Biology-driven discovery.
Life-changing medicines.

Corporate Overview

January 2024

NASDAQ: NGM

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Safe Harbor Statement

The following presentation contains forward-looking statements, including, but not limited to, statements regarding: NGM Bio's evolved strategy for aldafermin and NGM120 and focus on rare conditions with significant unmet needs; therapeutic potential of, potential indications for and/or planned and continued development of the product candidates in NGM Bio's pipeline, including NGM707, aldafermin, NGM120, NGM831 and NGM438; the planned timing of initiation, enrollment, data readouts and results of NGM Bio's clinical trials; the potential roles of ILT2 and ILT4 in cancer, including the potential to enable tumors to evade immune detection and the potential of a dual blockade of ILT2 and ILT4 to act additively to reverse suppression of immune cell signaling and be more effective than blockade of either receptor alone; the potential of NGM707 to be first-in-class and to reprogram ILT4-expressing myeloid cells and stimulate the activity of ILT2-expressing myeloid and lymphoid cells; the potential of NGM831, including the potential to inhibit interaction of ILT3 with fibronectin and other ligands; the potential of NGM438, including the potential to inhibit LAIR1 and block interactions with all known ligands including collagens, and to be first-in-class; the potential of ELF to predict clinical outcome in patients with PSC; the potential of aldafermin to improve liver fibrosis in PSC patients; discussions with the FDA regarding the design of a potential registrational trial of aldafermin in PSC, including the proposed use of surrogate endpoints for potential accelerated approval; the potential to differentiate aldafermin from other late-stage drugs on breadth of applicability to the PSC patient population; the potential that targeting GFRAL ameliorates the metabolic and emetic effects caused by overstimulation of GFRAL neurons by excessive GDF15; the potential role of GDF15 in nausea, vomiting and HG; the potential of NGM120, including the potential to inhibit GDF15/GFRAL and reduce nausea and vomiting in patients; NGM Bio's potential exploration of NGM120 in a proof-of-concept study for the treatment of HG, including ongoing discussions with the FDA on an acceptable toxicology package; and other statements that are not historical fact. 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The forward-looking statements contained in the following presentation are made only as of the date hereof or as of the dates indicated in the forward-looking statements, even if they are subsequently made available by NGM Bio on its website or otherwise. NGM Bio undertakes no obligation to update or supplement any forward-looking statements after the date hereof, or to update the reasons why actual results may differ or differ materially from those anticipated in the forward-looking statements.

NGM Bio is Focusing Development Efforts on NGM707, While Exploring Opportunities to Develop Aldafermin in PSC and NGM120 in HG

Program	Mechanism	Indication	Preclinical	Phase 1	Phase 2	Status
CLINICAL DEVELOPMENT PRIORITIES						
NGM707 + pembrolizumab	ILT2/ILT4 Dual Antagonist + PD-1 Antagonist Antibody	Advanced solid tumors		PHASE 1/2		Ongoing
Aldafermin	FGF19 Analog	Primary Sclerosing Cholangitis (PSC)			PHASE 2 *Discussing design of a potential registrational trial of aldafermin in PSC with FDA, including the use of proposed surrogate endpoints with goal of accelerated approval	In Discussion with FDA*. Received Orphan Drug Designation
NGM120	GFRAL Antagonist Antibody	Hyperemesis Gravidarum (HG)		PHASE 2 PLANNING **In discussion with the FDA on an acceptable toxicology package to support clinical trials		In Discussion with FDA**
OTHER CLINICAL PROGRAMS[1]						
NGM831 + NGM438 + pembrolizumab	ILT3 + LAIR1 + PD-1 Antagonist Antibodies	Advanced solid tumors		PHASE 1 PART 1C		Target Completion in 1H24
NGM120 + gemcitabine + nab-paclitaxel	GFRAL Antagonist Antibody + chemotherapy	PDAC			PHASE 2	Enrollment Complete. No Further Plans in Oncology

[1]In 2023, NGM completed the Phase 2 CATALINA study of NGM621 (Anti-Complement C3 Antibody) in geographic atrophy and completed Phase 1 dose finding studies of each of NGM831 and NGM438, alone and in combination with pembrolizumab. Merck, which had licensed NGM313 (FGFR1c/KLB Agonist Antibody) in 2019, terminated its Phase 2b study in NASH F2/F4 and returned the molecule to NGM

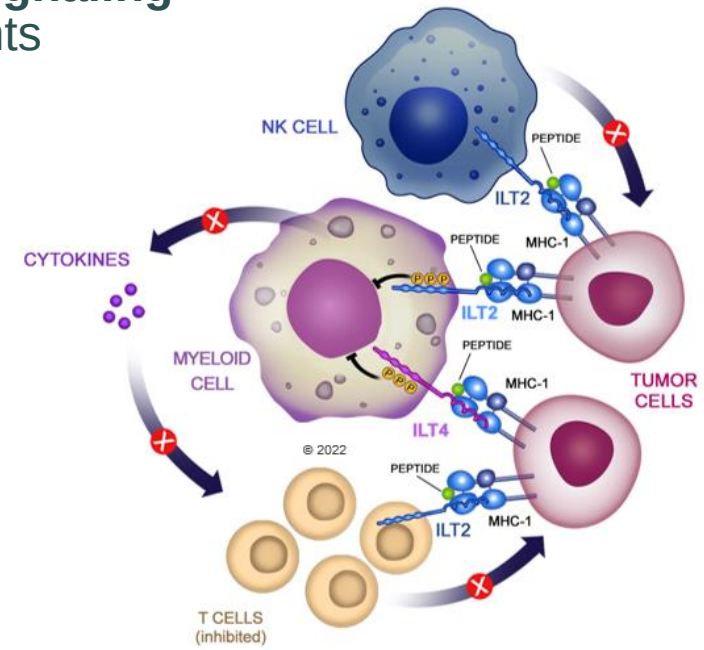
F2/4 = stage 2/4 liver fibrosis; FGF19 = fibroblast growth factor 19; GFRAL = glial cell-derived neurotrophic factor receptor alpha-like; ILT2/3/4 = immunoglobulin-like transcript 2/3/4; KLB = beta-klotho; LAIR1 = leukocyte-associated immunoglobulin-like receptor 1; NASH = non-alcoholic steatohepatitis; PDAC = pancreatic ductal adenocarcinoma

NGM Bio's Myeloid Reprogramming Strategy to Treat Solid Tumors

NGM707, NGM831, NGM438

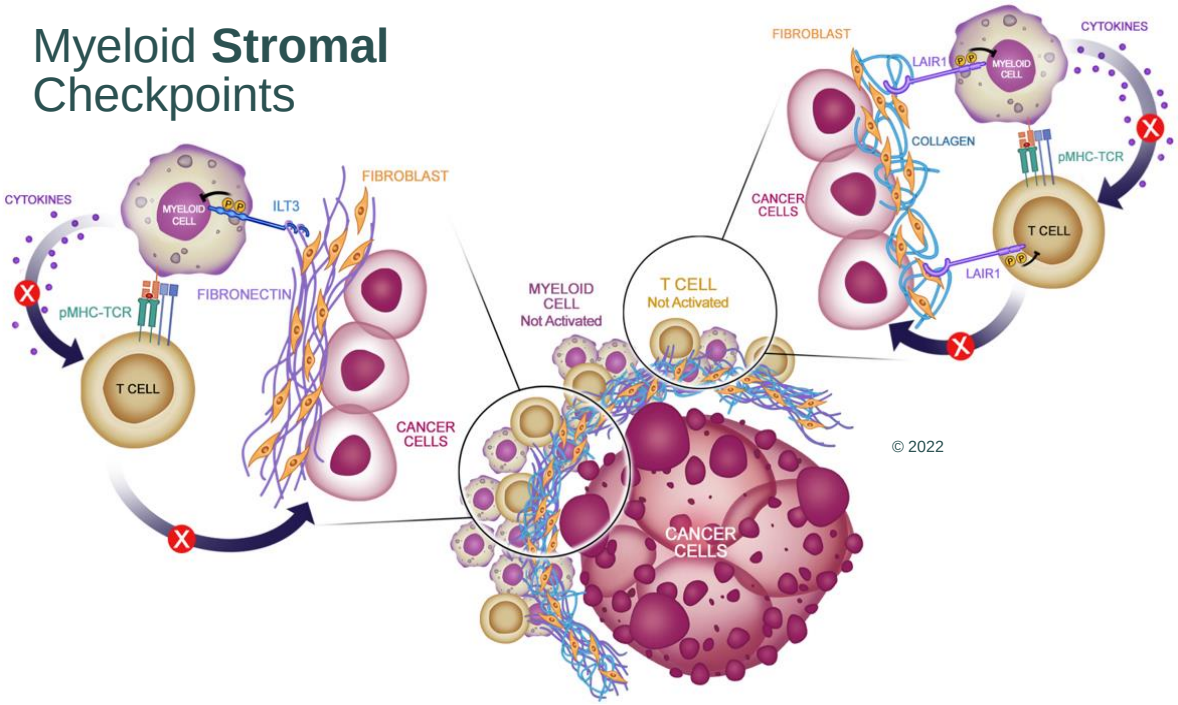
NGM Bio is Targeting Inhibitory Receptors on Myeloid Cells to Attempt to Restore Immune Response Against Tumors

Myeloid **Signaling** Checkpoints



NGM707	Potential first-in-class dual antagonist antibody inhibiting **ILT2** and **ILT4**

Myeloid **Stromal** Checkpoints



NGM831	Novel **ILT3** antagonist antibody inhibiting interaction with fibronectin[1] and other ligands
NGM438	Potential first-in-class antagonist antibody inhibiting **LAIR1**, blocking interactions with all known ligands including collagens

[1]NGM identified fibronectin, an extracellular matrix protein that forms a fibrillar network within the tumor stroma, as a ligand of ILT3 (Paavola *et al*., Cancer Immunology Research 2021)

NGM Bio is Nearing Completion on Multiple Cohorts That Will Inform Future Clinical Development in 2024

	NGM707	NGM831	NGM438
Phase 1 Part 1a *(Monotherapy dose finding)*	✓ **Enrollment complete** 1 PR in melanoma[1], 8 patients with target lesion reductions out of 35 response-evaluable patients	✓ **Enrollment complete**	✓ **Enrollment complete**
Phase 1 Part 1b *(Dose finding with pembrolizumab)*	✓ **Completing enrollment** 4 confirmed PRs, including 1 pathological CR[2], out of 37 response-evaluable patients[3]	✓ **Enrollment complete**	✓ **Enrollment complete**
Phase 1 Part 1c *(Dose finding for triplet combination: NGM831, NGM438 and pembrolizumab)*		✓ **Completing enrollment in Phase 1 Part 1c**	

PR=partial response; CR=complete response

[1]Melanoma patient has been on study since August 2022; [2]One patient had significant target lesion reduction that allowed subsequent surgical resection of all gross residual disease and confirmed pathological CR with no active tumor cells and ctDNA below detection [3] Data as of November 6, 2023

NGM707 in Advanced Solid Tumors

ILT2 and ILT4: Key Myeloid and Lymphoid Checkpoints and Their Potential Roles in Cancer

Upregulated in certain cancer types[1-5]

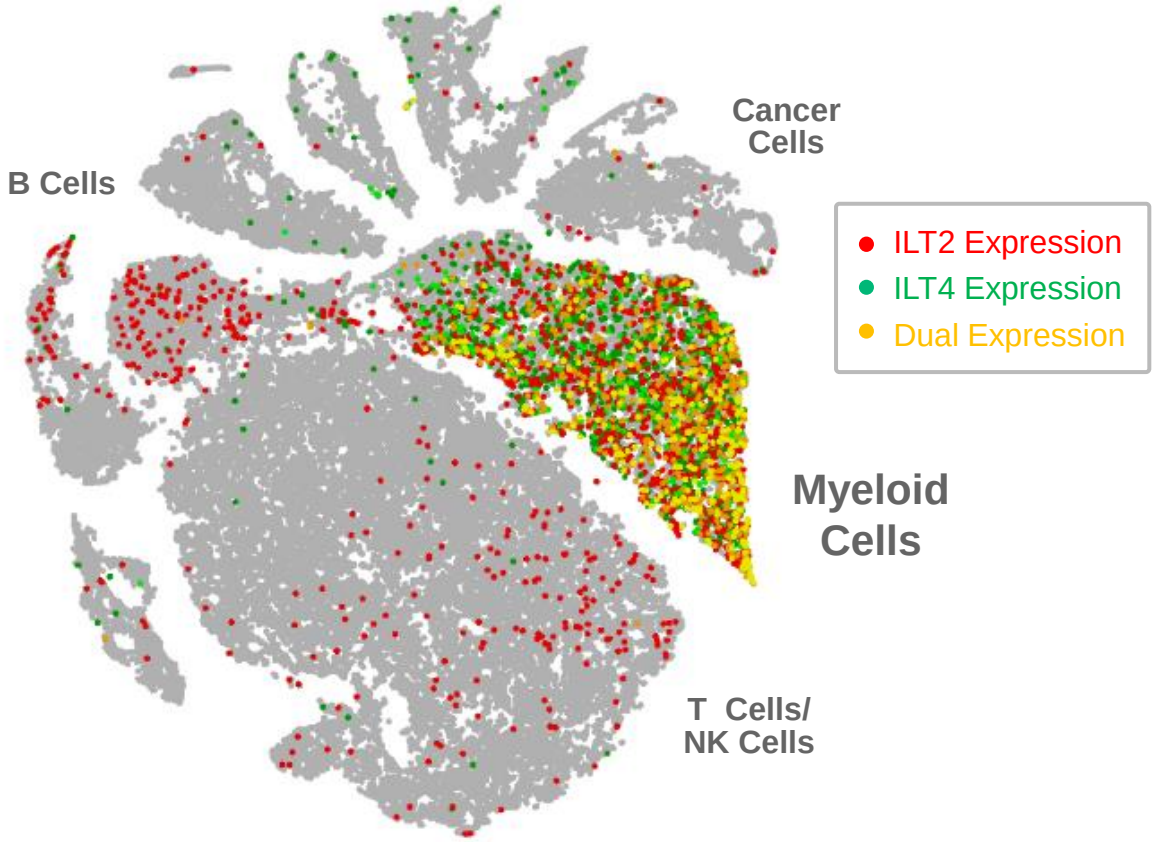
- ILT2 and ILT4 receptors are expressed on myeloid cells (APCs, MDSCs, macrophages, granulocytes) in the tumor microenvironment
- ILT2 additionally exhibits expression on natural killer (NK) cells, B cells and a subset of highly cytolytic T cells

Restrict anti-tumor immunity and promote a tolerogenic state

- By suppressing anti-tumor immune responses, ILT2 and ILT4 may enable tumors to evade immune detection

Contribute to T cell checkpoint inhibitor resistance[6]

- ILT2 and ILT4 are upregulated on macrophages in the tumor microenvironment of certain cancer patients that are non-responders to T cell checkpoint inhibitor therapy



Within the myeloid cluster there are distinct subsets of cells: ILT2+, ILT4- / ILT2-, ILT4+ / ILT2+, ILT4+

Blocking both ILT2 and ILT4 may provide further benefit

[1]Tirosh *et al., Science*, 2016; [2]Li *et al., Nat Genet*, 2017; [3]Puram *et al., Cell*, 2017; [4]Azizi *et al., Cell*, 2018; [5]Lambrechts *et al., Nat Med*, 2018; [6]Sade-Feldman *et al., Cell*, 2018
APC = Antigen-presenting Cell; MDSC = Myeloid-derived Suppressor Cell

NGM707 is a Dual Antagonist Antibody Designed to Inhibit ILT2 and ILT4

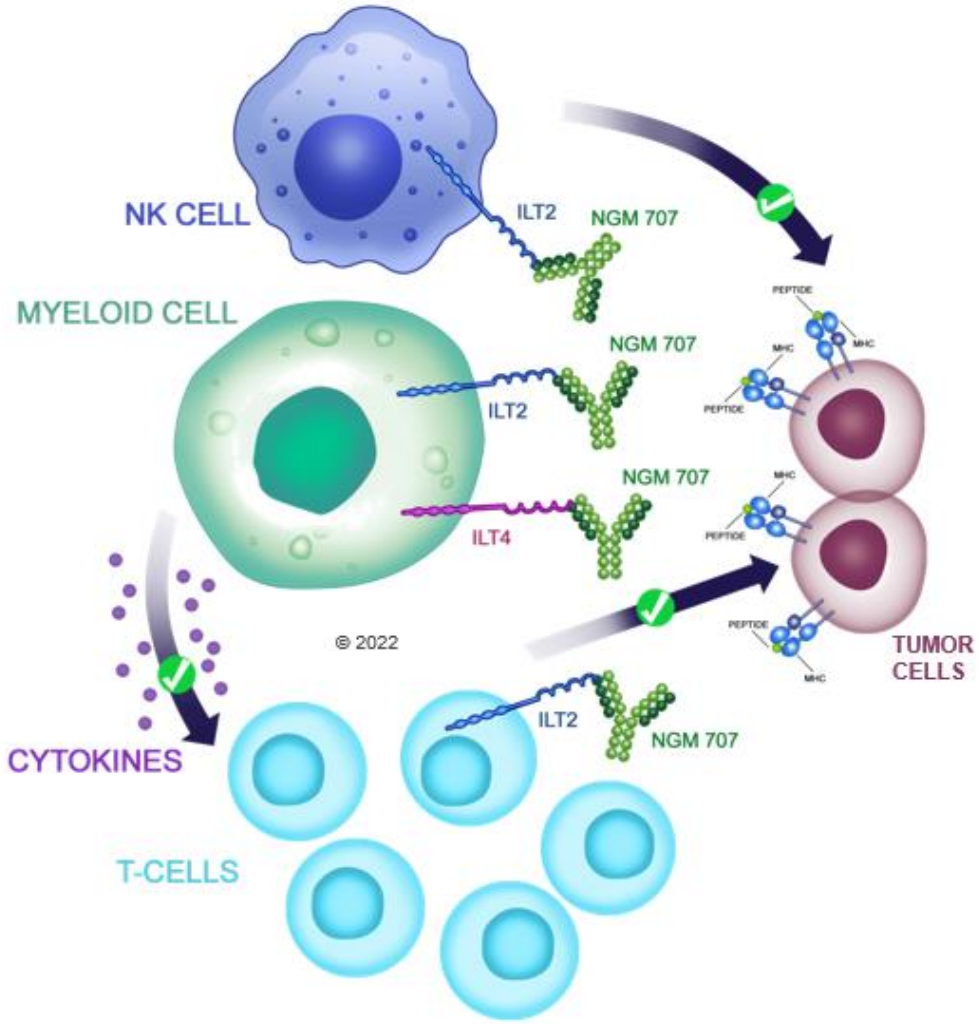
Potent, potential first-in-class antibody targeting the myeloid-enriched inhibitory receptors ILT2 (LILRB1) and ILT4 (LILRB2)

Potential to reprogram ILT4-expressing myeloid cells and stimulate the activity of ILT2-expressing myeloid and lymphoid cells

Preclinical studies of NGM707 suggest that:

- ILT4 blockade reverses myeloid cell immune suppression
- ILT2 blockade promotes tumor cell killing by NK and CD8+ T cells as well as tumor cell phagocytosis by macrophages
- **Dual blockade of ILT2 and ILT4 may act additively to reverse suppression of immune cell signaling and be more effective than blockade of either receptor alone**

Phase 1 Part 1a completed enrollment. Phase 1 Part 1b enrollment is ongoing



Preliminary Findings of Ongoing NGM707 Phase 1/2

	Phase 1 Part 1a[1]	Phase 1 Part 1b[1]
Cohort	Monotherapy dose finding	Combination dose finding with pembrolizumab
Dose levels tested	7 NGM707 dose levels (6, 20, 60, 200, 600, 1200, 1800 mg)	4 NGM707 dose levels (200, 600, 1200, 1800 mg)
Enrollment	41	46 (5 crossover from monotherapy)
Indication	Advanced solid tumors	Advanced solid tumors
Median prior lines of therapy	4	3
Most common tumor types	CRC, Melanoma, NSCLC, Cervical, PDAC	CRC, Gastric, NSCLC, PDAC, SCCHN
Primary endpoint (Safety and Tolerability)	• NGM707 was generally **well-tolerated**	• NGM707 + pembro was generally **well-tolerated**
	• 46% TRAEs any grade, 5% Grade 3+	• 41% TRAEs any grade, 4% Grade 3+
Secondary endpoint (Efficacy)	• Of 35 response-evaluable patients[2], best overall responses: – 1 partial response in melanoma (71% reduction) – 10 stable disease (incl. 1 NCR/NPD) – 8 patients with reduced lesion size	• Of 37 response-evaluable patients[2], best overall responses: – 4 confirmed partial responses (incl. 1 pathological CR[3]) – 12 stable disease – 9 patients with reduced lesion size
	• Preliminary evidence of myeloid reprogramming was seen in peripheral blood and in tumor biopsies in mono and combo • Exploratory endpoints: serum cytokines/chemokines, flow cytometry immunophenotyping, tumor RNA-sequencing, tumor immunohistochemistry	

[1]Data as of November 6, 2023 [2]Those completing at least one on-treatment scan [3]One patient had significant target lesion reduction that allowed subsequent surgical resection of all gross residual disease and confirmed pathological CR with no active tumor cells and ctDNA below detection
CRC = Colorectal Cancer; NSCLC = Non-small Cell Lung Cancer; PDAC = Pancreatic Ductal Adenocarcinoma; SCCHN = Squamous Cell Carcinoma of Head and Neck; TRAE = Treatment-related Adverse Events; CR = Complete Response; NCR = Not Complete Response; NPD = Not Progressive Disease

Preliminary Data from Phase 1 Part 1a Trial of NGM707 Monotherapy

NGM707 monotherapy appears to be generally well tolerated at all dose levels. In advanced or metastatic solid tumor cohort, early signals of anti-tumor activity were observed. Preliminary evidence of myeloid reprogramming was seen in peripheral blood and in tumor biopsies

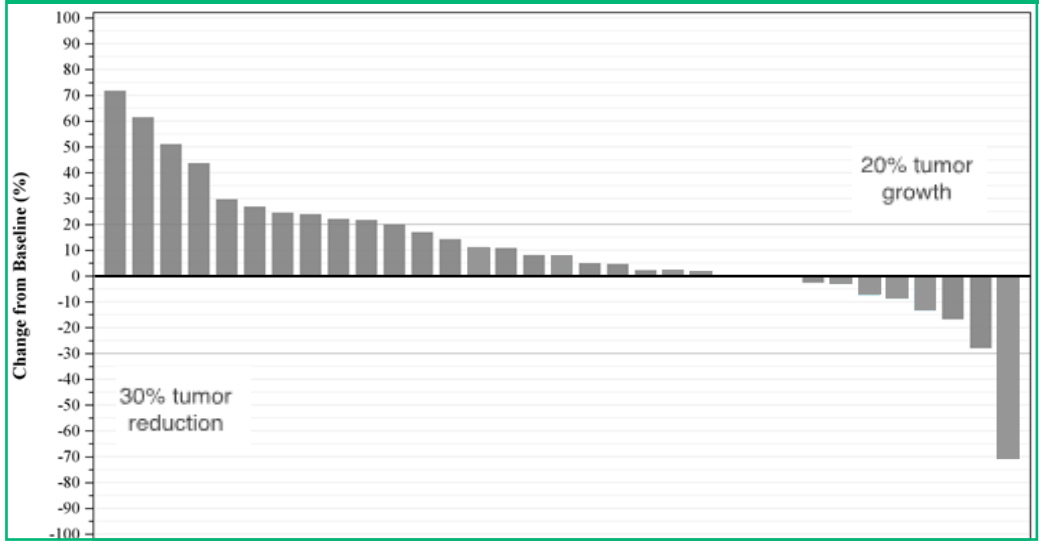
NGM707 was generally well tolerated[1]:

- Treatment-related adverse events (any grade/grade ≥3) occurred in 46% (5%) of patients
- One dose-limiting toxicity of pneumonitis (G5) in a patient with pulmonary metastasis was observed at 600 mg
- A maximum tolerated dose was not reached; the maximum administered dose was 1800 mg

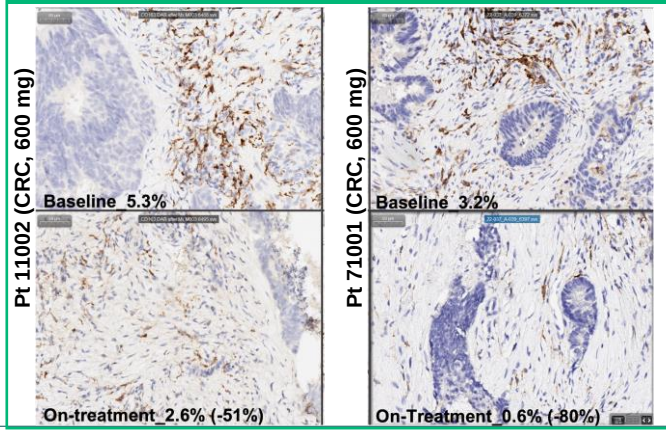
Encouraging early signals observed:

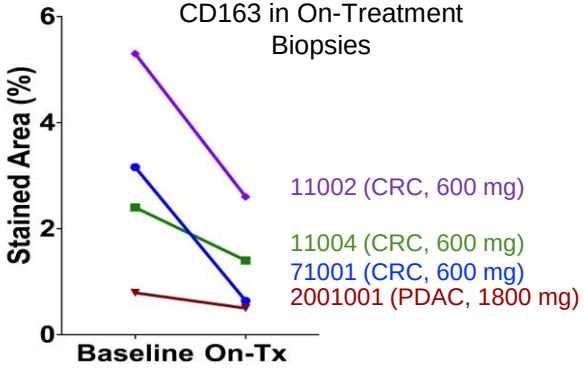
- Of 35 response-evaluable patients[2], best overall responses[1] are 1 partial response, 10 stable disease (including 1 non-complete response/non-progressive disease) patients. Eight patients had reduced target lesion size incl. a maximum decrease in 1 patient of 71%
- Preliminary evidence of myeloid reprogramming was seen in peripheral blood and in tumor biopsies

NGM707 Ph1a - Target Lesion Assessment



Preliminary Evidence of Myeloid Reprogramming

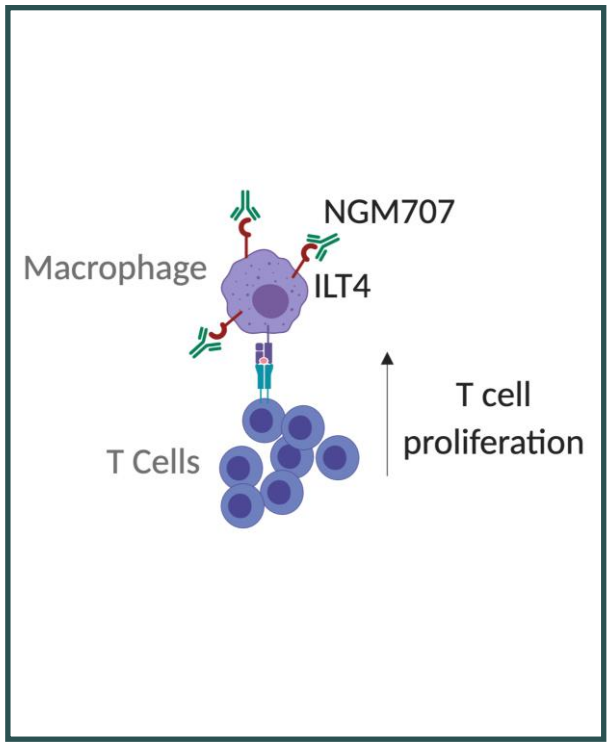


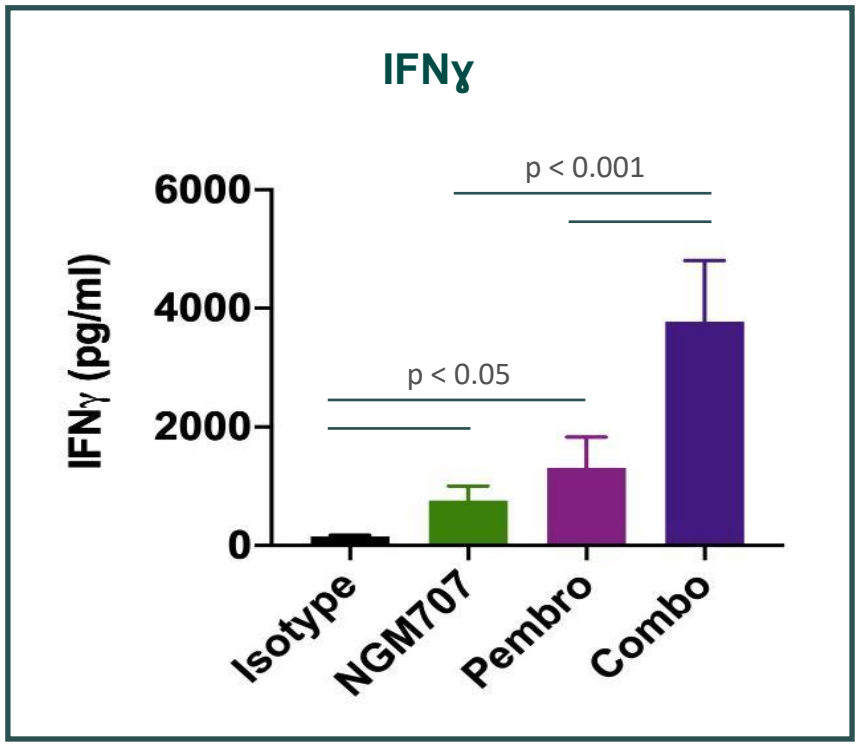


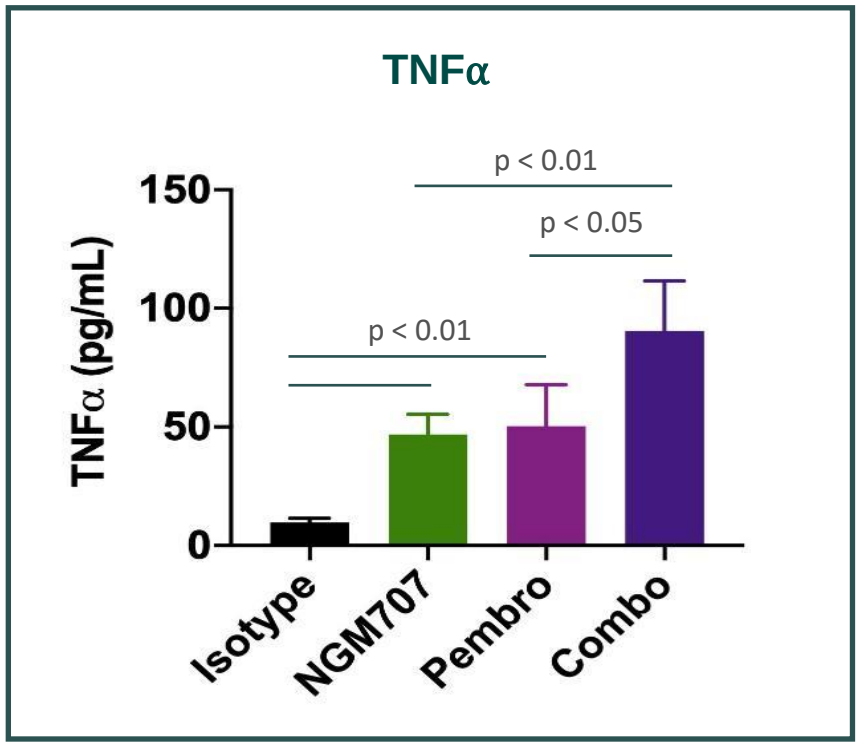
[1]Data as of November 6, 2023; [2]Those completing at least one on-treatment scan
G5 = Grade 5

Strong Biological Rationale for Combining NGM707 and Anti-PD-1: Enhance T Cell Activation and Cytokine Secretion

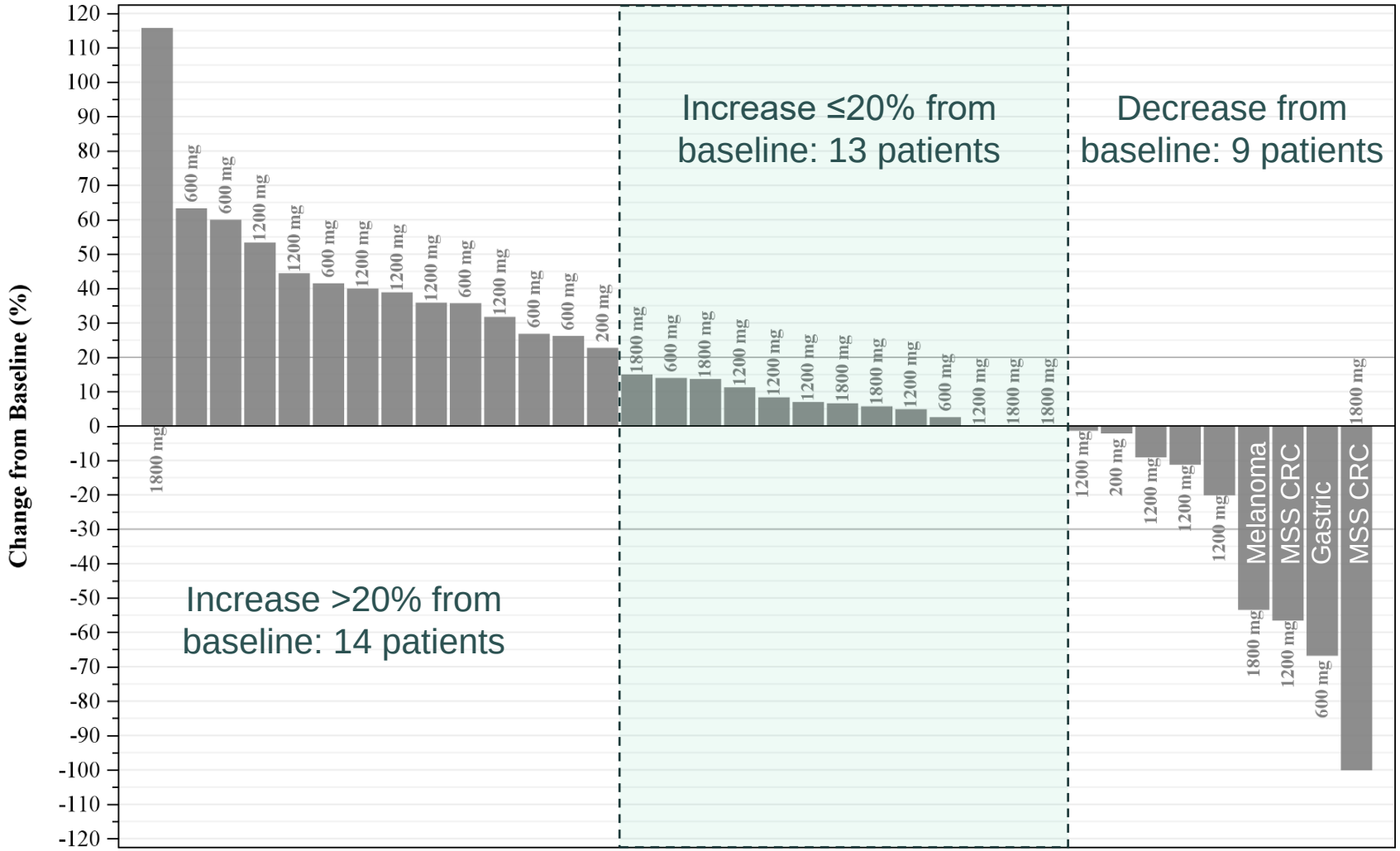
- Combination of NGM707 and pembrolizumab leads to an **additive** increase in T cell activation and cytokine secretion in preclinical studies
- Study involved monocytes from two individuals differentiated into macrophages and tested in mixed lymphocyte reactions with T cells from 12 individuals







Ongoing NGM707 Phase 1 Part 1b: Target Lesion Reduction Observed in 25% of Patients[1]



Data as of November 6, 2023

[1]One response-evaluable patient is not shown on this waterfall. They had a RECIST assessment result, but the target lesion information is missing

Patient Vignette from NGM707 Phase 1 Part 1b Trial: Durable Response in MSS CRC Adenocarcinoma Leading to Pathological Complete Response

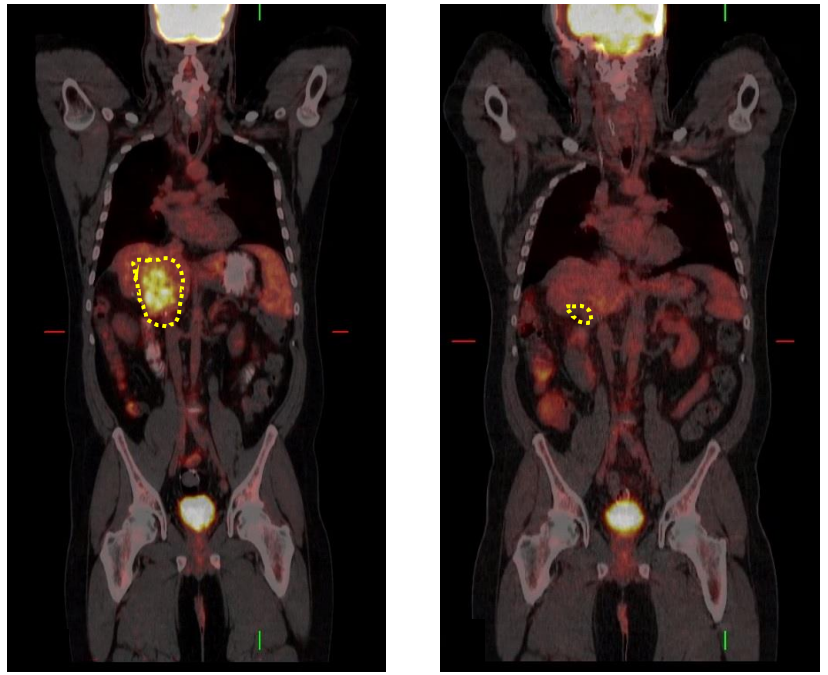
41-Year-Old Male Receiving NGM707 (1800 mg) and Pembro as 4th Line Therapy

Background

- Primary tumor in **colon**. Target lesions in **liver** and **adrenal** gland
- Prior **salvage liver surgery** and multiple lines of **chemo** (*e.g.,* FOLFOXIRI + beva)
- MSS, PD-L1 CPS 1%, PD-L1 TPS 0%, TMB low

Outcomes

- **Deep and durable PR**[1]
- **Pathological CR** after surgical resection of target lesions at 48 weeks
- **Ongoing therapy** (65 weeks[2] on treatment)



Baseline | Pre-surgery

No tumor cells found in resected tissue after surgery

Peripheral Blood Biomarkers Show Evidence of Myeloid Reprogramming

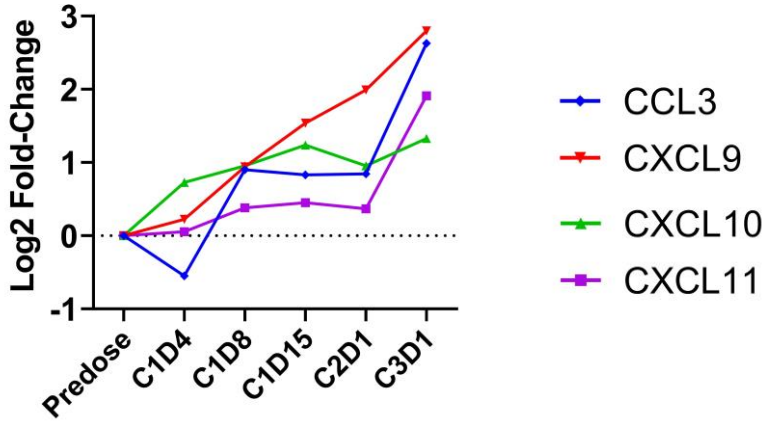


Figure 1. Increased cytokines associated with immune cell recruitment

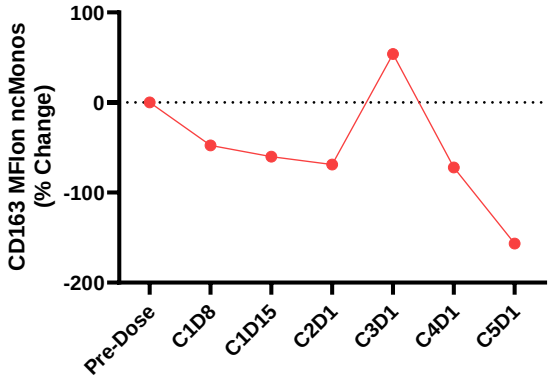


Figure 2. Decreased CD163 (marker of immune suppression) on non-classical monocytes

[1]Duration of response of 10.7 months as of December 18, 2023; [2]Data cutoff as of December 18, 2023
Beva = Bevacizumab; MSS = Microsatellite Stability Biomarker; TMB = Tumor Mutation Burden; PR = Partial Response; CR = Complete Response
Results from this selected patient may not be typical or predictive of final trial results and should be viewed with caution

Summary of Preliminary Findings from Ongoing NGM707 Phase 1 Part 1b

- Phase 1 Part 1b combination dose escalation study of NGM707 and pembrolizumab initiated in late 2022. Enrollment is expected to complete in the first half of 2024
- NGM707 and pembrolizumab was found to be generally well-tolerated at all 4 dose levels of NGM707. Maximum tolerated dose (MTD) was not reached
- Overall, 4 confirmed PRs (including 1 pathological CR[1]) across tumor types and 12 SD out of 37 response-evaluable patients, signifying a 11% ORR and 43% DCR
- 3 out of 4 patients with confirmed PRs have liver metastases
  - Patients with liver metastases, which are associated with immune suppression and lower survival rates, tend to have a reduced response to immunotherapy
- 2 confirmed MSS CRC PRs (including 1 pathological CR[1]) and 2 SD out of 8 response-evaluable MSS CRC patients[2] (25% ORR, 50% DCR)
  - Anti-PD-1/PD-L1 monotherapies have shown low or no benefit in MSS CRC patients

[1]One patient had significant target lesion reduction that allowed subsequent surgical resection of all gross residual disease and confirmed pathological CR with no active tumor cells and ctDNA below detection
[2]Eight response evaluable CRC patients excludes one CRC patient with undetermined MSS status
Data as of November 6, 2023
Source: Lee *et. al,, Immuno-Oncology Insights* 2022; Zlotnick *et al., Cancers (Basel)* 2023; Kawazoe *et. al., Annals of Oncology* 2023; Sahin *et. al., ASCO Educational Book* 2022

NGM831 and NGM438 in Advanced Solid Tumors

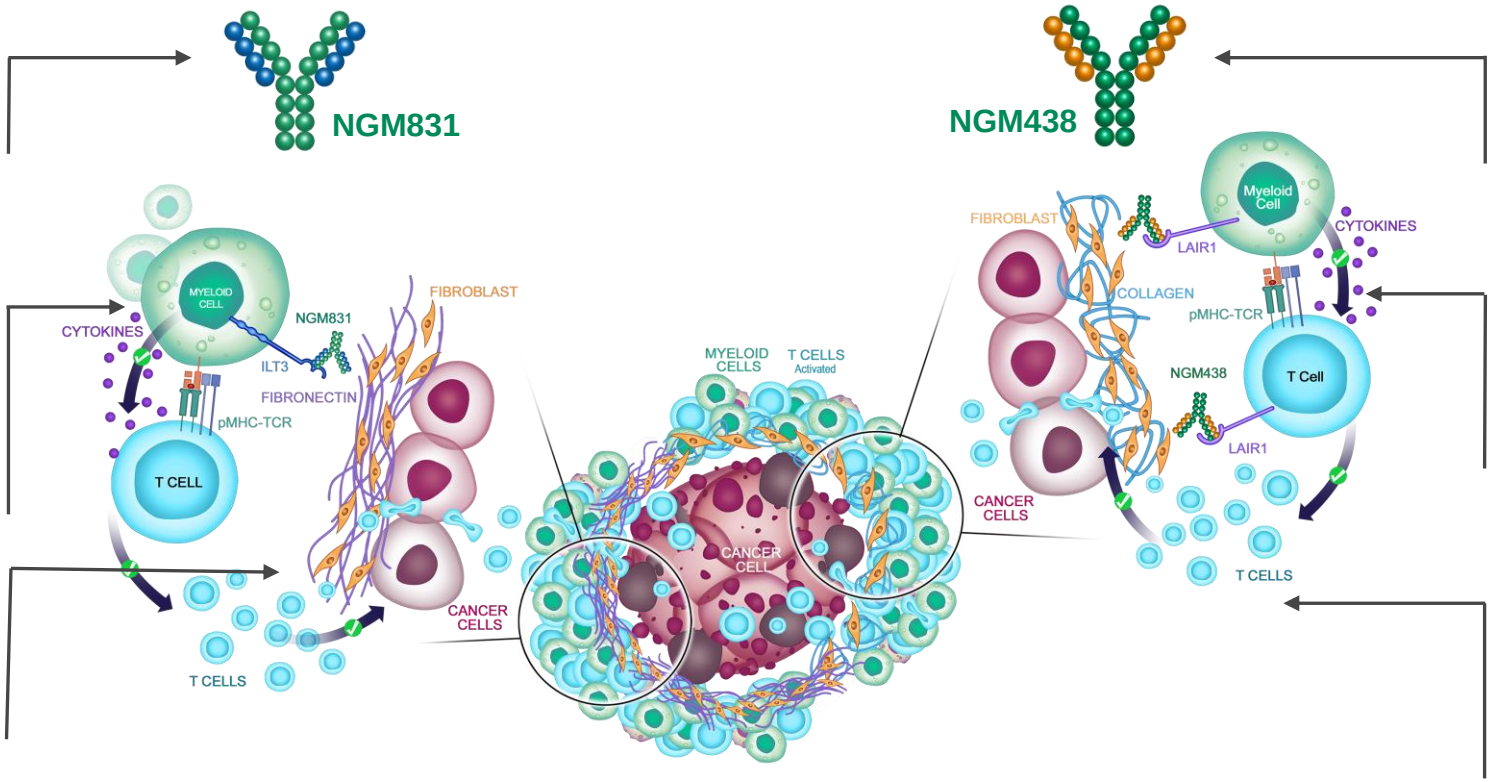
NGM831 and NGM438 Synergize to Inhibit Stroma-Mediated Immunosuppression in the Tumor Microenvironment

The ILT3-Fibronectin and LAIR1-Collagen Interactions Are "Stromal Checkpoints" Through Which the Extracellular Matrix Communicates with Immune Cells and Inhibits Immune Responses

NGM831 is an antagonist of ILT3

ILT3 is a **fibronectin**-binding inhibitory immunoreceptor that is highly expressed on suppressive myeloid cells

Fibronectin is an abundant component of the tumor stroma and is upregulated in numerous tumor types



NGM438 is a first-in-class antagonist antibody of LAIR1

LAIR1 is a **collagen**-binding inhibitory signaling receptor that is expressed on suppressive myeloid cells as well as T cells, B cells, and NK cells[1-2]

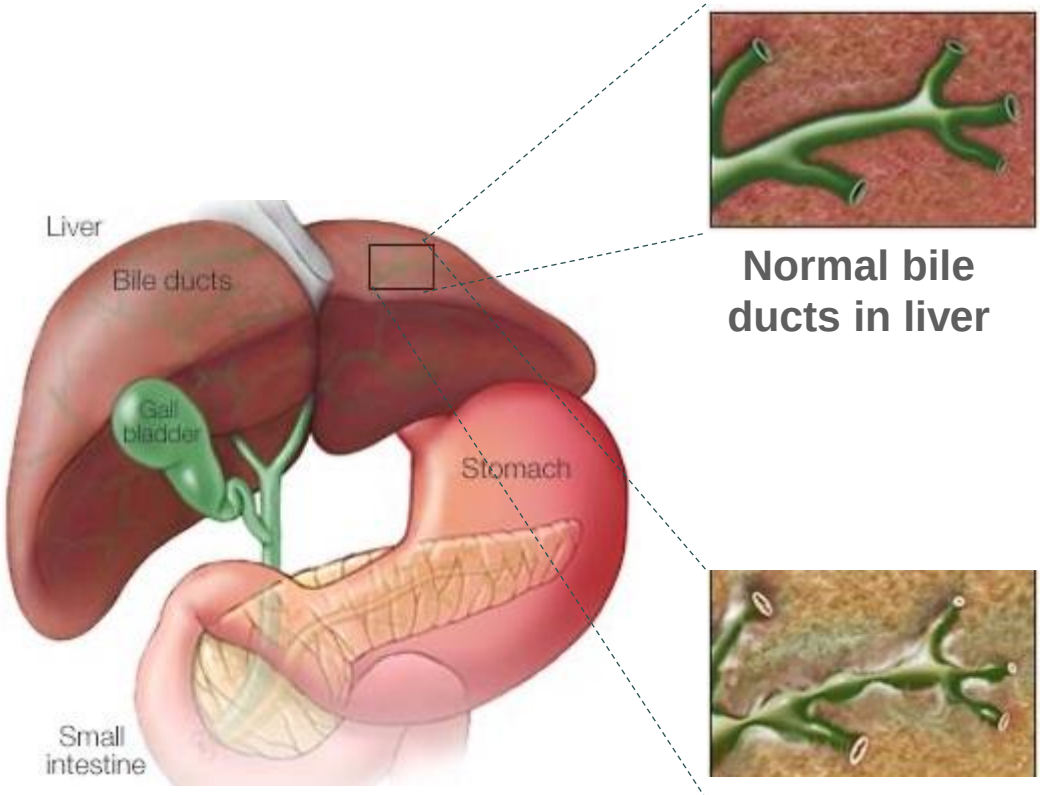
Collagen is an abundant component of the tumor stroma that contributes to immune exclusion and promotes immune checkpoint resistance in the TME[3-4]

NGM is completing enrollment of a Phase 1 study of NGM831 + NGM438 + pembro

[1]Meyaard *et al., Immunity*, 1997; [2]Guo *et al., Trans Med*, 2020; [3]Chakravarthy *et al., Nature Comm* 2018; [4]Peng *et al., Nature Comm*, 2020

Aldafermin in PSC

Primary Sclerosing Cholangitis (PSC) is a Rare Liver Disease Characterized by Inflammation and Fibrosis of the Bile Ducts



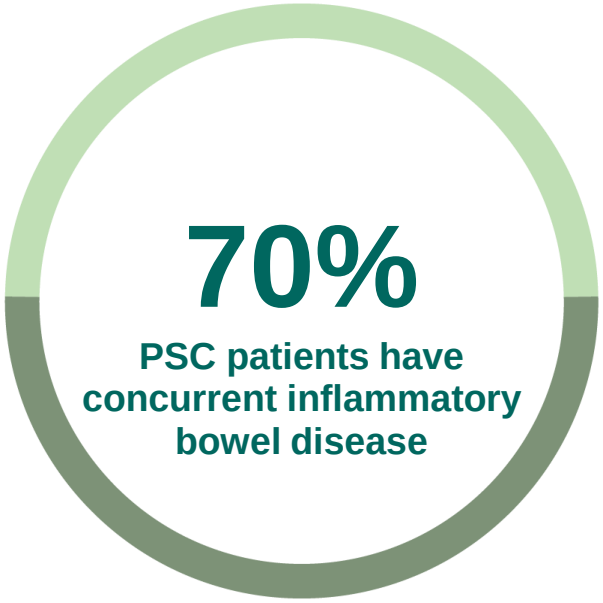
Normal bile ducts in liver

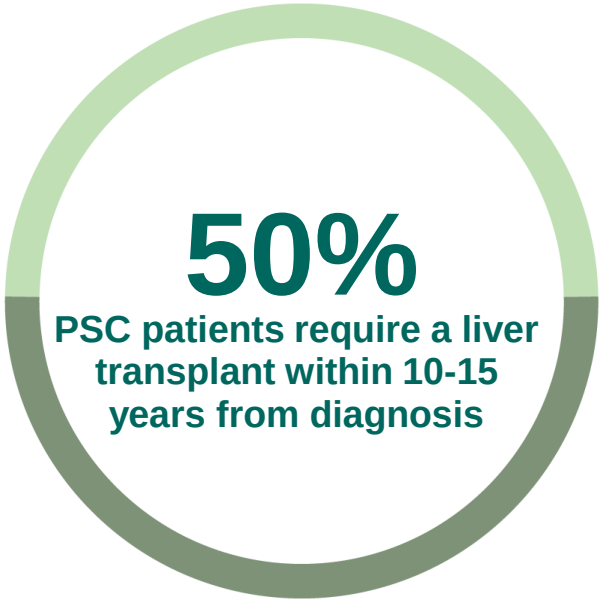
PSC – inflammation and scar tissue destroy the bile ducts

- PSC is characterized by **inflammation** and **fibrosis** of the bile ducts (hardening or narrowing of the bile duct walls), which obstructs the flow of bile
- In the short term, bile acids accumulate in the liver, leading to damage to cells, recurrent cholangitis and cirrhosis
- In the long term, the bile acid accumulation can lead to loss of liver function, end-stage liver disease and cancer
- Common symptoms include fatigue, pruritus (including severe itching), jaundice, abdominal pain, depression and enlarged liver
- There are no FDA-approved therapies. The only curative treatment is liver transplant, however, disease may recur in transplant cases

Source: PSC Partners; Mayo Clinic; Karlsen *et al. Journal of Hepatology* 2017

Significant Unmet Need in PSC

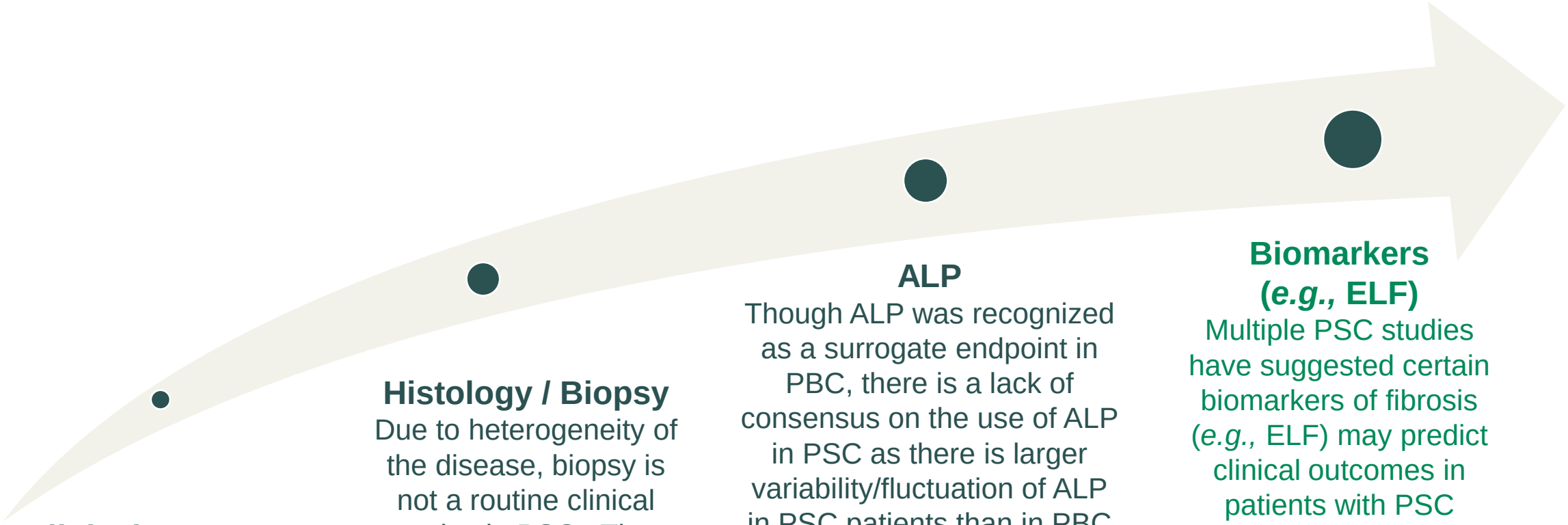




There are no FDA-approved therapies for treating PSC

[1]Similar patient prevalence in the US and EU5; Patient prevalence in PSC, as is common for rare diseases with no approved therapies, may be underestimated
Source: Cooper *et al.* 2023; De Vries *et al.* 2018; Horsley-Silva *et al.* 2017; Tabibian *et al*. 2018

PSC Regulatory Path Has Historically Been Difficult Due to Lack of Surrogate Endpoints and Requirement for Outcomes Studies



Clinical Outcomes
Difficult due to the infrequent number of events and limited patient population, resulting in a long trial duration

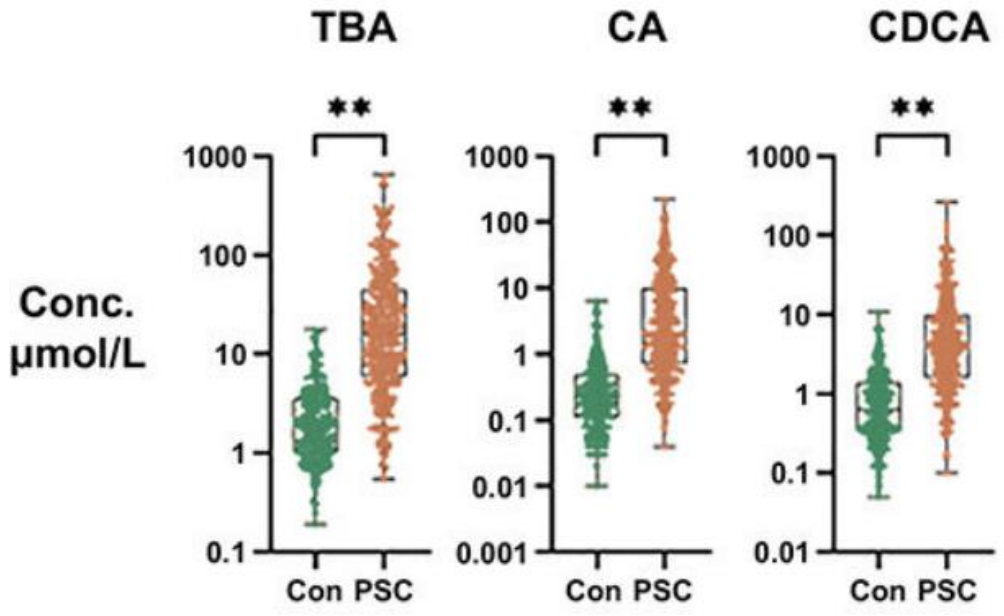
Histology / Biopsy
Due to heterogeneity of the disease, biopsy is not a routine clinical practice in PSC. There is a risk of damaging the remaining healthy tissue during the biopsy

ALP
Though ALP was recognized as a surrogate endpoint in PBC, there is a lack of consensus on the use of ALP in PSC as there is larger variability/fluctuation of ALP in PSC patients than in PBC patients

Biomarkers (*e.g.*, ELF)
Multiple PSC studies have suggested certain biomarkers of fibrosis (*e.g.,* ELF) may predict clinical outcomes in patients with PSC

ALP = Alkaline Phosphatase; ELF = Enhanced Liver Fibrosis; PBC = Primary Biliary Cholangitis
Source: Ponsioen *et al.*, 2016; Bowlus *et al.*, 2022; Trivedi *et al.*, 2021; Muir *et al.*, 2019

PSC Patients Have Been Shown to Have Higher Levels of Serum Bile Acids *vs.* Healthy Controls[1]



In the real-world study, average total bile acids in:
PSC patients: 16.34 µmol/L
Healthy controls: 1.90 µmol/L

In a real-world study consisting of both PSC patients and healthy controls, total bile acids and individual bile acids were significantly higher in PSC patients

Development Rationale:

Aldafermin suppresses bile acid production which may improve liver fibrosis in PSC patients

[1]Healthy volunteers
TBA = Total Bile Acids; CA = Cholic Acid; CDCA = Chenodeoxycholic Acid; Con = Controls
Source: Mousa *et. al., Hepatology* 2021

Previous PSC Studies Suggest ELF Predicts Clinical Outcomes

ELF Overview

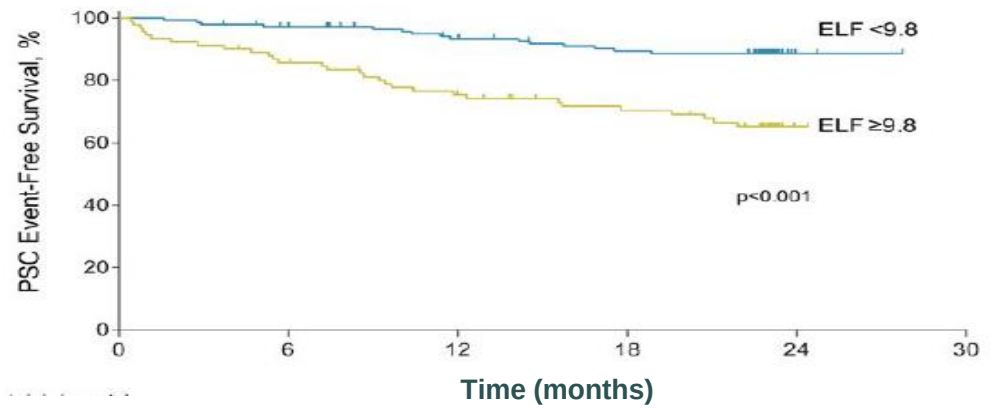
- Enhanced Liver Fibrosis (ELF) is a serum-based non-invasive test and is the first and only FDA-cleared non-invasive test for NASH prognostic assessment
- ELF measures direct markers of fibrosis: HA, PIIINP and TIMP-1
- ELF score is determined by an equation that is composed of those three markers of fibrosis

$$ELF = 2.278 + 0.851 \times \ln(C_{HA}) + 0.751 \times \ln(C_{PIINP}) + 0.394 \times \ln(C_{TIMP\text{-}1})$$

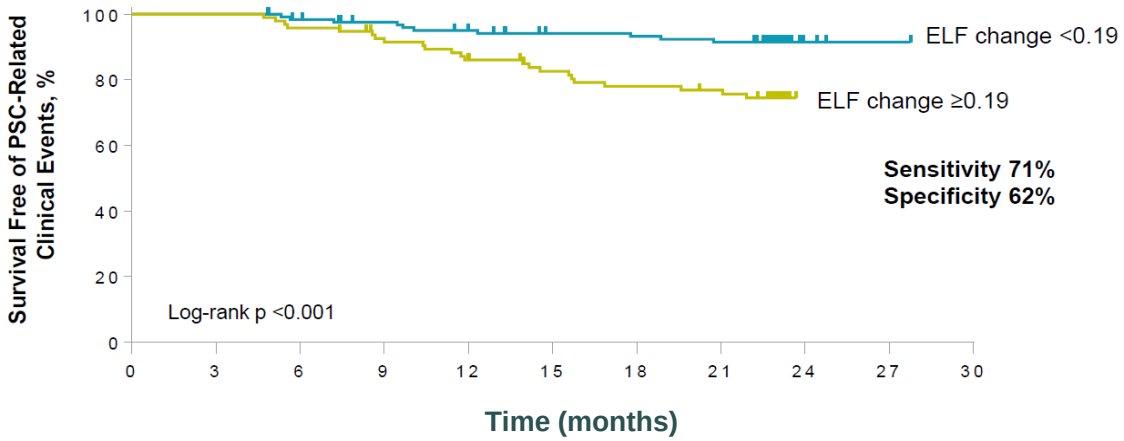
ELF in PSC

- ELF has been seen to potentially predict clinical outcomes in multiple PSC studies
  - Simtuzumab study of 234 PSC patients with compensated liver disease where the study identified the optimal threshold for baseline ELF to predict PSC-related clinical events was **≥9.8** (sensitivity 68%, specificity 67%) and that the optimal threshold for change in ELF at week 12 to predict events was **0.19** (sensitivity 69%, specificity 62%)
  - Norwegian study in 305 well-characterized PSC patients where higher ELF scores were associated with shorter survival
  - Multicenter, international, retrospective study of 534 PSC patients where ELF test potentially predicted clinical outcome (Hazard ratio 1.31; 95% confidence interval [1.05-1.65]; *P*=.018) and the ELF biomarker can be used to discriminate between PSC patients with and without a clinical outcome endpoint

Simtuzumab Study - Baseline ELF



Simtuzumab Study - ELF Change of 0.19 Predicts PSC – Related Clinical Events

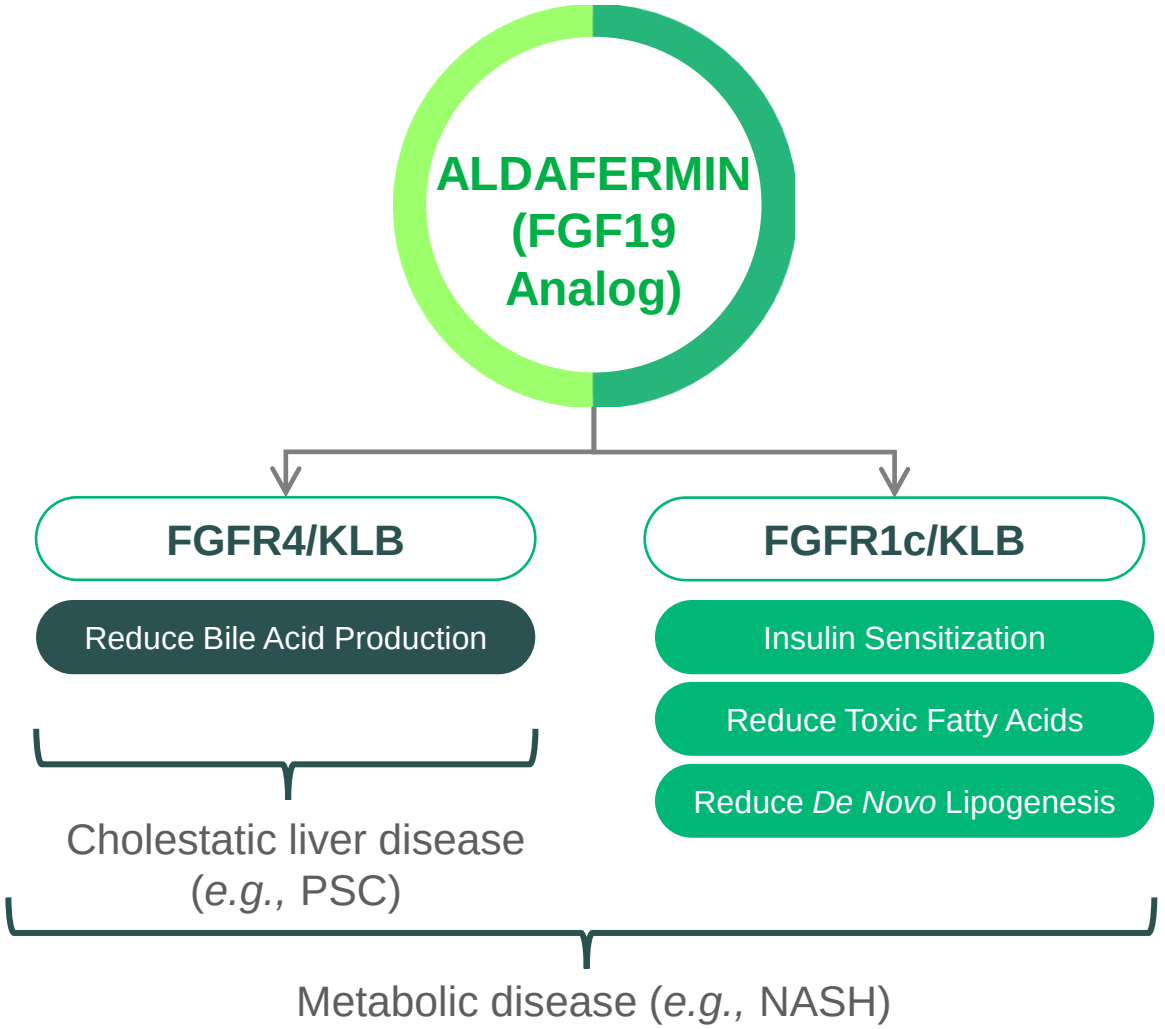


HA = Hyaluronic Acid; PIIINP = Procollagen III Amino Terminal Peptide; TIMP-1= Tissue Inhibitor of Metalloproteinase-1

Source: Muir *et al., Hepatology* 2019; Vesterhus *et al., Hepatology* 2015; de Vries *et al., Hepatology* 2017

Aldafermin is Well Positioned for Development in PSC

- Aldafermin is an investigational engineered analog of the **human hormone FGF19** that is dosed once daily as a subcutaneous injection
- FGF19 impacts two separate receptor complexes, resulting in a unique MOA with **anti-fibrotic** and **anti-inflammatory effects**:
  - FGFR4/KLB regulates bile acid synthesis
  - FGFR1c/KLB impacts components of metabolic dysregulation
- Aldafermin found to be **well-tolerated in over 800 patients** treated across indications (PSC, PBC, BAM, F2/F3/F4 NASH)
- NGM has received **Orphan Drug Designation** from the FDA and the EMA for aldafermin for the treatment of PSC and is in discussion with the FDA on the design of a potential **registrational trial in PSC, including using proposed surrogate endpoints**



MOA = Mechanism of Action; FGF19 = Fibroblast Growth Factor 19; FGFR4 = Fibroblast Growth Factor Receptor 4; FGFR1c = Fibroblast Growth Factor Receptor 1c; KLB = Beta-klotho; F2/3/4 = Stage 2/3/4 Liver Fibrosis; BAM = Bile Acid Malabsorption

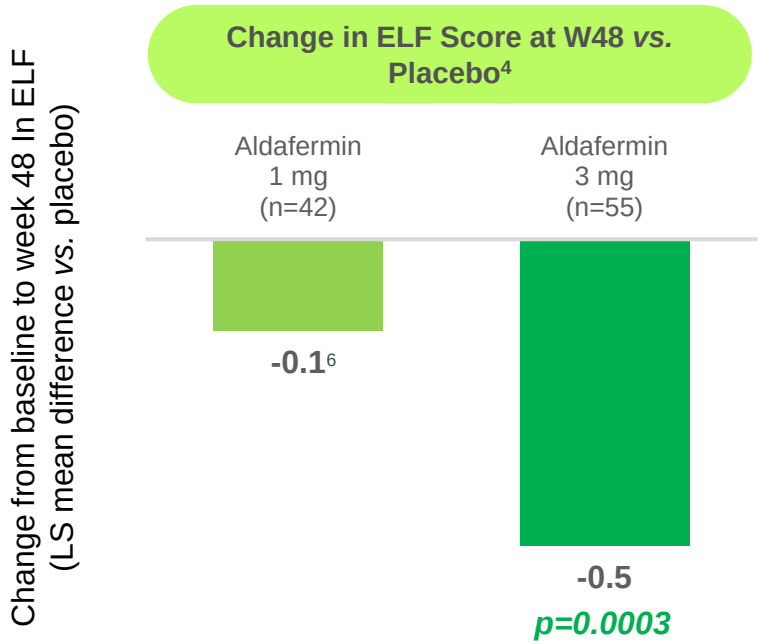
In 2023, NGM Bio Shared Positive Readout of Phase 2b ALPINE 4 Trial of Aldafermin in Compensated Cirrhosis Due to NASH (F4)

Study Design

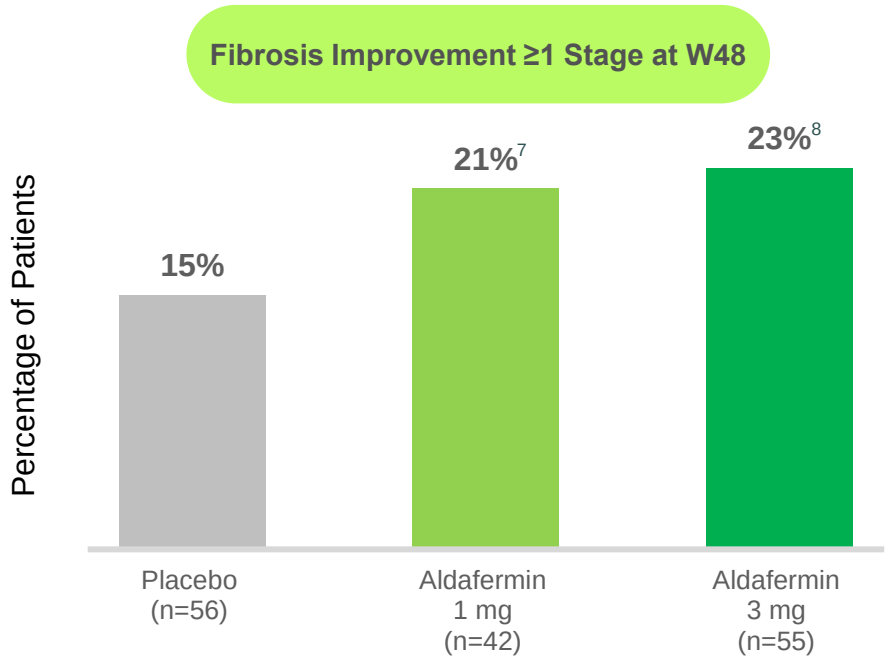
- 160 patients randomized to four arms (aldafermin 0.3 mg[1], 1 mg, 3 mg or placebo)
- Key inclusion criteria: NASH with compensated cirrhosis (NASH CRN fibrosis stage 4)[2]
- Primary endpoint: change from baseline in ELF at 48 weeks
- Key secondary endpoint[3]: fibrosis improvement of ≥1 stage at 48 weeks

Results

Primary Endpoint: ELF

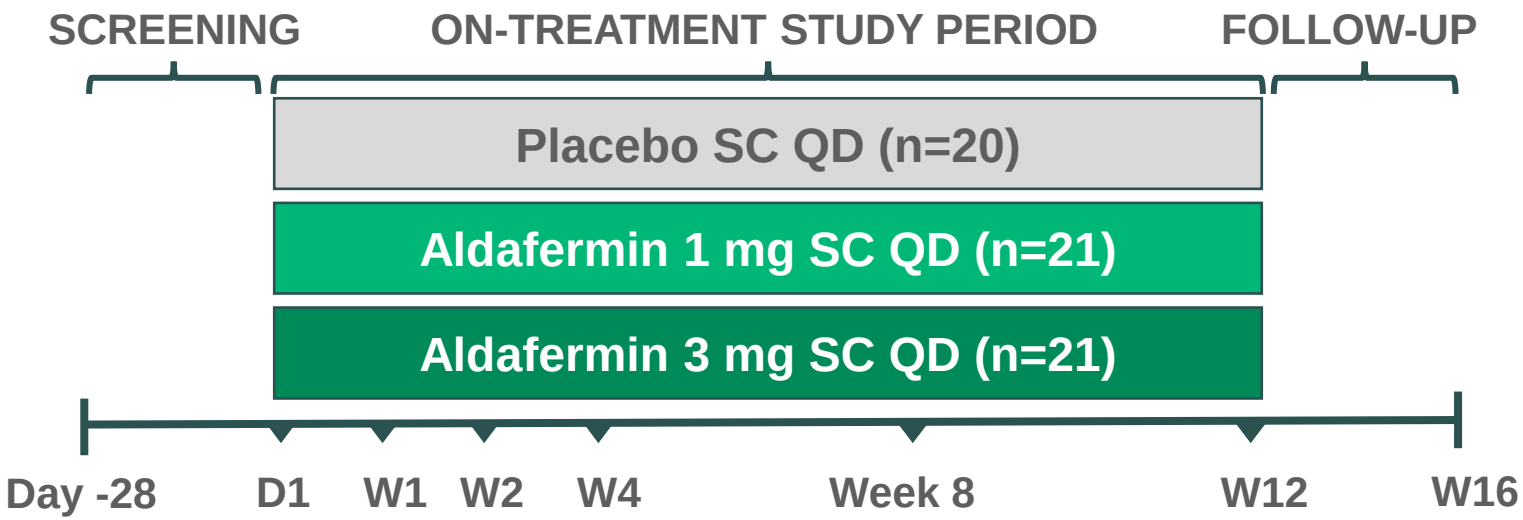


Secondary Endpoint: Fibrosis Reversal[5]



[1] 0.3mg dose discontinued during trial following enrollment of 7 patients to limit patients' exposure to suboptimal dose
[2] Up to 10% of subjects with a clinical diagnosis of NASH cirrhosis were allowed to enroll [3] Study was not powered for histologic endpoints (fibrosis improvement ≥1-stage by NASH CRN criteria);
[4] Placebo cohort ELF score increased 0.3 between baseline and W48; [5] Defined as patients who have an improvement in liver fibrosis by ≥1 stage from baseline to W48;
[6] p-value=0.31 for change in ELF at W48 for 1 mg aldafermin; [7] p-value=0.39 for fibrosis improvement ≥1 stage for 1 mg aldafermin; [8] p-value=0.36 for fibrosis improvement ≥1 stage for 3 mg aldafermin
Analysis was performed in the intention-to-treat (ITT) population; NASH CRN=Nonalcoholic Steatohepatitis Clinical Research Network

Completed Phase 2 Trial of Aldafermin in PSC

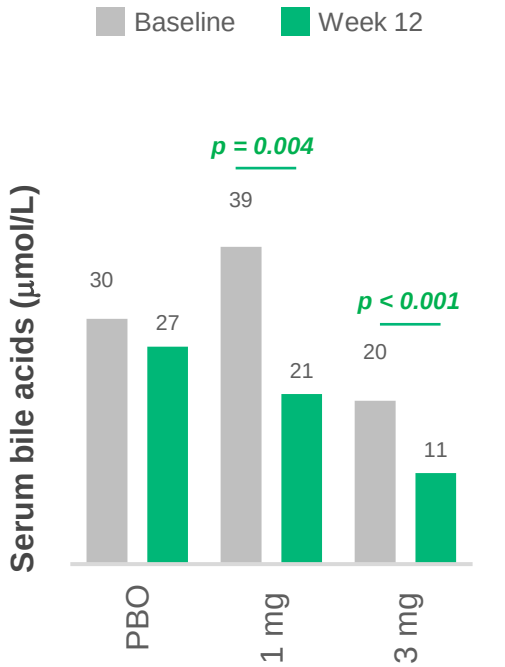


- Randomized (1:1:1), double-blinded, placebo controlled
- 62 subjects randomized at 27 sites in Europe and US
- Confirmed diagnosis of PSC by EASL Guidelines
  - Included subjects with features of AIH, small duct disease, stable dominant strictures and compensated cirrhosis
- ALP >1.5 x ULN, total bilirubin <2.5 mg/dL, ALT/AST <5 x ULN
- Stratified across groups by UDCA use
- Key endpoints:
  - Primary endpoint (was not met): Mean change in ALP from Baseline at Week 12
  - Secondary and exploratory endpoints: ELF, Pro-C3, ALT/AST

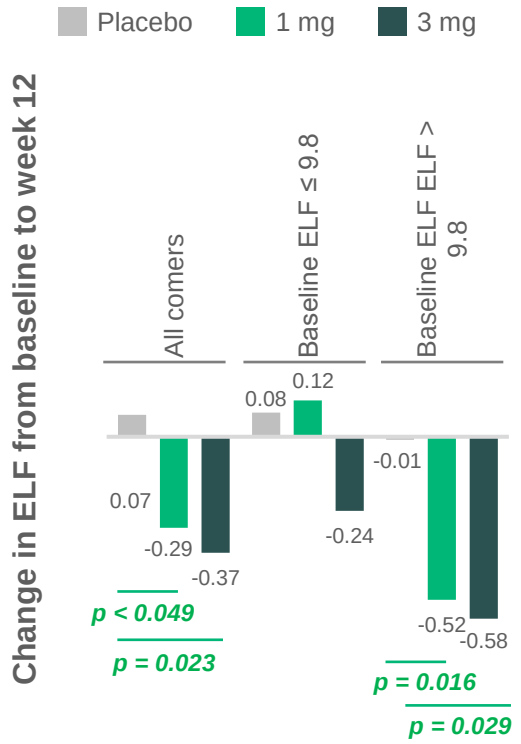
SC = Subcutaneous; QD = Once a Day; ALP = Alkaline Phosphatase; ALT = Alanine Transaminase; AST = Aspartate Transaminase; ULN = Upper Limit of Normal; UDCA = Ursodeoxycholic Acid; EASL = European Association for the Study of Liver; AIH = Autoimmune Hepatitis; Pro-C3 = Neo-epitope-specific Type III Collagen
Source: Hirshfield *et al. Journal of Hepatology* 2019

Although Primary Endpoint (ALP) Was Not Met in Prior Aldafermin PSC Trial, Dose-Dependent Reductions in Multiple Endpoints Were Seen

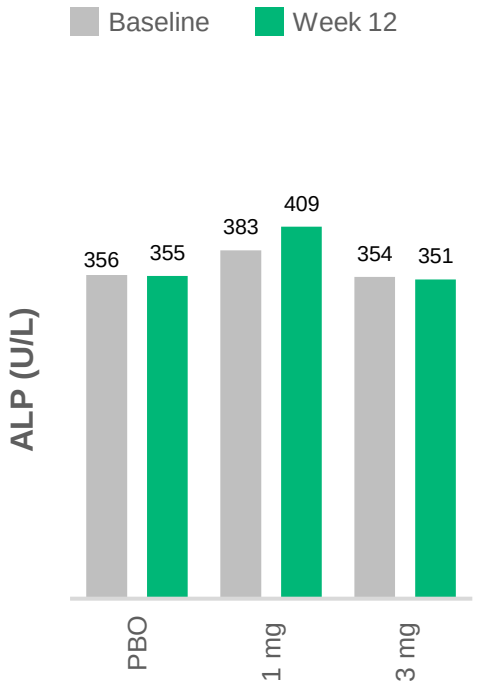
Bile Acids



ELF



ALP

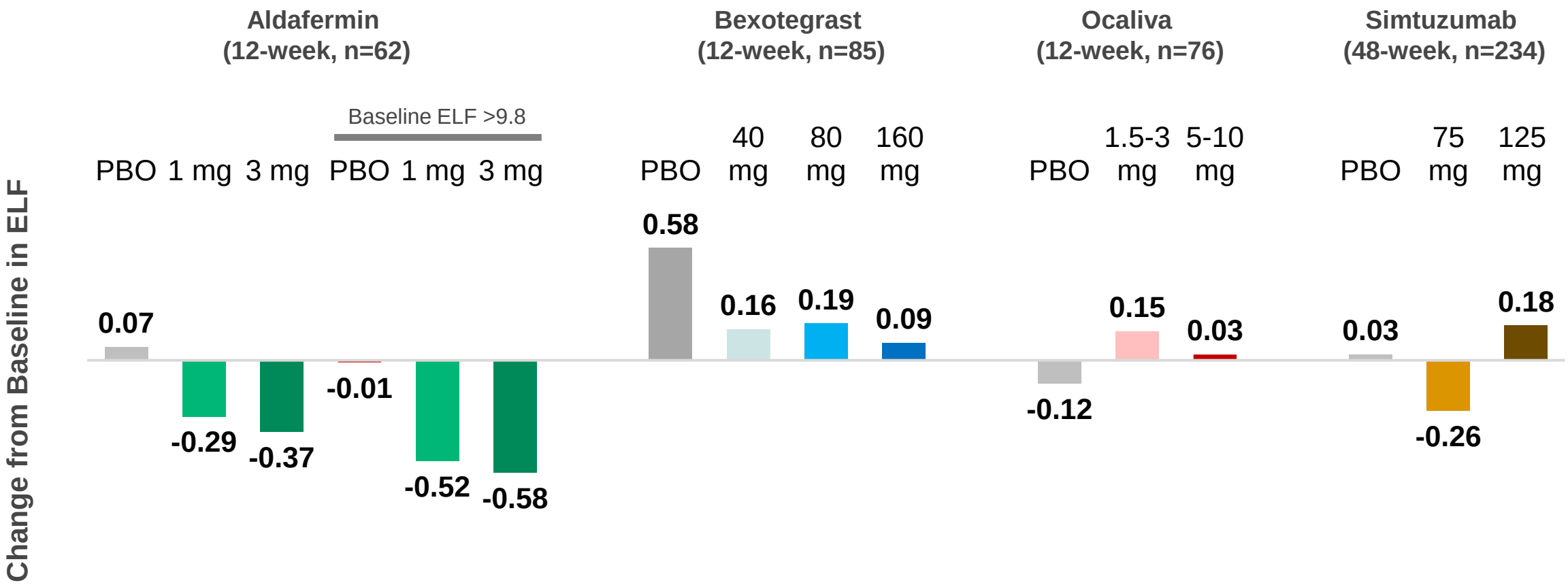


PBO = Placebo; NRS = Numerical Rating Scale
Source: Hirshfield *et al*. Journal of Hepatology 2019; Mayo *et al.*, *EASL* 2019

Dose-Dependent Reductions of Biomarkers Were Seen in Prior Aldafermin PSC Trial

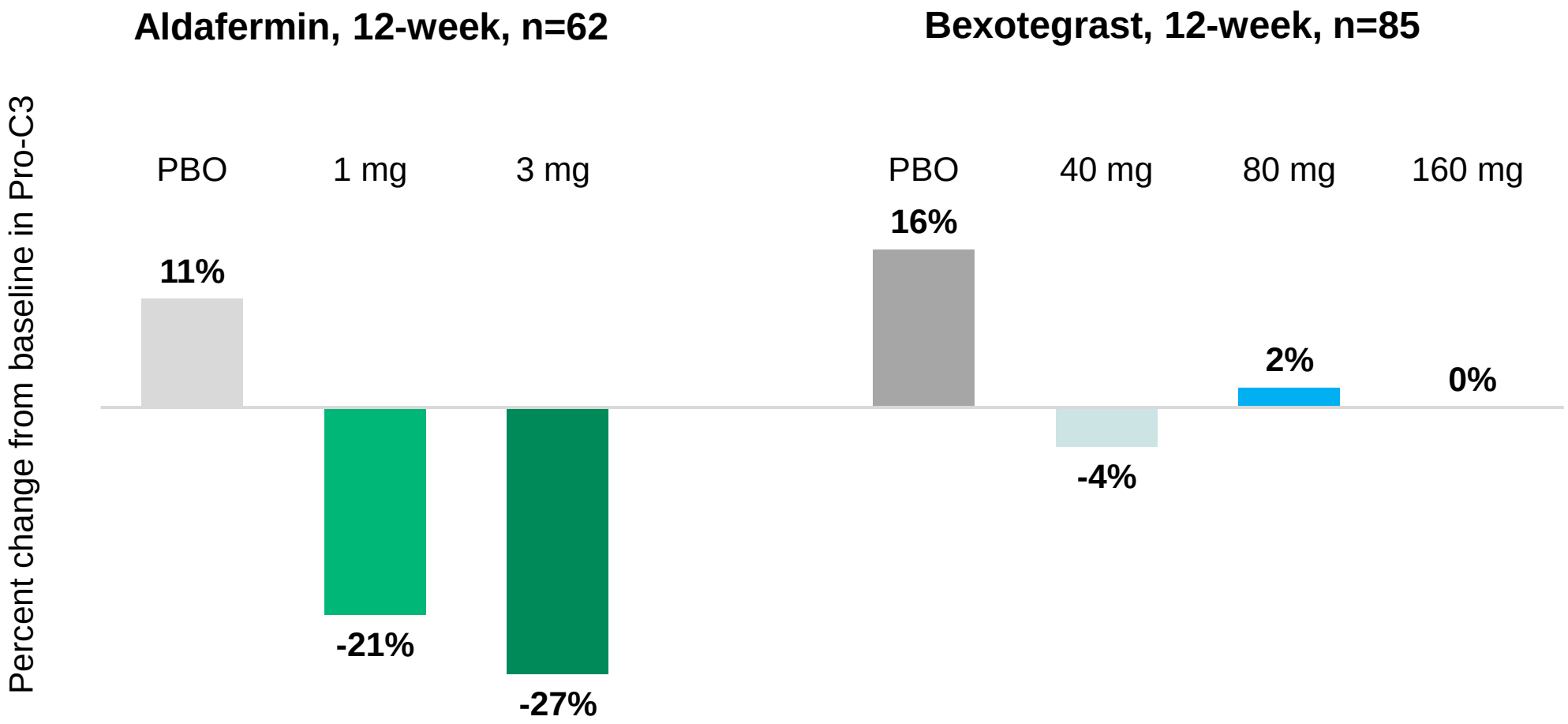
C4 = 7α-hydroxy-4-cholesten-3-one
Source: Hirshfield *et al. Journal of Hepatology* 2019

Aldafermin Demonstrated Dose-Dependent Reduction of Enhanced Liver Fibrosis (ELF) Biomarker in Prior PSC Trial



Source: Hirshfield *et al. Journal of Hepatology* 2019; Muir *et al.*, *Hepato*l 2019; Kowdley *et al.*, *J Hepato*l 2020; Company press releases and presentations
Certain data in this presentation are based on a cross-trial comparison and are not based on head-to-head clinical trials. Cross trial comparisons are inherently limited and may suggest misleading similarities or differences in outcomes. Results of head-to-head comparisons may differ significantly from those set forth herein.

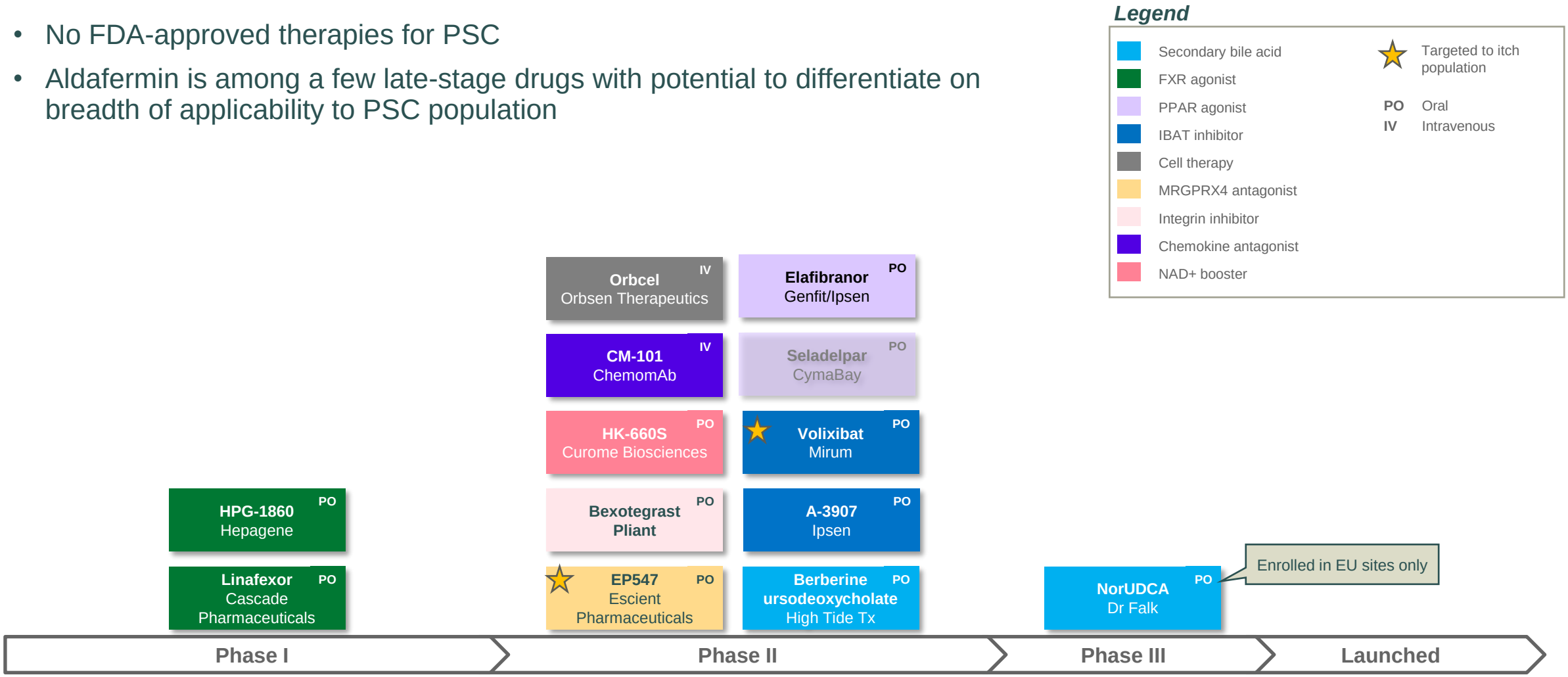
Aldafermin Also Significantly Decreased Pro-C3 (Marker of Fibrogenesis) in Prior PSC Trial



Source: Hirshfield *et al. Journal of Hepatology* 2019; Company press releases and presentations
Certain data in this presentation are based on a cross-trial comparison and are not based on head-to-head clinical trials. Cross trial comparisons are inherently limited and may suggest misleading similarities or differences in outcomes. Results of head-to-head comparisons may differ significantly from those set forth herein.

Development Landscape in PSC

- No FDA-approved therapies for PSC
- Aldafermin is among a few late-stage drugs with potential to differentiate on breadth of applicability to PSC population



FXR = Farnesoid X Receptor; PPAR = Peroxisome Proliferator-activated Receptor; IBAT = Ileal Bile Acid Transporter; MRGPRX4 = MAS Related GPR Family Member X4; NAD = Nicotinamide adenine dinucleotic

Source: Clinicaltrials.gov; Citeline; company websites; Trauner *et al. EASL* 2023

NGM120 for Hyperemesis Gravidarum

Significant Unmet Need in Hyperemesis Gravidarum (HG)

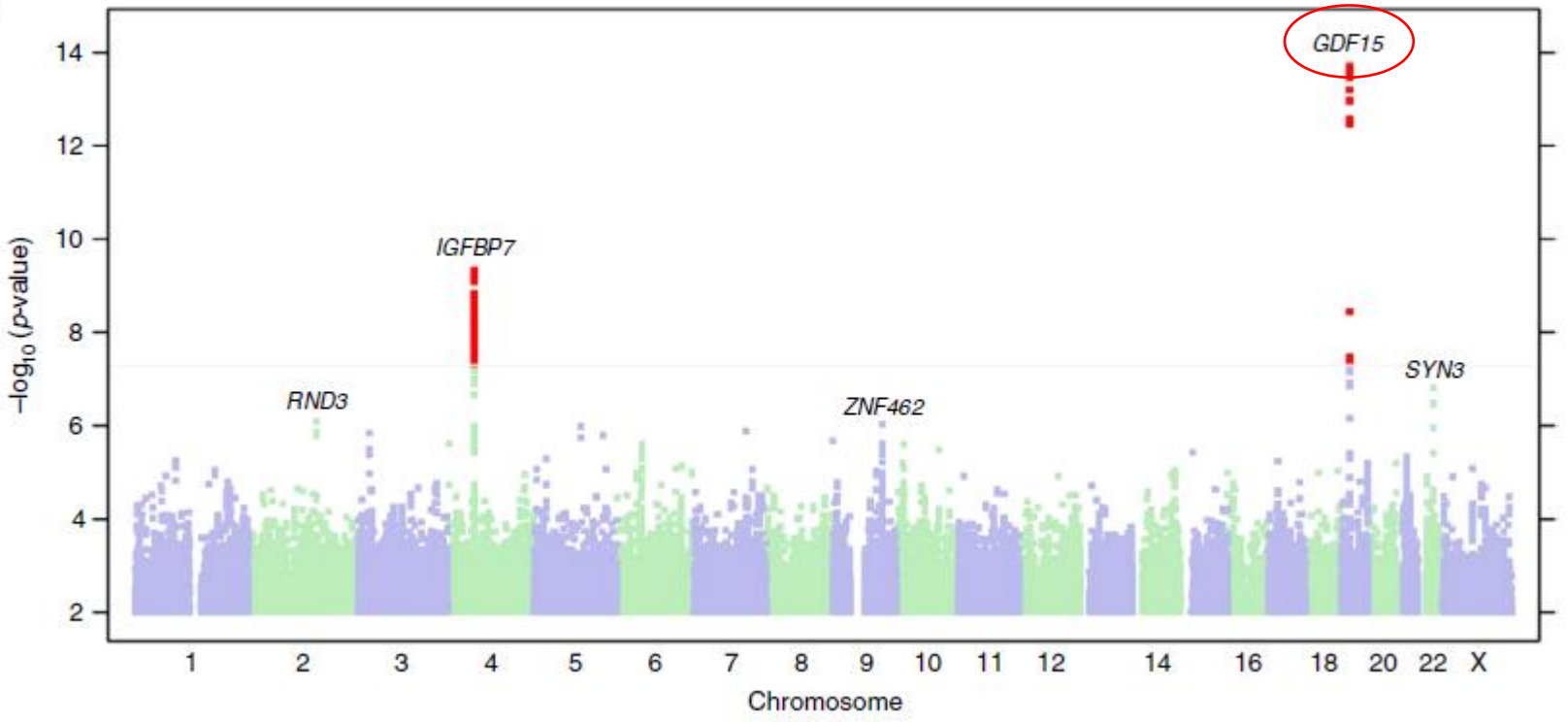
Photo credit: HER Foundation

- Hyperemesis gravidarum (HG) is a severe condition that affects ~100-150K patients in the US each year
- Characterized by intractable nausea and vomiting during pregnancy (which results in dehydration, debility, weight loss and malnutrition), HG takes a significant physical and psychosocial toll on patients. Consequently, HG can also lead to higher rates of fetal loss, preeclampsia, pre-term birth, low birth weight and malnutrition for the fetus
- HG patients may experience symptoms requiring hospitalization throughout the entire pregnancy and HG typically recurs in subsequent pregnancies
- HG is the second leading cause of hospitalization in pregnancy (second to pre-term labor) and is one of the costliest pregnancy complications to treat

Source: CDC; Fejzo *et. al. Nature Disease Primers 2019,* Einarson *et. al*. *J Popul Ther Clin Pharmacol*. 2013; Fiaschi *et. al. Human Reproduction* 2016; Fejzo *et. al., BJOG* 2022

Human Genetics Study Identifies GDF15 as a Risk Factor for HG

GDF15 Variants Associated with HG by GWAS



- In a Genome-Wide Association Study (GWAS) conducted with female participants, 1,306 participants had received IV fluids for nausea and vomiting during pregnancy (NVP), considered to be the HG cohort, and 15,756 participants reported no NVP symptoms
- The genetic locus most significantly associated with HG included the gene encoding GDF15

IV = intravenous
Source: Fejzo *et al. Nature Communications* 2018

NGM120 is an Antagonist Antibody Inhibiting GFRAL, the Receptor for GDF15

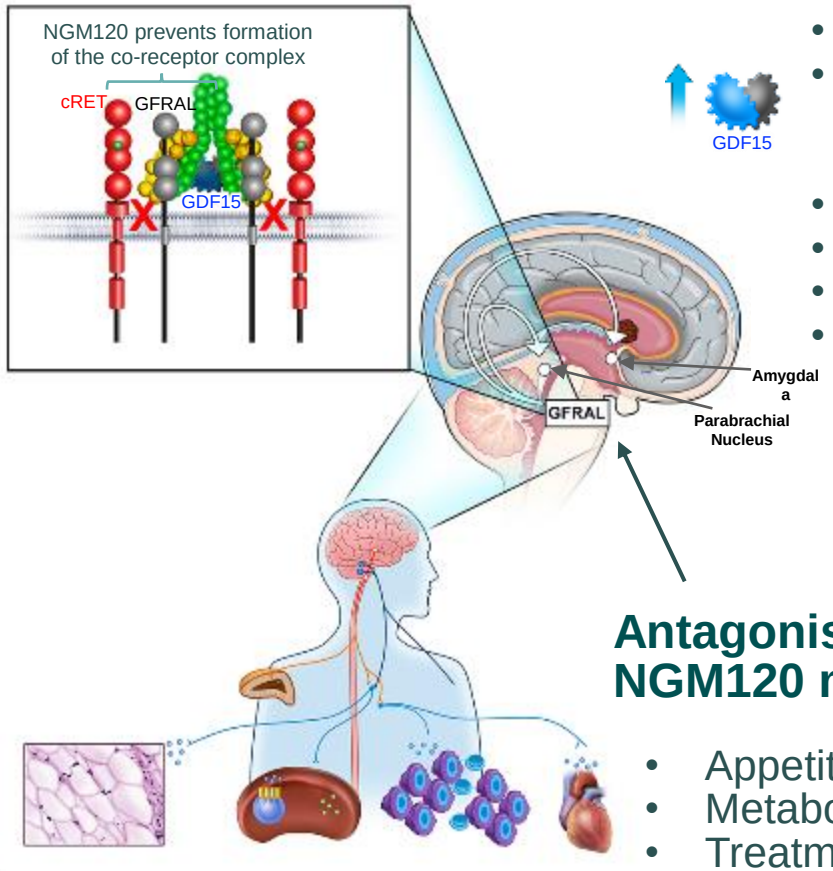
NGM was the first to identify GDF15's cognate receptor, GFRAL, and its associated signaling pathway[1]

Targeting GFRAL has the potential to ameliorate the metabolic and emetic effects caused by overstimulation of GFRAL neurons by excessive GDF15

Preclinical studies demonstrated that NGM120 can:

- Prevent cisplatin-induced GDF15-mediated weight loss in rodents
- Reduce cisplatin-induced weight loss and emesis in a cynomolgus monkey model

- **In healthy subjects**, NGM120 was generally well tolerated at 10-400 mg (single dose) and 10-200 mg (repeat dose, Q4W)
- **In a Phase 2 study of NGM120 in PDAC patients,** NGM120 was generally well tolerated at subcutaneous doses of 30 mg and 100 mg, every 3 or 4 weeks
- NGM is exploring initiation of a Phase 2 **proof-of-concept study** for the treatment of HG and is engaged in ongoing discussions with the FDA on an acceptable toxicology package



GDF15 levels are increased by:

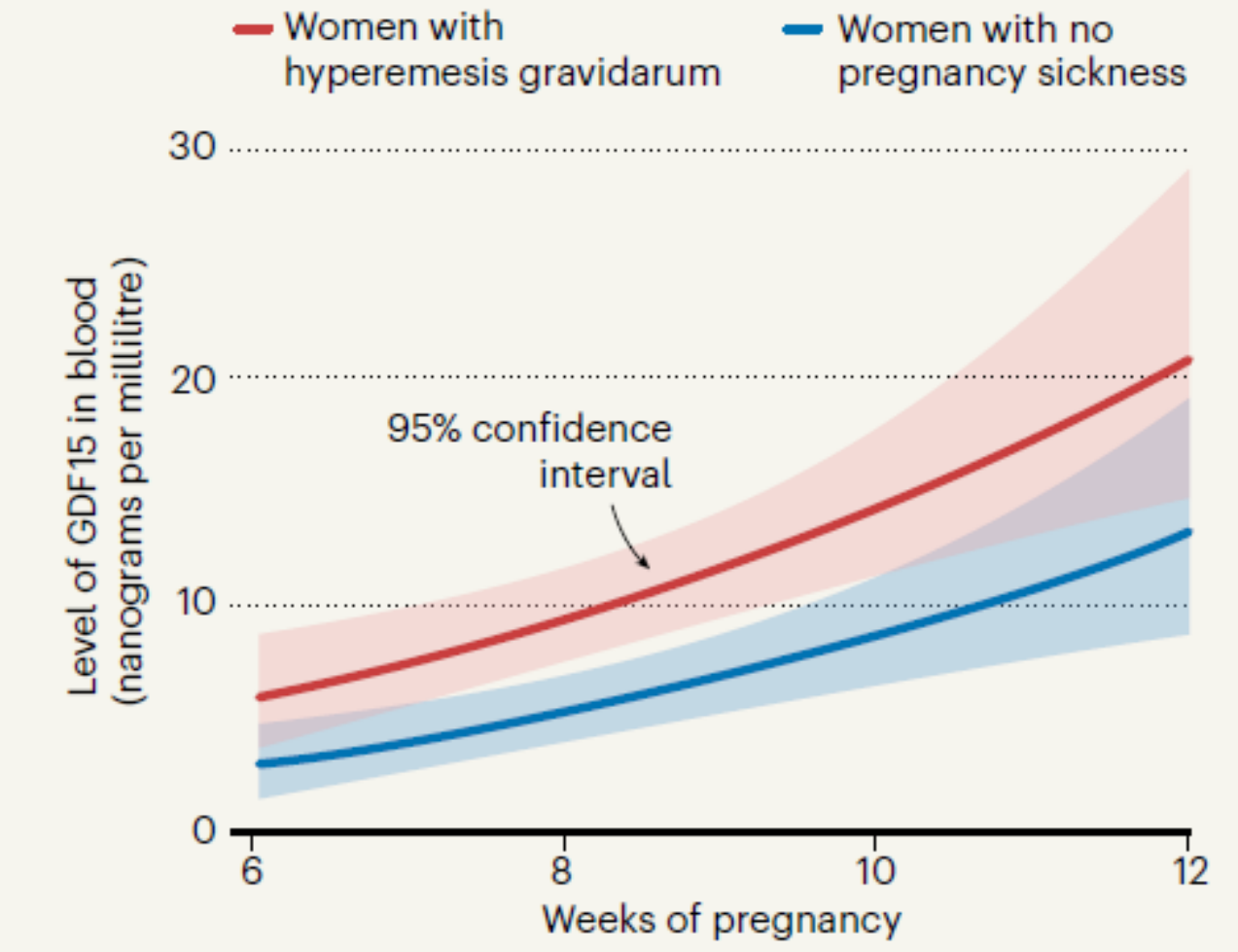
- Pregnancy
- Prolonged Nutritional Stress and Deficits
- Tumor
- Chemotherapy
- Infection
- Other stressors

Antagonism by NGM120 may result in:

- Appetite Regulation
- Metabolic Regulation
- Treatment of HG

[1]Hsu *et. al., Nature* 2017; Lockhart *et. al., Endocr Rev.* 2020
GFRAL = GDNF Family Receptor Alpha-Like

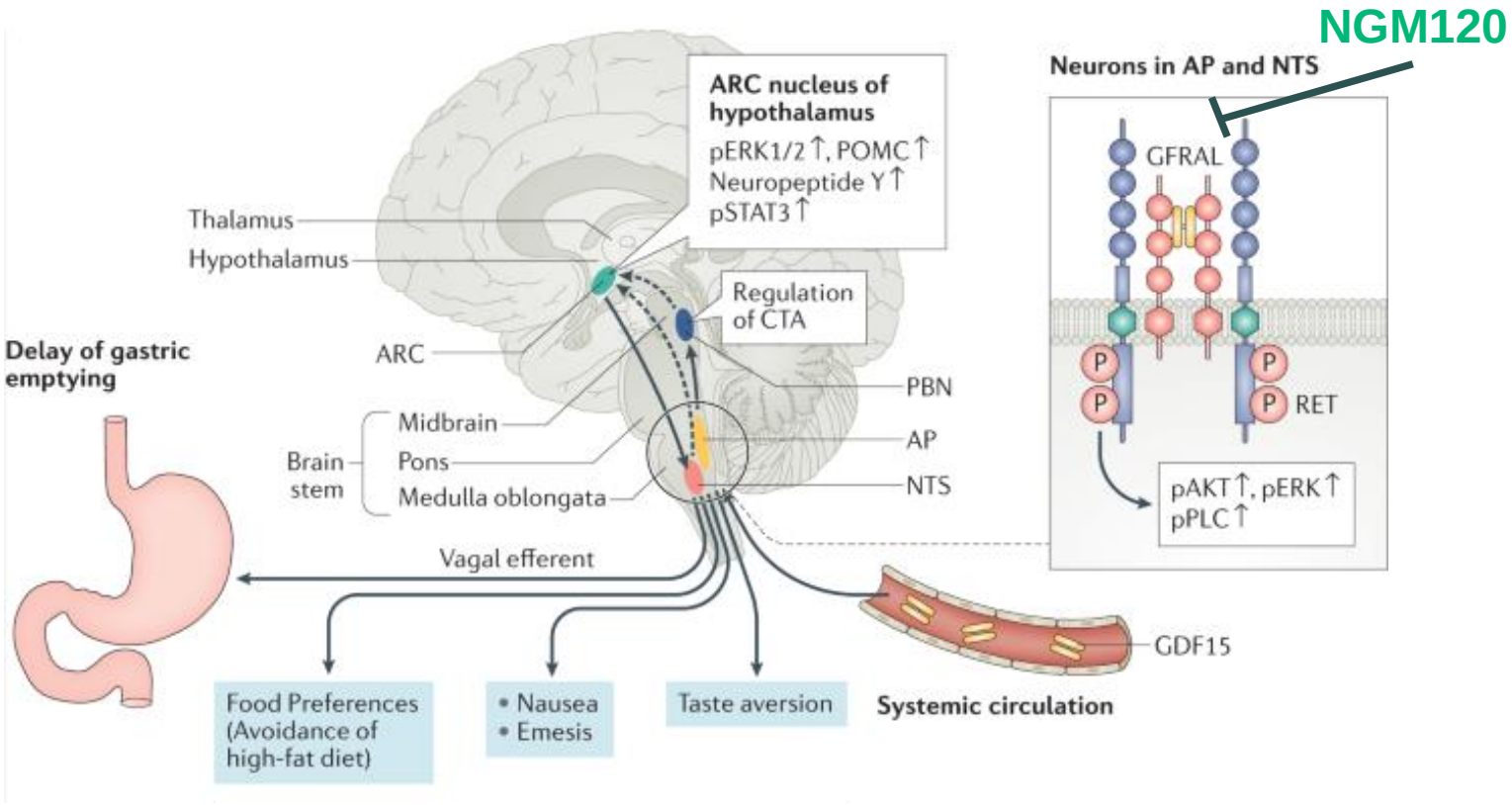
Fejzo *et. al.* Found that GDF15 Levels Increase Steadily in Early Pregnancy and are Higher in Women Who Experience NVP and HG



- In pregnancy, GDF15 levels increase steadily in the first 12 weeks
- On average, pregnant women who experience nausea, vomiting or HG have higher levels of GDF15 in their bloodstream

Source: Fejzo *et al., Nature* 2023; Hughes and Freathy *Nature* 2023

Inhibition of GDF15/GFRAL by NGM120 is Hypothesized to Reduce Nausea and Vomiting



- NGM120 inhibits GFRAL, which is exclusively expressed in the hindbrain in the area postrema (AP) and nucleus tractus solitarius (NTS)
- The AP, located outside of the blood brain barrier, is a well-known chemoreceptor trigger zone for nausea and vomiting
- GDF15 dosing has been shown to trigger vomiting in preclinical studies, including with non-human primates (NHP) and musk shrews
- In a clinical study of NGM395 (GDF15 analog), there was evidence of a dose-dependent increase in frequency and severity of nausea and vomiting

Source: Wang *et al., Nature Reviews Endocrinology* 2021; Breen *et. al., Cell Metabolism* 2020

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