Disclaimer

Certain statements contained in this presentation and in the accompanying oral presentation as well as subsequent discussion (if any) may constitute forward-looking statements. Examples of such forward-looking statements include statements regarding Ascletis' research and discovery, pre-clinical and clinical programs and plans of candidate drugs, the conduct of clinical trials and expected data readouts, planned commercial product launches, the advancement of and anticipated clinical development, regulatory milestones and commercialization of Ascletis' medicines and drug candidates. Actual results may differ materially from those indicated in the forward-looking statements as a result of various important factors, including Ascletis' ability to demonstrate the efficacy and safety of its drug candidates; the clinical results for its drug candidates, which may not support further development or marketing approval; actions of regulatory agencies, which may affect the initiation, timing and progress of clinical trials and marketing approval; Ascletis' ability to achieve commercial success for its marketed medicines and drug candidates, if approved; Ascletis' ability to obtain and maintain protection of intellectual property for its medicines and technology; Ascletis reliance on third parties to conduct drug development, manufacturing and other services; Ascletis' limited experience in obtaining regulatory approvals and commercializing pharmaceutical products and its ability to obtain additional funding for operations and to complete the development and commercialization of its drug candidates and achieve and maintain profitability; the impact of the COVID-19 pandemic on Ascletis' clinical development, regulatory, commercial and other operations, as well as those risks discussed in the section entitled "Major Risk Factors, Uncertainties and Risk Control" in Ascletis' most recent Annual Report filed with the Hong Kong Stock Exchange. The performance and results achieved by Ascletis in this presentation and in the accompanying oral presentation as well as subsequent discussion (if any) are historical in nature, and past performance is no guarantee of the future results.

The information, statements and opinions contained in this presentation and in the accompanying oral presentation as well as subsequent discussion (if any) do not constitute an offer to sell or solicitation of any offer to subscribe for or purchase any securities or other financial instruments or any advice or recommendation in respect of such securities or other financial instruments in any jurisdiction. In particular, this presentation is not an offer of securities for sale nor a solicitation of an offer to buy securities.

This presentation is provided for investment purposes only. All information in this presentation is as of the date of this presentation, and Ascletis undertakes no duty to update such information unless required by law, and no liability in the event that any of the forward-looking statements or opinions do not materialize or turn out to be incorrect.

This presentation and the accompanying oral presentation contain data and information obtained from third-party studies and internal company analysis of such data and information. Ascletis has not independently verified the data and information obtained from these sources. Forward-looking information obtained from these sources is subject to the same qualifications noted above.

GANNEX

A Member of the Ascletis Group

Corporate Presentation

Gannex Pharma Inc.

Dec 2023

About Gannex

Gannex, a wholly-owned company of Ascletis Pharma(Hong Kong listed public biotech, HK.1672), is dedicated to the R&D and commercialization of new drugs in the field of NASH and PBC. Gannex has two clinical stage NASH drug candidates with global rights against two different targets – THRβ and FXR and one clinical stage PBC drug candidate

NASH/PBC Clinical Pipeline

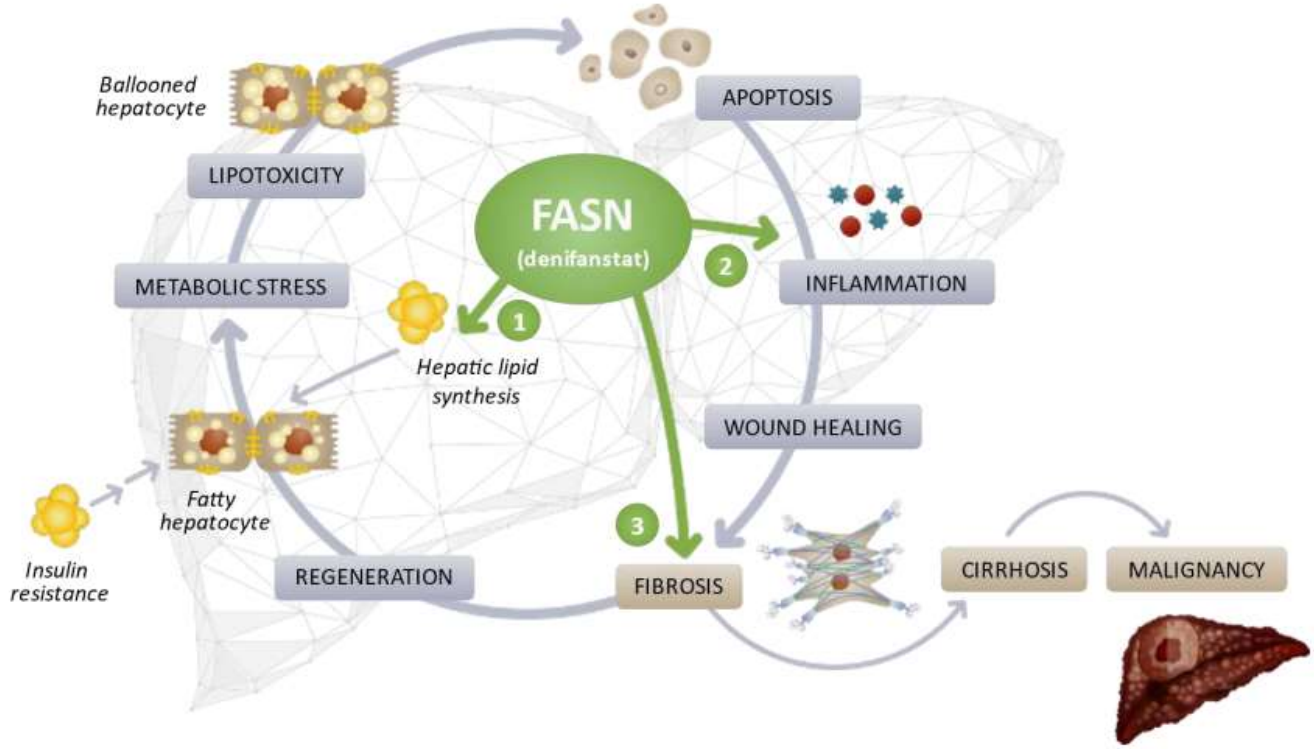
Target	Candidate	Commercial rights	Pre-IND	IND	Phase I	Phase II	Phase III	■ Competitiveness and next catalysts
FASN	ASC40 (NASH)	Great China	*U.S. FDA Fast Track*					■ First-in-class FASN inhibitor for NASH treatment ■ Phase IIb biopsy readouts expected in 1Q2024
THRβ	ASC41 (NASH)	Global	*52 weeks, Biopsy*					■ Potentially second-in-class THRβ agonist to market after resmetirom (Madrigal) ■ Interim 12-week liver fat reduction (MRI-PDFF) and fibrosis biomarkers expected in 4Q2023
THRβ + FXR	ASC43F FDC (NASH)	Global						■ First-in-class, dual targets to THRβ and FXR ■ 52 week Phase IIa/IIb in biopsy confirmed NASH patients authorized by FDA
FXR	ASC42 (PBC)	Global						■ Low pruritus rate after 12-week treatment (10mg, QD) ■ Phase II data expected by the end of 2023

ASC40 NASH

ASC40 (Denifanstat) : Differentiated Mechanism Believed to Target Key Drivers of NASH

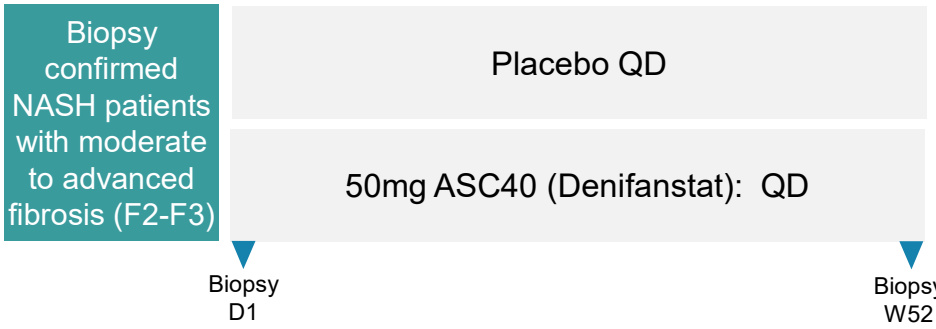
Denifanstat has independent mechanisms designed to:

1. Block **steatosis** via inhibiting de novo lipogenesis in hepatocytes
2. Reduce **inflammation** via preventing immune cell activation
3. Blunt **fibrosis** via inhibiting stellate cell activation



ASC40 (Denifanstat) Phase IIb Clinical Trial Design

FASCINATE-2 Phase IIb trial design



Primary Endpoints

- *≥ 2-point improvement in NAS (Nonalcoholic fatty liver disease (NAFLD) Activity Score) that results from reduction of necro-inflammation (inflammation or ballooning), or*
- *improvement in fibrosis.*

- *Biopsy confirmed F2-F3 NASH patients • 52 weeks, 2:1 50mg or placebo, double-blind • Fully enrolled: 168 patientsin U.S., Canada, and Europe • Prespecified interim analysis of the first 52 patients with MRI-PDFF >8%*

Secondary Endpoints

- *Improvement in liver fibrosis ≥1 stage without worsening of NASH (Bx) • Digital AI pathology • Interim MRI-PDFF: absolute decrease, % change from baseline, % pts ≥30% (responders)*

*A baseline signature of metabolites involving the gut-liver axis predicts MRI-PDFF response to FASN inhibitor TVB-2640: results from the FASCINATE-1 study, The European Association for the Study of the Liver (EASL) 2022, June 25, 2022. Virtual Conference.

Interim Data from Phase IIb Clinical Trial: 67% of Patients Reduced Liver Fat by More Than 30%

ASC40 50 mg (n=30)	ASC40 50 mg (n=30)	Placebo (n=22)	P-value vs placebo
Relative reduction in liver fat	*- 34.1%*	*- 1.5%*	*p<0.001*
≥30% reduction of liver fat (responder rate)	*67%*	*18%*	*p<0.01*
ALT (U/L)	*- 16.5*	*- 4.0*	*p<0.05*
Dual liver fat & ALT responder >30% + >17U/L decrease	*37.0%*	*9.0%*	*p<0.05*
PRO-C3	*– 8.2%*	*-1.5%*	*p<0.05*
Enhanced liver fibrosis (ELF) score*	*- 0.34*	*- 0.02*	*p<0.05*
LDL cholesterol (mg/dL)	*-12.4*	*0.0*	*p<0.05*
FGF21	*+73.1%*	*+ 0.9%*	*p<0.01*

**approximately half of denifanstat responders decreased liver fat by ≥50%*

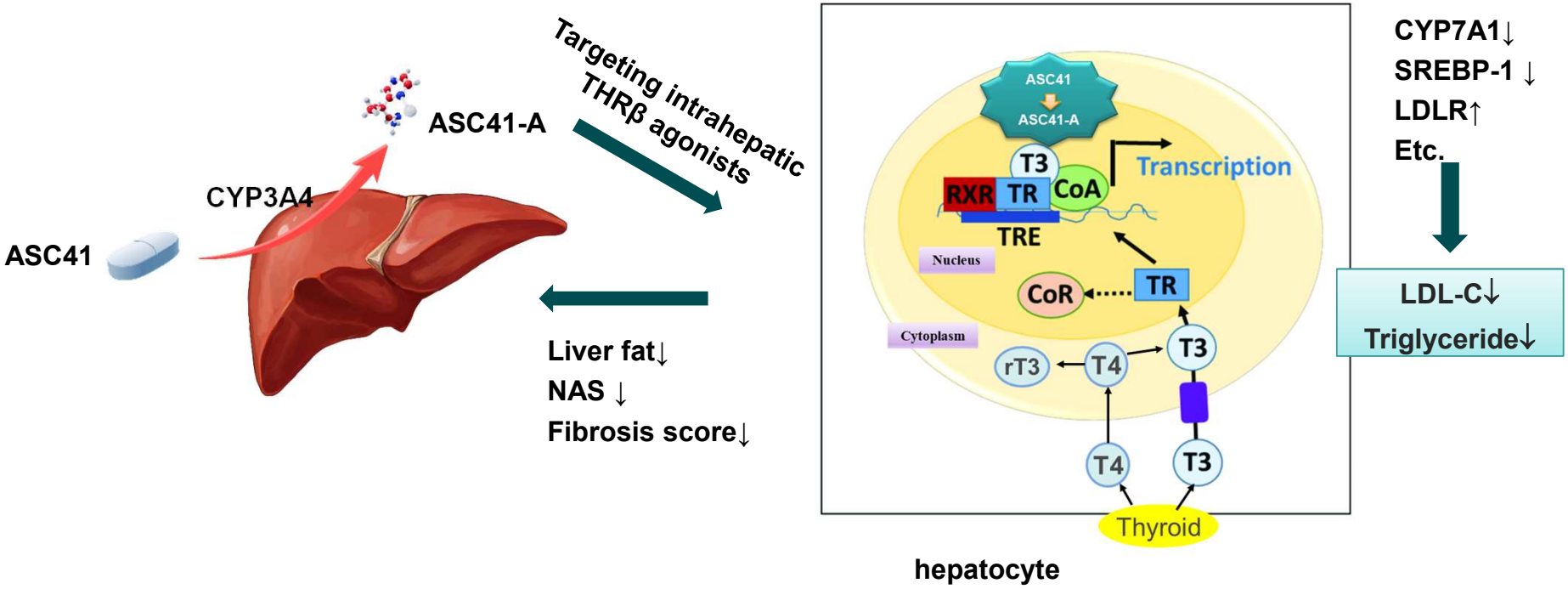
■ **Phase IIb biopsy readouts expected in 1Q2024**

Sagimet Biosciences Presents Positive Phase 2b FASCINATE-2 Clinical Trial Interim Data for Denifanstat for the Treatment of NASH at EASL Congress 2023 - Sagimet Biosciences

ASC41 NASH

ASC41: A Liver Targeting Thyroid Hormone Receptor Beta (THRβ) Agonist

- ASC41 is a liver targeted small molecule which is converted to its active metabolite ASC41-A - a potent and selective THRβ agonist



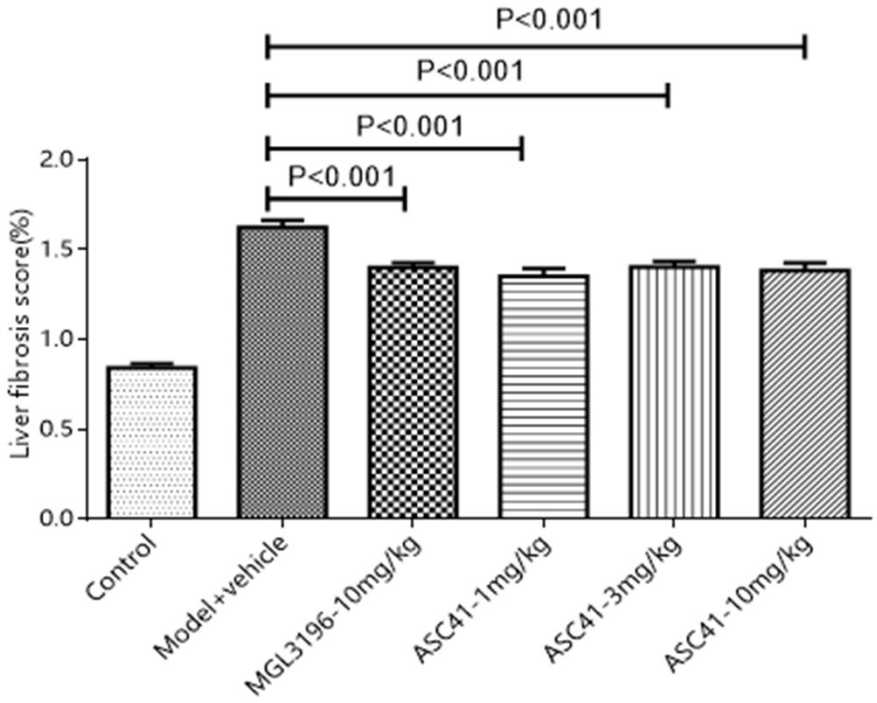
Source: Gannex data

ASC41: A Potential Second-in-Class THRβ Agonist

- ASC41 is a liver-targeted prodrug, and its active metabolite is a selective THRβ agonist.
- In two NASH animal models, at 1/10 dose of MGL-3196, ASC41 demonstrated the same improvement of liver steatosis, inflammation and fibrosis.
- Commercially ready oral tablet formulation developed with in-house proprietary technology
- 2 Phase I completed
  - Single doses (1, 2, 5, 10, 20 mg) and 14 day multiple doses (1, 2, 5 mg) in 65 subjects with elevated LDL-C > 110 mg/dL
  - Food effect in 12 healthy subjects
- 2 US bridging studies completed: no significant difference in drug exposure among Chinese and Americans
- 1 Phase Ib study completed
  - 28 day, 10 mg in 20 overweight and obese subjects with elevated LDL-C > 110 mg/dL
- Based on above studies, 2 mg and 4 mg once-daily doses have been selected for a 52-week Phase II trial in biopsy-confirmed NASH patients
  - First patient dosed in Oct, 2022

Source: 1. Jinzi J. Wu et al, AASLD 2021 abstract 1851; 2. Jinzi J. Wu et al, EASL 2021 abstract PO-1851;

ASC41 in HFD+CHOL Mouse Model Decreased Liver Fibrosis



- MGL-3196 is a THR-beta agonist from Madrigal
- HFD + CHOL: 60% high fat + 1.25% cholesterol + 0.5% cholate.

Conclusions

◆ ASC41's 1 mg/kg group lowered liver fibrosis score by 25%.

Phase 1 Results of ASC41, Significant Lipid Lowering

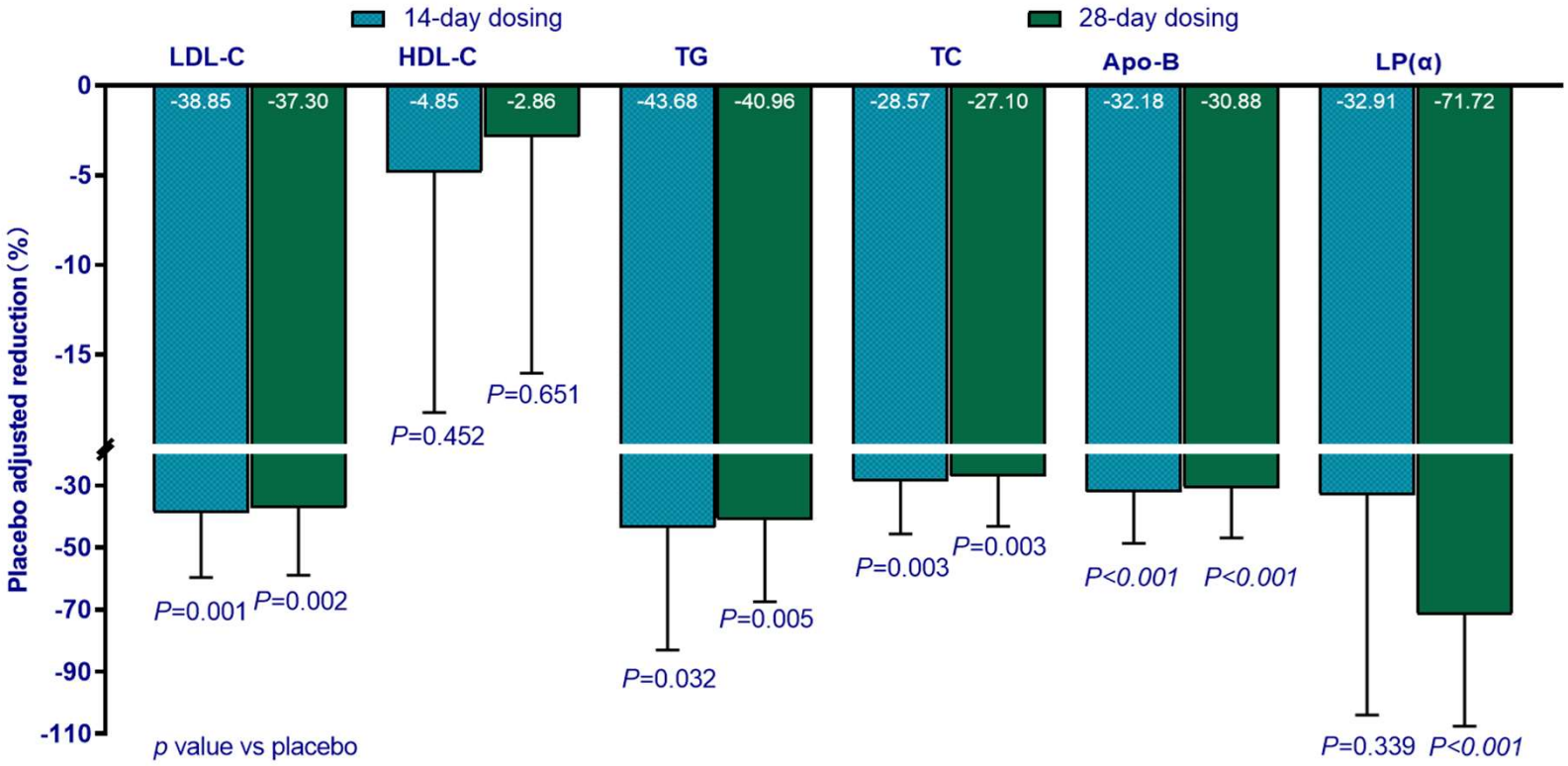
Placebo-adjusted relative change (mean) from baseline after 14 days of once daily oral dosing of ASC41 tablets

	1 mg (n=12)	2 mg (n=12)	5 mg (n=12)
Placebo-adjusted LDL-C reduction P-value vs placebo	-0.42% p=0.947	-11.94% p=0.052	-19.99% p=0.002
Placebo-adjusted triglyceride reduction P-value vs placebo	-39.43% p=0.002	-31.06% p=0.029	-34.49% p=0.015
Placebo-adjusted TC reduction P-value vs placebo	-1.48% p=0.766	-8.53% p=0.142	-10.71% p=0.030
Placebo-adjusted HDL-C reduction P-value vs placebo	8.11% p=0.135	-2.54% p=0.668	-0.22% p=0.962

Source: Wu J.J et al EASL 2021, abstract #1851

Positive Phase Ib Results in Overweight and Obese Subjects

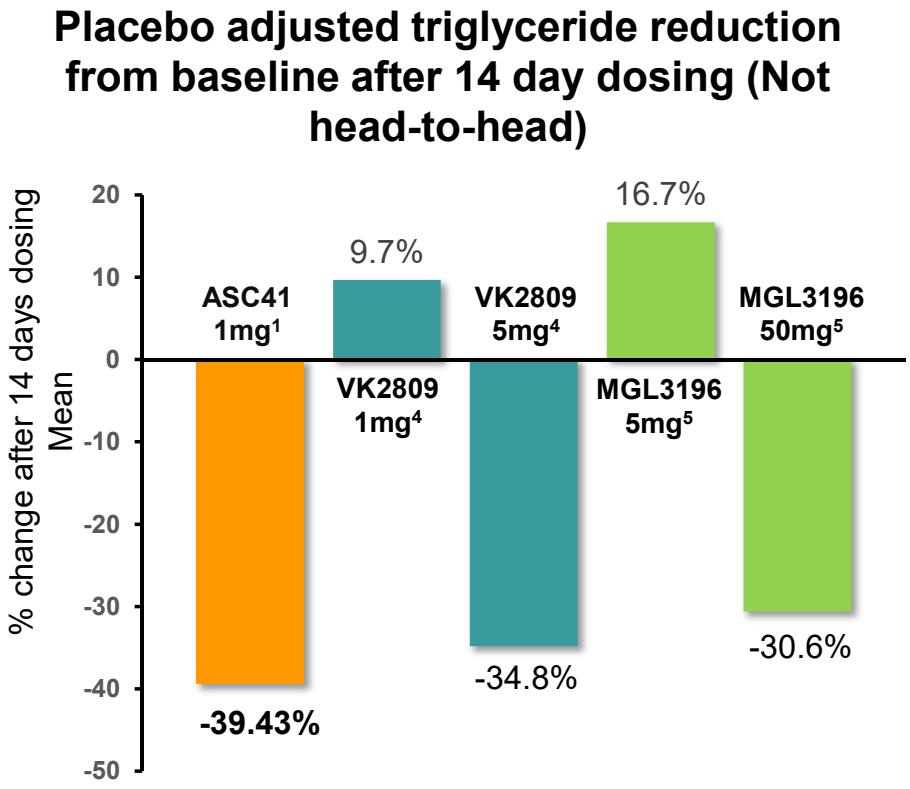
Placebo-adjusted relative change (mean) from baseline after 14 or 28 days of once daily oral dosing of 10 mg ASC41 tablets in overweight and obese subjects



Source: A Phase Ib Study to Evaluate the Safety, Tolerability and Pharmacokinetics of ASC41, a THR-β Agonist, for 28-days in Overweight and Obese Subjects with Elevated LDL-C, a Population with Characteristics Of NAFLD. 72th American Association for the Study of Liver Diseases (AASLD)& The Liver Meeting ®, November 12-15, 2021. Virtual Conference.

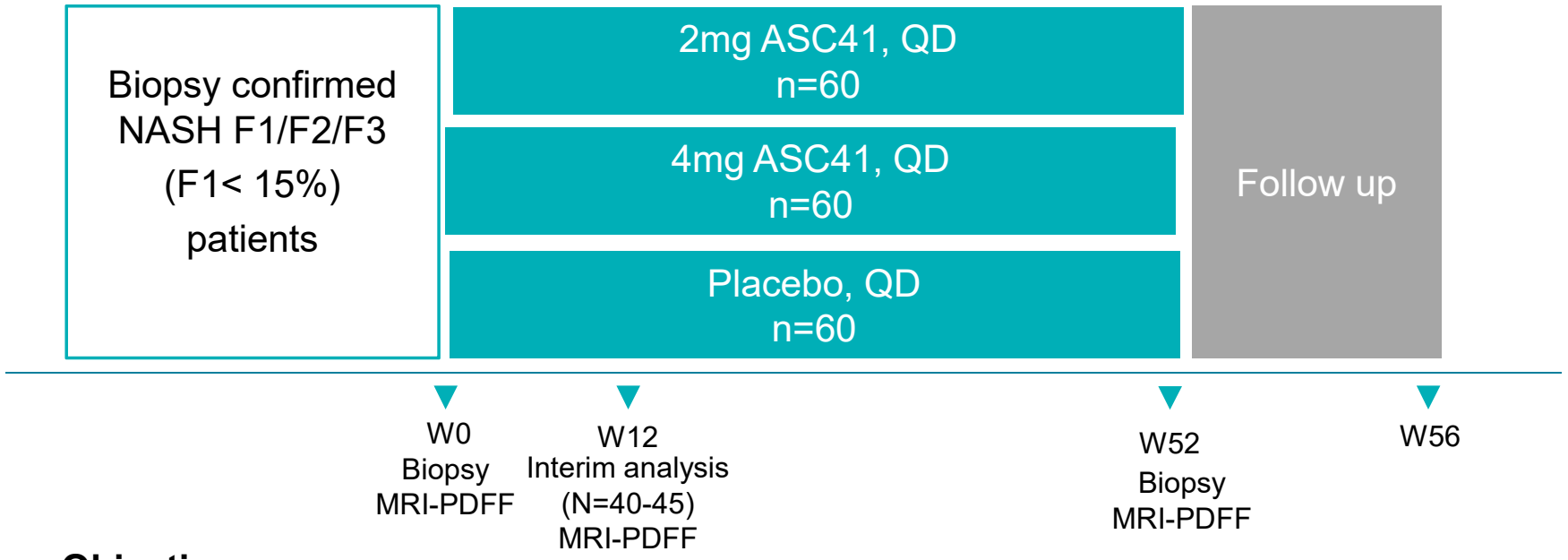
THRβ Differentiations: Gannex VS Viking and Madrigal

	Gannex ASC41[1]	Viking VK2809[2,3]	Madrigal MGL3196[4]
Oral formulation	Tablet, room temp storage, commercially ready	Capsule, refrigerated	Tablet, room temp storage, commercially ready
Dosing frequency	Once a day	Once every two days	Once a day
DDI	-	+	-
Human dose needed for > 30% TG reduction[5,6]	1 mg	2.5 mg	50 mg



Source: 1.EASL 2021 Abstract No. PO-1851 2.EASL2020 Abstract No. AS073. 3. VK2809-202: Informed Consent Form, Iowa Diabetes and Endocrinology Research Center. 4.Stephen A Harrison et al. Resmetirom (MGL-3196) for the treatment of non-alcoholic steatohepatitis: a multicentre, randomised, double-blind, placebo-controlled, phase 2 trial. www.thelancet.com Published online November 11, 2019 https://doi.org/10.1016/S0140-6736(19)32517-6 5.VK2809 data presented at the 2016 Meeting of the American College of Cardiology 6.Taub et al. Lipid lowering in healthy volunteers treated with multiple doses of MGL-3196, a liver-targeted thyroid hormone receptor-b agonist. Atherosclerosis 230 (2013) 373e380

ASC41: Phase II Ongoing Study for NASH in China



Primary Objective

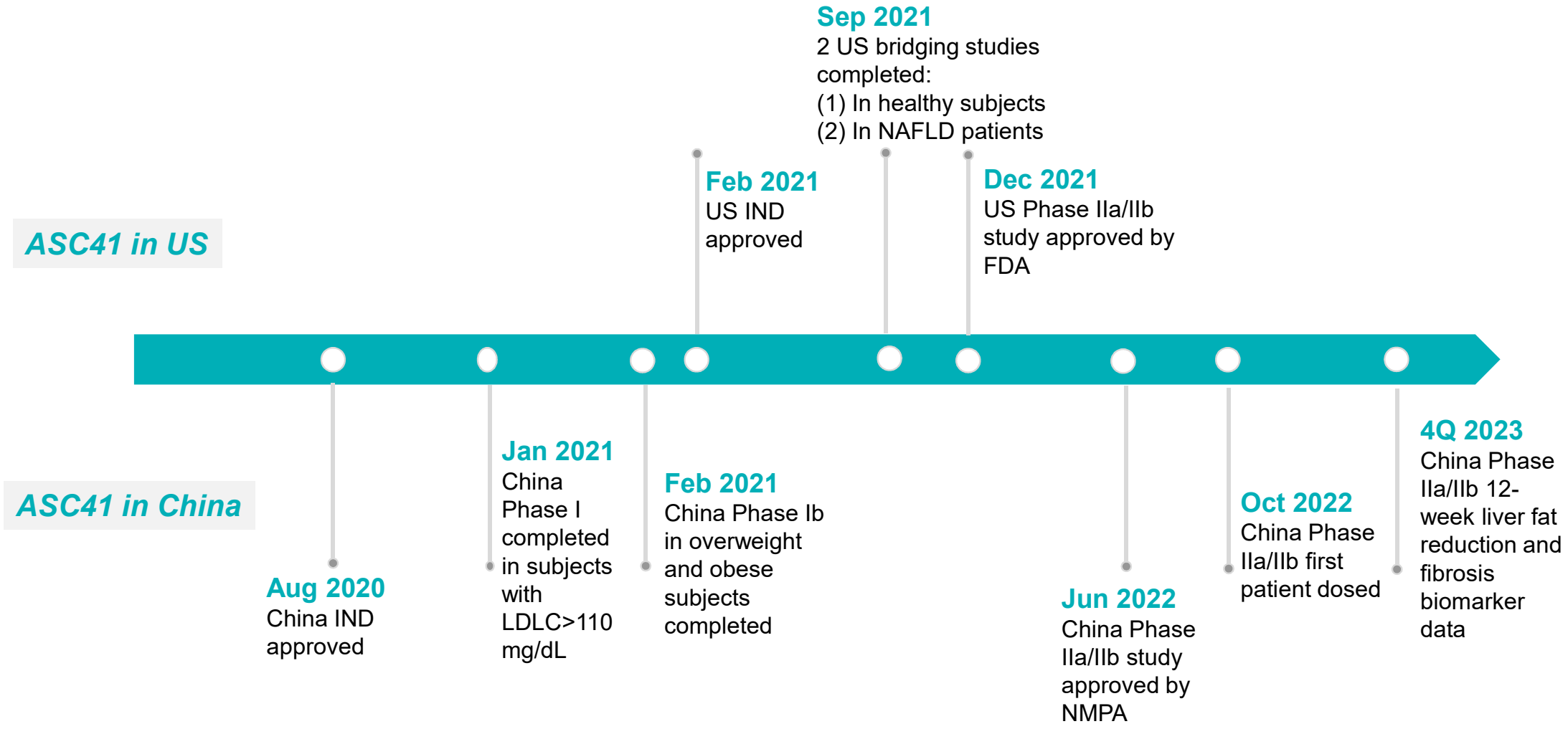
To evaluate the effect of ASC41 compared with placebo in noncirrhotic patients with NASH and F1, F2, and F3 fibrosis at Week 52 by a histological reduction in NAS ≥ 2 points that results from reduction of necroinflammation (inflammation or ballooning) without worsening fibrosis.

Secondary objectives

1. NASH resolution; 2. Fibrosis improvement.

First biopsy-confirmed patient dosed in Oct, 2022. Interim analysis (MRI-PDFF etc) expected in 4Q2023

ASC41 NASH Timeline Till 2023

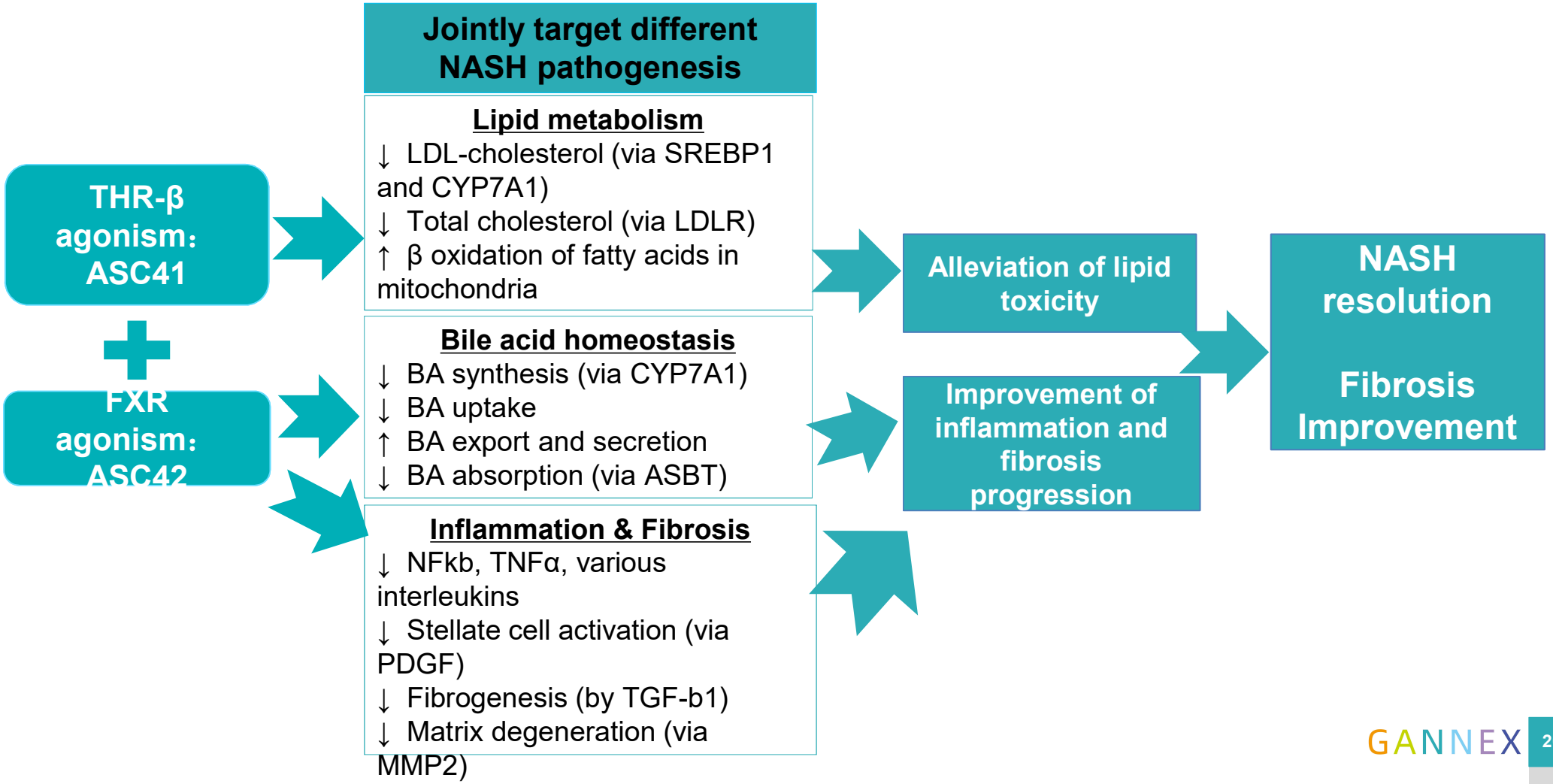


ASC41 vs VK2809 vs Resmetirom (MGL3196) in US: Strong patent protection

Category	Ascletis: ASC41		VIKING: VK2809		Madrigal: MGL-3196	
Crystal Patent	YES	Publication No. :WO2022067602A1 Filing Date: 2020-09-30	YES	Publication No. :US20210024554A1 Filing Date : 2019-03-18	YES	1.Patent No:US9266861B2 Filing Date:2013-09-17 2.Publication No. :US20210122740A1 Filing Date:2019-07-02
Formulation Patent	YES	1.Publication No. : US20210308155A1 Filing Date :2021-3-25 2.Patent No.: US11583502B2 Filing Date: 2021-4-16	NO	No published patent has been searched	NO	No published patent has been searched
Synthesis Patent	YES	Patent No. : US11292805B2 Filing Date: 2021-4-15	NO	No published patent has been searched	YES	Patent No. : US9266861B2 Filing Date：2013-09-17
Compound Patent	NO Irrelavant	-	YES Irrelavant	Patent No. : US7829552B2 Expiration Date: 2026-10-20	YES irrelavant	Patent No. : US7452882B2 Expiration Date: 2026-09-12
Method-Use Patent	NO	-	YES	1.Patent No. : US10130643B2 Filing Date：2006-05-26 2.Patent No. : US10925885B2 Filing Date：2006-05-26 3.Patent No. : US11202789B2 Filing Date：2017-11-17 4.Publication No. : US20200179412A1 Filing Date：2018-06-04 5.Publication No. US20220016137A1 Filing Date：2019-12-04	YES	1.Patent No. : US9968612B2 Filing Date：2013-09-17 2.Patent No. : US11090308B2 Filing Date：2017-10-18 3.Patent No. : US20210330675A1 Filing Date：2021-07-07

ASC43F (ASC41+ASC42)
NASH

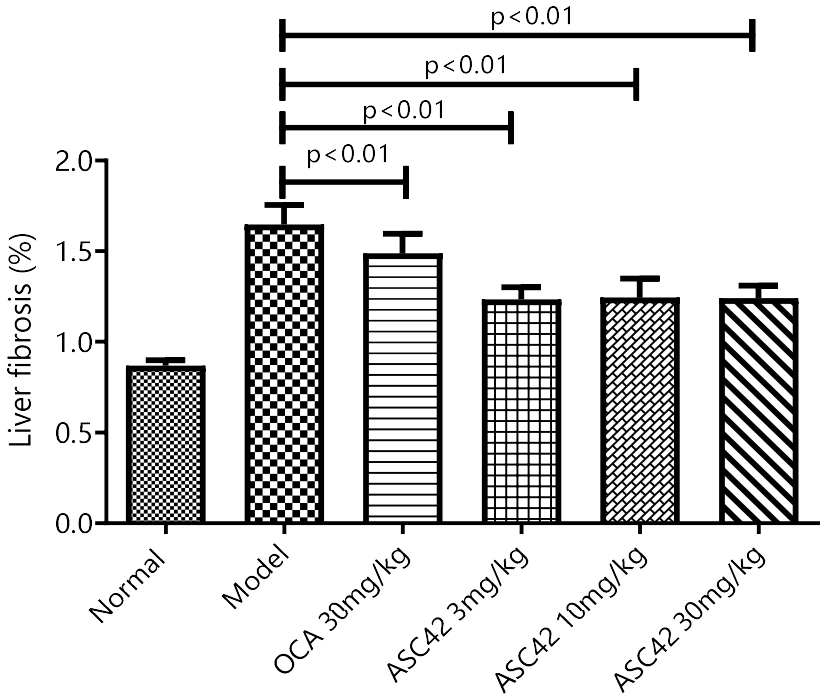
Mechanism of ASC43F(ASC41+ASC42) against NASH



ASC43F- First-in-Class Dual Targeting (THRβ +FXR) Fixed-dose Combination

- An in-house developed, first-in-class dual targeting fixed-dose combination (FDC) of ASC41 (a THRβ agonist) and ASC42 (an FXR agonist)
- THRβ agonism has demonstrated both NASH resolution and fibrosis improvement, while FXR agonism has shown primarily anti-fibrotic, as well as anti-inflammatory effects. The combination of ASC41 and ASC42 may complement the advantages of these two agents
- U.S. FDA IND approval in November 2021
- U.S. Phase I trial completed in January 2022
  - ASC43F was safe and well tolerated in healthy volunteers
  - PK parameters of ASC41 and ASC42 from ASC43F are similar to those of ASC41 and ASC42 monotherapy
- 52-week phase IIa/IIb in biopsy-confirmed NASH patients approved by US FDA

ASC42 in DEN+HFD Rat Model



- DEN, diethylnitrosamine to promote liver fibrosis
- HFD, high fat diet that contains 60KCal% fat
- OCA, obeticholic acid

■ ASC42 at the dose range of 3~30 mg/kg significantly inhibited the progress of liver fibrosis (both p<0.01), among which ASC42's 30 mg/kg group lowered cirrhosis by 28.0%.

Source: 1. Jinzi J. Wu et al, AASLD 2021 abstract 1851; 2. Jinzi J. Wu et al, EASL 2021 abstract PO-1851;

ASC42 Phase I Study Results

Dose	5 mg QD	15 mg QD	50 mg QD
Incidence rate of pruritus during 14 days treatment (%)	0	0	57
AUC on Day 14 (ng.h/mL)	196	1752	10970
Safety margin for pruritus versus 50 mg (fold)	56	6.3	NA
LDL-C change from baseline on Day 14 (%, Median)	-6.6	2.43	-14.8
FGF19 on Day 14 versus baseline (%)	471	1780	3970
C4 reduction on Day 14 (%)	53	91	96
ALT change from baseline on Day 14 (U/L, Median)	-1.0	-2.5	3

Doses ranging between 5 and 15 mg were selected for the Phase II studies based on the study data from this Phase I study, including the fact that there were no pruritic events after 14 days of dosing associated with these doses.

Source: Jinzi J. Wu et al, AASLD 2021 abstract 1854;

Improvement of PBC biomarkers FGF19 and C4 by ASC42 versus Ocaliva

Biomarker change relative to baseline	ASC42 (QD, 14 days) (ASC42-I-CTP-01)[1]		OCA (QD, 21 days) (NCT01625026)[2,3]
Dosage	5mg	15mg	25mg
FGF19: relative to baseline %	471%	1780%	147%
C4: inhibition %	53%	91%	91%
Side effect: Pruritus	0	0	70% (10mg)

Conclusion: ASC42 is potentially better or comparable in treatment of PBC could be to that of Ocaliva with less side effect of pruritus.

Source: 1. Jinzi J. Wu et al, AASLD 2021 abstract 1854; 2. Al-Dury, S., et al.[J] J Hepatol, (2019).DOI: 10.1016/j.jhep.2019.06.011;3. Kowdley, K. V., et al.[J] Hepatology, (2018).DOI: 10.1002/hep.29569

ASC42 Demonstrated Minimal Pruritis in a 12 Week HBV Phase II Study

DESIGN

- ASC42 10mg or 15mg qd + PEG-IFN α-2a 180μg QW+ETV 0.5mg qd
- 15 patients per treatment arm for 12 weeks

RESULTS

- In CHB patients, 12-week treatment of 10 mg ASC42 in combination of PEG-IFN-α-2a and ETV was safe and well-tolerated and showed minimum and mild pruritus (6.7%).

Comparison of incidences of pruritus of ASC42 with other FXR agonists and PEG-IFN α-2a

	ASC42 10 mg (N = 15)	Obeticholic Acid 10 mg (N = 653)	PEG-IFN α-2a 180 μg (N = 271)	Cilofexor 30 mg (N = 40)	Tropifexor 140 μg (N = 50)
Patient type	CHB	NASH	CHB	NASH	NASH
Treatment duration	12 weeks	18 months	48 weeks	48 weeks	48 weeks
Pruritus, number of patients (%)	1(6.7)	183 (28)	26 (10)	8 (20)	20 (40)

Source: Jinzi J. Wu et al, EASL abstract SAT-201

Phase I PK parameters of ASC43F versus monotherapy ASC42 and ASC41 in healthy volunteers

	ASC42 Tablet (5mg*3)	ASC41 Tablet (5mg*1)		ASC43F Tablet (ASC41 5mg + ASC42 15mg)		
	ASC42	ASC41	ASC41-A	ASC42	ASC41	ASC41-A
C_{max} (ng/mL)	363 (29.5)	4.69 (35.2)	23.9 (32.9)	254 (84.2)	3.60 (53.7)	28.4 (20.3)
T_{max} (h)	2.50	1.00	4.00	3.00	1.00	4.00
$t_{1/2}$ (h)	8.11 (22.9)	6.75 (33.2)	15.5 (26.8)	7.84 (36.9)	7.28 (74.3)	14.7 (14.2)
AUC_{0-t} (ng·h/mL)	1631 (27.0)	16.7 (54.9)	442 (52.9)	1580 (72.4)	20.8 (65.4)	527 (26.7)
AUC_{0-inf} (ng·h/mL)	1635 (26.9)	23.8 (37.5)	455 (53.2)	1584 (72.3)	23.8 (64.9)	546 (28.2)

Conclusion:

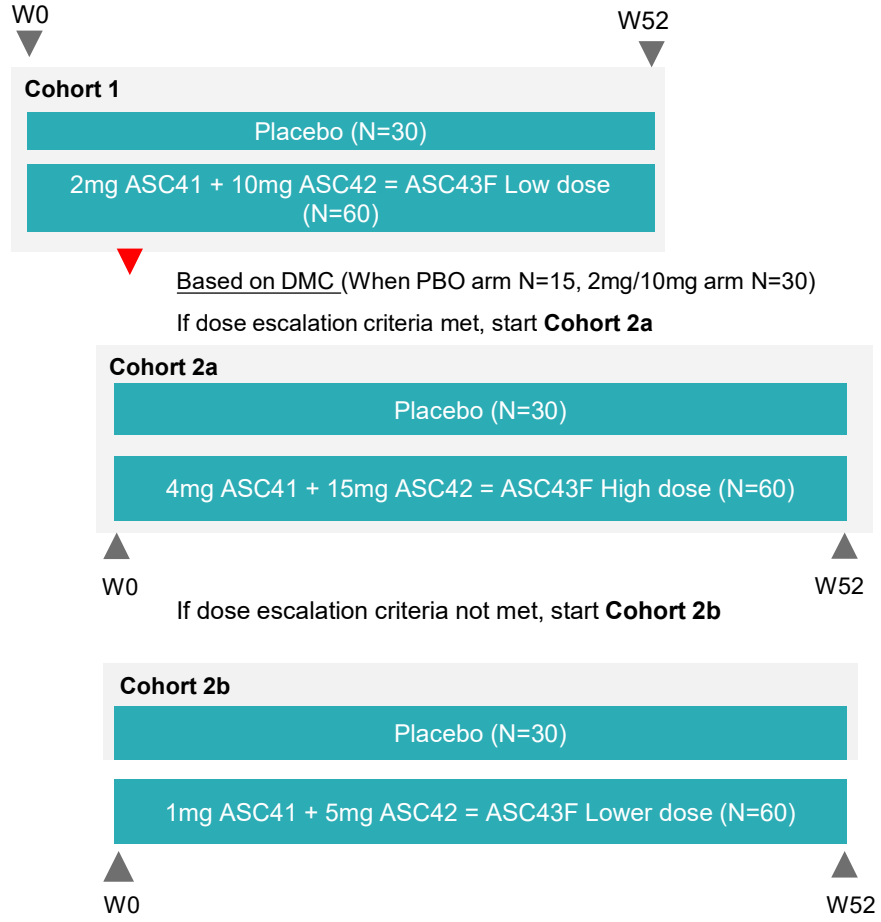
- Phase I study demonstrated that ASC43F showed good tolerability and safety profiles
- PK parameters of ASC41/ASC41A and ASC42 from ASC43F were similar to those of ASC41 and ASC42 as monotherapy.
- One-pill, once-a-day FDC for NASH treatment, thus should improve patient compliance

Source: Jinzi J. Wu et al, AASLD 2021 abstract 2314.

US FDA approved 52-week phase IIa/IIb in biopsy-confirmed NASH patients : ASC43F Trial Design

A Seamless, Phase IIa/IIb Double-blind, Randomized, Multicenter, Placebo-controlled, Study to Evaluate the Safety, Tolerability and Efficacy of ASC43F, a fixed dose combination (FDC) oral tablet, in Adults with NASH

Biopsy Confirmed
NASH F1, 2, 3
Total Enrollment
N=90-180



Primary Objective

To evaluate the effect of ASC43F compared with placebo in noncirrhotic patients with NASH and F1, F2, and F3 fibrosis at Week 52 by a histological reduction in NAS ≥ 2 points that results from reduction of necroinflammation (inflammation or ballooning) without worsening fibrosis.

Secondary objectives

1. NASH resolution; 2. Fibrosis improvement.

Published ASC42 Patents/Applications : Strong patent protection

Category	Filing Date	Publication No.	Patent Application	Countries Patent Authorized
Compound Patent and NASH/PBC use patent	2020-10-12	US20220388997A1	Globally	China
Use Patent： HBV	2020-10-12	US20230165843A1	Globally	China
Combo Patent (ACS41+ASC40)	2022-07-05	WO2023280150A1 （PCT）	Globally	-
Formulation Patent	2021-09-09	WO2023035181A1 （PCT）	Globally	-

ASC43F patent applications not published yet.

Gannex Has a Strong THRβ Portfolio for NASH

- ASC41: Oral, once daily drug candidate targeting THRβ
  - Positioned to be a second-in-class THRβ Agonist to market with best-in-class potential
  - Interim 12-week liver fat reduction and fibrosis biomarkers data expected in 4Q2023
  - US NDA filing expected 1H2029
- ASC43F: Oral, once daily fixed dose combination drug candidate targeting THRβ and FXR
  - Positioned to be a first-in-class fixed dose combination drug candidate to market
  - 52-week phase IIa/IIb protocol in biopsy-confirmed NASH patients approved by US FDA
  - US NDA filing expected 2H2030

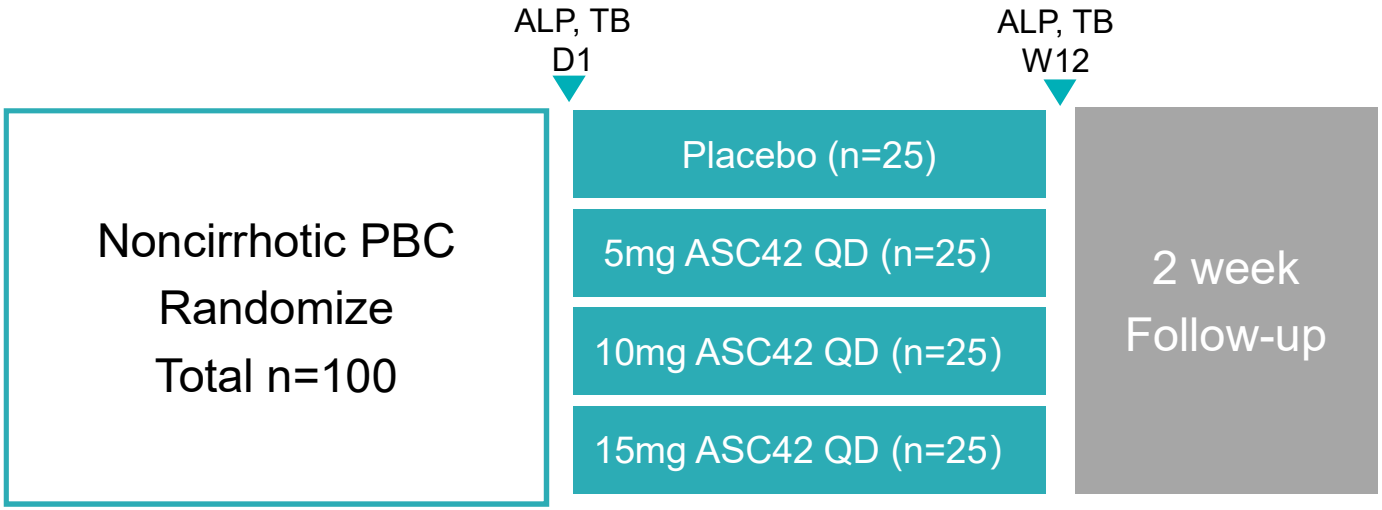
ASC42 FXR
For Primary Biliary Cholangitis (PBC)

ASC42: A Novel Non-steroidal, Selective, Potent FXR Agonist For PBC

- Potentially best-in-class, **Significantly less pruritus** at human therapeutic doses
- U.S. FDA IND approval
- U.S. FDA Fast Track Designation in NASH
- U.S. Phase I trials completed: Single ascending doses and multiple ascending doses, Food effect
- Phase II trial in HBV patients showed minimum and mild pruritus (6.7%).
- Phase II clinical trial for PBC topline results expected by the end of 2023

ASC42 PBC Phase II Study Design +/- UDCA

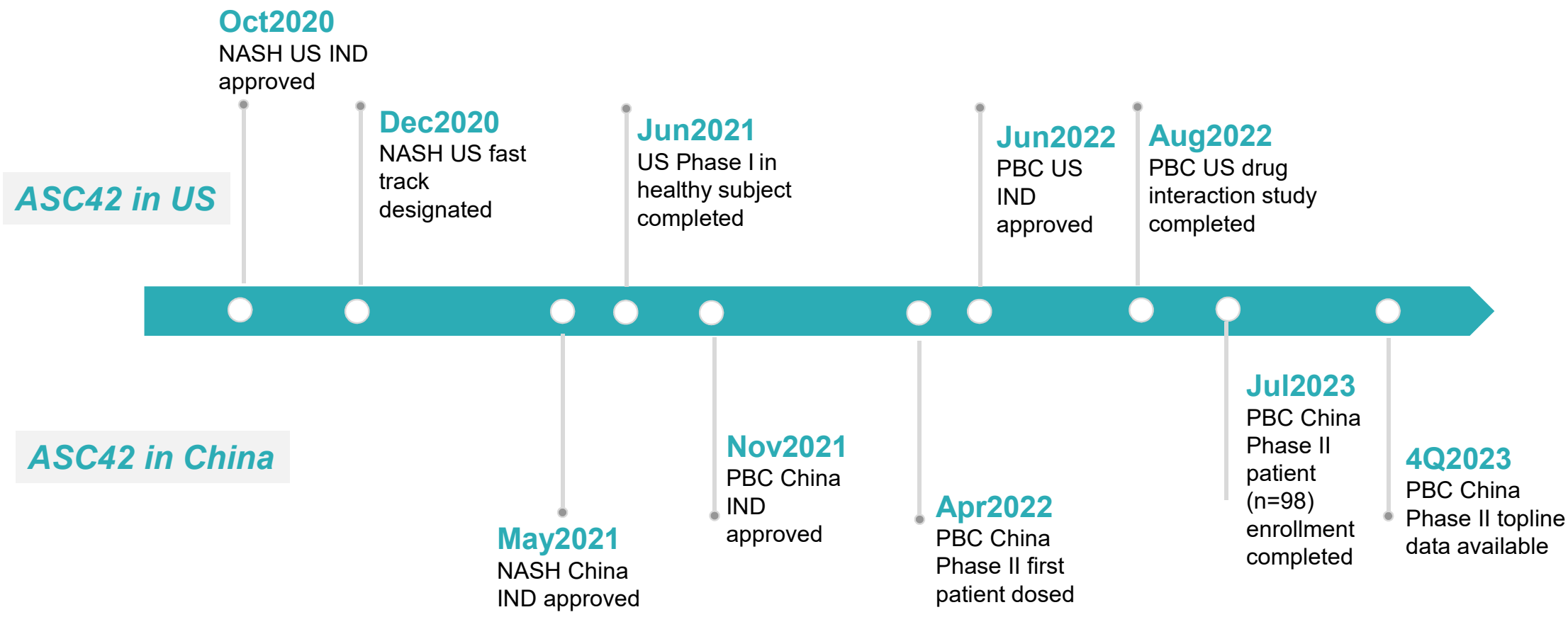
If on UDCA, Continue on UDCA



****Patient population***
1. inadequate response to UDCA
2. unable to tolerate UDCA

Primary endpoints:
% patients achieving: 1) ALP ≤ 1.67x ULN, 2) a minimum ALP reduction of ≥ 15% from baseline and 3) total bilirubin ≤ULN

ASC42 NASH/PBC timeline



THANKS

GANNEX