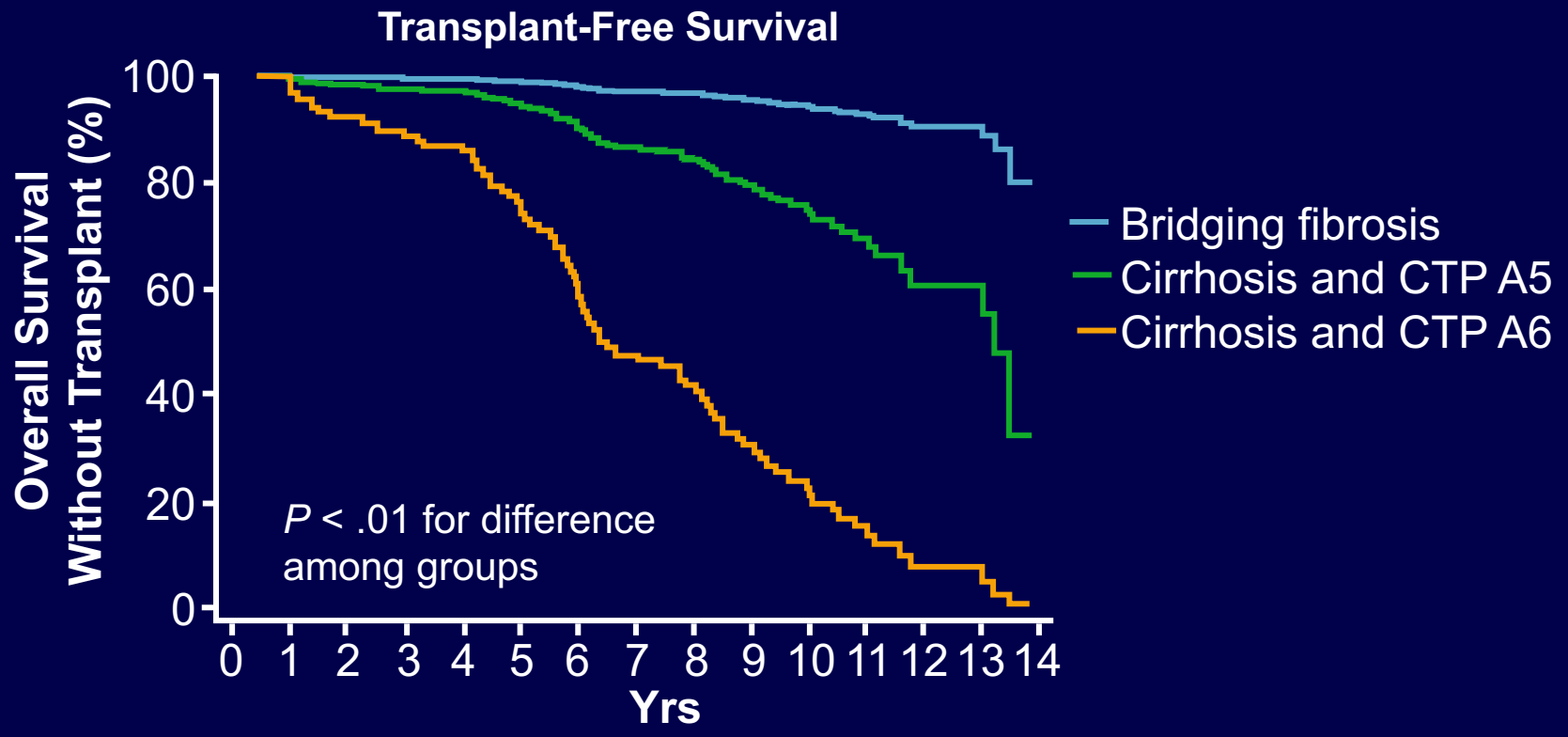
Parameter	Finding
F3-F4 fibrosis prevalence in NAFLD pts, %‡	19.5
F3-F4 fibrosis relative risk in NAFLD pts (95% CI)‡	
▪ White vs Hispanic pts	1.02 (0.94-1.11)
▪ White vs black pts	1.10 (1.00-1.22)

*Based on 9 studies, n = 343,393.
†Based on 10 studies, n = NR (subset of 16,083).
‡Based on 11 studies, n = NR (subset of 16,083).

Rich NE, et al. AASLD 2017. Abstract 57.
Rich NE, et al. Clin Gastroenterol Hepatol. 2017;[Epub ahead of print].

Association Between Fibrosis Severity and Clinical Outcomes in Advanced NAFLD

- International, prospective cohort study of clinical outcomes in NAFLD pts with compensated cirrhosis or biopsy-proven bridging fibrosis from 1995-2013 (N = 458)



*Adjusted by age, center, sex, and race/ethnicity.

Vilar-Gomez E, et al. AASLD 2017. Abstract 60. Reproduced with permission.

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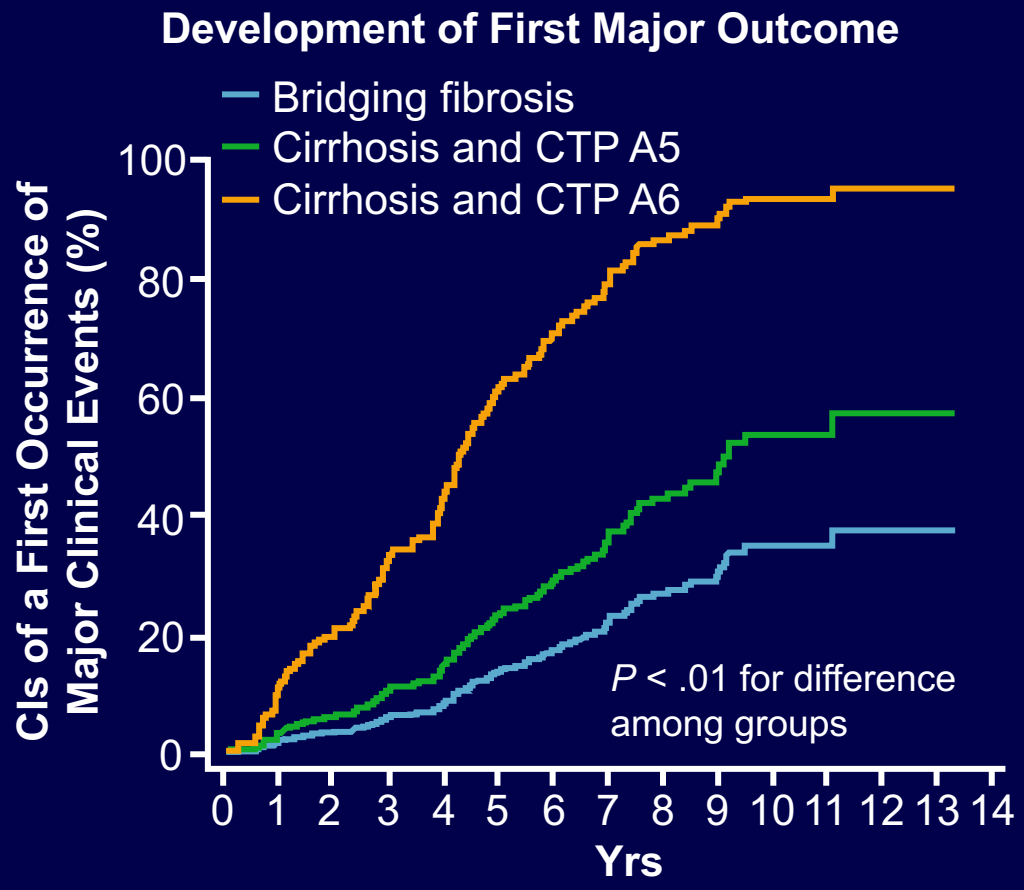
Association Between Fibrosis Severity and Clinical Outcomes in Advanced NAFLD



Clinical Outcome	F3 Fibrosis (n = 159)	Cirrhosis CTP-A5 (n = 222)	Cirrhosis CTP-A6 (n = 77)
1st event occurrence, n (%)	26 (16)	63 (28)	52 (66)
▪ Hepatic decompensation, n/N (%)	5/26 (19)	37/63 (59)	44/52 (85)
▪ HCC, n/N (%)	2/26 (8)	12/63 (19)	8/52 (15)
▪ Major vascular event, n/N (%)	9/26 (35)	4/63 (6)	0
▪ Nonhepatic malignancy, n/N (%)	10/26 (38)	10/63 (16)	0

Vilar-Gomez E, et al. AASLD 2017. Abstract 60. Reproduced with permission.

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Cardiovascular Risk in Pts With NAFLD

- Retrospective analysis of sex-related incidence of CV events in pts diagnosed with NAFLD (n = 3869) and matched controls (n = 15,209) in Rochester County, Minnesota, from 1997-2014
  - 122,758 PYFU; 1375 CV events, 1551 deaths
- Risk of CV event:
  - General population: 23% lower in women vs men
  - NAFLD population: not different between women vs men, suggesting NAFLD may attenuate the sex-based CVD risk "advantage" for women

Female:Male HR (95% CI)	NAFLD	Control
Any incident CV event	0.94 (0.80-1.11)	0.77 (0.69-0.85)

Allen A, et al. AASLD 2017. Abstract 55.

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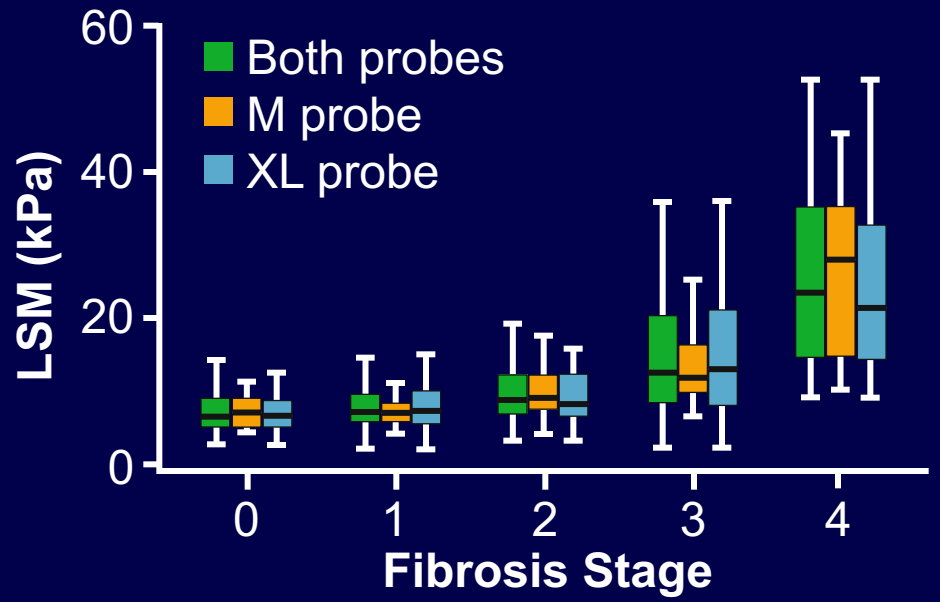
Diagnostic Performance of *FibroScan* for Assessing Liver Stiffness in NAFLD

- Multicenter, prospective study evaluating diagnostic performance of *FibroScan* for liver stiffness measurement in pts undergoing biopsy for suspected NAFLD (N = 374 analyzed)
- Fibrosis (biopsy): F0, 17%; F1, 23%; F2, 23%; F3, 29%; F4, 9%

***FibroScan* LSM Correlation With Fibrosis Stage**



AUROC (95% CI)		
≥ F2	≥ F3	≥ F4
0.77 (0.72-0.82)	0.80 (0.75-0.84)	0.89 (0.84-0.93)

- By multivariate analysis, only factor significantly influencing LSM was fibrosis stage

Eddowes P, et al. AASLD 2017. Abstract 184. Reproduced with permission.

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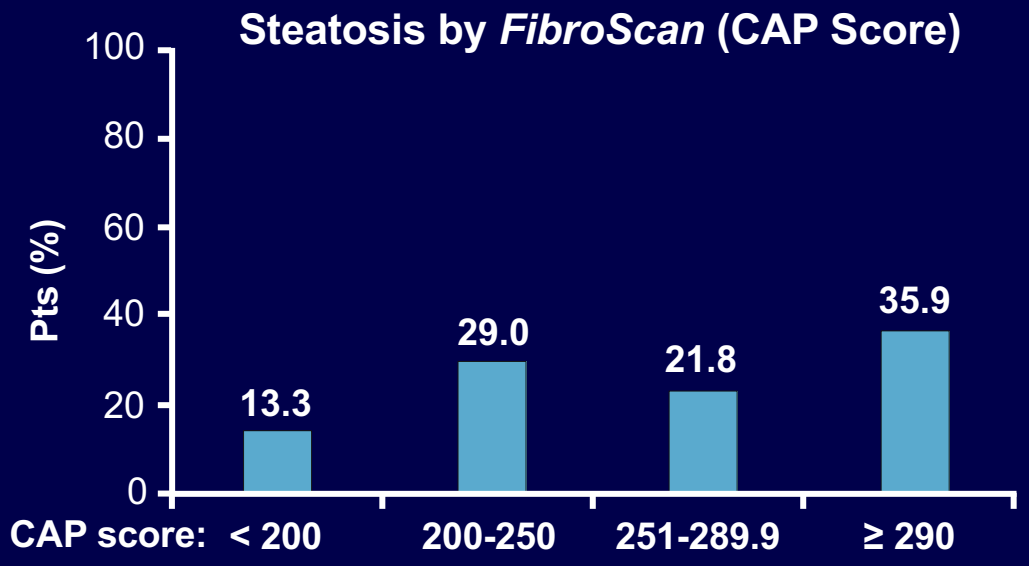
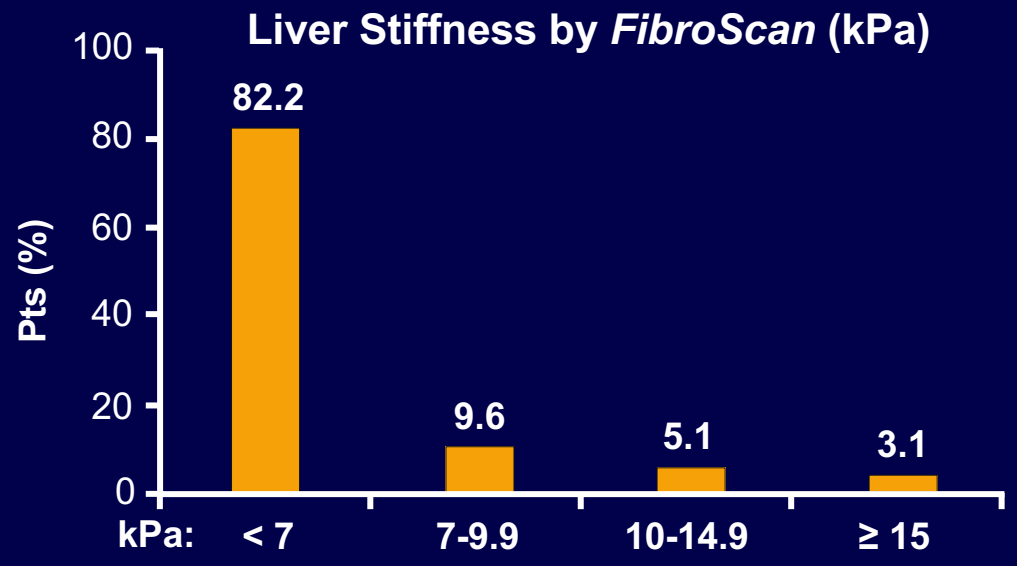
Assessment of Fatty Liver and Fibrosis Among Pts Attending Primary Care Clinic

- Single-center, *FibroScan*-based assessment of fatty liver and liver fibrosis in pts without liver disease history (N = 958)
  - Pts offered free *FibroScan*; most evaluated pts were female (64%), Hispanic ethnicity (85%), and above normal BMI (overweight, 36%; obese, 24%; morbidly obese, 19%)





- In pts with CAP ≥ 290 and complete NAFLD/NASH workup, diagnostic performance of *FibroScan* CAP and LSM will be compared with biopsy results

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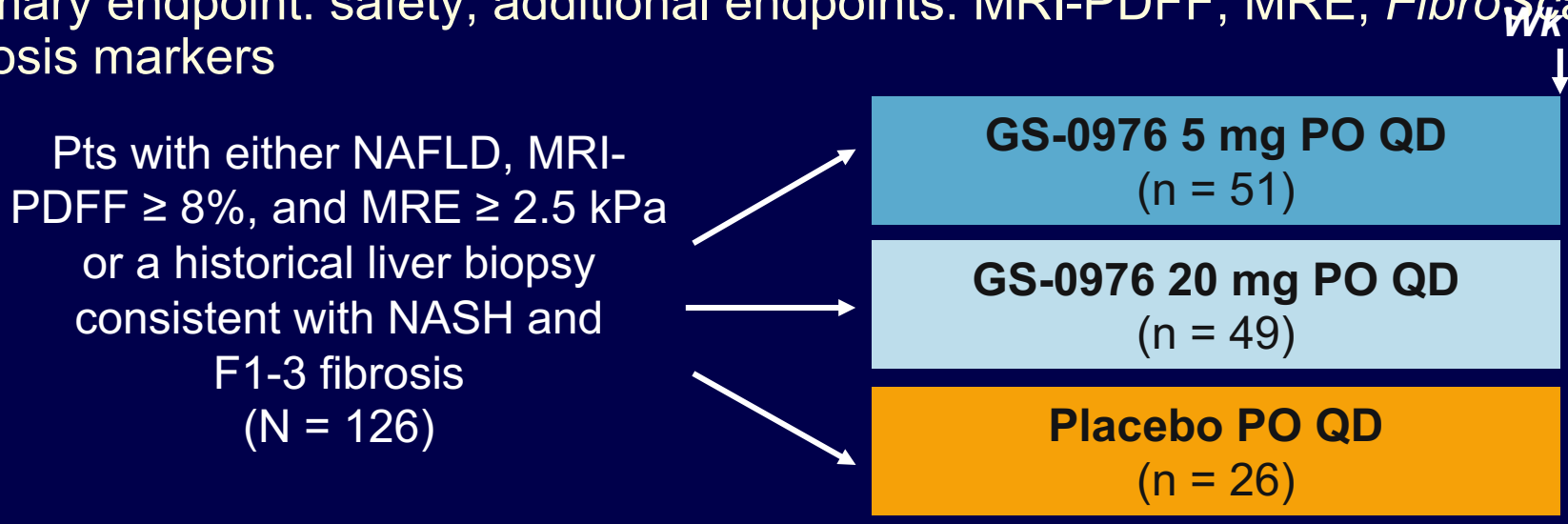
GS-0976: Acetyl-CoA Carboxylase Inhibitor in Pts With NASH

- No agents currently approved for treating NASH; numerous agents in phase II/III studies
- GS-0976: randomized, double-blind, placebo-controlled phase II study
  - GS-0976: liver-targeted inhibitor of acetyl-CoA carboxylase, which catalyzes rate-limiting step of de novo lipogenesis
  - Primary endpoint: safety; additional endpoints: MRI-PDFF, MRE, *FibroScan*, and serum fibrosis markers



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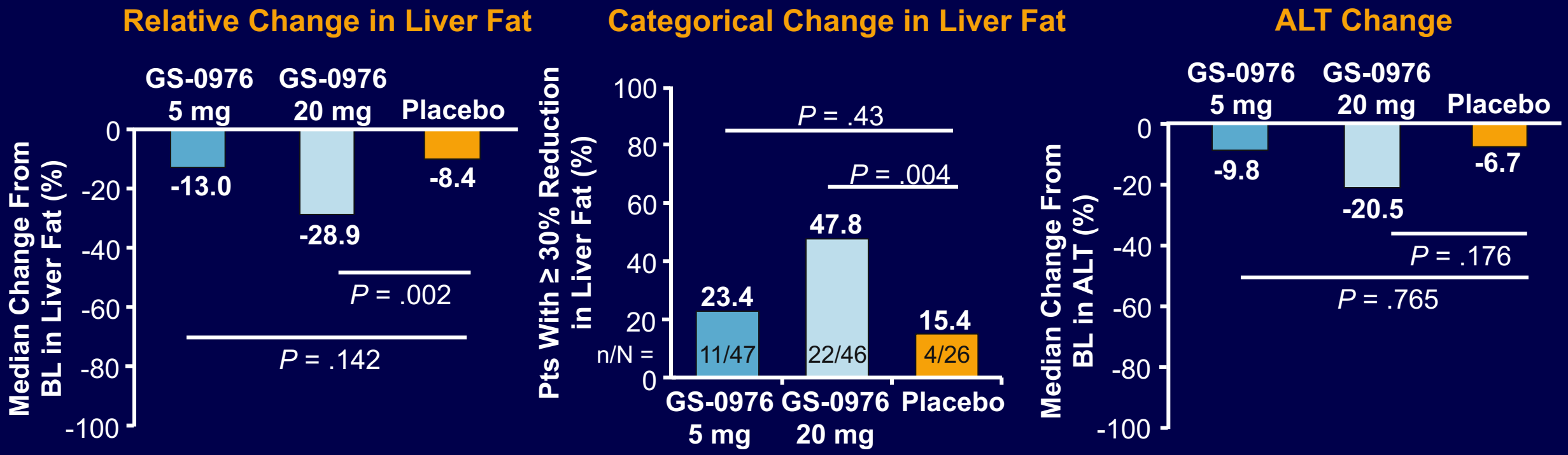
GS-0976: Key Efficacy Findings at Wk 12

- Statistically significant decrease in liver fat content with 20 mg, but not 5 mg, vs placebo by MRI-PDFF; no statistically significant decrease in ALT vs placebo



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GS-0976: Safety

Safety Event	GS-0976 5 mg (n = 51)	GS-0976 20 mg (n = 49)	Placebo (n = 26)
AE, n (%)	36 (71)	35 (71)	16 (62)
Serious AE, n (%)	2 (4)	2 (4)	0
Discontinuation for AE, n (%)	2 (4)	0	0
Median relative change in TG, %	13	11	-4
Asymptomatic grade 3/4 TG elevation, n (%)	9 (18)	7 (14)	0
▪ Grade 3 (> 500-1000 mg/dL)	5 (10)	5 (10)	0
▪ Grade 4 (> 1000 mg/dL)	4 (8)	2 (4)	0

- Grade 3/4 TG elevation predicted by BL TG > 250 mg/dL ($P < .001$)
- Of 16 pts with grade 3/4 TGs, n = 11 lowered TGs to < 500 mg/dL at Wk 12 (response to fibrate or fish oil, n = 4; resolution without treatment or study drug cessation, n = 7)

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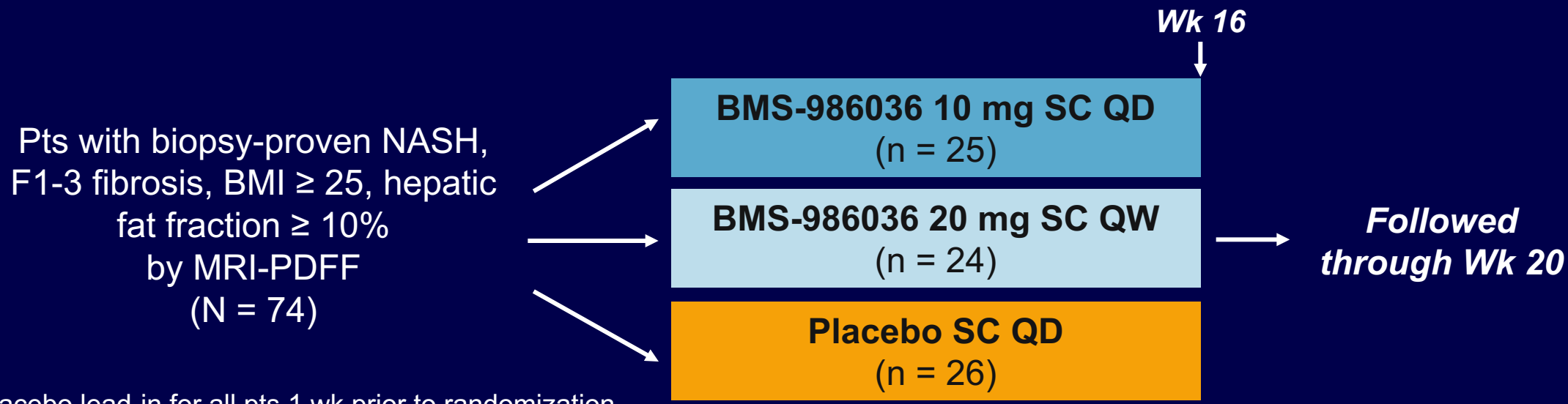
BMS-986036 in Pts With NASH, F1-3 Fibrosis

- Multicenter, randomized, double-blind, placebo-controlled phase II study
  - BMS-986036: pegylated analogue of metabolism regulator FGF21
  - Primary endpoint: absolute change in hepatic fat fraction



Placebo lead-in for all pts 1 wk prior to randomization.
Planned N = 90; enrollment ended early due to significant effect of BMS-986036 on primary endpoint in preplanned interim analysis at Wk 8.

Sanyal A, et al. AASLD 2017. Abstract 182.

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BMS-986036 for Pts With NASH: Key Efficacy Findings

- Significant reduction in liver fat content vs placebo by MRI-PDFF at Wk 16

Endpoint, n (%)	BMS-986036		Placebo (n = 25)
	10 mg QD (n = 23)	20 mg QW (n = 22)	
Absolute change in hepatic fat fraction from baseline, % (*P* value vs placebo)	-6.8 (< .001)	-5.2 (< .01)	-1.3
Pts with ≥ 30% reduction in hepatic fat fraction, % (*P* value vs placebo)	56 (.02)	54 (.03)	24

- Improvements in ALT and AST with BMS-986036 vs placebo through Wk 16

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BMS-986036 for Pts With NASH: Key Safety Data

- HDL levels improved from BL with BMS-986036 QD and QW vs no meaningful changes from BL with placebo; BMS-986036 QD only arm to show mean reduction in LDL from BL
- No deaths, treatment-related serious AEs, or AE-related discontinuations

Event, n (%)	BMS-986036		Placebo (n = 26)
	10 mg QD (n = 25)	20 mg QW (n = 24)	
Serious AEs	1 (4)	1 (4)*	1 (4)
AEs in > 10% of pts			
▪ Diarrhea	3 (13)	5 (22)	2 (8)
▪ Nausea	4 (16)	3 (13)	2 (8)
▪ Frequent bowel movements	5 (20)	0	0
Grade 3 lab abnormalities†	1 (4)	2 (8)	2 (8)

*Pt randomized in error; received small amount of 20 mg BMS-986036 on Day 1, should have received placebo.
†ALT elevation, n = 4; glucose elevation, n = 1 (in 20 mg QW arm).

Sanyal A, et al. AASLD 2017. Abstract 182.

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