Biology-driven discovery.
Life-changing medicines.

Corporate Overview

January 2022

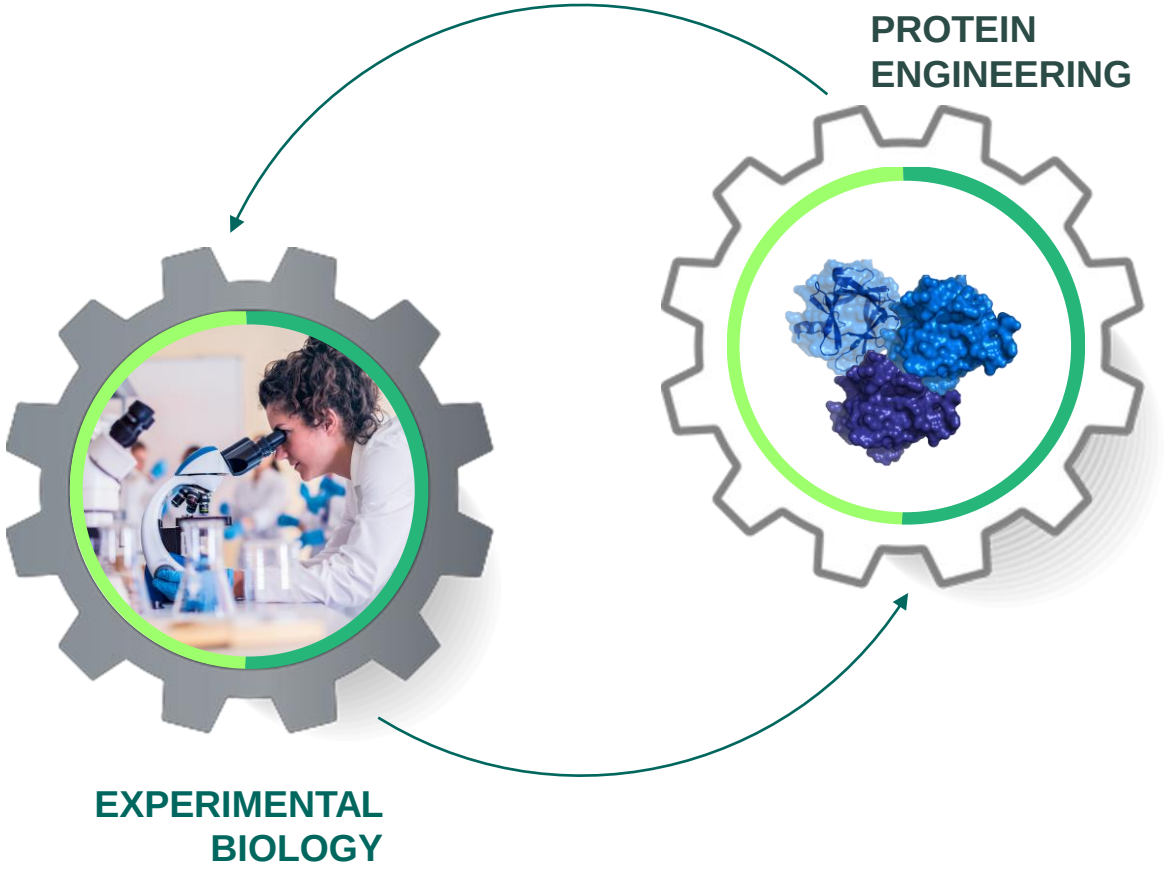
NASDAQ: NGM

©2022 NGM Bio

Safe Harbor Statement

The following presentation contains forward-looking statements, including, but not limited to, statements regarding potential indications for, planned and continued development of, and therapeutic potential of, product candidates in NGM Bio's pipeline, including NGM120, NGM707, NGM831, NGM438, NGM621, MK-3655 and aldafermin; the planned timing of initiation, enrollment, data readouts and results of NGM Bio's clinical trials, including with respect to topline data for NGM621; potential future late-stage development of product candidates in NGM Bio's pipeline, including NGM621 and aldafermin; the potential activity, complementarity, safety, tolerability and efficacy of NGM's product candidates, including the potential of NGM Bio's oncology product candidates to become next-generation treatment options; NGM Bio's belief that myeloid cell reprogramming can be an important additional approach to augment anti-tumor immunity and that its portfolio of product candidates provide multiple opportunities to harness that biology; the design of NGM Bio's and Merck's clinical trials of NGM's product candidates; the preliminary findings in the Phase 1a/1b study of NGM120 providing encouraging initial signals of anti-cancer activity; the preliminary findings in the Phase 1a study of NGM707 and anticipated timing thereof; the availability and anticipated timing of topline data from Phase 2 CATALINA study of NGM621 in patients with geographic atrophy; the continuation of the Phase 2b ALPINE 4 trial of aldafermin; potential activities under NGM's amended collaboration with Merck and the potential receipt of milestone and royalty payments by NGM under the amended collaboration with Merck; the potential roles of regulating the GDF15/GFRAL pathway and ILT2, ILT4, ILT3 and LAIR1 in cancer, the powerful biology of the GDF15 pathway, the potential consequences of ILT2, ILT4, ILT3 and LAIR1 blockade and the opportunity for next generation myeloid checkpoint inhibitors to address limitations of existing immunotherapies; and the potential for NGM621 to have an opportunity for differentiation for the treatment of geographic atrophy and opportunities to achieve category leadership, and its potential for every 8-week dosing; potential option exercises by Merck under NGM Bio's amended collaboration with Merck; NGM Bio's opportunities for value creation and its ability to deliver powerful treatments; NGM Bio's strategy, including its myeloid reprogramming strategy, and potential impact of its portfolio prioritization; NGM Bio's potential near-term catalysts; and any other statements other than statements of historical facts. Because such statements deal with future events and are based on NGM Bio's current plans, objectives, estimates and expectations, they are subject to various significant risks and uncertainties and actual results, performance and achievements and the timing of events could differ materially from those described in or implied by the statements herein. Such risks and uncertainties include, without limitation, those associated with the costly and time-consuming pharmaceutical product development process and the uncertainty of clinical success, including risks related to failure or delays in successfully initiating, enrolling or completing clinical studies, the risk that NGM Bio's ongoing or future clinical studies in humans may show that NGM Bio's product candidates are not tolerable or effective treatments, the risk that preclinical studies or modeling may not be indicative of results in future human clinical trials, the risk that preliminary results from clinical studies may not be predictive of the final results of such studies, the risk that success in earlier-stage clinical studies does not ensure that later clinical trials evaluating NGM Bio's product candidates will generate the same results or otherwise provide adequate data to demonstrate the effectiveness and safety of such product candidates, and the risk that others may discover, develop or commercialize products before or more successfully than NGM Bio, including in NASH and/or geographic atrophy (GA); the ongoing COVID-19 pandemic which has adversely affected, and could materially and adversely affect in the future, NGM Bio's business and operations, including NGM Bio's ability to timely supply, initiate, enroll and complete its ongoing and future clinical trials; the time-consuming and uncertain regulatory approval process, including the risk that NGM Bio or Merck, as applicable, may not receive marketing approvals for any of NGM Bio's product candidates in a timely manner, or at all; seeking and maintaining protection of intellectual property; NGM Bio's reliance on third party manufacturers and delays or problems in the manufacture or testing of product candidates; NGM Bio's dependence on its amended collaboration with Merck for the development and potential commercialization of product candidates falling within the scope of the amended collaboration and its ability to maintain the amended collaboration, including the risk that if Merck were to breach or terminate the amended collaboration or Merck's development funding obligations thereunder, NGM Bio would not obtain all of the anticipated financial and other benefits of the amended collaboration, and the development and/or commercialization of NGM Bio's product candidates falling within the scope of the amended collaboration could be delayed, perhaps substantially; the sufficiency of NGM Bio's cash resources, including to fund development programs that fall outside of the narrower scope of NGM Bio's amended collaboration with Merck, and need for additional capital; and other risks and uncertainties affecting NGM Bio and its research and development programs, including those described under the caption "Risk Factors" and elsewhere in NGM Bio's quarterly report on Form 10-Q for the quarter ended September 30, 2021 filed with the United States Securities and Exchange Commission (SEC) on November 4, 2021 and future filings and reports of NGM Bio with the SEC. 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.

Our Approach Integrates Biology and Protein Engineering Expertise into the Drug Discovery and Development Process

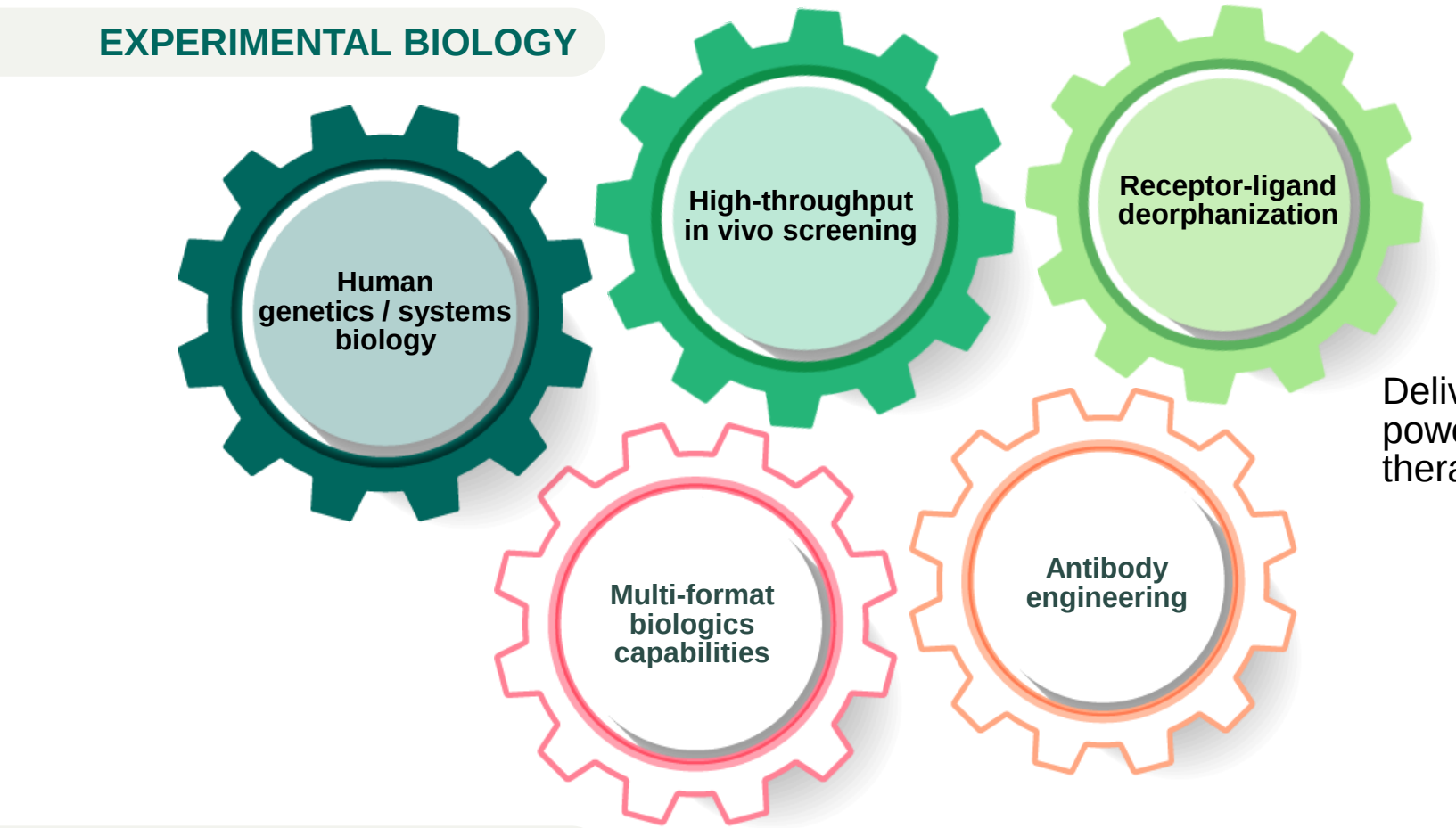


Delivering powerful therapies

EXPANSIVE PIPELINE

- **3** Therapeutic Areas
- **7** Disclosed Programs
- **5** Programs in Clinical Development
- **4** Ph2/Ph2b Studies Ongoing

Reproducible Drug Discovery Process Has Been Applied Successfully Across Biological Frontiers



Delivering powerful therapies

7 Disclosed Programs

- NGM 707
- NGM 831
- NGM 621
- MK 3655
- NGM 438
- NGM 120
- Aldafermin

Our Expansive Pipeline

[1] Phase 1a cohort = monotherapy; Phase 1b cohort = in combination with standard-of-care treatment of gemcitabine + Nab-paclitaxel
[2] At NGM's option at Phase 3
NASH = non-alcoholic steatohepatitis; FGF = fibroblast growth factor; KLB = klotho beta; C3 = Component 3; GFRAL = glial cell-derived neurotrophic factor receptor alpha-like; ILT2 = Immunoglobulin-like transcript 2; ILT4 = Immunoglobulin-like transcript 4; ILT3 = Immunoglobulin-like transcript 3; LAIR1 = Leukocyte-associated immunoglobulin-like receptor 1; F2/F3/F4 = stage 2 or 3 or 4 liver fibrosis; PoC = proof of concept

Looking Forward to Multiple Program Milestones in 2022

Program	Mechanism	Status	Anticipated Milestones
NGM621 Geographic Atrophy	Anti-Complement C3 Antibody	Ph2 CATALINA trial fully enrolled	**Topline Ph2 CATALINA data readout in 4Q22**
NGM707 Advanced Solid Tumors	ILT2/ILT4 Dual Antagonist Antibody	Ph1/2 trial enrolling	**Initial Ph1a clinical data readout in 2H22**
NGM831 Advanced Solid Tumors	ILT3 Antagonist Antibody	Preclinical	**Initiation of Ph1 trial in 1Q22**
NGM438 Advanced Solid Tumors	LAIR1 Antagonist Antibody	Preclinical	**Initiation of Ph1 trial in 2Q22**
NGM120 Cancer and Cachexia	GFRAL Antagonist Antibody	Ph2 trial enrolling Ph1a/1b trial ongoing	**Additional Ph1a/1b clinical data readouts in 2H22**
Aldafermin Cirrhotic NASH	FGF19 Analog	Ph2b ALPINE 4 trial enrolling	**Last Patient In (LPI) in 1Q22**
MK-3655 Non-cirrhotic NASH	FGFR1c/KLB Agonist Antibody	Merck-led global Ph2b trial enrolling	**Ongoing enrollment**

NGM Bio's Myeloid Reprogramming Strategy to Treat Solid Tumors

NGM707, NGM831, NGM438

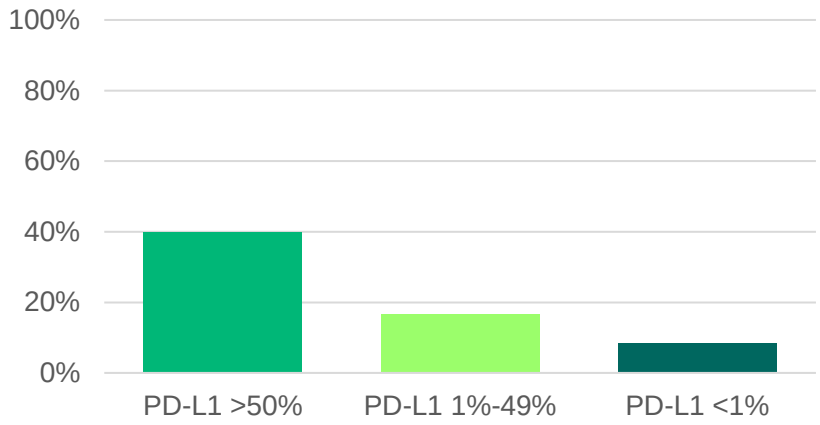
While T Cell Checkpoint Inhibitors Have Advanced the Treatment of Cancer, There is Opportunity to Improve Breadth / Depth of Response

Breadth of response: Patient response to PD-1 therapies are limited and dependent on PD-L1 expression levels

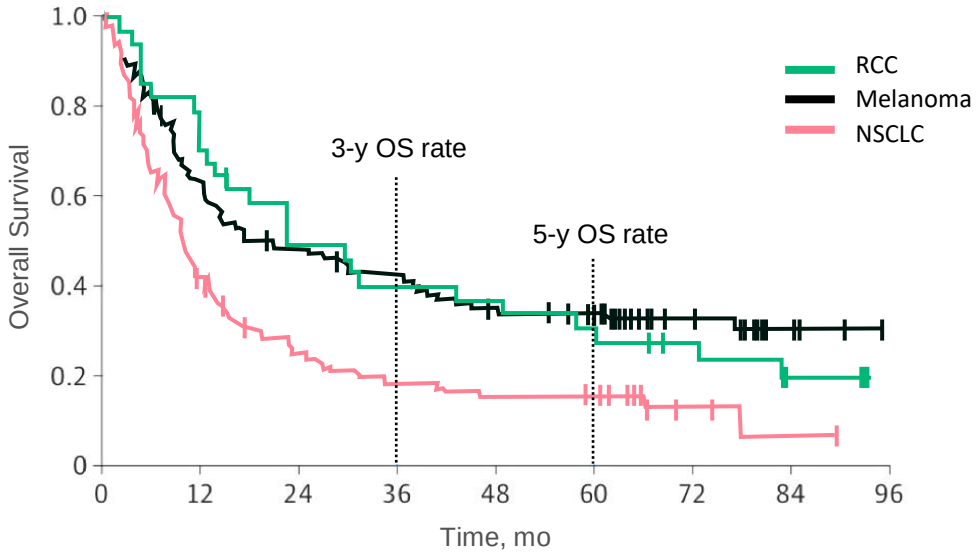
Depth of response: Amongst responders there is opportunity to increase duration of response

PD-1 Response Rate Dependent on PD-L1 Expression

ORR (%) to PD-1 Antagonist in Advanced NSCLC



Long-term survival following nivolumab treatment in melanoma, RCC, NSCLC

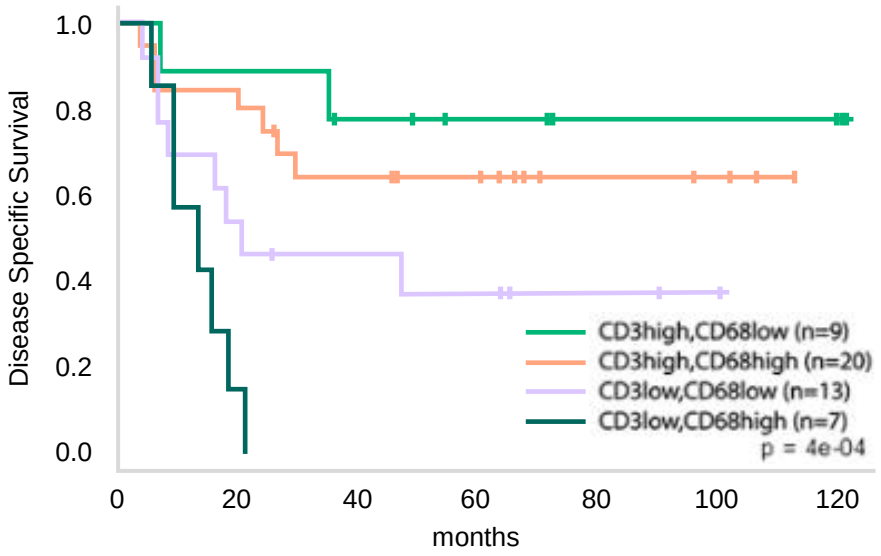


Man et al. *JNCI Spectrum*, 2021; Topalian, Sznol *et al.* - JAMA Oncol 2019
PD-1 = programmed cell death protein 1; PD-L1 = programmed death-ligand 1; ORR = overall response rate; NSCLC = non-small cell lung cancer; RCC = renal cell carcinoma

Modulation of Myeloid Checkpoint Inhibitors Has the Potential to Be a Next Wave in Immuno-oncology Treatment

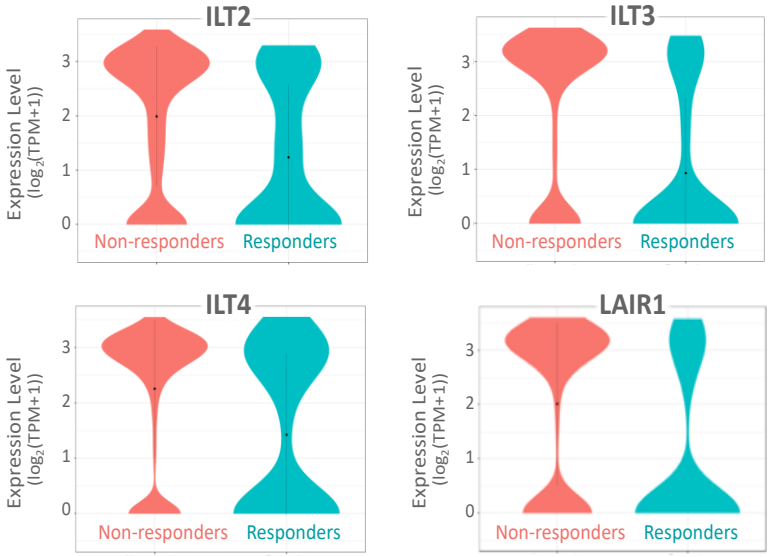
Myeloid-enriched tumors have poor prognosis

High CD68:CD3 ratio is associated with poor survival



Elevated ILT2, ILT3, ILT4, LAIR1 expression in macrophages from CPI R/R melanoma tumors

T cell CPI responders (blue) have lower levels of ILT2, ILT3, ILT4 and LAIR1 expression

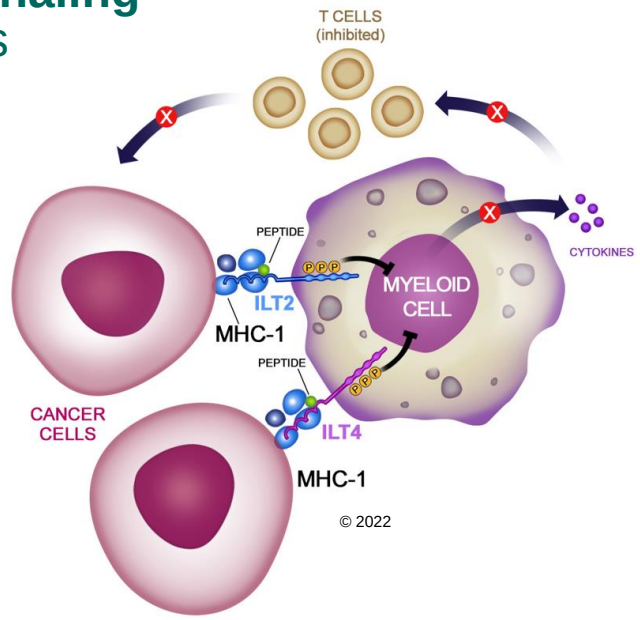


Significant opportunity for next generation myeloid checkpoint inhibitors to address limitations of existing immunotherapies

Sjodahl, 2014; Wang *et al.,* 2018, Sade-Feldman, *et al.*, 2019, GSE120575 – scRNAseq analysis
CD68 = Cluster of Differentiation 68; CD3 = Cluster or Differentiation 3; CPI = check-point inhibitors; R/R = relapsed/refractory

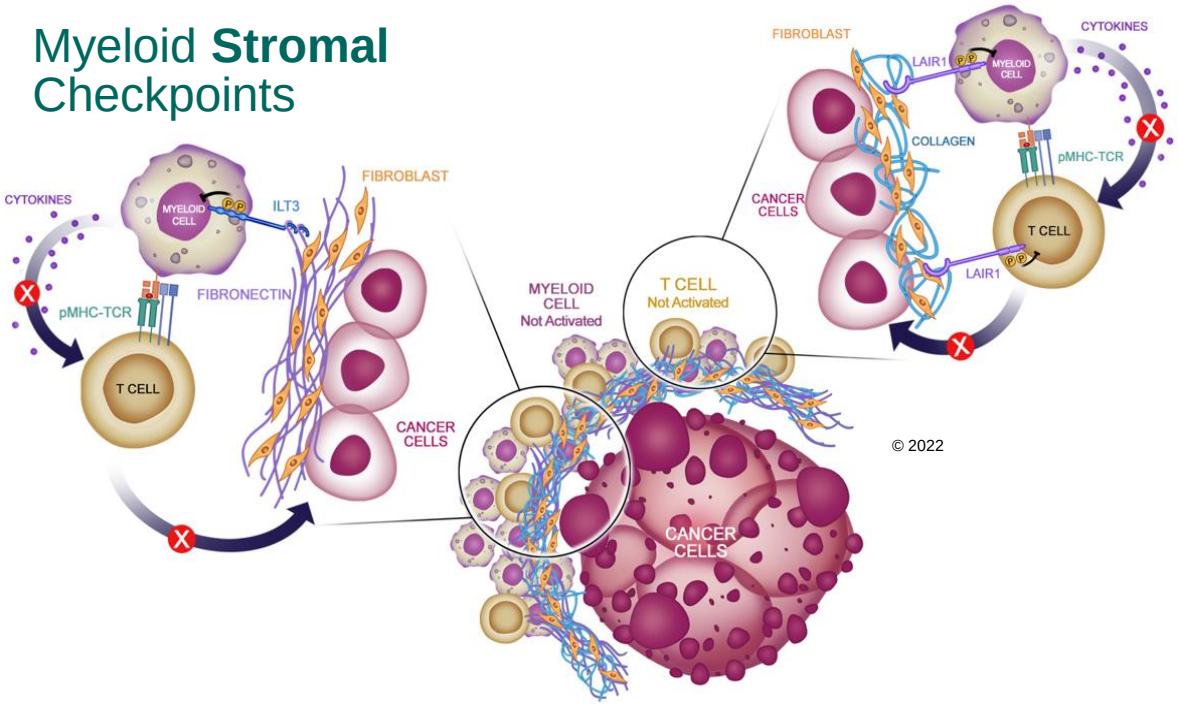
NGM Bio is Targeting Inhibitory Receptors on Myeloid Cells to Attempt to Restore Immune Response Against Tumors

Myeloid **Signaling** Checkpoints



NGM707	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	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).

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]

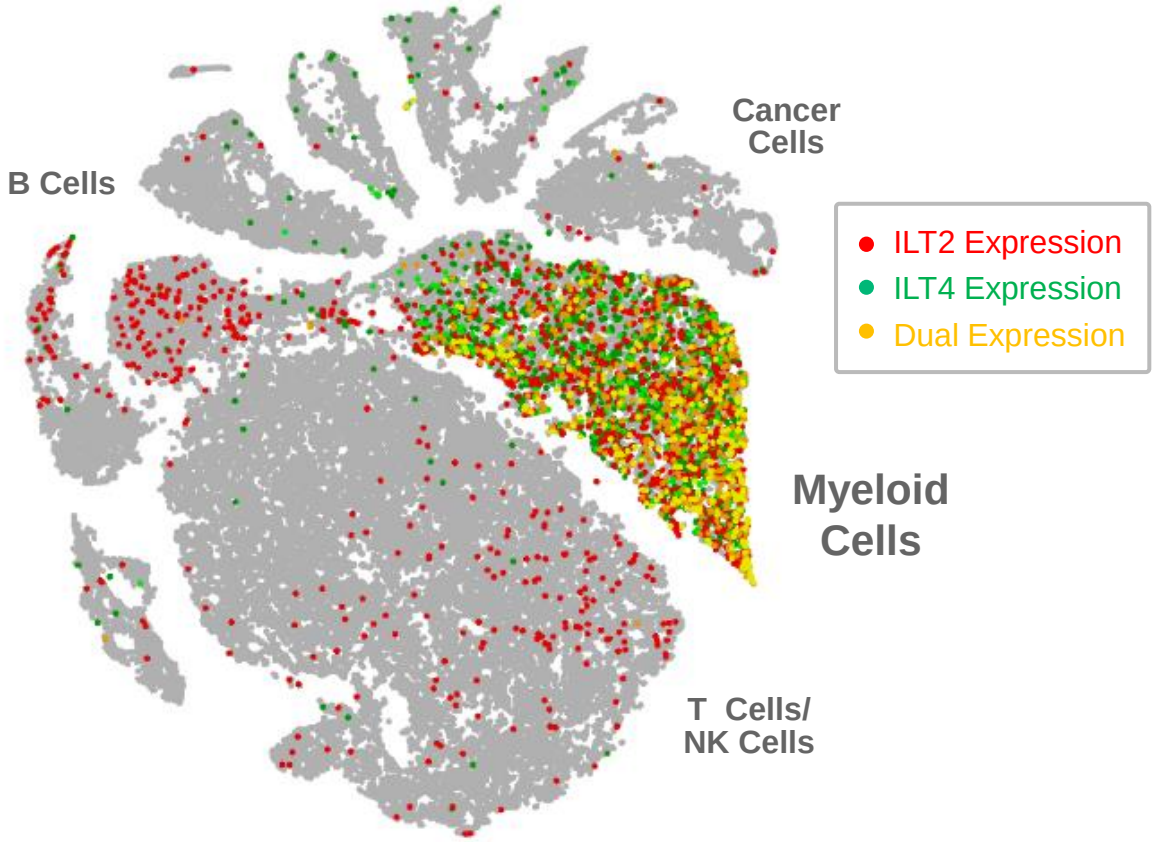
- ILT2 and ILT4 receptors are expressed on myeloid cells (APCs, MDSCs, macrophages, granulocytes) in the tumor microenvironment
- ILT2 additionally exhibits expression on natural killer (NK) cells, B cells and a subset of highly cytolytic T cells

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 that Entered the Clinic in 2021

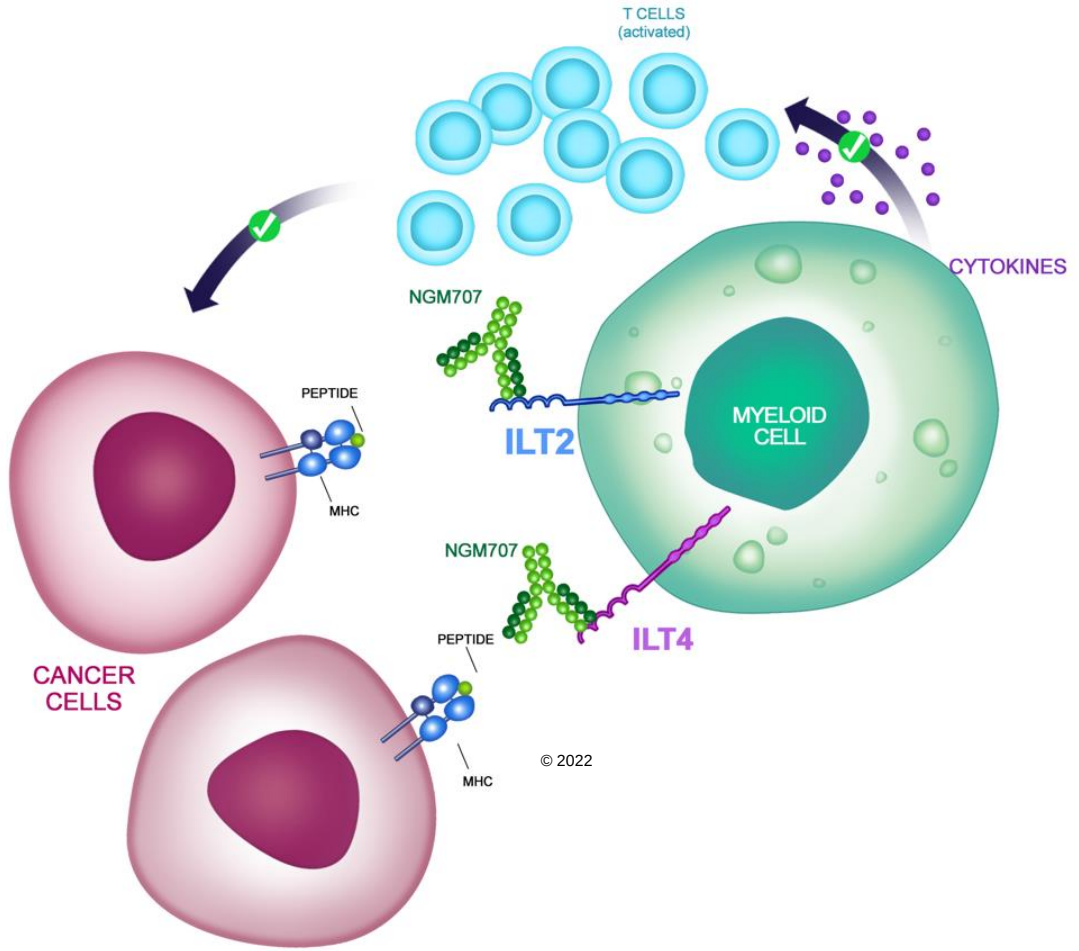
Potent, first-in-class antibody targeting the myeloid-enriched inhibitory receptors ILT2 (LILRB1) and ILT4 (LILRB2)

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**

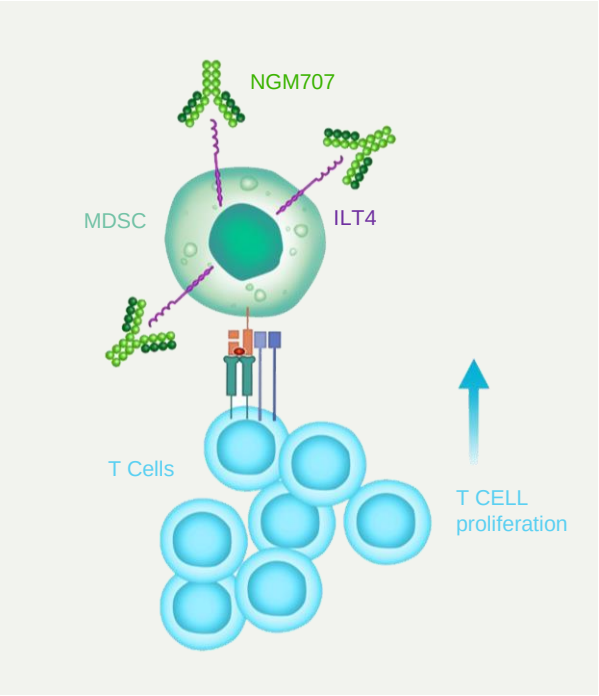
Ph1/2 first-in-human trial of NGM707 initiated in mid-2021



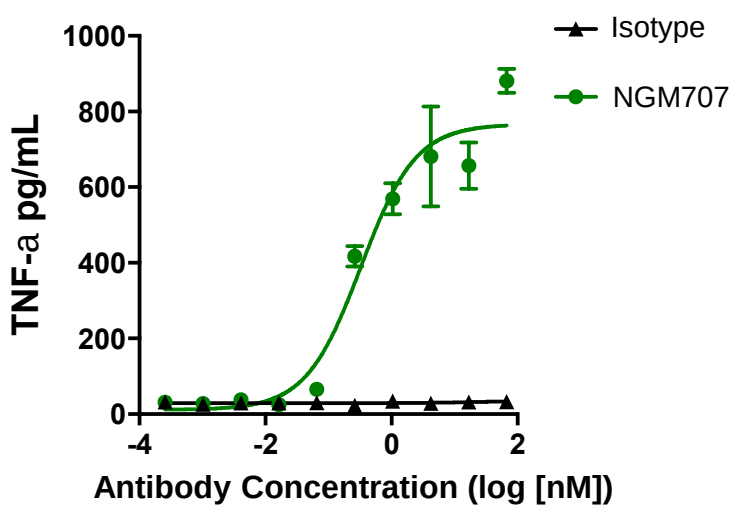
NK = Natural killer; FC = fragment crystallizable; MHC = major histocompatibility complex; TNF = Tumor necrosis factor; IL = interleukin; GM-CSF = Granulocyte-macrophage colony-stimulating factor; LILRB = Leukocyte immunoglobulin-like receptor subfamily B

ILT4 Blockade Reprograms Tumor-conditioned Myeloid-derived Suppressor Cells (MDSC)

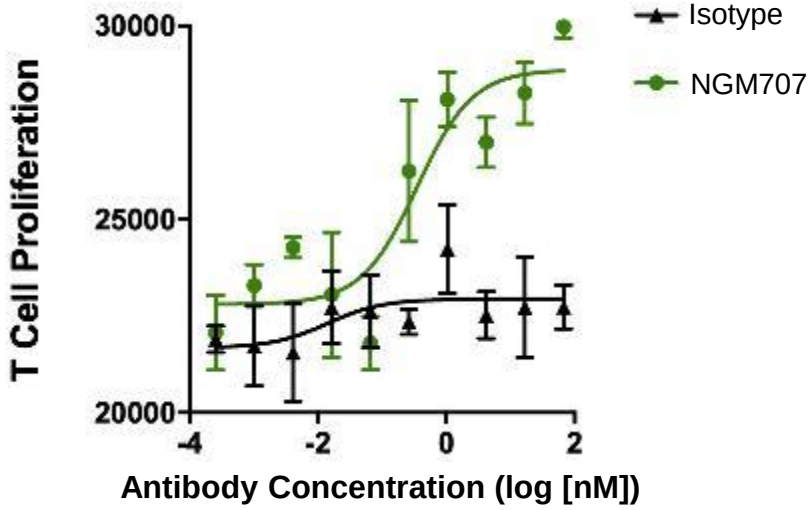
ILT4 antagonism enhances T cell activity and proliferation



T Cell Activation



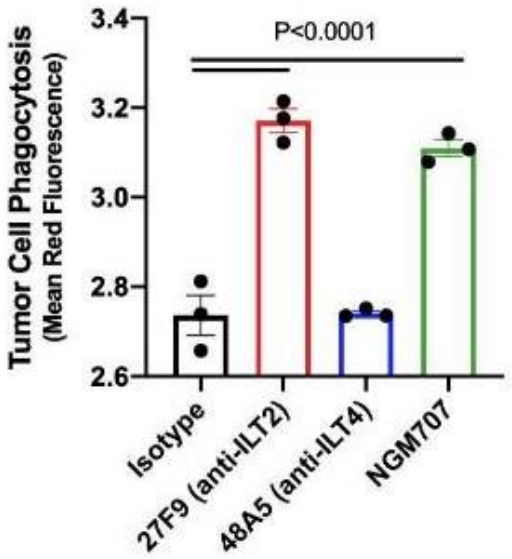
T Cell Proliferation



MDSC generated from monocytes using cancer cell-conditioned media (OVISE cells)
MLR performed by mixing MDSC with allogeneic T cells

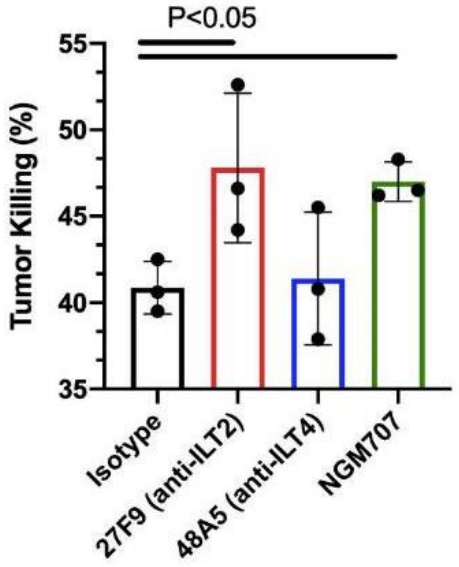
ILT2 Blockade Enhances Macrophage Phagocytosis of Tumor Cells, CD8+ T Cell Cytolytic Activity and Primary NK Cell Killing Activity

Tumor Cell Phagocytosis



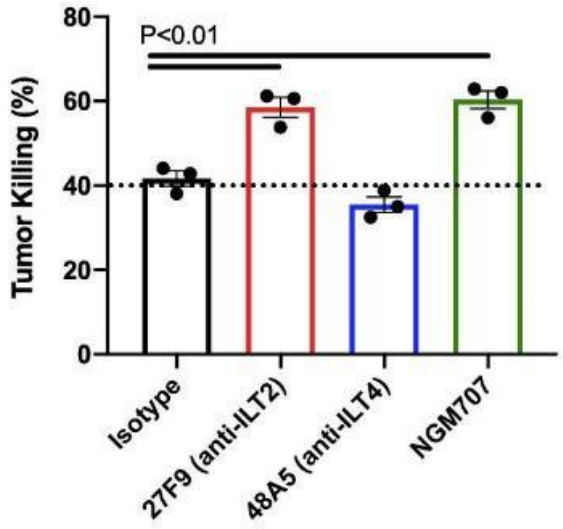
- Macrophage phagocytosis may increase tumor killing and potentially drive antigen spread
- Activity is specific to ILT2/MHC-I interaction despite ILT4 co-expression on macrophages

NGM707 enhanced CD8+ T cell cytolytic activity against a tumor B cell line expressing HLA-G



- ILT2 is expressed on (TEMRA CD8+ T cells)
  - TEMRA cells represent a highly cytolytic T cell subset
  - Expression distinct from PD-1, TIM3, LAG3 expression on exhausted T cells

NGM707 enhances primary NK cell tumor killing activity



- ILT2 blockade enhances primary NK cell killing activity

NGM Bio unpublished data
HLA-G = human leukocyte antigen G; TIM3 = T cell immunoglobulin and mucin domain-containing protein 3 ; LAG3 = lymphocyte-activation gene 3

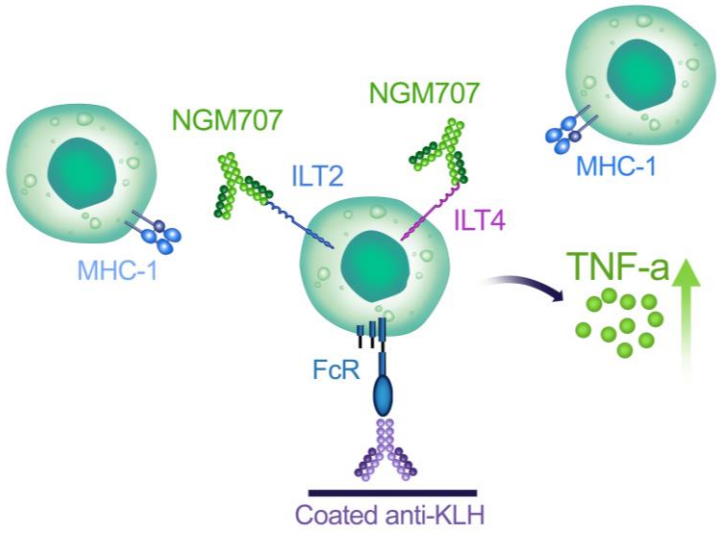
Preclinical Models Suggest That ILT2 and ILT4 Blockade May Act Additively to Enhance Myeloid Cell Activation

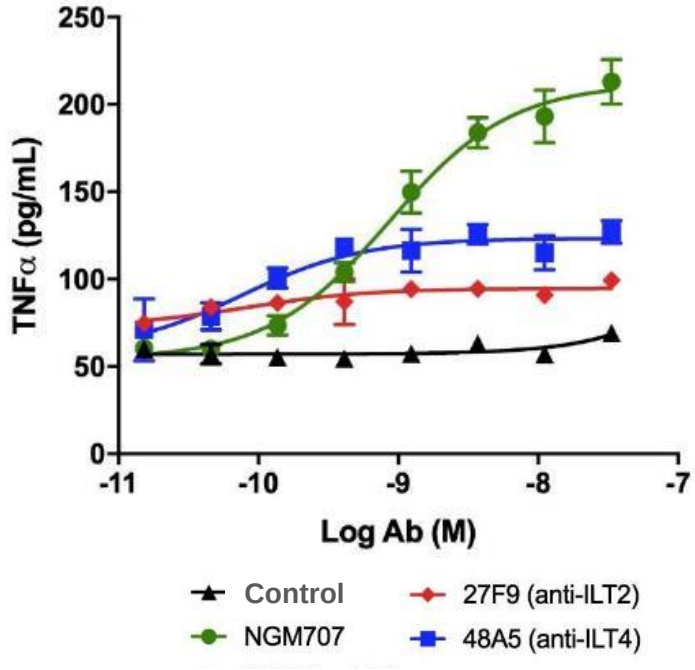
Fc receptors represent key stimulatory receptors on myeloid cells

- Inhibition of Fc receptor signaling by ILT2 and ILT4 promotes a suppressive myeloid cell phenotype

Dual blockade of ILT2 and ILT4 strongly potentiates Fc receptor signaling

- Blockade of ILT2 or ILT4 alone leads to a modest increase in Fc receptor signaling





Dual blockade of ILT2 and ILT4 may be more effective than blockade of either receptor alone in reversing suppression of Fc receptor signaling

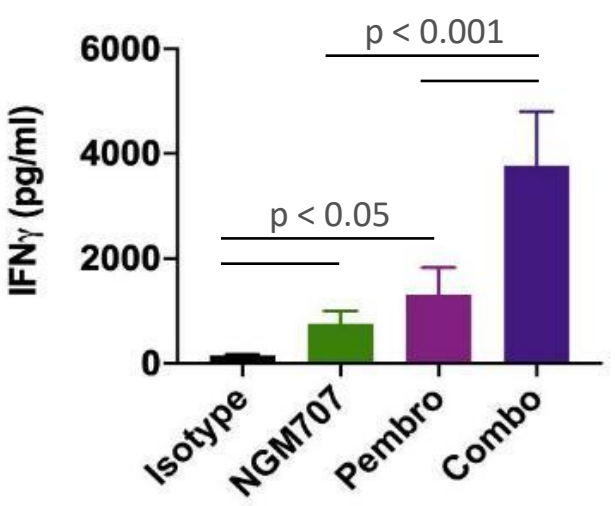
NGM707 and Pembrolizumab Acted Additively to Enhance T Cell Activation in a Mixed Lymphocyte Reaction

NGM707 or pembrolizumab alone **modestly** enhanced T cell activation and increase in cytokine secretion (IFNɣ, IL-2, TNFa, GM-CSF)

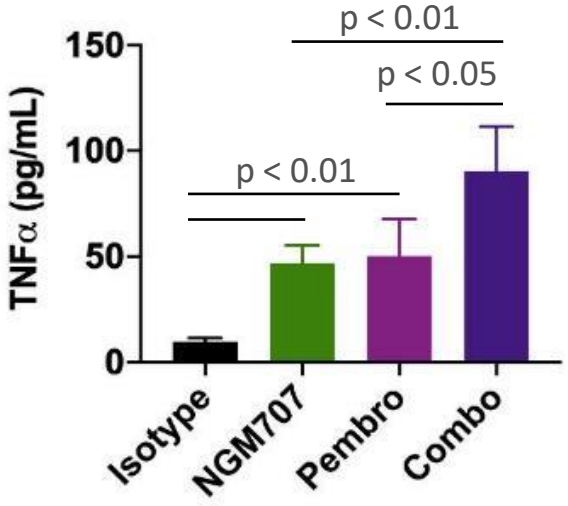
Combination of NGM707 and pembrolizumab led to an **additive** increase in T cell activation and cytokine secretion

Monocytes from two individuals differentiated into macrophages and tested in mixed lymphocyte reactions with T cells from 12 individuals

IFNɣ



TNFα



NGM Bio unpublished data
IFNɣ = Interferon Gamma; TNFα = Tumor Necrosis Factor alpha, Pembro= pembrolizumab, Combo = pembrolizumab and NGM707

17

NGM831 in Advanced Solid Tumors

ILT3: Key Stromal Checkpoint and its Potential Roles in Cancer

ILT3 is a fibronectin-binding inhibitory immune receptor that is highly expressed on tumor-associated myeloid cells

- Particularly high expression on tolerogenic dendritic cells, myeloid-derived suppressor cells and M2 macrophages

Fibronectin is an extracellular matrix protein that forms a fibrillar network within the tumor stroma

ILT3 is upregulated in several tumor types and is associated with poor survival[1,2]

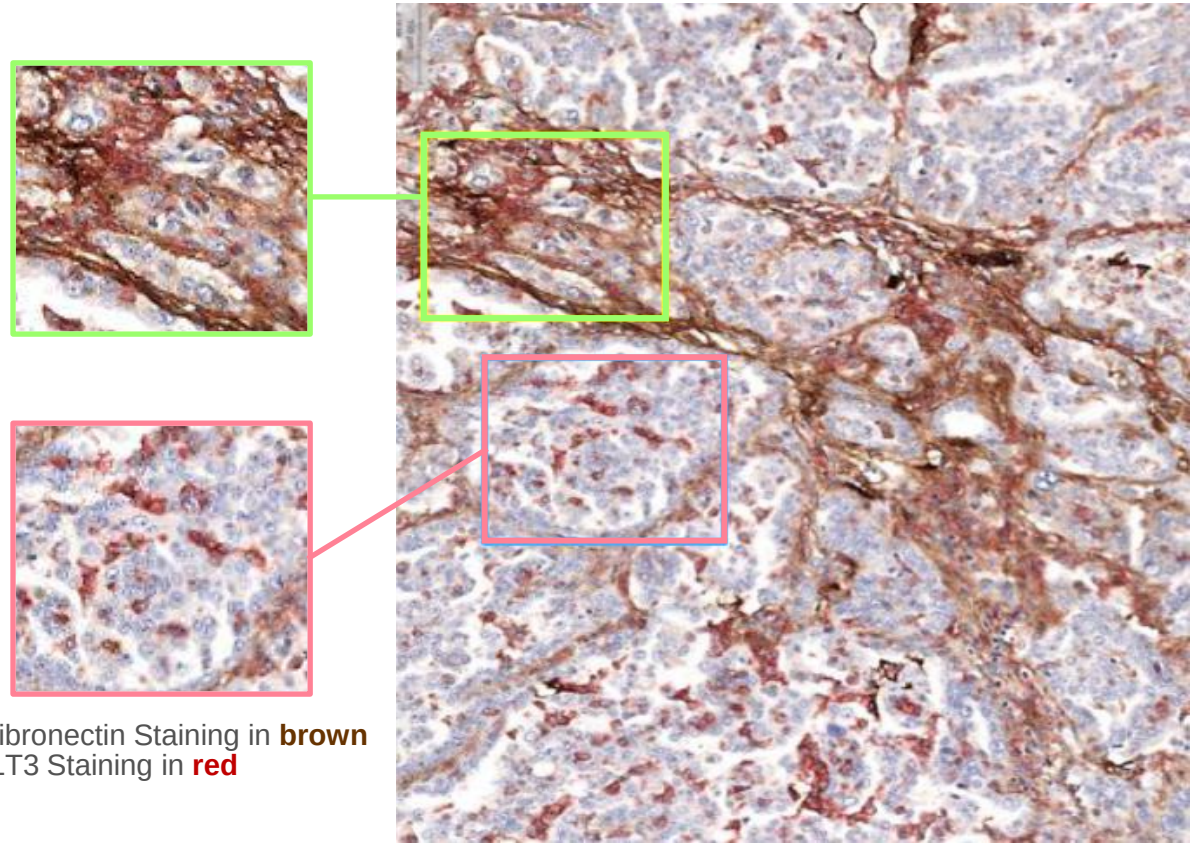
Fibronectin has been shown to be upregulated in multiple cancers and is associated with tumor progression[3,4]

ILT3-fibronectin interactions may form a stromal checkpoint within the tumor microenvironment

- May actively suppress myeloid cell function and inhibit antitumor immunity

ILT3 and Fibronectin Enrichment in Ovarian Tumors

Two populations of ILT3-positive myeloid cells:
1. Myeloid cells “trapped” in fibronectin (yellow box)
2. Myeloid cells infiltrating into tumor cell mass (blue box)



Fibronectin Staining in **brown**
ILT3 Staining in **red**

1 = L de Goeje et al, OncoImmunology 2015; 2 = Liu et al, Pathology - Research and Practice, 2018; 3 = Saito et al, Molecular Medicine Reports, 2008; 4 = Niknami et al, EXCLI Journal, 2017

NGM831 is an Antagonist Antibody Designed to Inhibit ILT3 That is Anticipated to Enter the Clinic in 2022

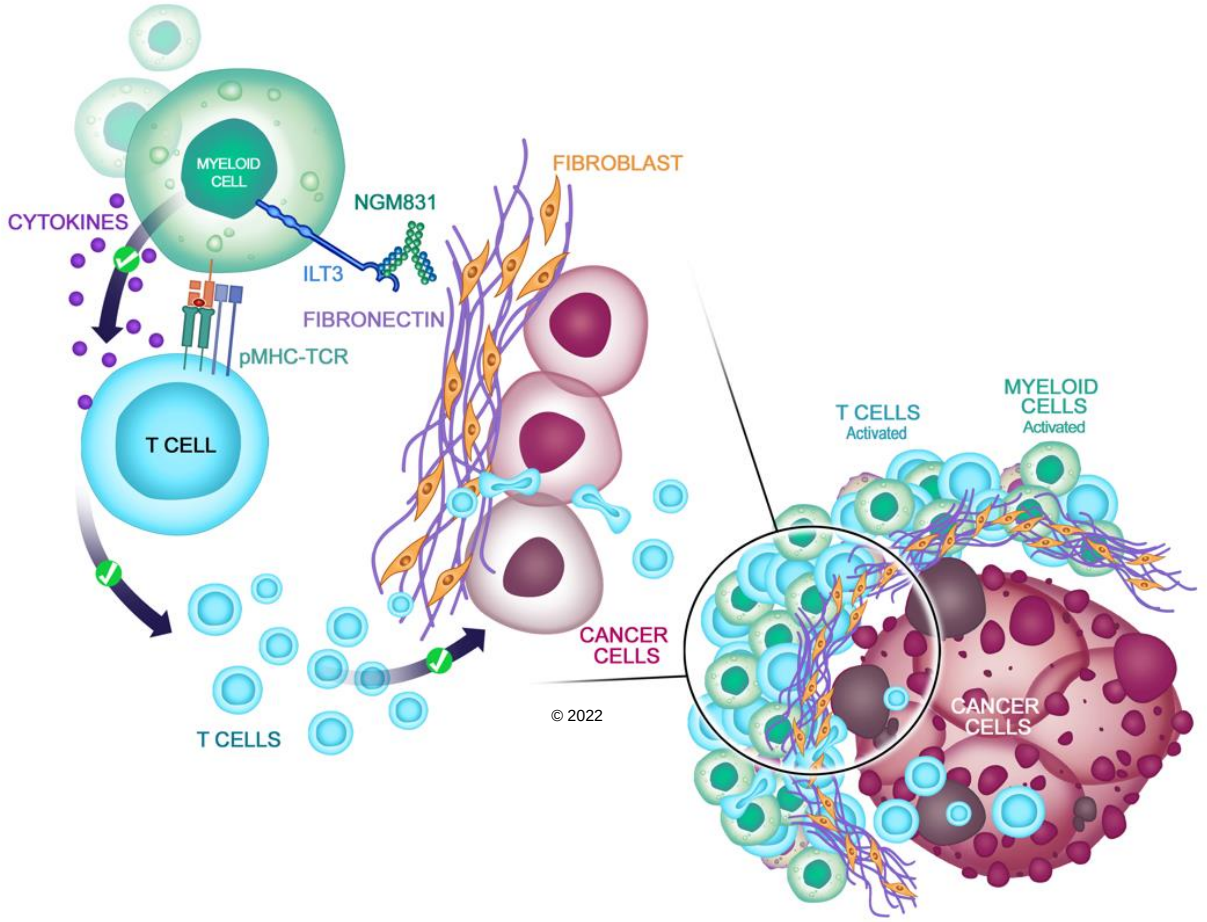
Potent antibody targeting the myeloid-enriched inhibitory receptor ILT3 (LILRB4)

Potential to reprogram ILT3-expressing suppressive myeloid cells and mediate signals from the extracellular matrix that promote myeloid cell suppression

Preclinical studies suggest that NGM831 may:

- Reprogram tolerogenic dendritic cells into stimulatory cells
- Enhance Fc Receptor activity
- Enhance T cell activation and infiltration of tumors

Plan to initiate first-in-human trial of NGM831 in 1Q22



ILT3 Blockade Reverses Fibronectin-mediated Inhibition of Fc Receptor Activity

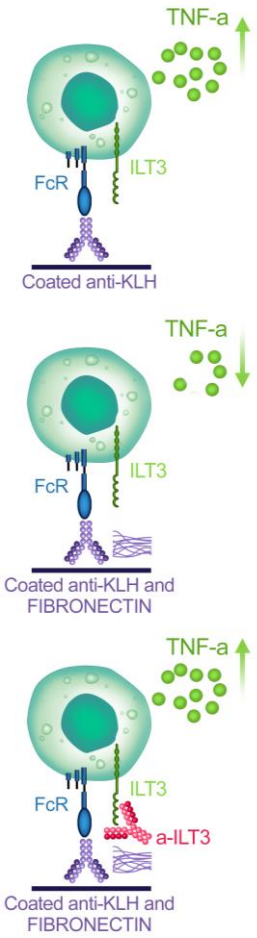
ILT3 blockade restores the ability of dendritic cells to respond to Fc Receptor activation in the presence of fibronectin

- ILT3-fibronectin interaction inhibits the activity of the Fc receptor, a key stimulatory receptor. This inhibition is reversed with ILT3 blockade.

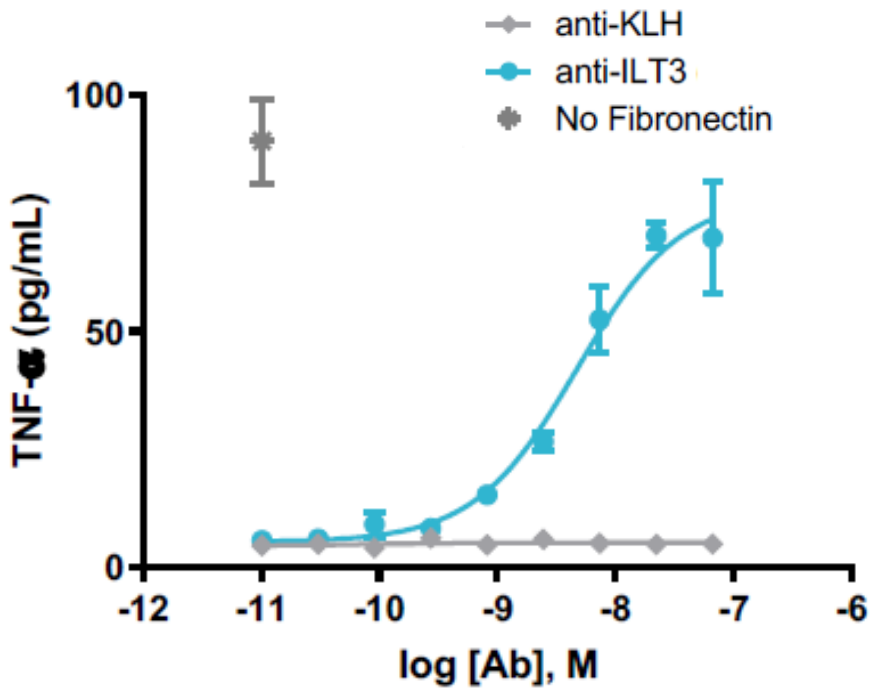
1 DCs stimulated with anti-KLH secrete TNF-α

2 TNF-α secretion is inhibited by platebound fibronectin

3 Inhibition of FcR signaling by fibronectin is blocked by ILT3



TNF-α Secretion by Monocyte-Derived Dendritic Cells

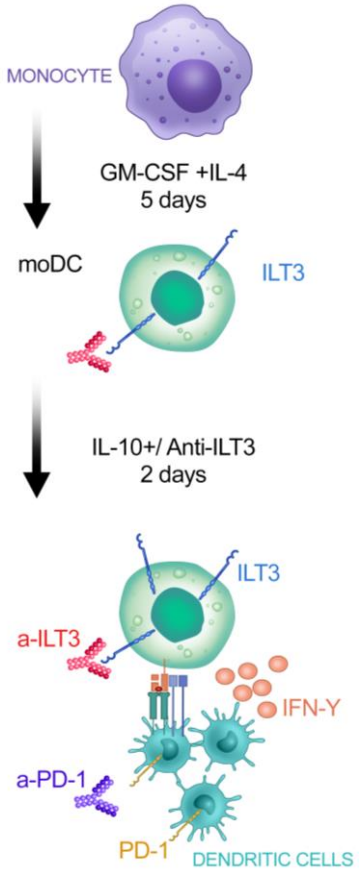


Paavola et al, *Cancer Immunology Research,* 2021

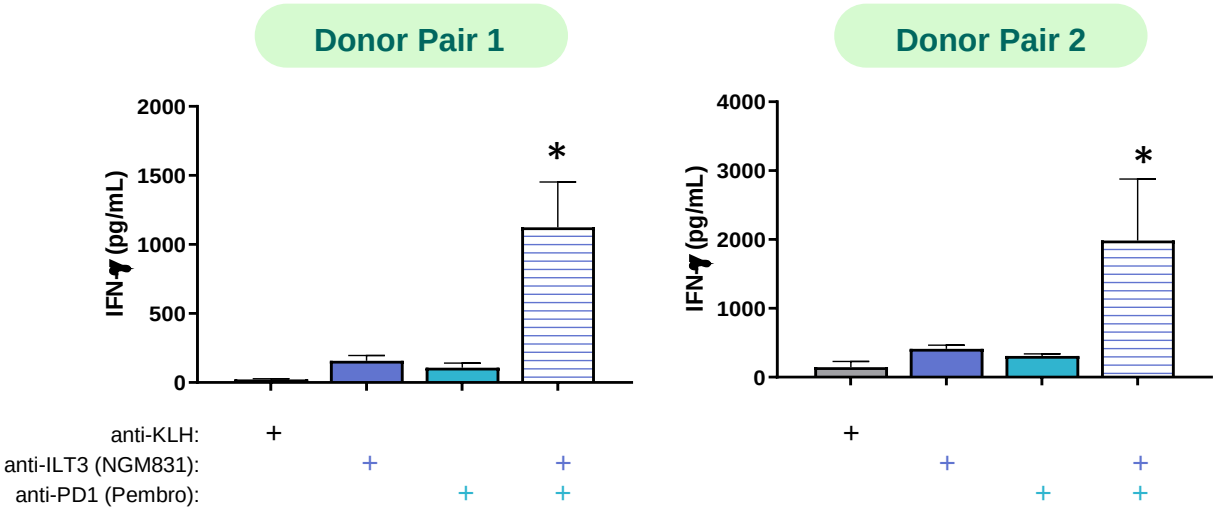
Preclinical Models Suggest that NGM831 and Pembrolizumab May Act Synergistically to Enhance T Cell Activation

In preclinical study NGM831 significantly enhanced the stimulatory capacity of tolerized dendritic cells in combination with pembrolizumab

NGM831 T cell activation was on par with anti-PD1



T Cell Cytokine Secretion



* P < 0.01 *vs*. each Ab alone

NGM Bio unpublished data
moDC = Monocyte-Derived Dendritic Cells; GM-CSF = Granulocyte-macrophage colony-stimulating factor; IL = interleukin

NGM438 in Advanced Solid Tumors

LAIR1: Key Stromal Checkpoint and its Potential Roles in Cancer

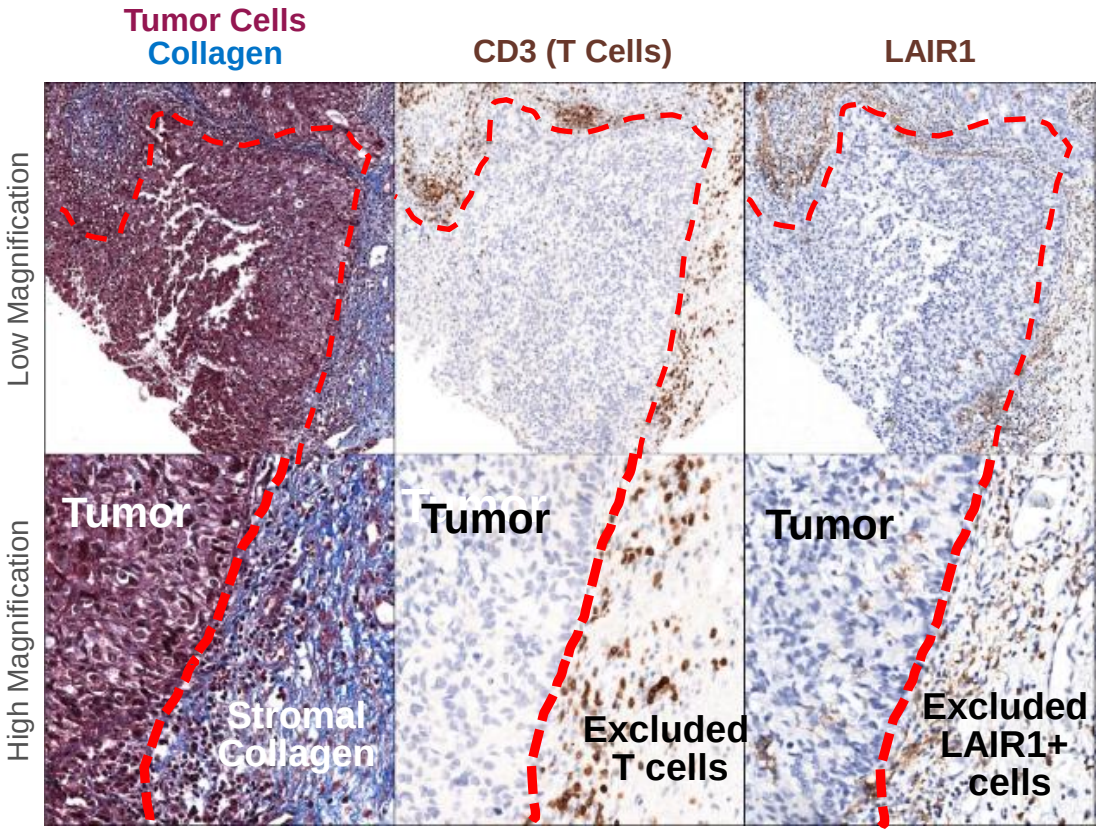
LAIR1 is a collagen-binding inhibitory signaling receptor expressed on immune cells: T cells, B cells, NK cells and myeloid cells[1-2]

LAIR1 and collagens are upregulated in certain cancer types[3-7] and impose signal-based immune suppression[8-9]

- Collagens act as a stromal checkpoint to physically impede anti-tumor immunity
- Co-localization of LAIR1-expressing immune cells and stromal collagen may impose signaling-based immune suppression

Stromal derived factors, such as collagen expression, and LAIR1-expressing myeloid cells are associated with poor responses to checkpoint inhibitors

LAIR1-Expressing Immune Cells in Pancreatic Tumor



Pancreatic ductal adenocarcinoma tumor section

1. Meyaard, Immunity, 1997; 2. Guo, Trans Med, 2020, 3. Cao, 2015, Biochem Biophys Res Commun; 4. Wang, Exp Ther Med, 2016; 5. Wu, CP Cancer, 2018; 6. Yang, Head & Neck, 2018 7. Jingushi, Onc. Reports, 2018; 8. Peng, Nat Comm, 2020; 9. Lijun, Oncoimmunology, 2020

NGM438 is an Antagonist Antibody Designed to Inhibit LAIR1 That is Anticipated to Enter the Clinic in 2022

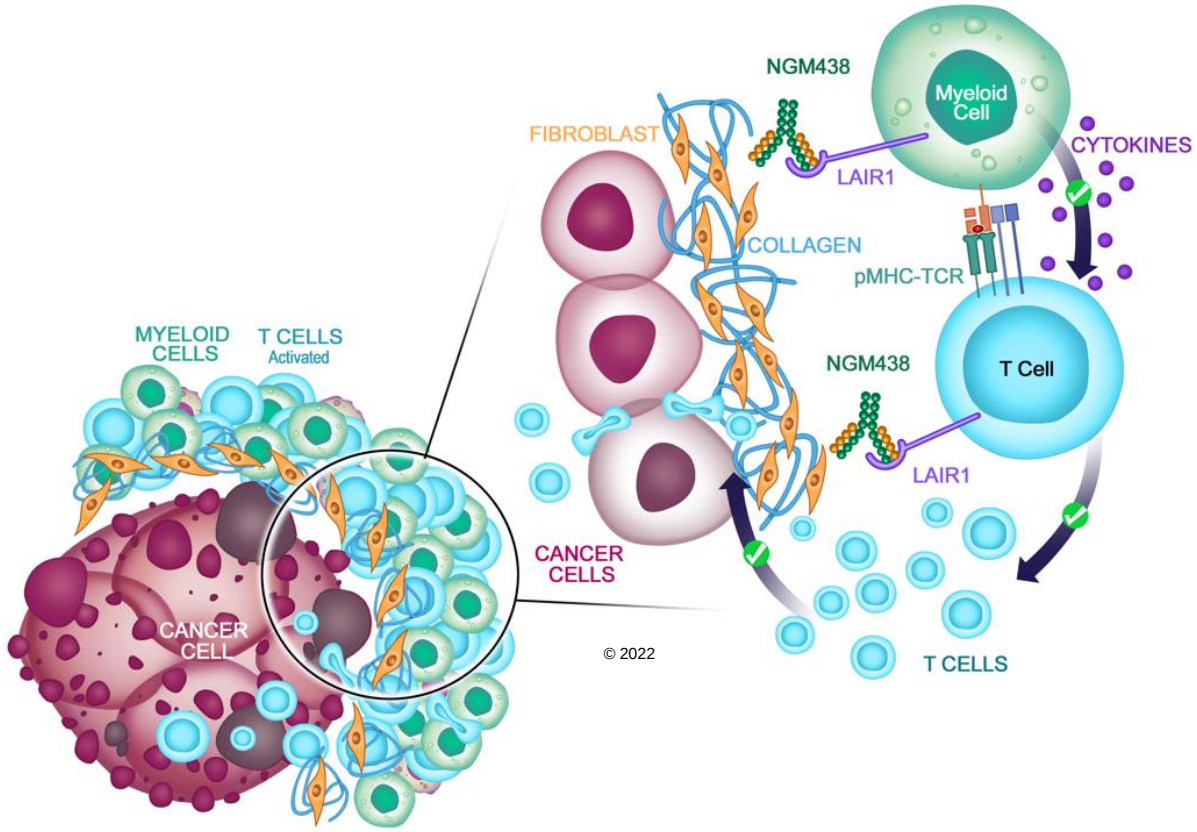
Potent, first-in-class antibody targeting the myeloid-enriched inhibitory receptor LAIR1

Potential to reprogram LAIR1-expressing suppressive myeloid cells within the tumor via disruption of collagen-LAIR1 mediated immune cell signaling

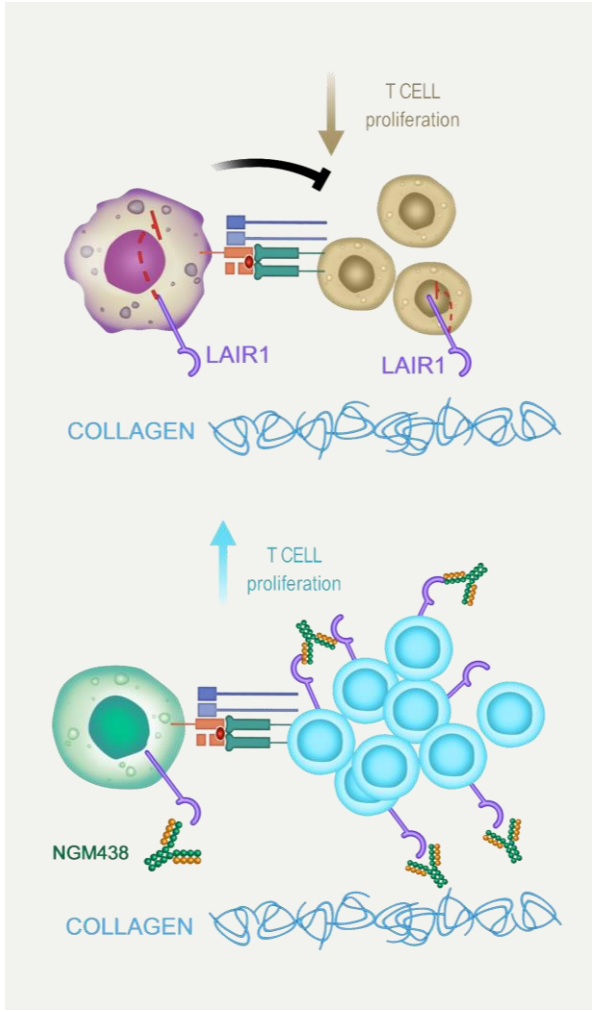
Preclinical studies suggest that NGM438 may:

- Reverse collagen mediated suppression of myeloid cells to a stimulatory phenotype
- Stimulate inflammatory cytokine production in myeloid and T cells
- Reprogram collagen suppressed myeloid cells to stimulate T cell activation
- Enhance cellular proliferation of collagen suppressed T cells

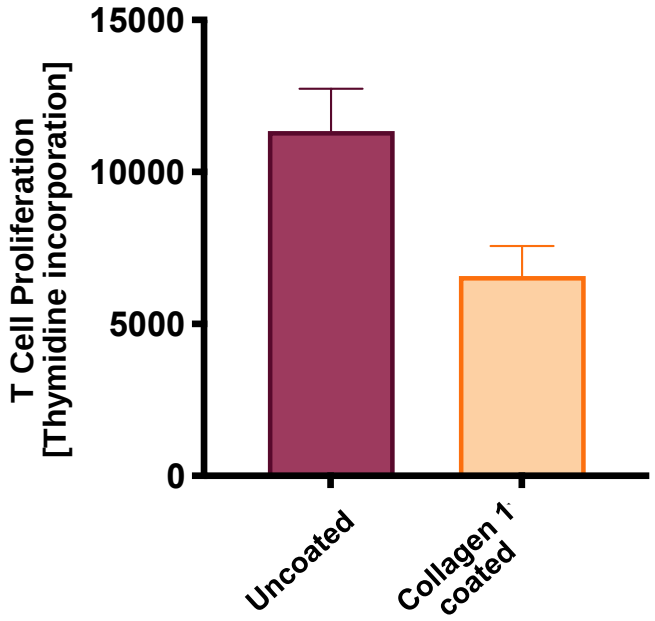
Plan to initiate first-in-human trial of NGM438 in 2Q22



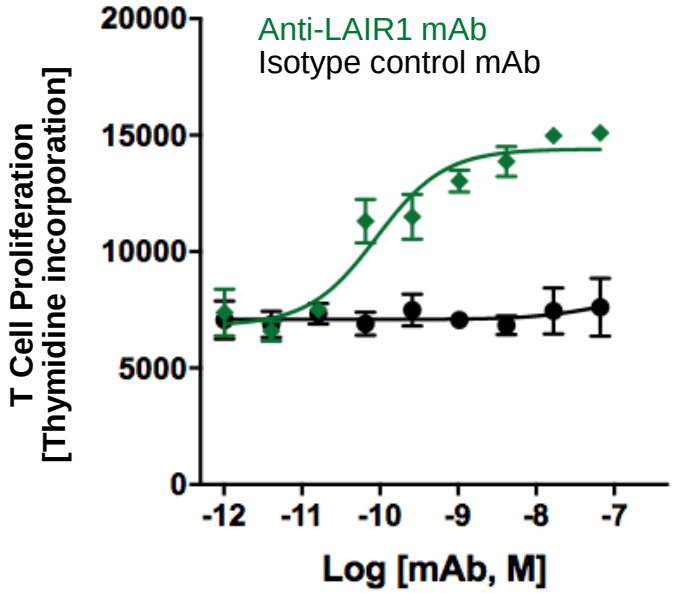
LAIR1 Blockade Reverses Suppression of Myeloid Cells by Collagen Leading to Enhanced T Cell Proliferation



T Cell Proliferation (MLR) is Suppressed by Collagen



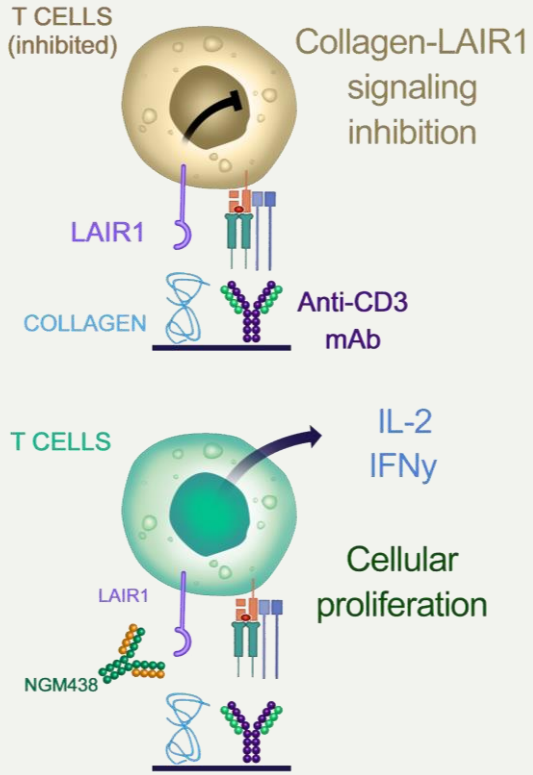
NGM438 Reverses T Cell Proliferation Suppression in a Dose-Dependent Manner



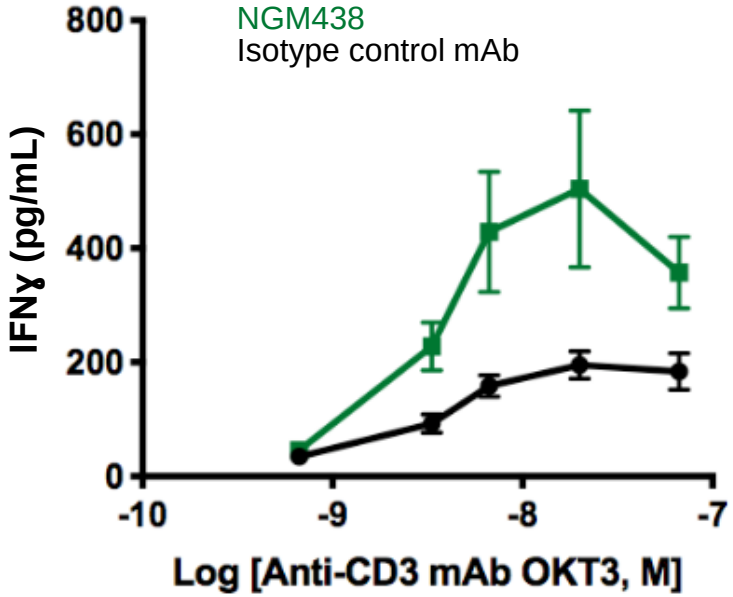
NGM Bio unpublished data
donor-mismatched monocyte-derived dendritic cells and T cells were incubated on plates coated with human collagen type 1 with soluble antagonists in the media
MLR = Mixed Lymphocyte Reaction; mAb = monoclonal antibodies

NGM438 Enhanced Inflammatory Cytokine Secretion and T Cell Proliferation in Collagen-suppressed T Cells

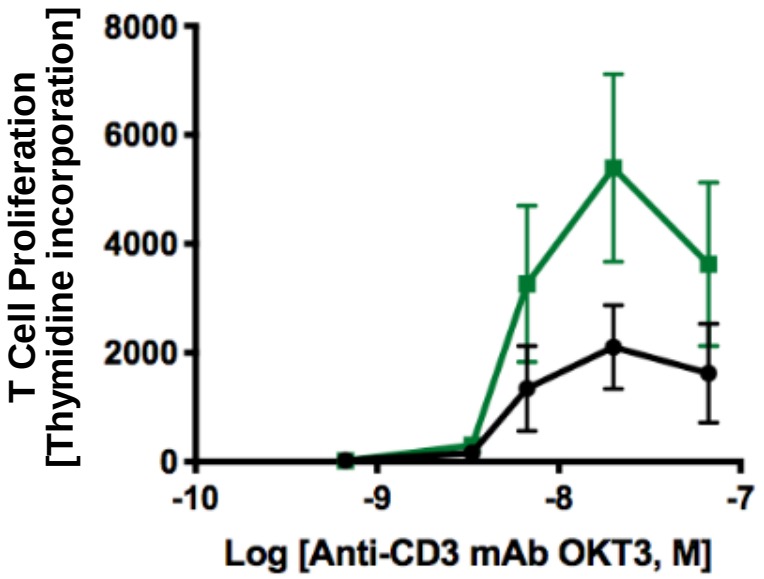
Collagen-Suppressed T cell Assay



NGM438 Promotes Th1 Cytokine Production



NGM438 Enhances T Cell Proliferation *in vitro*

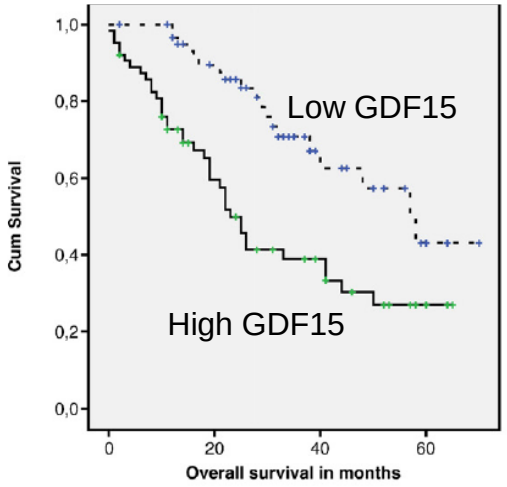


NGM Bio unpublished data
Human T cells incubated on plates coated with collagen and range of anti-CD3 stimulating mAb with single concentration of soluble antagonist mAbs in the media
Th1 = T cell helper 1

NGM120 for the Treatment of Cancer and Cancer-Related Cachexia

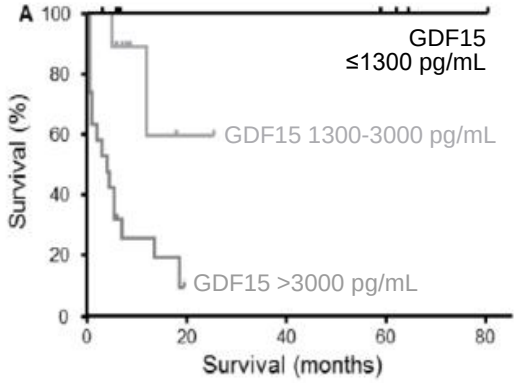
Elevated GDF15 Levels are Linked to Shorter Patient Survival in Multiple Cancer Types

Ovarian Cancer



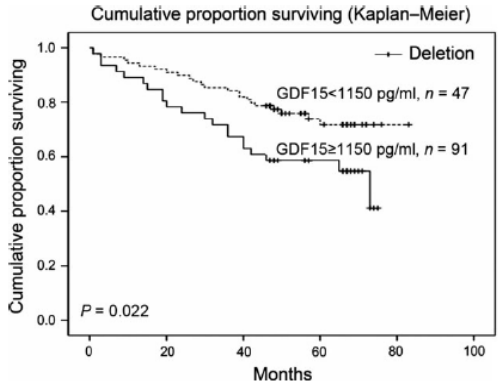
Staff *et al*., *Gynecologic Oncology* 2010

Prostate Cancer



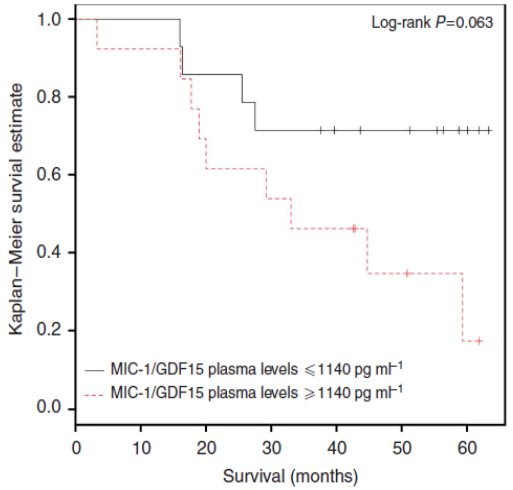
Winand *et al.*, *J Mol Biomark Diagn* 2014

Colorectal Cancer (CRC)



Li *et al*., *J Cell Mol Med*_2016

Esophageal Cancer



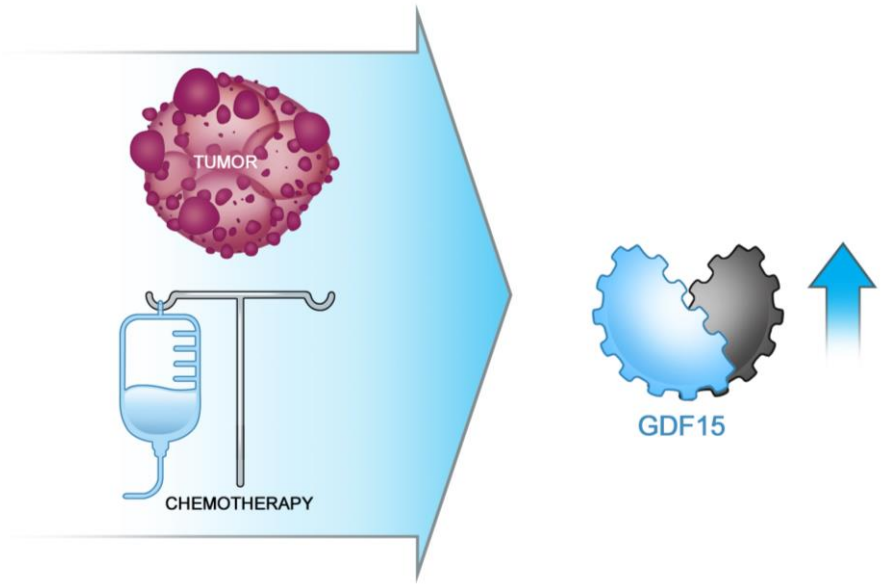
Fisher *et al., BJC* 2015

Preclinical research suggests the GDF15/GFRAL pathway may play a role in promoting tumor-associated immune regulation, metabolic regulation and appetite suppression

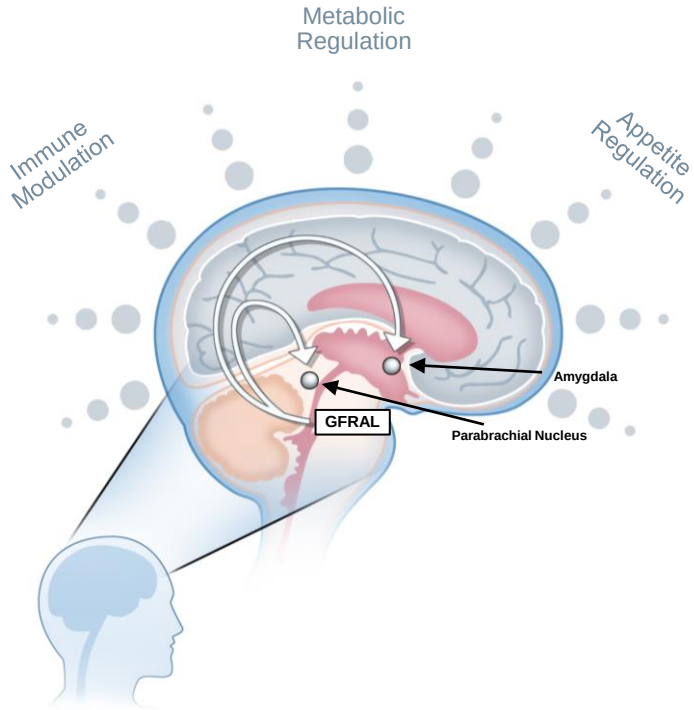
There are Multiple Mechanisms by Which the GDF15-GFRAL Pathway May Impact Tumor Growth

As a result of NGM's discovery of the GFRAL receptor, we developed novel insights into the mechanism of action of GDF15 and the function of the GDF15/GFRAL interaction

GDF15 levels are increased by **tumors, chemotherapy,** infection, inflammation and other stressors



GDF15-GFRAL pathway is involved in immune modulation, metabolic regulation and appetite



Hsu et al., *Nature,* 2017

The Unmet Medical Need for Pancreatic Cancer is High

About 60,000 will be diagnosed with pancreatic cancer in the U.S. in 2021

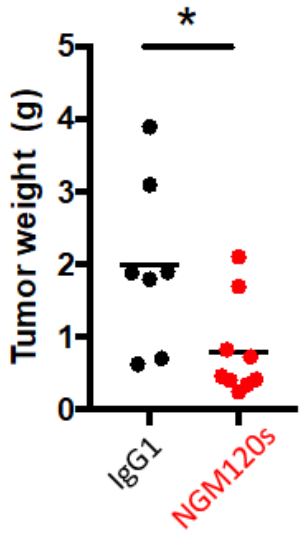
Pancreatic cancer is seldom detected early when it's most curable because symptoms often don't develop until after it has spread to other organs

Pancreatic cancer accounts for about 3% of all cancers in the U.S. and about 7% of all cancer deaths

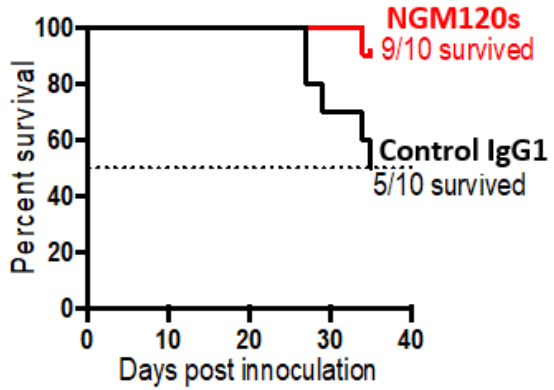
1-year survival rate of 18% of all stages of the disease reflecting the fact that tumors progress rapidly and advanced stage of disease at diagnosis[1]

NGM120 Reduces Tumor Growth and Improves Survival in a Pancreatic Tumor Model[2]

Tumor Weight



Survival



Tumor Model

- **Orthotopic syngeneic tumor model**
- **Pancreatic cancer**
- **NGM120 dosed weekly**

American Cancer Society, Key Statistics for Pancreatic Cancer, 2021
Mayo Clinic, Pancreatic Cancer Overview
[1]"Addressing the challenges of pancreatic cancer: future directions for improving outcomes" Pancreatology, 2015; pancreatic ductal adenocarcinoma
[2]NGM Unpublished Data

NGM120 is an Antagonist Antibody Designed to Inhibit GFRAL

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

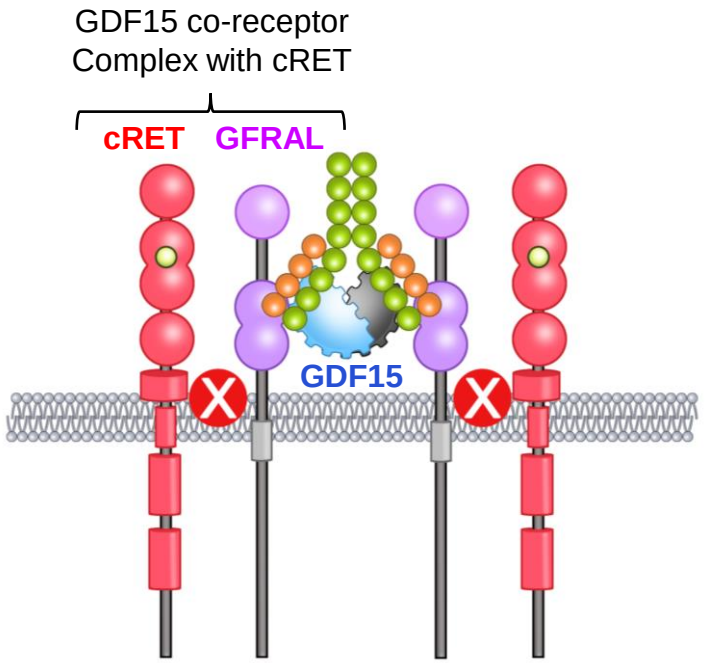
Potential to regulate the GDF15/GFRAL pathway in the brain stem that is known to signal feeding and the autonomic nervous system

GDF15 is upregulated in cancer and associated with poor prognosis. Preclinical studies suggest that NGM120 may:

- Reduce tumor growth and improve survival in syngeneic orthotopic pancreatic tumor model
- Reverse human tumor-induced body weight loss in mice

- **Ph1a/1b NGM120 dose escalation study in patients with cancer; preliminary data presented at the European Society for Medical Oncology (ESMO) in 3Q21**
- **Ph2 NGM120, placebo-controlled expansion in patients with metastatic pancreatic cancer initiated in 1Q21**

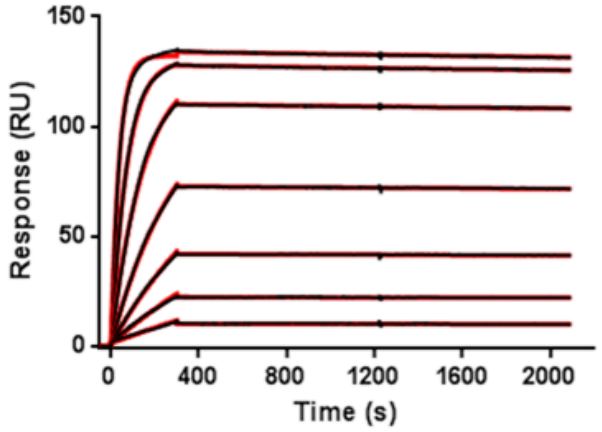
NGM120 prevents formation of the co-receptor complex



[1] *Hsu et. al., Nature 2017*

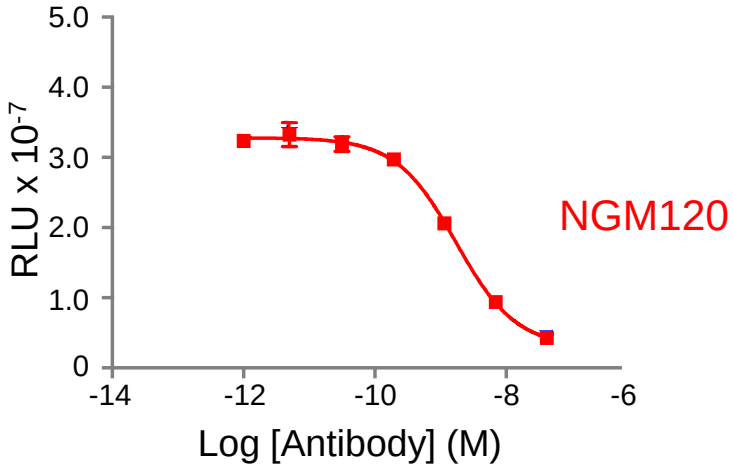
NGM120 Binds with High Affinity to GFRAL and Acts as a Non-Competitive Antagonist of Receptor-Mediated Signaling

NGM120 binding to GFRAL (Biacore)

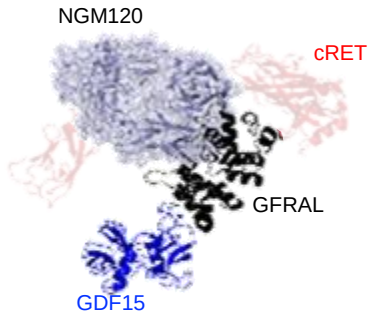


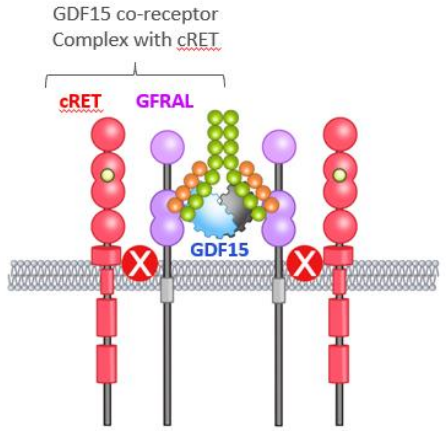
K_D=785 fM
(KinExA)

Inhibition of GDF15 signaling by NGM120



NGM120 prevents formation of the co-receptor complex





Suriben et. al.., Nat Med 2020

Preliminary Results of Phase 1a/1b Study of NGM120 in Patients with Advanced Solid Tumors as Presented at ESMO 2021

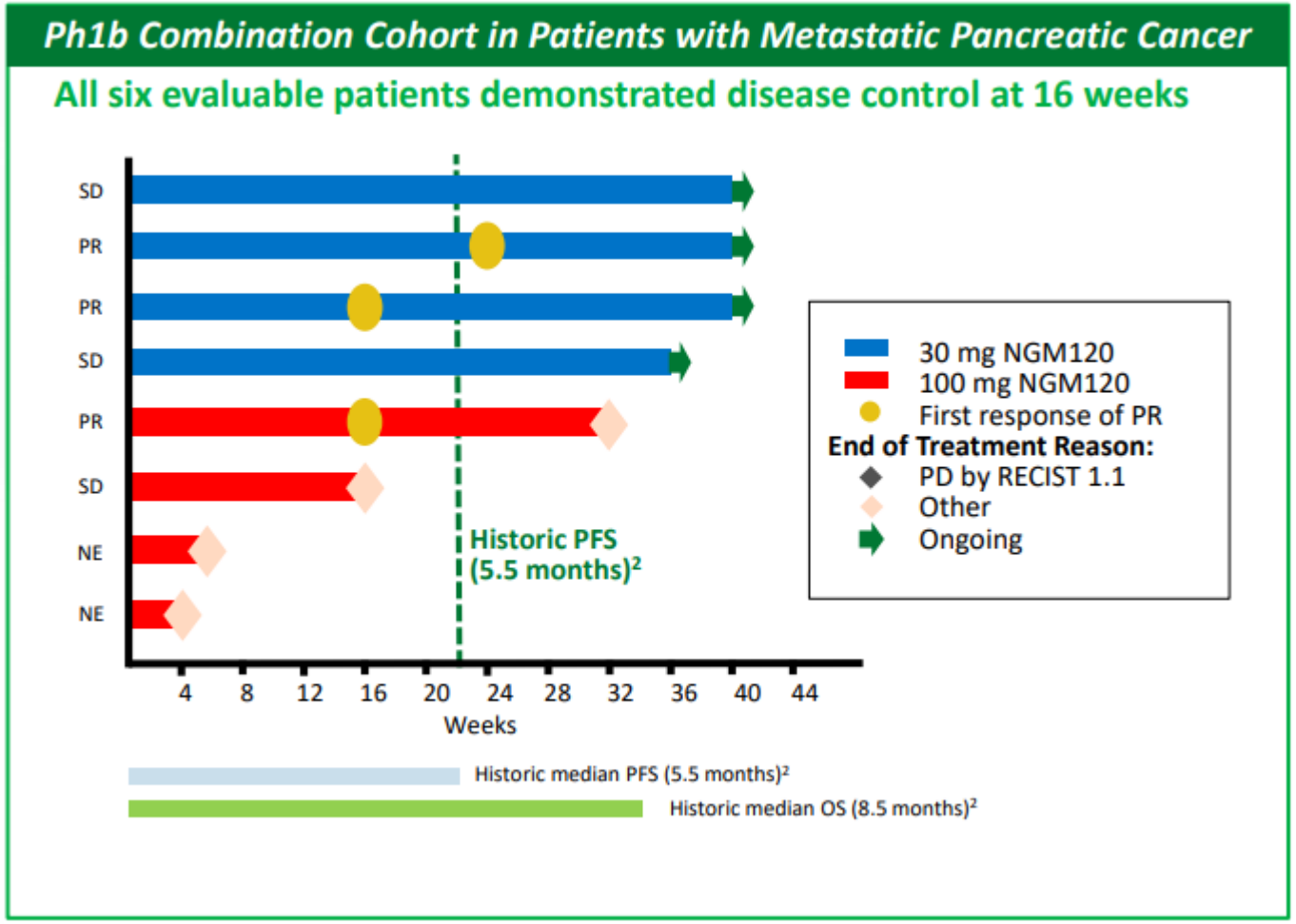
Treatment with NGM120[1] was well tolerated in the study with no dose-limiting toxicities as monotherapy or in combination with Gem/Nab-P

In Ph1a monotherapy cohort in advanced solid tumors, five stable disease (SD) (5/20, 25%) observed, but no objective responses observed

PK exposure increased with dose

In Ph1b cohort in combination with Gem/Nab-P (pancreatic cancer), all six CT-evaluable patients treated with NGM120 demonstrated disease control at 16 weeks with three partial responses (PR) and three SD for ESMO preliminary results

- As of the July 26, 2021 data cut-off for ESMO, four patients continued to exhibit PR/SD beyond 36 weeks
- As of September 16, 2021 three of those four patients remain on drug, exhibiting PR (two patients) and SD (one patient), beyond 44 weeks



Preliminary results presented at European Society of Medical Oncology (ESMO) Virtual Congress 2021; NGM plans to report final results from the Phase 1a and Phase 1b cohorts once all patients have completed treatment and follow-up per protocol
1 NGM120 30 mg and 100 mg ; 2 Von Hoff DD, et al. N Engl J Med. 2013
SD = Stable Disease; PR = Partial Response; NE = Nonevaluable

Preliminary Results of Phase 1a/1b Study of NGM120 in Patients with Advanced Solid Tumors

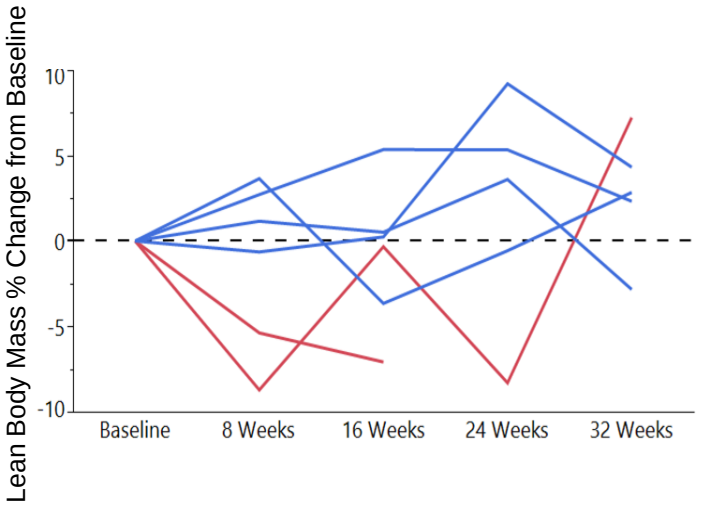
Increases in lean body mass and body weight were observed in a subset of the patients in both the NGM120 monotherapy[1] and combination[2] settings

In Ph1a cohort, four patients experienced increases in lean body mass >3.5% at Week 8

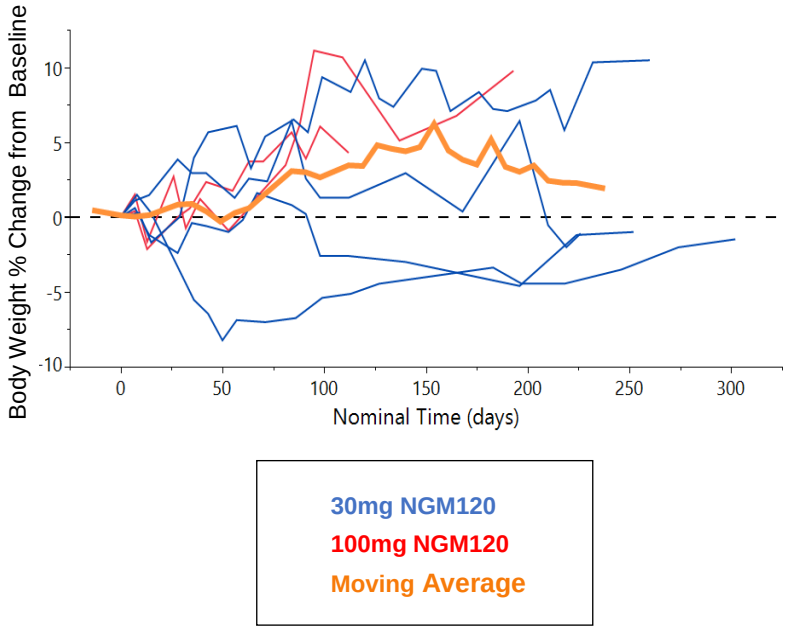
In Ph1b cohort, all six evaluable patients showed a 4% average maximal increase in lean body mass, and four of the six evaluable patients exhibited >5% maximum body weight gain

Ph1b Combination Cohort in Patients with Metastatic Pancreatic Cancer

Six Evaluable Patients Exhibited a 4% Average Maximal Increase in Lean Body Mass



4/6 Evaluable Patients Exhibited >5% Maximum Body Weight Gain



Preliminary results presented at European Society of Medical Oncology (ESMO) Virtual Congress 2021; NGM plans to report final results from the Phase 1a and Phase 1b cohorts once all patients have completed treatment and follow-up per protocol

1 Treatment with NGM120 30mg and 100mg, once every 3 weeks, subcutaneous

2 Treatment with NGM120 30mg and 100mg, once every 4 weeks, subcutaneous, in combination with gemcitabine + Nab-paclitaxel

NGM120 in Clinical Development

ADVANCING CLINICAL DEVELOPMENT

- ✓ **Completed** single (n=48) and multiple (n=44) ascending dose cohorts in first-in-human healthy volunteer studies

- ✓ **Phase 1a/1b** preliminary findings presented at ESMO 2021

- ✓ **Phase 2** expansion study initiated in metastatic pancreatic cancer patients (PINNACLES)
  - Randomized, single-blind (investigator-blinded), placebo-controlled, multi-center expansion study
  - Patients will be randomized to either NGM120 or placebo in combination with gemcitabine/Nab-paclitaxel
  - Assessment of both cancer and cancer-related cachexia endpoints
    - Overall response rate (ORR), progression-free survival (PFS), overall survival (OS), body weight change, lean body mass change, patient reported outcomes and functional status changes
  - Study initiated in March 2021

NGM621 in Geographic Atrophy

Geographic Atrophy (GA) is an Age-Related, Progressive Retinal Degenerative Disease Associated with Irreversible Loss of Vision

GA is characterized by **progressive and irreversible loss** of photoreceptors, retinal pigment epithelium (RPE) and choriocapillaris

GA is typically **bilateral and lesion enlargement** results in irreversible blindness

GA **affects ~5+ million people** globally and ~ 1+ million people in the US[1]

GA disease progression, and accompanying vision decline, **may lead to loss of independence, poorer quality of life,** depression and an increased incidence of falls and fractures

Neurodegenerative Disease of the Retina

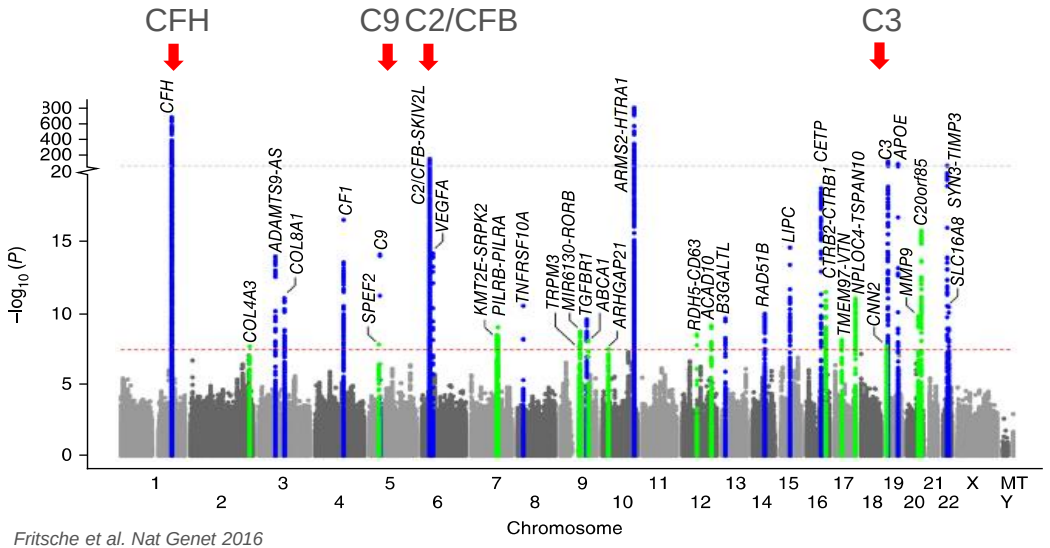
No FDA-approved treatments

ngmBIO

[1]Boyer et al, Retina 2017
Chakravarty U et al. Ophthalmology. 2018 Jun;125(6):842-849.

Evidence Strongly Supports the Pathological Role of Dysregulated Complement Activity in GA

Genetic Evidence

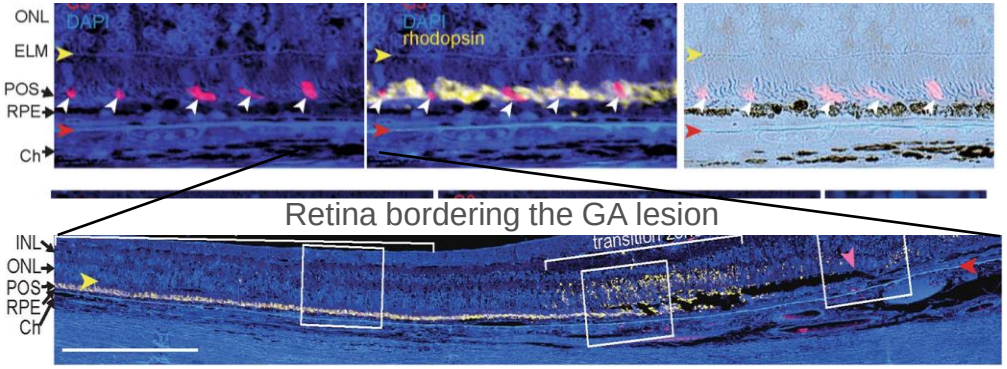


Fritsche et al. Nat Genet 2016

Variants in the complement pathway account for the majority of the known genetic risk for GA/AMD

Histopathological Evidence

C3 Deposition on Photoreceptors Precedes their Degeneration in Human GA Eyes

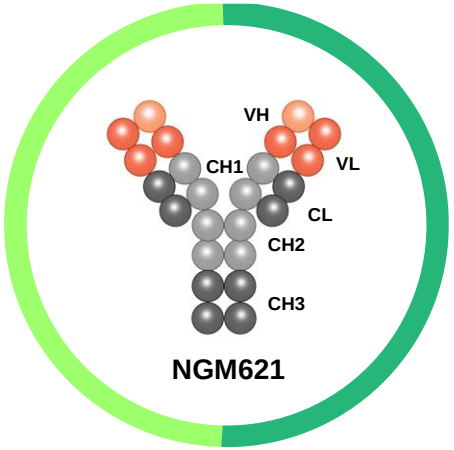


Katschke et al. Sci. Reports 2018

Pathological activation of complement system is strongly implicated in development and progression of GA

AMD = Age-related macular degeneration

NGM621: A Potent Anti-Complement C3 Antibody



NGM621 MOLECULE ATTRIBUTES

Type	Humanized IgG1 monoclonal antibody
Target	Complement C3
MW	~150 kDa
Affinity	K_D = 340pM
Effector Function	Fc mutations eliminating effector function

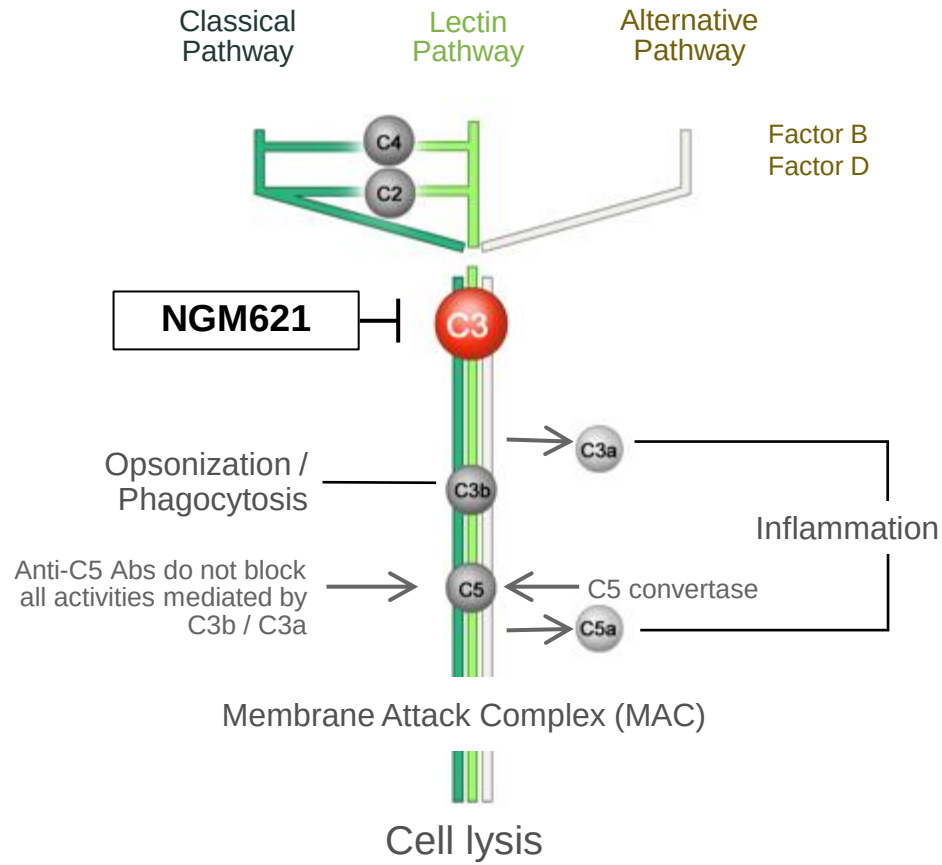
SCIENTIFIC RATIONALE: **NGM621 FOR GEOGRAPHIC ATROPHY**

Dysregulated activation of the complement system has been implicated in the onset and progression of geographic atrophy

C3 is a central component of the complement system, which helps orchestrate the body's response to infection and maintains tissue homeostasis

NGM621 is a novel monoclonal antibody that potently inhibits C3, blocking all complement pathways

COMPLEMENT CASCADE



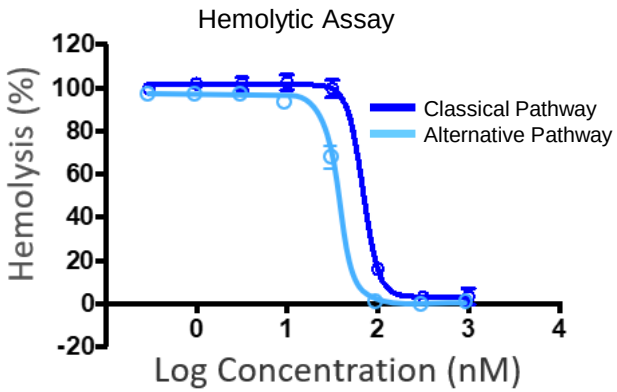
IgG1 = immune globulin G1

Existing Clinical Data Validates Complement Cascade as a Target for Treating GA and Leaves Room for Improvement and Differentiation

Potential NGM621 Differentiation in Geographic Atrophy (GA)

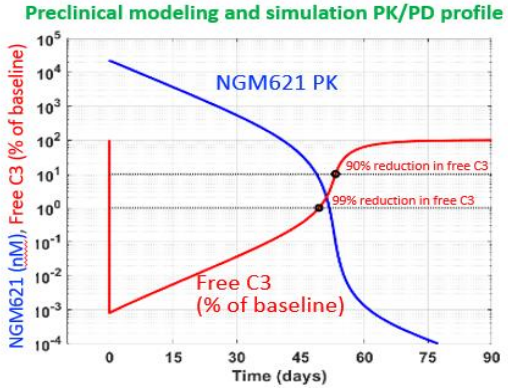
1 Efficacy.

As a monoclonal antibody, NGM621 potentially has superior potency compared with other approaches for targeting C3



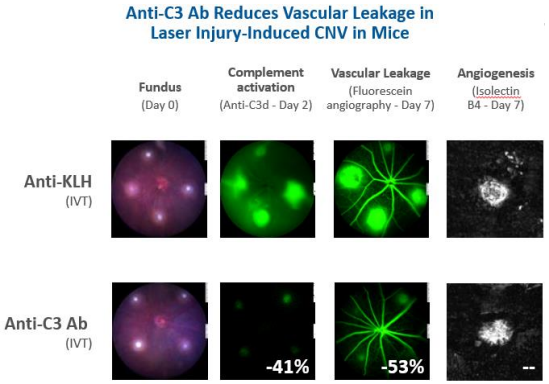
2 Dosing frequency.

Preclinical pharmacokinetic modeling, coupled with results from Ph1 testing, suggests NGM621 may allow for extended, every 2 month dosing



3 Safety.

Opportunity to differentiate on safety if CNV events do not show a dose-related trend relative to sham



Multiple areas of opportunity for NGM621 to achieve category leadership

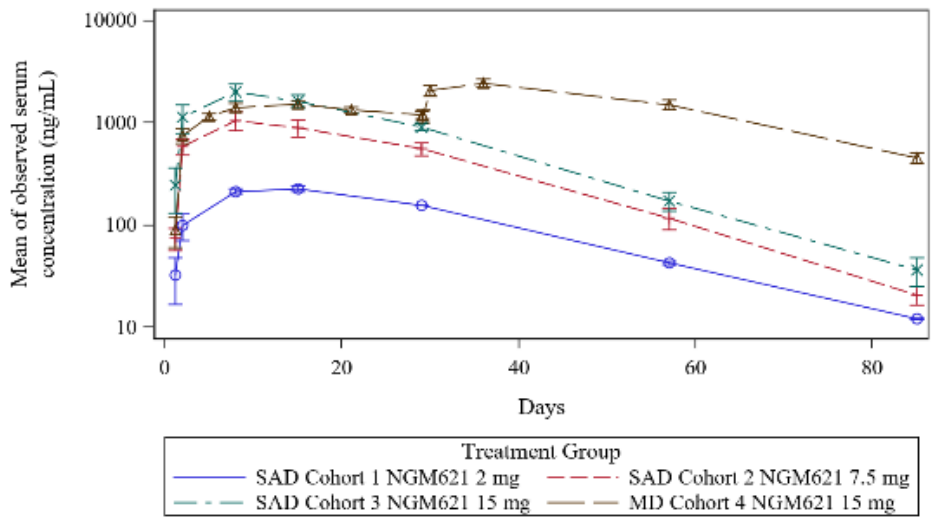
CNV = choroidal neovascularization; Anti-KLH = anti-keyhole limpet hemocyanin
Wei-Sheng Chen et. al. ARVO 2020
Alexander Loktev et. Al. ARVO 2020

NGM621 Human Serum PK Profile and Ocular PK/PD Preclinical Modeling Supports Potential for Every 8-Week IVT Dosing Regimen

PHASE 1 SERUM PK POST-IVT SINGLE & REPEAT DOSING

- The serum PK of NGM621 was linear and dose-proportional with low accumulation following every 4-week repeat IVT dosing
- NGM621 serum exposure was below concentrations that produce systemic complement inhibition at the highest IVT dose of 15 mg
- All subjects were ADA negative at all timepoints

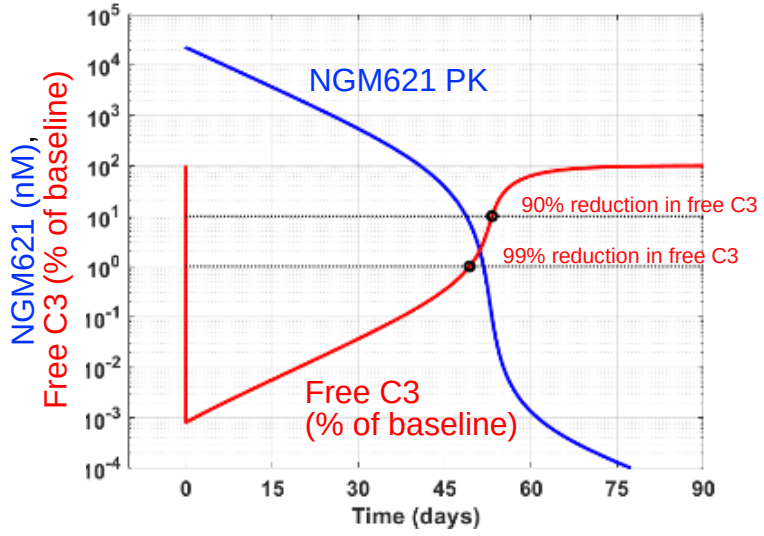
Mean Serum Concentration-time Profile post-IVT of NGM621



OCULAR PK/PD PRECLINICAL MODELING

- Preclinical modeling suggests that NGM621 may achieve >90% C3 target engagement in the eye for 7 weeks following a single IVT dose of 15 mg based on a PK/PD model
- We believe that PK/PD preclinical modeling support a potential every 8-week IVT dosing regimen at the 15 mg dose level

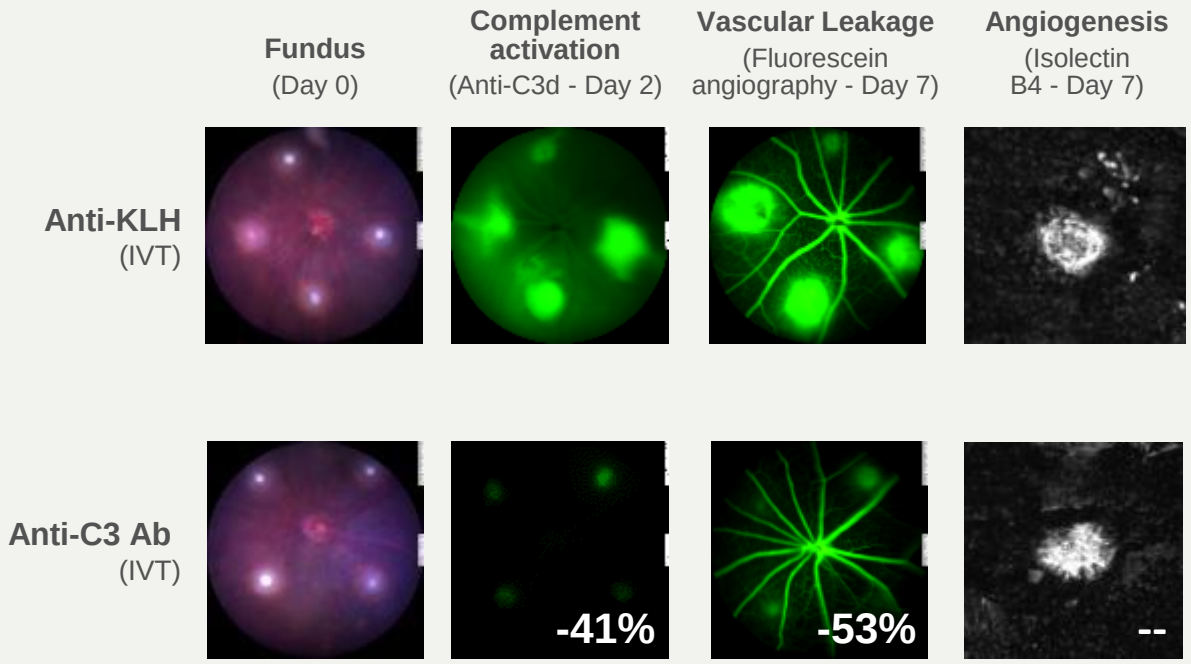
Preclinical modeling and simulation PK/PD profile



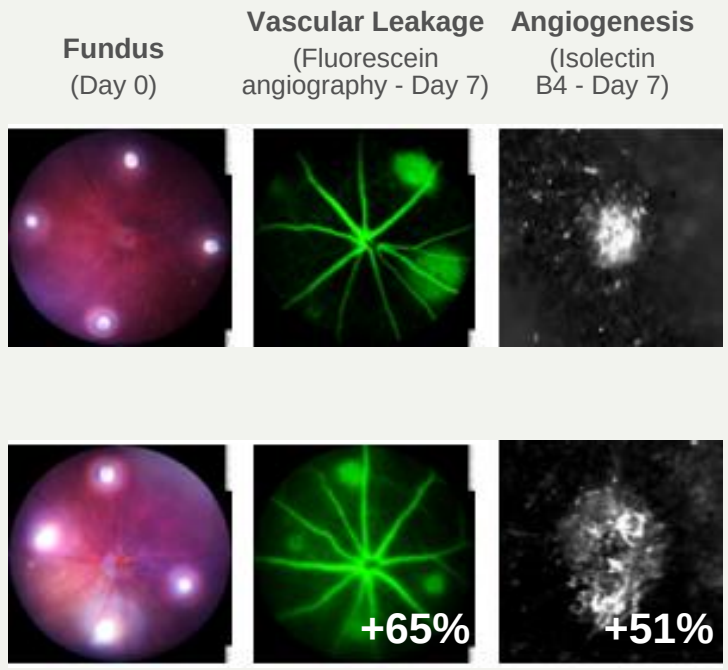
ADA = anti-drug antibodies; IVT = intravitreal; MD = multidose cohort; PD = pharmacodynamics; PK = pharmacokinetic; SAD = single ascending dose cohort..

Preclinical Data Shows PEG Can Exacerbate CNV Post-Laser Injury

Anti-C3 Ab Reduces Vascular Leakage in Laser Injury-Induced CNV in Mice



PEG-40KD Exacerbates Laser Injury-induced CNV in Mice



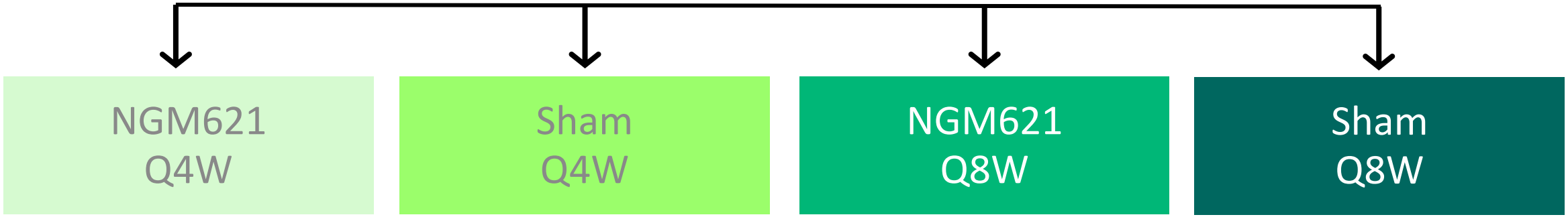
The absence of PEG may provide a safety profile advantage for NGM621

Wei-Sheng Chen et. al. ARVO 2020

Phase 2 CATALINA Trial is Fully Enrolled with Topline Data Expected in 4Q22

Patients With GA Secondary to AMD; N = 320[1,2]

Randomly assigned 2:1:2:1

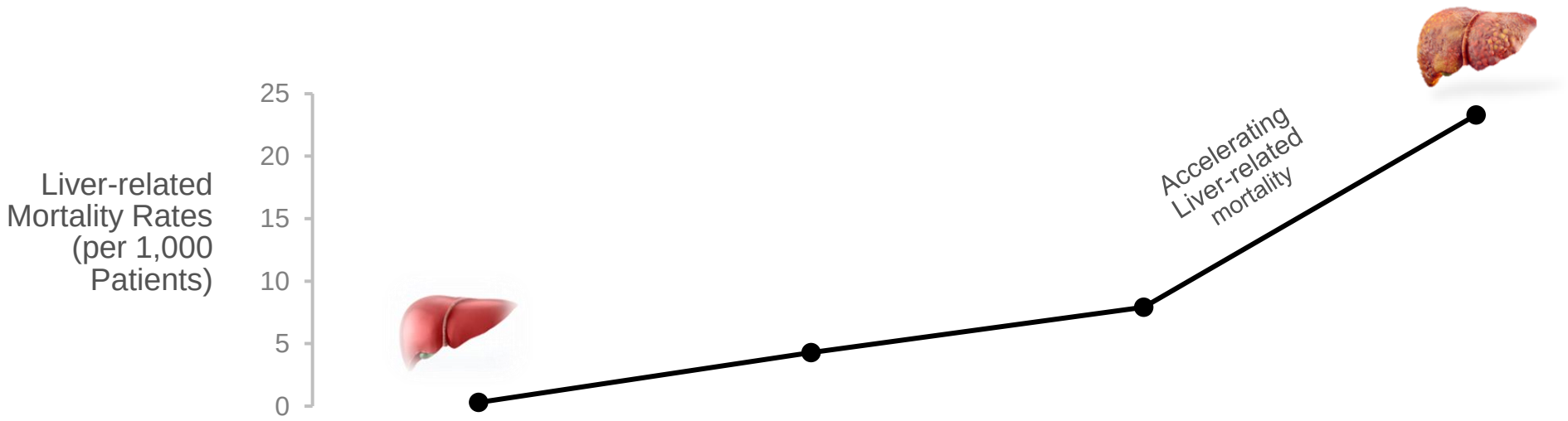


Primary Objective	To evaluate the efficacy, based on rate of change in GA lesion area as measured by fundus autofluorescence, and safety after 52 weeks of NGM621 IVT injections administered Q4W or Q8W compared with sham control in patients with GA
Design	Multicenter, randomized, double-masked, sham-controlled, Phase 2 study

[1]NGM Corporate Press Release July 22, 2021; [2]ClinicalTrials.gov NCT04465955
IVT = intravitreal; Q4 = every 4 weeks; Q8 = every 8 weeks.

MK-3655 and aldafermin in NASH

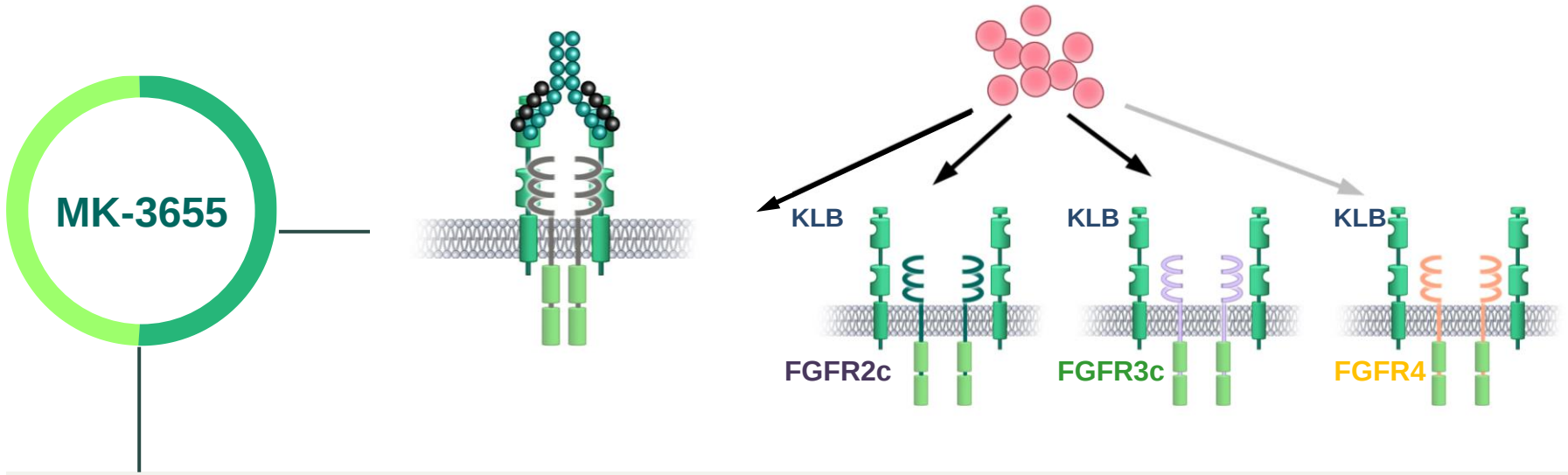
NASH: A Serious and Growing Disease, with Negative Outcomes Linked to Severity of Fibrosis



FIBROSIS STAGE	F0/F1	F2	F3	F4
US Prevalence – 2020	11.0M	4.1M	2.6M	1.6M
US Prevalence – *2030 (Est'd)*	12.9M	6.1M	4.5M	3.5M
Liver-related Mortality Ratio (vs. F0)	1x / 1.4x	9.6x	**16.7x**	**42.3x**

Sources: Dulai et al, Hepatology 2017, 65(5):1557-1565; Singh et al, Clin Gastroenterol Hepatol. 2015, 13(4): 643–654; Estes et al, Hepatology 2018, 67(1): 123-133; Angulo et al., Gastroenterology 2015;149:389-397; Ekstedt et al., Hepatology 2015;61:1547-1554; Sanyal et al., Hepatology. 2019 Dec;70(6):1913-1927

MK-3655 (formerly NGM313) for the Treatment of F2/F3 NASH

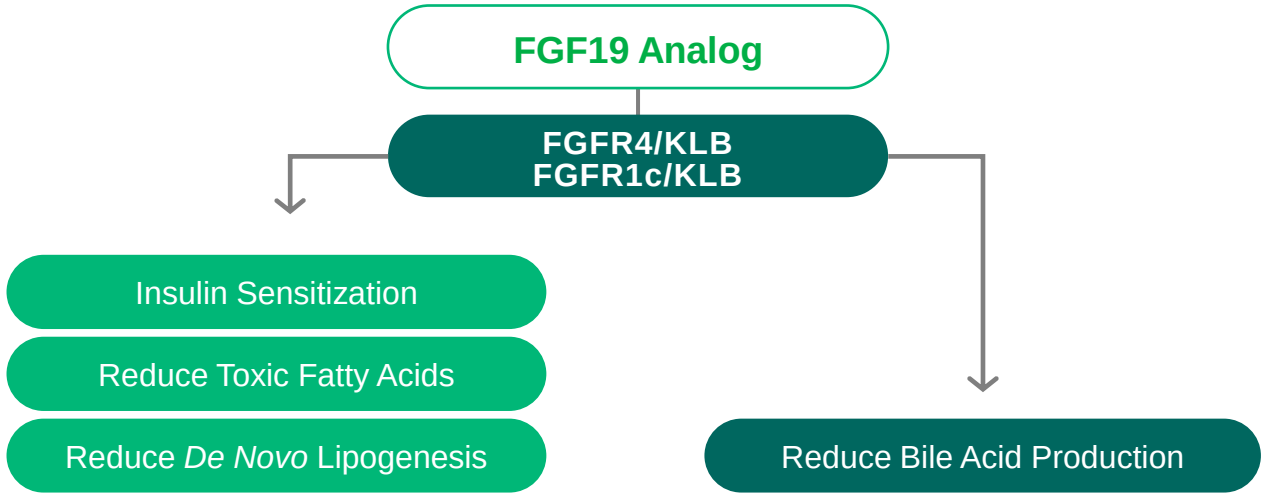


FGF21 analogs have demonstrated variable clinical efficacy in metabolic syndrome patients; native ligand has potential for safety liabilities

- Agonistic antibody that selectively activates FGFR1c / KLB to regulate energy metabolism
- Potential to be once-monthly injectable insulin sensitizer for treatment of NASH
- Completed Ph1 SAD/MAD study in obese, insulin resistant subjects and Ph1b study in subjects with NAFLD
- Single dose of MK-3655 resulted in **significant reductions in liver fat content and improvement in metabolic markers** based on preliminary data from a Ph1b study in obese, insulin resistant subjects with NAFLD **after five weeks**

Merck exercised its option and licensed MK-3655 (formerly NGM313) and initiated a Ph2b trial, funded by Merck, during 4Q20; NGM to receive milestones and double-digit royalties or up to 50% profit/cost share at NGM's option at end of Phase 3

Aldafermin for the Treatment of F4 NASH



Aldafermin is an engineered analog of the human hormone FGF19 that is dosed once daily as a subcutaneous injection

FGF19 is significantly downregulated in NASH patients, which causes downstream increases in bile acid production. Moreover, total serum bile acids significantly increases progressively as liver fibrosis stage increases[1]

Aldafermin's mechanism of action may be particularly well suited for in patients with NASH with F4 and compensated cirrhosis

- The primary MoA of aldafermin (FGF19 analog) is inhibition of CYP7A1, via FGFR4, to strongly reduce bile acid synthesis and improve fibrosis in the liver
- Secondarily, aldafermin activates the peripheral FGF1c/KLB pathway to improve insulin sensitization and decrease lipotoxicity

The 24-week Phase 2b ALPINE 2/3 trial in F2/F3 NASH patients showed a lack of significant fibrosis improvement

- This result was unexpected given the consistency of histology findings previously seen with aldafermin in our adaptive four-cohort Phase 2 study as well as in ALPINE 2/3 aldafermin achieved statistical significance on multiple non-invasive measurements

Currently continuing enrollment in our ongoing 48-week Phase 2b ALPINE 4 study to understand the profile of aldafermin in patients with F4 NASH and compensated cirrhosis

ngmBIO

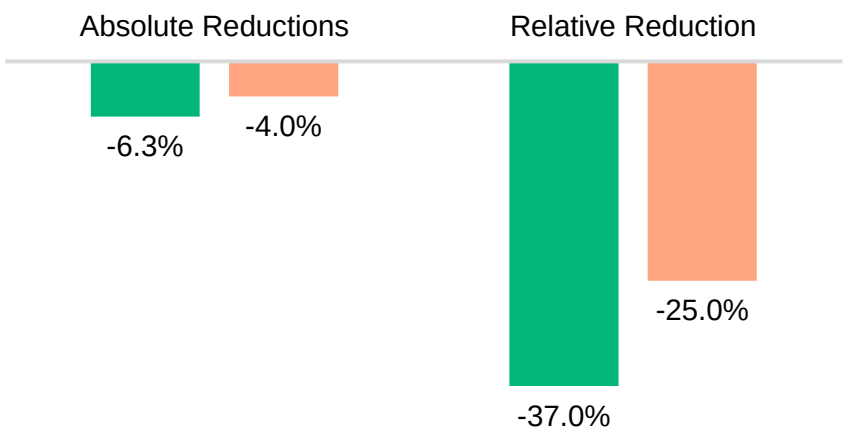
1 = Caussy et al, Aliment Pharmacology 2019

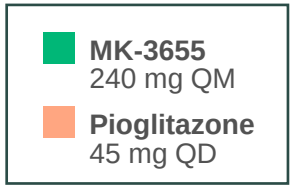
MK-3655 for the Treatment of NASH

Significant Reductions in Liver Fat Content and Improvement in Metabolic Markers (HbA1C) After 5 Weeks

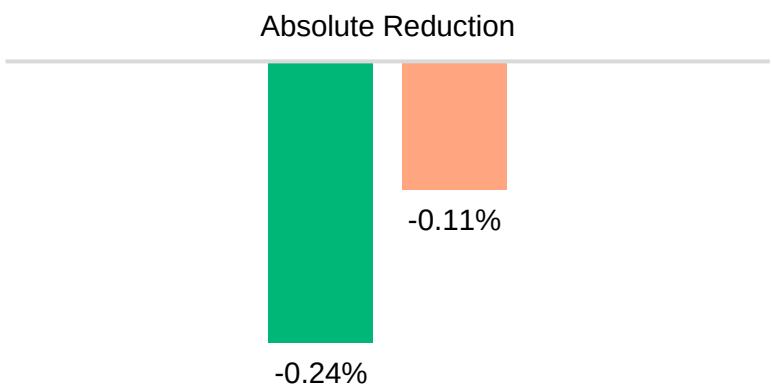
Phase 1b Study in Obese, Insulin Resistant Subjects with NAFLD

Liver Fat Content





HbA1c



MK-3655

Statistically Significant Improvements in
ALT and AST, Triglycerides, HDL-C, LDL-C, PRO-C3

Pioglitazone

Increased Body Weight by 2.3kg
(*vs.* 1.6kg increase for MK-3655 – no edema/fluid retention)

Phase 1b Safety: MK-3655 was Well Tolerated and Adverse Events (AEs) Were Generally Comparable to Placebo

MK-3655 Safety Results

- Favorable safety and tolerability profile consistent with other MK-3655 studies
- All AEs were mild in severity
- No Serious Adverse Events (SAEs) or Grade 2/3/4 AEs
- No pattern of AEs or organ system AEs of note
- No hypoglycemia

- Most common AEs (>10%) were injection site reaction (12%) and increased appetite (12%)
- No evidence of safety issues that have been associated with FGF21 analogues in clinical development
  - No tremor, no GI side effects, no effects on cortisol, no blood pressure changes

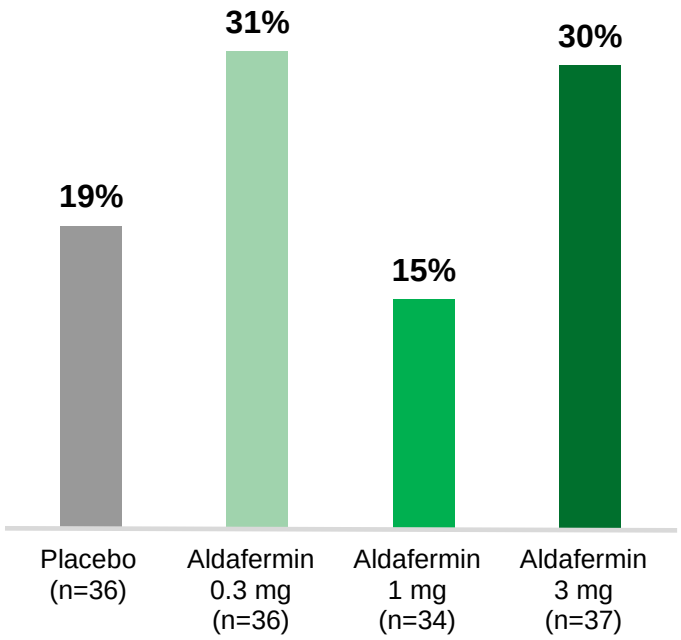
EASL 2019, Oral Abstract, DePaoli et.al.

ALPINE 2/3 for Aldafermin: Efficacy Results on FDA Guided Histological Endpoints

Fibrosis Reversal

Fibrosis Improvement ≥1 Stage with No Worsening of NASH[1] at W24

(% of patients)

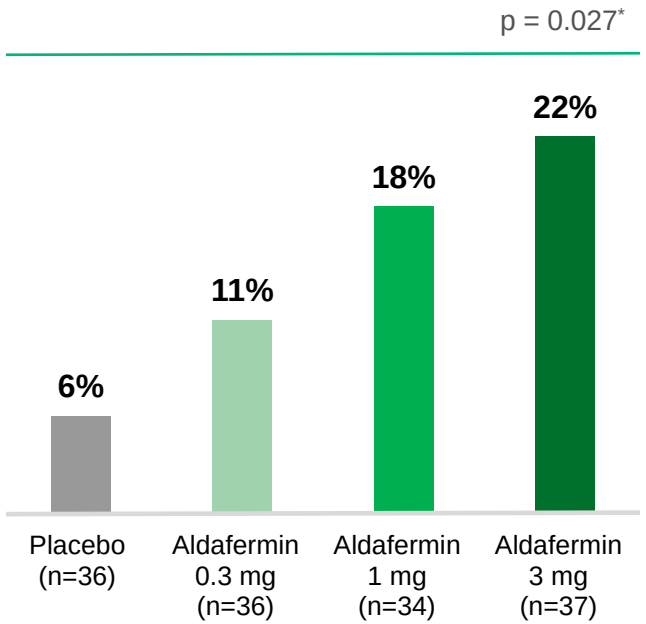


[1] Defined as patients who have an improvement in liver fibrosis by ≥1 stage with no worsening of NASH (no worsening of steatosis, lobular inflammation or hepatocyte ballooning grade) from baseline to W24

NASH Resolution

Resolution NASH with No Worsening of Fibrosis[2] at W24

(% of Patients)

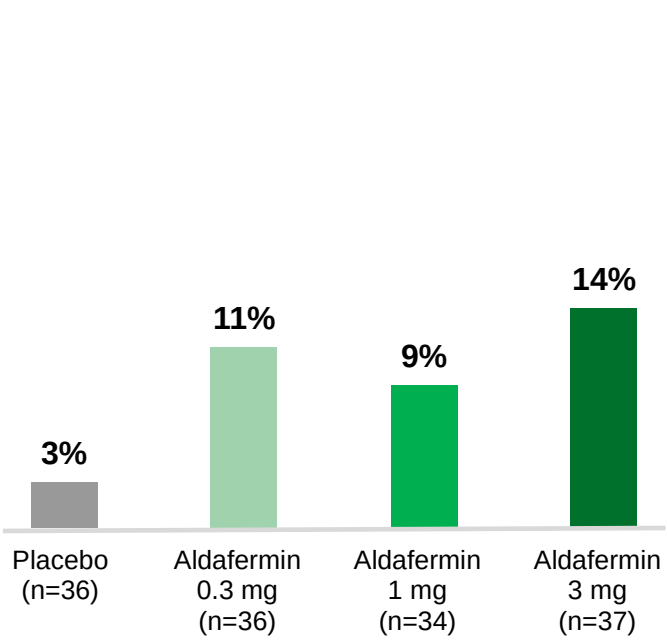


[2] Defined as patients having a NAS score of 0 or 1 for inflammation and 0 for ballooning, with no worsening of fibrosis (no progression of NASH CRN fibrosis stage) from baseline to W24

Fibrosis Improvement and NASH Resolution

Fibrosis Improvement and NASH Resolution[3] at W24

(% of Patients)



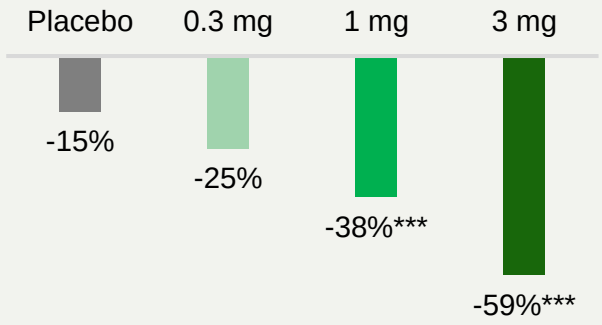
[3] Defined as patients who have an improvement in liver fibrosis by ≥1 stage AND have a NAS score of 0 or 1 for inflammation and 0 for ballooning and no worsening of steatosis at W24

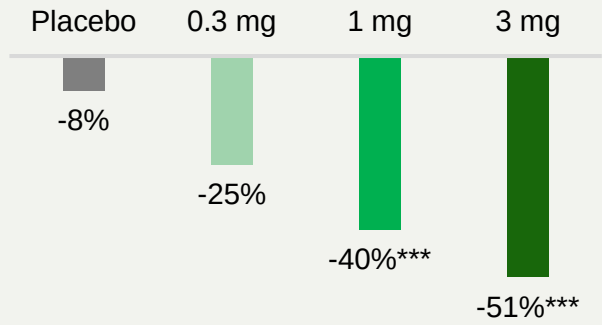
Population with paired biopsies. *Only the 3 mg dose met statistical significance in NASH resolution.

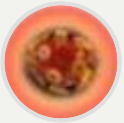
FDA Draft Guidance: Noncirrhotic Nonalcoholic Steatohepatitis With Liver Fibrosis: Developing Drugs for Treatment DECEMBER 2018

ALPINE 2/3 Topline: Consistent Results Across Non-Invasive Measures

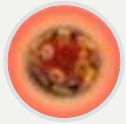
Change in Relative LFC by MRI-PDFF from Baseline



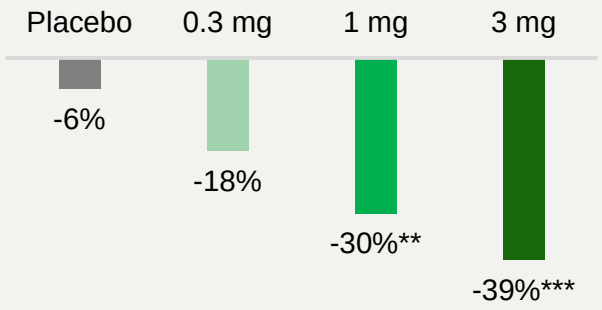


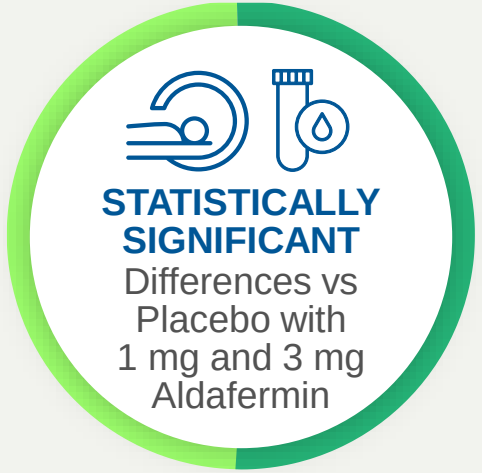


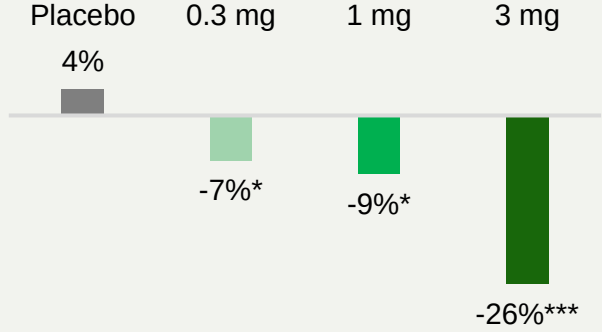
Change in Relative ALT from Baseline



Change in Relative AST from Baseline







Change in Relative Pro-C3 from Baseline

*p≤0.05; **p≤0.01; ***p≤0.001
Intent-to-treat population. Relative values are calculated as mean change from baseline.

ALPINE 2/3: Aldafermin was Well Tolerated with AEs Generally Comparable to Placebo

ALPINE 2/3

Treatment Emergent Adverse Event (TEAE) Classification	Placebo (N=43)	Aldafermin 0.3 mg (N=43)	Aldafermin 1 mg (N=42)	Aldafermin 3 mg (N=43)
Any TEAE	36 (83.7%)	30 (69.8%)	34 (82.9%)	38 (88.4%)
Drug-related TEAE	14 (32.6%)	13 (30.2%)	20 (48.8%)	21 (48.8%)
Serious TEAE	3 (7.0%)	1 (2.3%)	4 (9.8%)	1 (2.3%)
Drug-related TEAE leading to discontinuation	2 (4.7%)	1 (2.3%)	1 (2.4%)	1 (2.3%)
TEAE leading to death	0	0	1 (2.4%)*	0

*determined unrelated to treatment by site investigator; occurred 30 days after the last confirmed aldafermin dose

MedDRA Preferred Term (≥10%)	Placebo (N=43)	Aldafermin 0.3 mg (N=43)	Aldafermin 1 mg (N=41)	Aldafermin 3 mg (N=43)
Diarrhea	6 (14.0%)	3(7.0%)	5 (12.2%)	10 (23.3%)
Nausea	8 (18.6%)	5 (11.6%)	8 (19.5%)	7 (16.3%)
Upper Abdominal Pain	4 (9.3%)	5 (11.6%)	3 (7.3%)	2 (4.7%)
Headache	4 (9.3%)	6 (14.0%)	2 (4.9%)	4 (9.3%)
Constipation	2 (4.7%)	5 (11.6%)	1 (2.4%)	1 (2.3%)
Injection Site Erythema	0	0	4 (9.8%)	6 (14.0%)
Sinusitis	1 (2.3%)	0	5 (12.2%)	1 (2.3%)

- All SAEs were deemed unrelated to treatment by site investigator
- Aldafermin-induced LDL-C elevations safely and effectively managed by background statin regimen

Intent-to-treat population.
TEAE = treatment emergent adverse event; SAE = serious adverse event; LDL-C = low-density lipoprotein cholesterol

ngmBIO

Financial Overview

3Q21 and FY20 Financial Results

STATEMENT OF OPERATIONS (In thousands)	THREE MONTHS ENDED September 30, 2021[1] (unaudited)	FULL YEAR ENDED December 31, 2020
RELATED PARTY REVENUE	$18,575	$87,368
RESEARCH AND DEVELOPMENT EXPENSES	$38,714	$163,972
GENERAL AND ADMINISTRATIVE EXPENSES	$8,867	$27,229
TOTAL OPERATING EXPENSES	$47,581	$191,201
NET LOSS	**($28,865)**	**($102,487)**

BALANCE SHEET	September 30, 2021 (unaudited)	December 31, 2020
CASH, CASH EQUIVALENTS AND SHORT-TERM MARKETABLE SECURITIES	**$383.4M**	**$295.2M**

[1] See the Company's Quarterly Report on Form 10-Q for the quarter ended September 30, 2021 filed with the Securities and Exchange Commission (SEC) on November 4, 2021 and the Company's Annual Report on form 10K for the year ended December 31, 2020 filed with the SEC on March 15, 2021 for more complete financial information

©2021 NGMBio™ | All Rights Reserved.

This presentation, including the illustrations therein, is protected by copyright and other IP rights of NGM Bio and may not be copied or reprinted without the permission of NGM Bio.