Novel Biology. Powerful Medicines. Transformative Impact.

NGM Biopharmaceuticals, Inc.

Corporate Overview

August 2021

NASDAQ: NGM

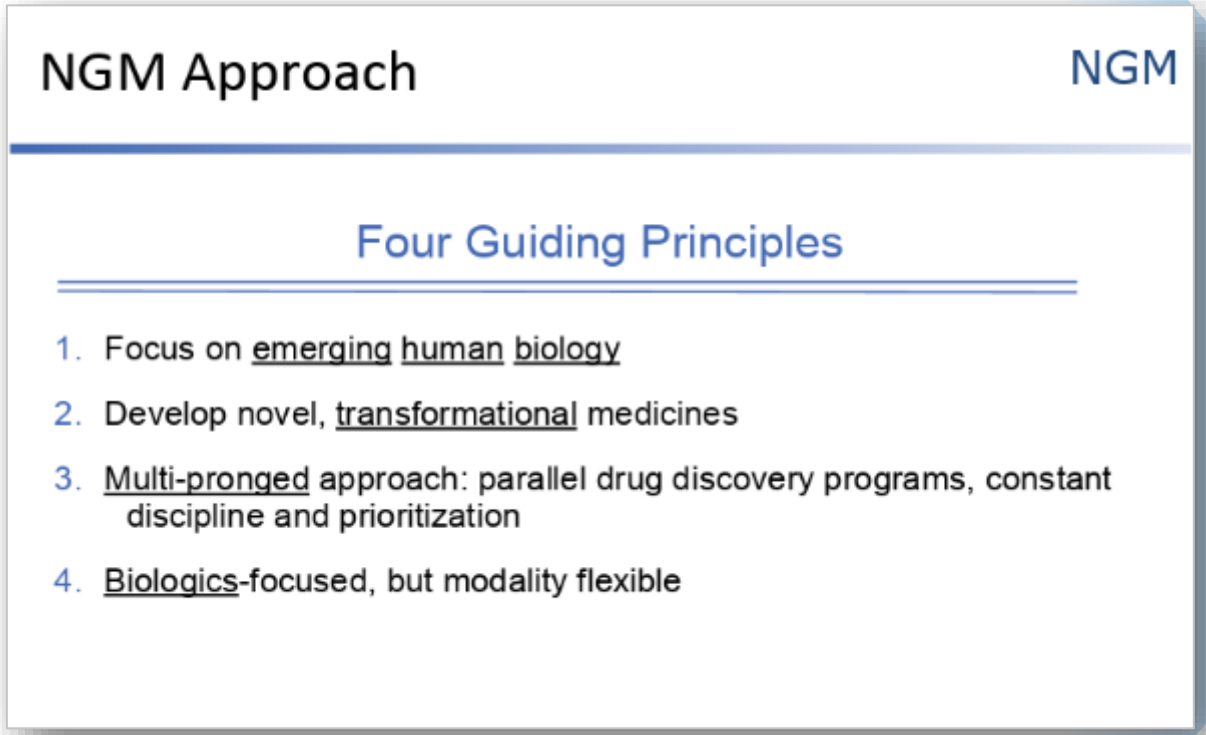
Safe Harbor Statement

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's pipeline, including NGM621, NGM120, NGM707, NGM831, NGM438, MK-3655 and aldafermin; the planned timing of initiation, enrollment, data readouts and results of NGM's clinical trials, including data readouts for NGM621 and NGM120; potential future late-stage development of product candidates in NGM's pipeline, including NGM621 and aldafermin; the potential activity, complementarity, safety, tolerability and efficacy of NGM's product candidates, including the potential for aldafermin's mechanism of action to be well suited for patients with NASH with stage 4 liver fibrosis (F4) and compensated cirrhosis, the potential for NGM621 to have a safety profile advantage due to the absence of PEGylation and its potential for every 8-week dosing, the potential roles of regulating the GDF15/GFRAL pathway and ILT2, ILT4, ILT3 and LAIR1 in cancer and the potential consequences of ILT2, ILT4, ILT3 and LAIR1 blockade; anticipated regulatory submissions and, actions and the timing thereof; potential option exercises by Merck under NGM's amended collaboration with Merck; NGM's vision to build an iconic biologics therapeutic company, its opportunities for value creation and its ability to deliver powerful or transformational treatments; and any other statements other than statements of historical facts. Because such statements deal with future events and are based on NGM'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 failures or delays in successfully initiating, enrolling or completing clinical trials; the risk that results obtained in NGM's clinical trials to date may not be indicative of results obtained in ongoing or future trials, including pivotal trials, including the risk that ongoing or future studies will not show that aldafermin and/or MK-3655 are tolerable or effective treatments for NASH patients or that NGM621 is a tolerable or effective treatment for geographic atrophy (GA); the risk that preclinical studies or modeling may not be indicative of results in future human clinical trials; the risk that clinical trials of NGM438, NGM831, NGM707 and NGM120 will not show that NGM438, NGM831, NGM707 and/or NGM120 are tolerable or effective treatments in cancer or cancer-related cachexia indications; the risk that others may discover, develop or commercialize products before or more successfully than NGM, including in NASH and/or GA; the ongoing COVID-19 pandemic which has adversely affected, and could materially and adversely affect in the future, NGM's business and operations, including NGM'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 or Merck, as applicable, may not receive marketing approvals for any of NGM's product candidates in a timely manner, or at all; seeking and maintaining protection of intellectual property; NGM's reliance on third party manufacturers and delays or problems in the manufacture of product candidates; NGM'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 would not obtain all of the anticipated financial and other benefits of the amended collaboration, and the development and/or commercialization of NGM's product candidates falling within the scope of the amended collaboration could be delayed, perhaps substantially; the sufficiency of NGM's cash resources, including to fund development programs that fall outside of the narrower scope of NGM's amended collaboration with Merck, and need for additional capital; and other risks and uncertainties affecting NGM and its research and development programs, including those described under the caption "Risk Factors" and elsewhere in NGM's quarterly report on Form 10-Q for the quarter ended June 30, 2021 and future filings and reports of NGM with the Securities and Exchange Commission. 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 on its website or otherwise. NGM 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.


NGMBio
Interrogating
HUMAN
BIOLOGY
Engineering
POWERFUL
BIOLOGICS
To
TRANSFORM
TREATMENT

Our Vision: Build a Next-Generation, Leading Biologics Company

Original Business Plan in 2008



REPEATABLE & SCALABLE

Today

DELIVER TRANSFORMATIVE TREATMENTS

- **3** Therapeutic areas
- **7** Disclosed programs
- **5** Programs in clinical development
- **4** Ph2/Ph2b studies ongoing

Progressing Our Expansive Pipeline

RETINAL					**Rights**
NGM621	Anti-Complement C3 Antibody	Geographic Atrophy	PHASE 2	Topline Data Expected 2H22	Merck option at PoC; if optioned, NGM to receive milestones + double-digit royalties **or** up to 50% profit/cost share[1]
ONCOLOGY					
NGM120	GFRAL Antagonistic Antibody	Cancer & Cancer-related Cachexia	PHASE 1A/1B	Ph1a/1b Interim Dose Finding Data Expected in 2H21	Global
		Metastatic Pancreatic Cancer & Cancer-related Cachexia	PHASE 2	Placebo-controlled Expansion Enrolling	Global
NGM707	ILT2/ILT4 Dual Antagonist Antibody	Advanced Solid Tumors	PHASE 1/2	Enrolling	Global
NGM831	ILT3 Antagonist Antibody	Advanced Solid Tumors	IND-ENABLING STUDIES	Ph1 Initiation Expected 1H22	Global
NGM438	LAIR1 Antagonist Antibody	Advanced Solid Tumors	IND-ENABLING STUDIES	Ph1 Initiation Expected 1H22	Global
LIVER & METABOLIC DISEASES					
MK-3655 (NGM313)	FGFR1c/KLB Agonistic Antibody	NASH F2/F3	PHASE 2B	Enrolling	Merck optioned at PoC; NGM to receive milestones + double-digit royalties **or** up to 50% profit/cost share[1]
Aldafermin	FGF19 Analog	NASH F4	PHASE 2B	Enrolling	Global

[1] *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; stage 2 or 3 or 4 liver fibrosis (F2/F3/F4); PoC = proof of concept

The NGM Team by the Numbers

100+
Researchers

200+
Employees

122,000 sq. ft. total footprint including **60,000 sq. ft.** of active lab space

A team that has contributed to **10+** commercially successful medicines

40+
Patents held

>$500M
Received from business development collaborations

As of December 31, 2020

Biology-Driven Drug Discovery: Connecting the Dots

BIOLOGY TECHNOLOGY MEDICINE

Empirical Approach: Integrated, **powerful,** multi-disciplinary platform

- Genetics: human and model systems
- Industrial-scale functional genomics
  - *in vivo*-based discovery
  - High content *in vitro* analysis
  - Orphan ligand-receptor matching
- Computational biology

Biology-Driven Drug Discovery: Designing Unique Solutions for Complex Problems

BIOLOGY

TECHNOLOGY

MEDICINE

Versatile Biologics Platform

Fit-for-purpose design

- Engineered proteins
- Engineered antibodies
- Multi-specific / multi-functional therapeutics

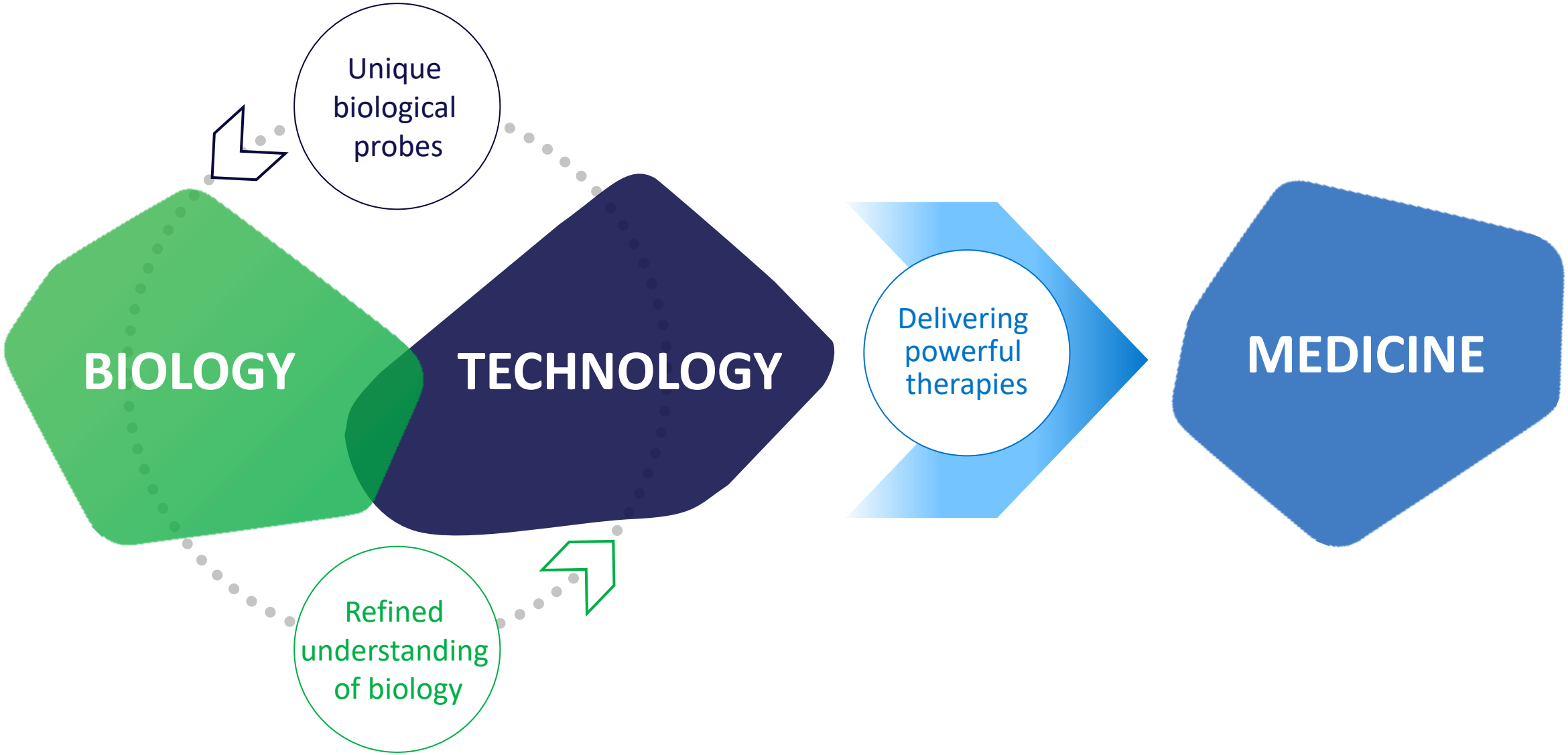
Flexible targeting

- Hormones
- Ligands / receptors
- Enzymes
- Transporters / ion channels
- Cell-cell interactions

Tunable pharmacology

- Antagonists / inhibitors
- Agonists / activators
- Modulators
- Biased ligands
- Multi
  - specificity
  - functionality
  - valency
- Tissue specificity

Biology-Driven Drug Discovery: Seamless Integration of Biology and Technology



NGM621 in Geographic Atrophy

Geographic Atrophy 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]

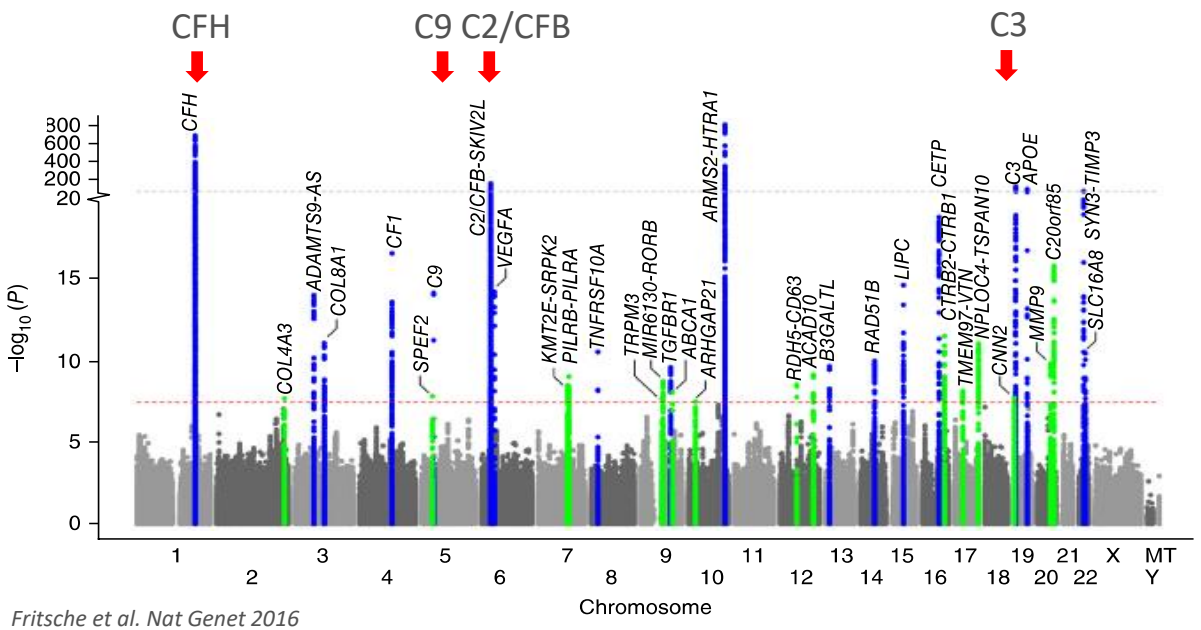
NEURODEGENERATIVE DISEASE OF THE RETINA

No FDA-approved treatments

1 = Boyer et al, Retina 2017
GA = geographic atrophy

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

GENETIC EVIDENCE

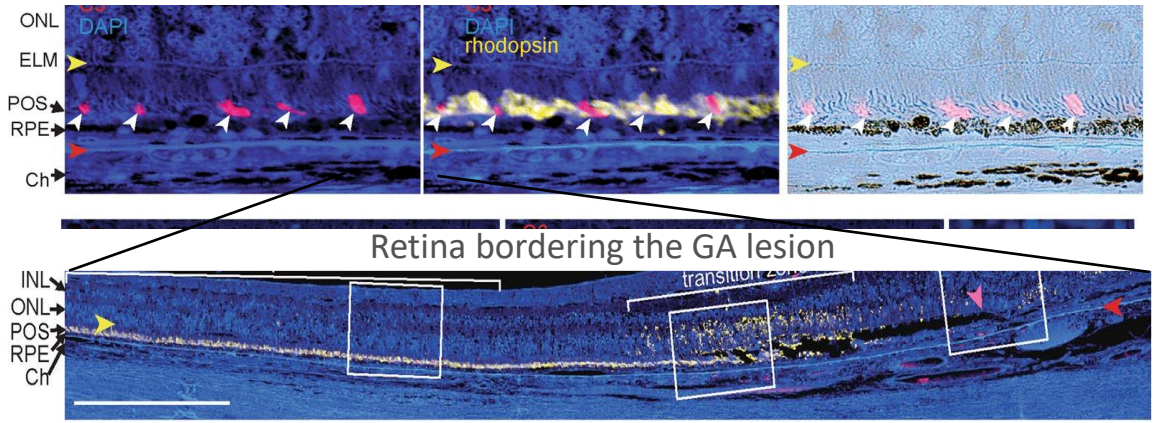


Fritsche et al. Nat Genet 2016

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



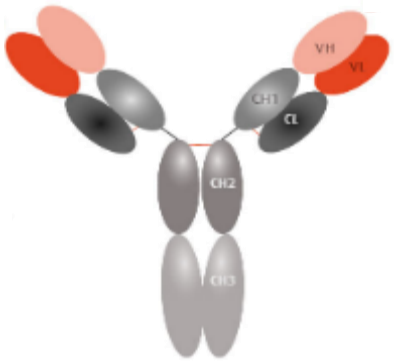
Katschke et al. Sci. Reports 2018

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

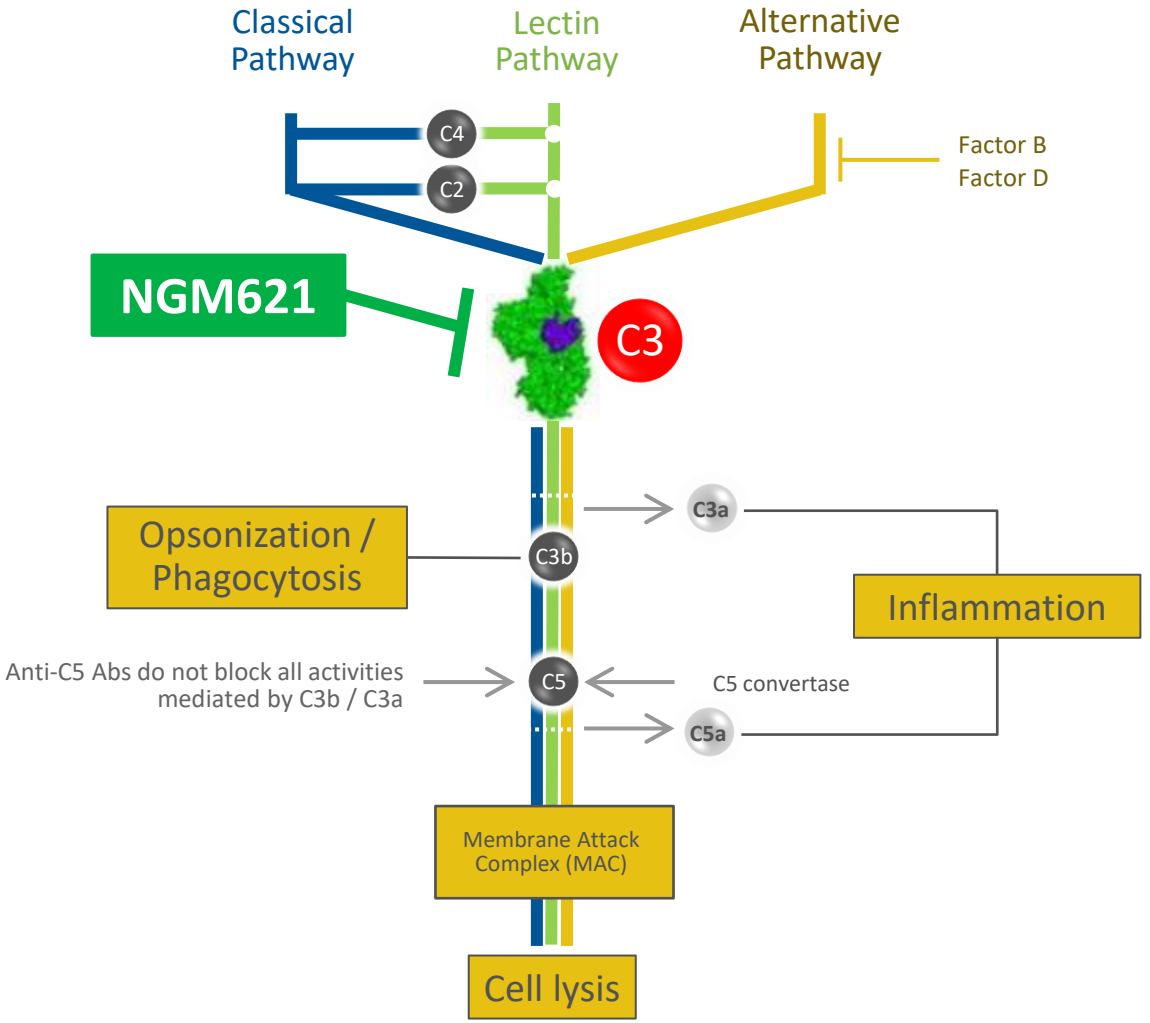
NGM621 MOLECULE ATTRIBUTES



NGM621

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

COMPLEMENT CASCADE



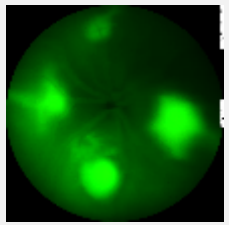
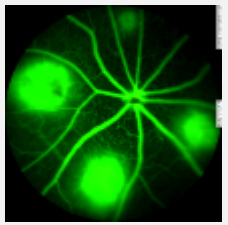
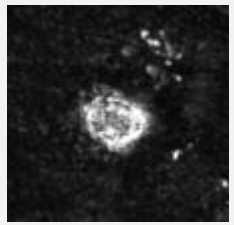
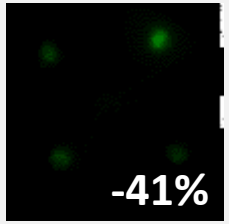
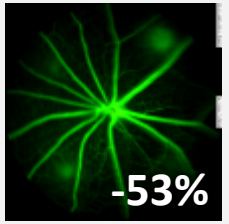
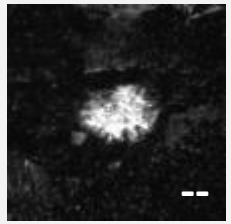
SCIENTIFIC RATIONALE: NGM621 FOR GEOGRAPHIC ATROPHY

- Complement dysregulation is implicated in GA/AMD; C3 is the central convergence point in the complement cascade
- NGM621 is a novel monoclonal antibody that potently inhibits C3, blocking all complement pathways, with the potential for extended dosing without PEGylation

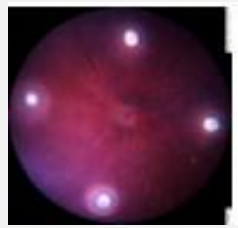
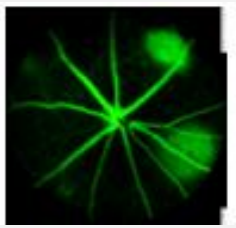
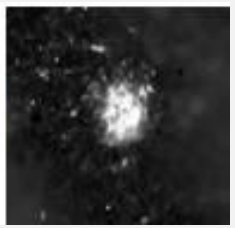
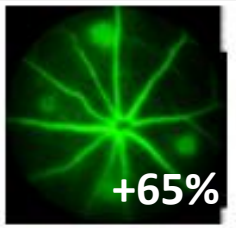
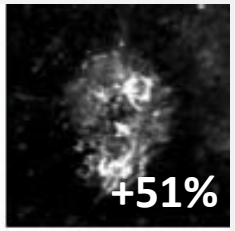
IgG1 = immune globulin G1; PEG = polyethylene glycol.

Preclinical Data Shows PEG Can Exacerbate CNV Post-Laser Injury

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

	Fundus (Day 0)	Complement activation (Anti-C3d - Day 2)	Vascular Leakage (Fluorescein angiography - Day 7)	Angiogenesis (Isolectin B4 - Day 7)
Anti-KLH (IVT)				
Anti-C3 Ab (IVT)				

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

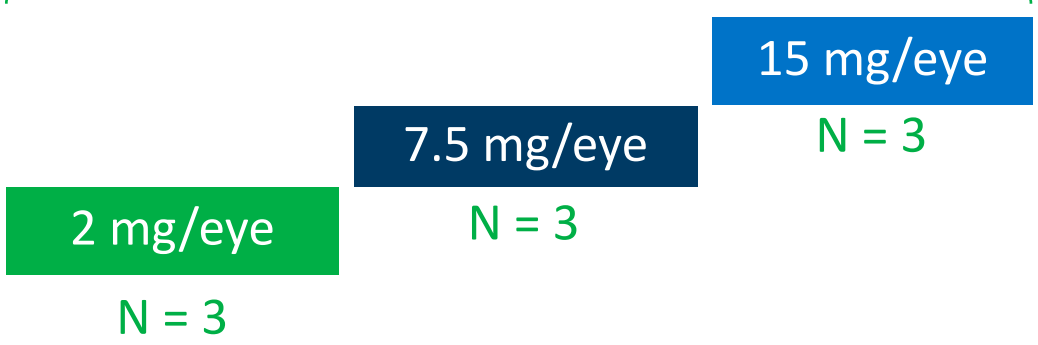
	Fundus (Day 0)	Vascular Leakage (Fluorescein angiography - Day 7)	Angiogenesis (Isolectin B4 - Day 7)
PBS (IVT)			
PEG-40kD (IVT)			

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

Wei-Sheng Chen et. al. ARVO 2020

Phase 1 Study Objectives and Design

SINGLE-ASCENDING DOSE COHORTS



MFD

MULTIDOSE COHORT

15 mg/eye
Q4W X 2

N = 6

STUDY OBJECTIVES

- **Primary:** To evaluate the safety and tolerability of single and multiple IVT injection(s) of NGM621 in patients with GA
- **Secondary:** To characterize the PK of single or multiple doses of and evaluate immunogenicity of NGM621 (serum ADA levels)

COHORT DESIGN

- 3 Single-Ascending Dose Cohorts of 2mg, 7.5mg, and 15mg
- 1 Multidose Cohort of 15mg NGM621 given twice, 4 weeks apart
- Patients dosed sequentially, followed for 12 weeks
- Safety review performed after sentinel patient dosed and prior to enrollment to subsequent cohorts

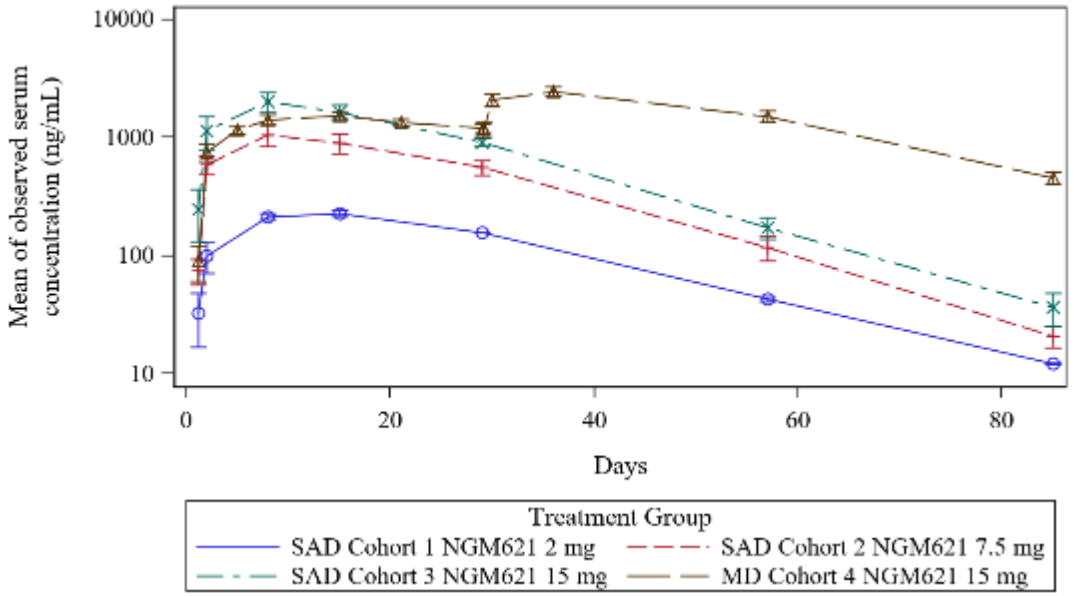
ADA = anti-drug antibodies; IVT = intravitreal; MFD = maximum feasible dose; PK = pharmacokinetics; Q4W = every 4 weeks.

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

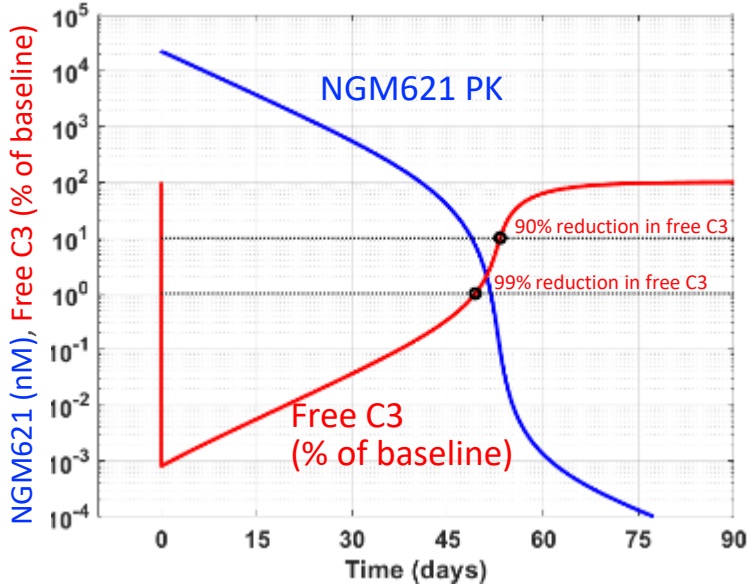
Mean Serum Concentration-time Profile post-IVT of NGM621



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..

Clinical Development of NGM621 has Rapidly Advanced

ADVANCING CLINICAL DEVELOPMENT

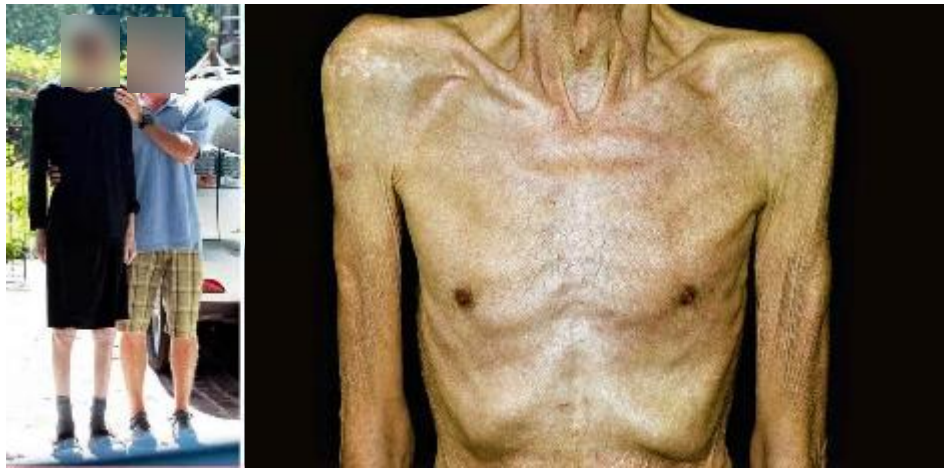
- ✓ **Phase 1** in GA patients successfully completed
  - ✓ Data presented at AAO in November 2020
  - ✓ NGM621 well tolerated
  - ✓ No drug-related adverse events or serious adverse events
  - ✓ No CNV developed in either eye

- ✓ **Phase 2** sham-controlled, double-masked study for GA (CATALINA)
  - Evaluating the safety and efficacy of intravitreal NGM621 dosed every 4 or 8 weeks compared to matched sham arms
  - 320 patients from US sites
  - Designed to be a potential Phase 3-enabling study
  - Enrollment completion in July 2021; topline data expected during 2H22

NGM120: An Inhibitor of GDF15 Signaling for the Treatment of Cancer and Cancer-Related Cachexia

Cancer-Related Cachexia (Cachexia)

- Common ‘wasting’ syndrome linked to many cancers
  - No FDA approved therapy
  - Significant contribution to morbidity and mortality
- Cachexia is estimated affect **to 60% to 80% of advanced cancer patients** and to be responsible for **~30% of all cancer deaths**[1]
- Weight loss exacerbated by many chemotherapies
  - Often linked to nausea and vomiting
  - May require dose reduction of chemotherapy
- Elevated GDF15 associated with both chemotherapy and cancer cachexia



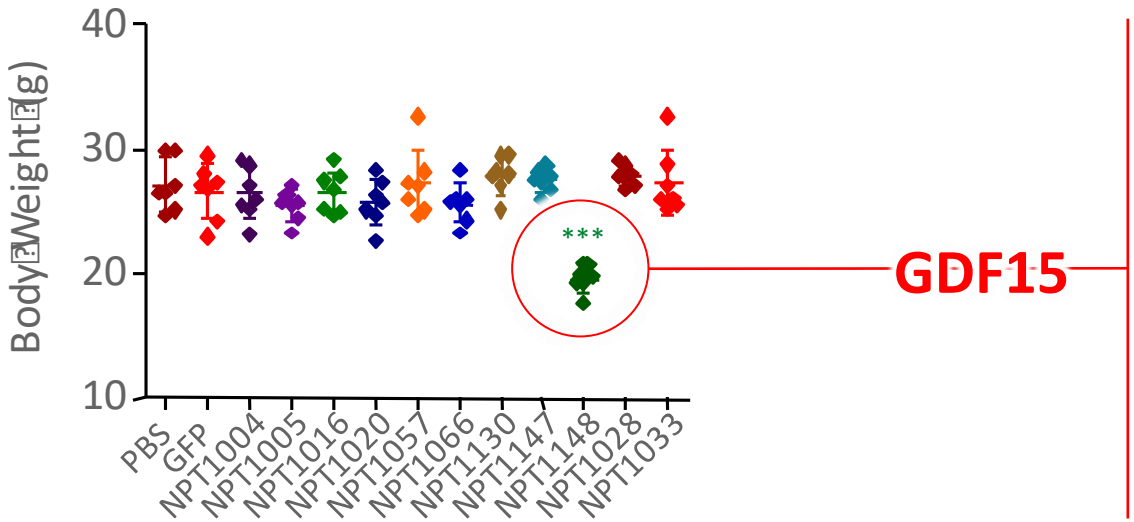
No FDA-approved treatments

1 = Haehling et al, J. Cachexia Sarcopenia Muscle, 2010

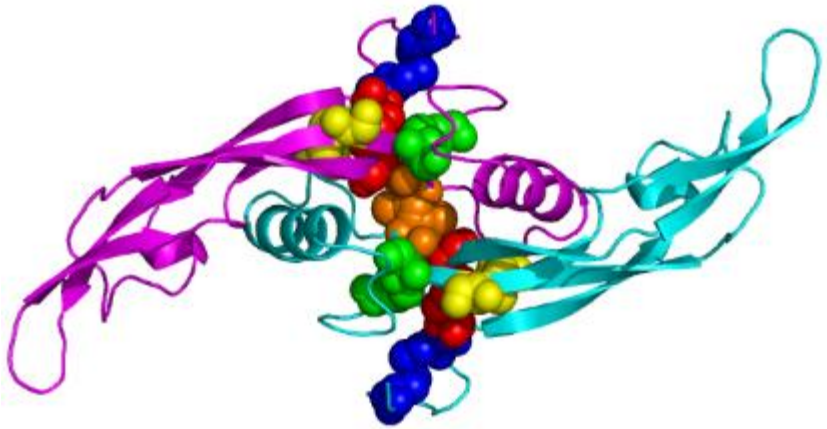
Growth Differentiation Factor 15 (GDF15): A Powerful Biological Pathway

- Identified GDF15 ‘wow biology’ through the systematic *in vivo* screening of >1000 secreted factors
- GDF15 is secreted by macrophages, endothelial cells, myocytes, adipocytes, and multiple tumor types in response to cellular stress
- GDF15 regulates feeding, metabolism and immune function

In-Vivo Systematic Screening of Human Genes using AAV*



GDF15



X-ray Crystal Structure of dimeric GDF15
(1.97Å resolution, NGM)

*AAV – Adeno-Associated Virus

NGM Unpublished Data
GDF = growth differentiation factor

NGM120 is an Antagonist Antibody Inhibiting GFRAL

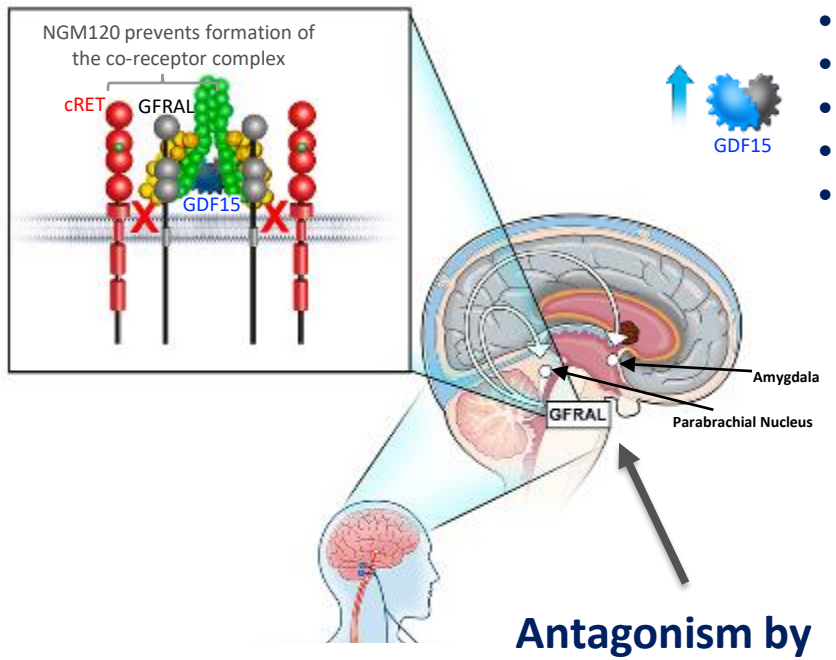
NGM was the first to identify GDF15's cognate receptor, GFRAL, and the associated signaling pathway[1]

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

Preclinical studies suggest that NGM120 may:

- Reverse human tumor-induced body weight loss in mice
- Reduce tumor growth and improves survival in syngeneic orthotopic pancreatic tumor model
- Prevent cisplatin-induced GDF15-mediated weight loss in mice

- **Ph1a/1b NGM120 dose-finding study in patients with cancer; interim data expected at ESMO in 3Q21**
- **Ph2 NGM120, placebo-controlled expansion in patients with metastatic pancreatic cancer initiated in 1Q21**



GDF15 levels are increased by:

- Tumor
- Chemotherapy
- Infection
- Inflammation
- Other stressors

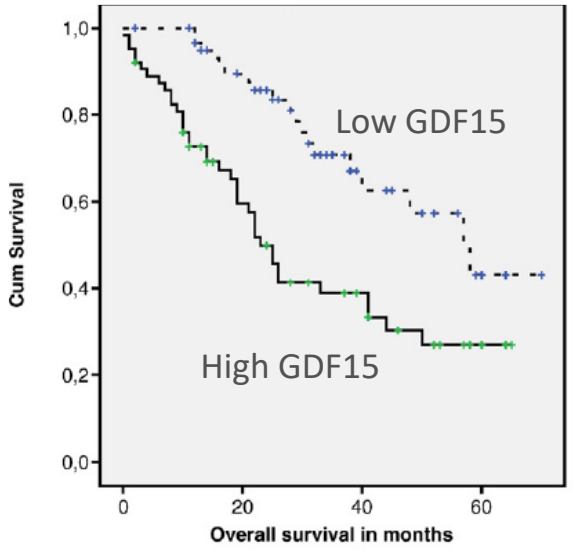
Antagonism by NGM120 may result in:

- Appetite Regulation
- Metabolic Regulation
- Immune Modulation

1 = Hsu et. al., Nature 2017; GFRAL = GDNF Family Receptor Alpha-Like

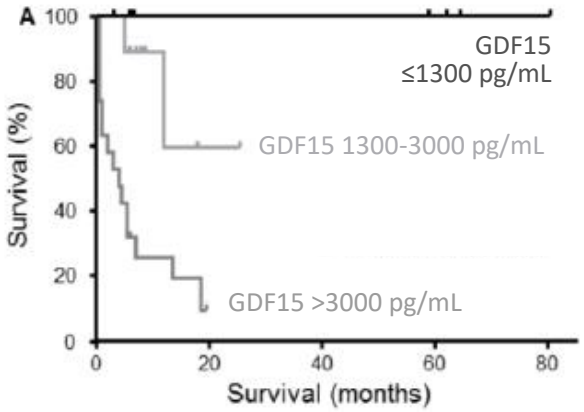
Elevated GDF15 Levels are Linked to Poor Survival in Multiple Cancers

Ovarian Cancer



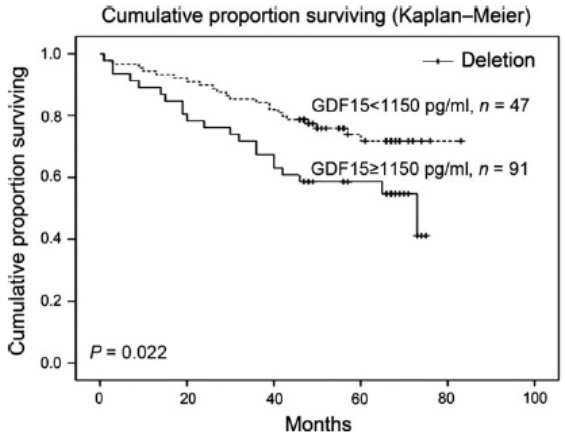
Staff *et al*., *Gynecologic Oncology* 2010

Prostate Cancer



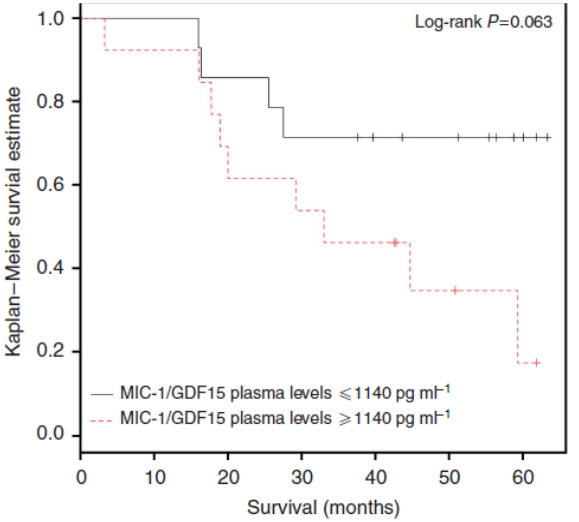
Winand *et al.*, *J Mol Biomark Diagn* 2014

Colorectal Cancer (CRC)



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

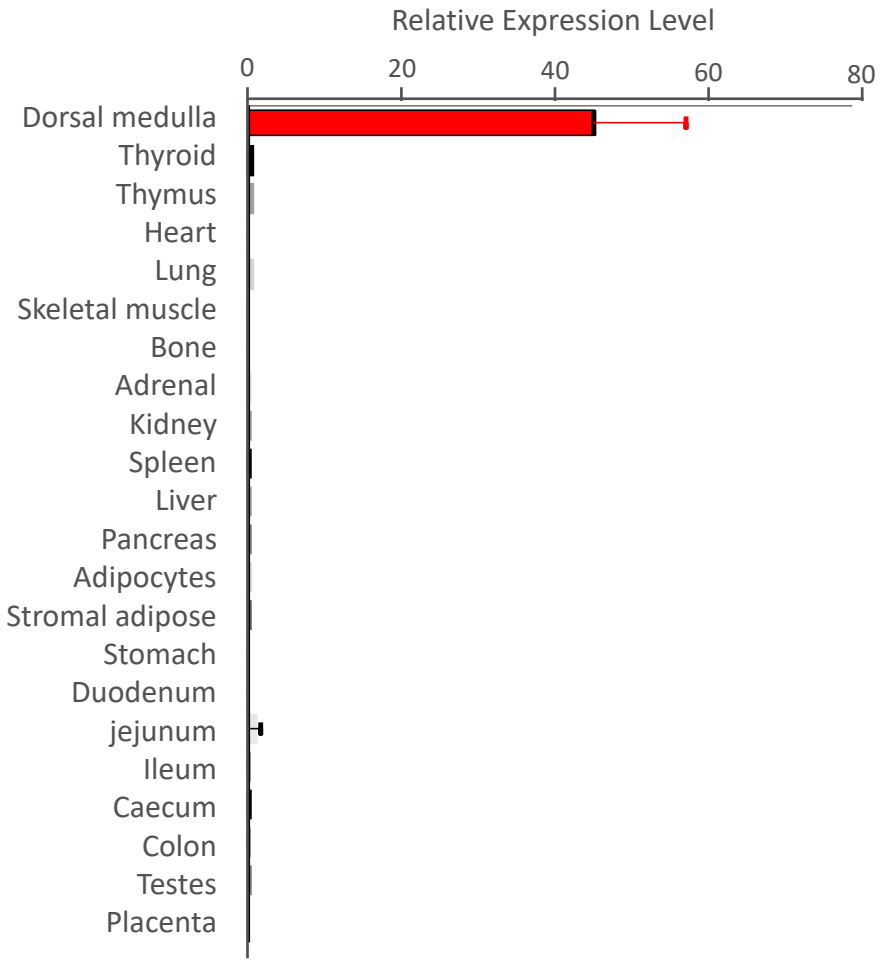
Esophageal Cancer



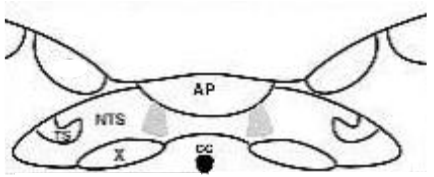
Fisher *et al., BJC* 2015

The GDF15 Receptor, GFRAL, is Exclusively Expressed in the Brain Stem

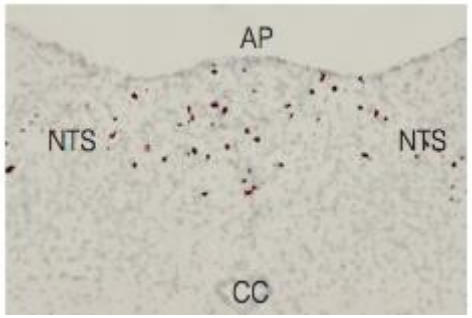
GFRAL Gene Expression Profile



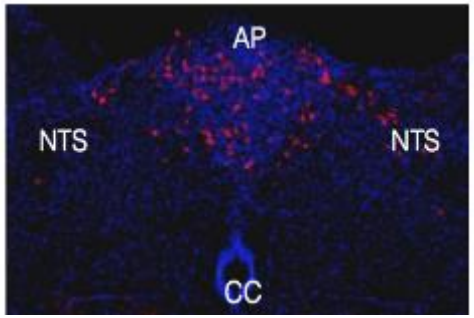
GFRAL Expression in the Dorsal Medulla, Exclusively Expressed in the Brain Stem



AP: Area Postrema
NTS: Nucleus of Solitary Tract
CC: Central Canal



in situ hybridization

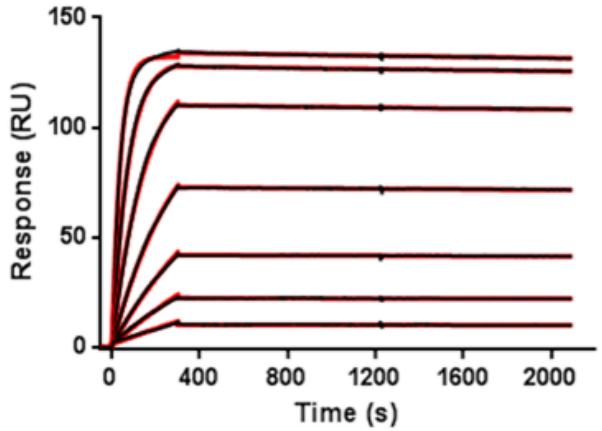


Immunofluorescence

Hsu et. al., Nature 2017

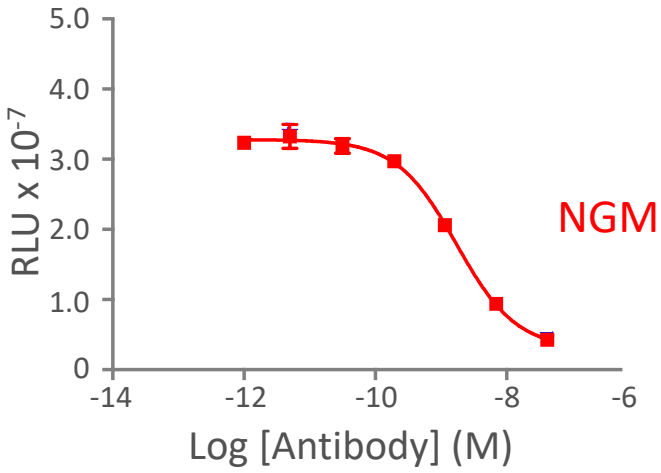
NGM120 Binds with High Affinity to GFRAL and Acts as a Non-Competitive Antagonist of Receptor-Mediated Signaling

NGM120 binding to GFRAL (Biacore)

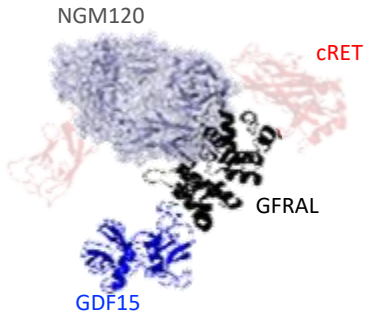


K_D=785 fM
(KinExA)

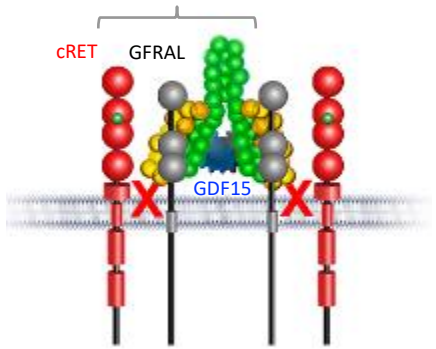
Inhibition of GDF15 signaling by NGM120



NGM120 prevents formation of the co-receptor complex



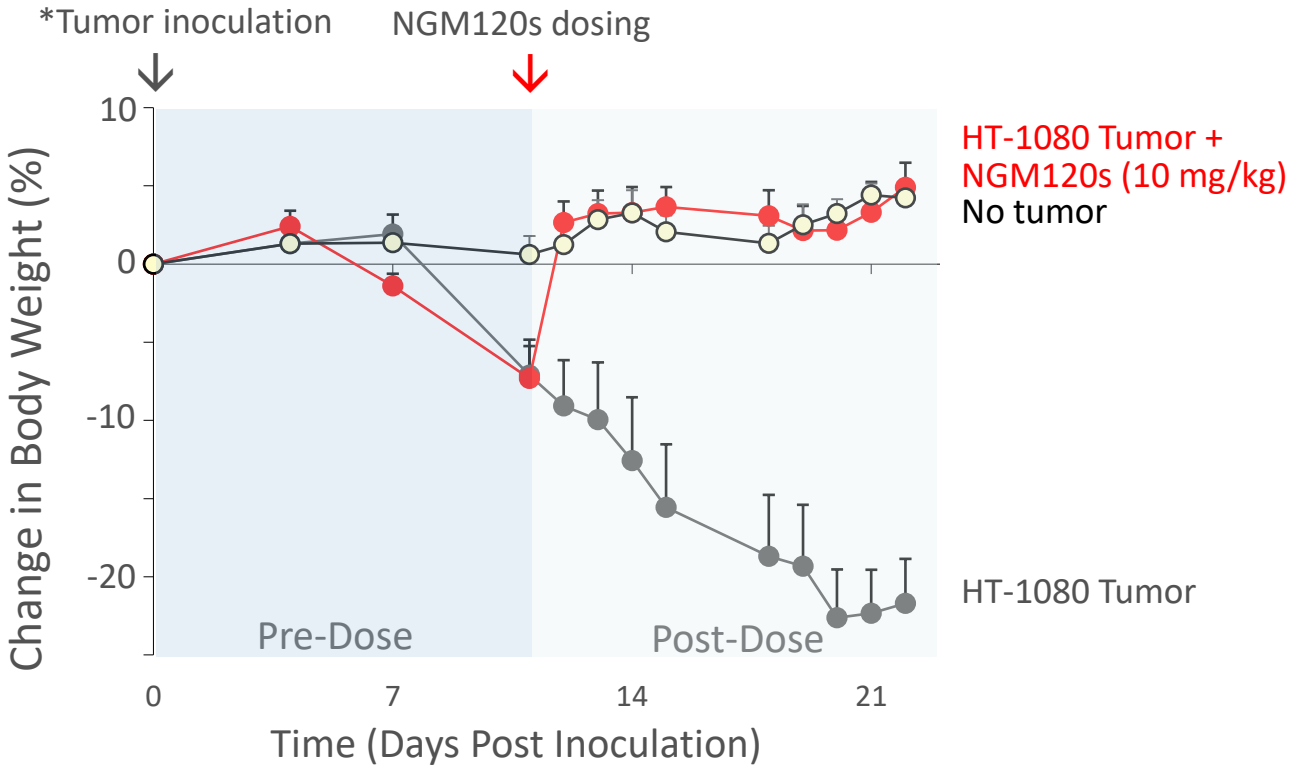
GDF15 co-receptor
Complex with cRET



Suriben et. al.., Nat Med 2020

NGM120s Rapidly Reversed Human Tumor-Induced Body Weight Loss in a Murine Cachexia Model

A Murine Model of Cancer Cachexia Syndrome



Study Model: Tumor-bearing (HT-1080 (Human fibrosarcoma cell line)) SCID mice; (n=6/group)

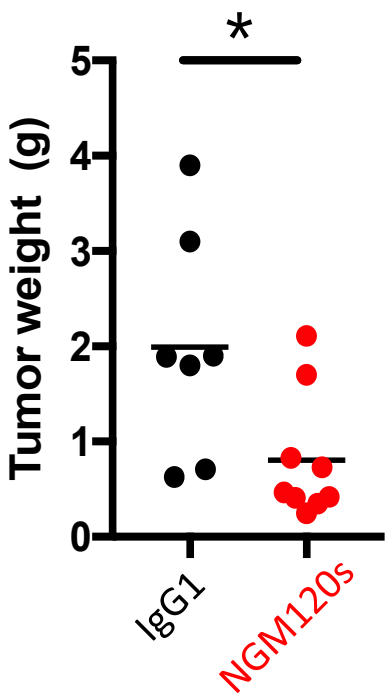
Suriben et. al.., Nat Med 2020; NGM120s is a Tool Compound for NGM120

NGM120s Reduced Tumor Growth and Improved Survival in a Pancreatic Tumor Model

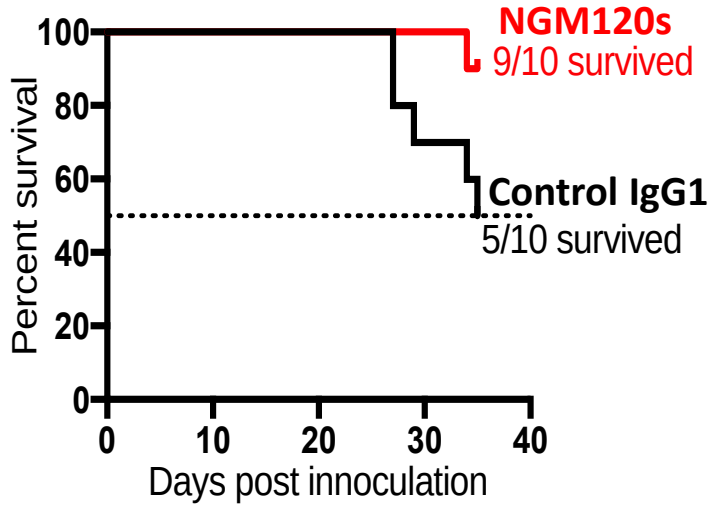
Tumor Model

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

Tumor Weight



Survival

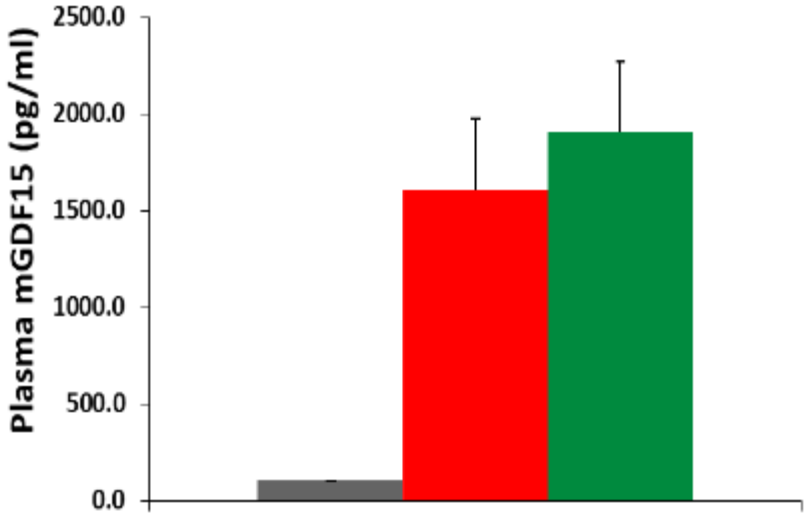


NGM Unpublished Data
NGM120s is a Tool Compound for NGM120

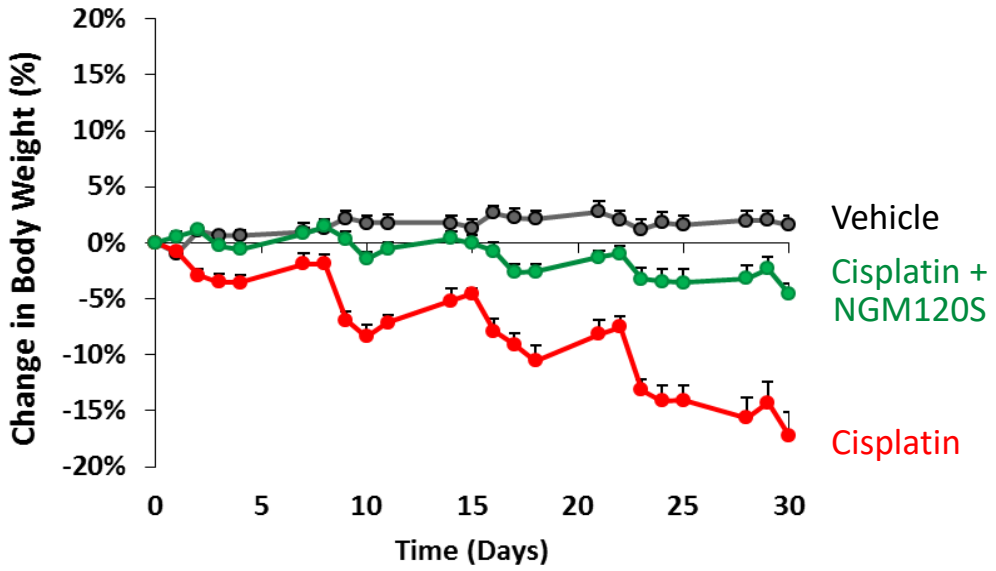
NGM120s Prevented Cisplatin-induced GDF15-mediated Weight Loss in Mice

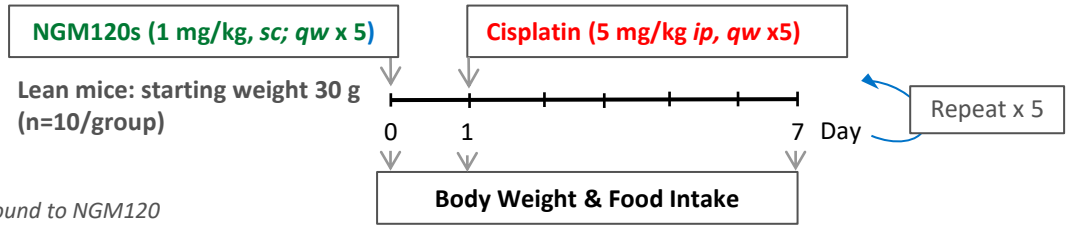
- Administration of NGM120 also preserved lean body mass and muscle function in mice treated with cisplatin

Plasma GDF15 Levels



Change in Body Weight





NGM Unpublished Data
NGM120s is a Tool Compound to NGM120

NGM120 in Clinical Development to Treat Cancer and Cancer-Related Cachexia

- Completed single (n=48) and multiple (n=44) ascending dose cohorts in first-in-human healthy volunteer studies
  - Well-tolerated at all doses
  - No serious adverse events or adverse event of interest
  - $T_{1/2}$ approximately 35 days
- Plan to present interim data from dose finding studies for Ph1a monotherapy in patients with select solid tumors (n=18) and Ph1b in combination with gemcitabine and Abraxane® (paclitaxel protein bound) in patients with metastatic pancreatic cancer (n=8) at ESMO 2021
- Phase 2 expansion study in metastatic pancreatic cancer patients initiated in 1Q21
  - Randomized, single-blind (sponsor unblinded), placebo-controlled, multi-center trial (n=60)
  - Patients will be randomized 1:1 to receive either NGM120 or placebo monthly in combination with the first-line standard of care, gemcitabine and Abraxane
  - Assessment of both cancer and 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.

NGM707 in Advanced Solid Tumors

NGM707 is a Dual Antagonist Antibody Inhibiting ILT2 and ILT4

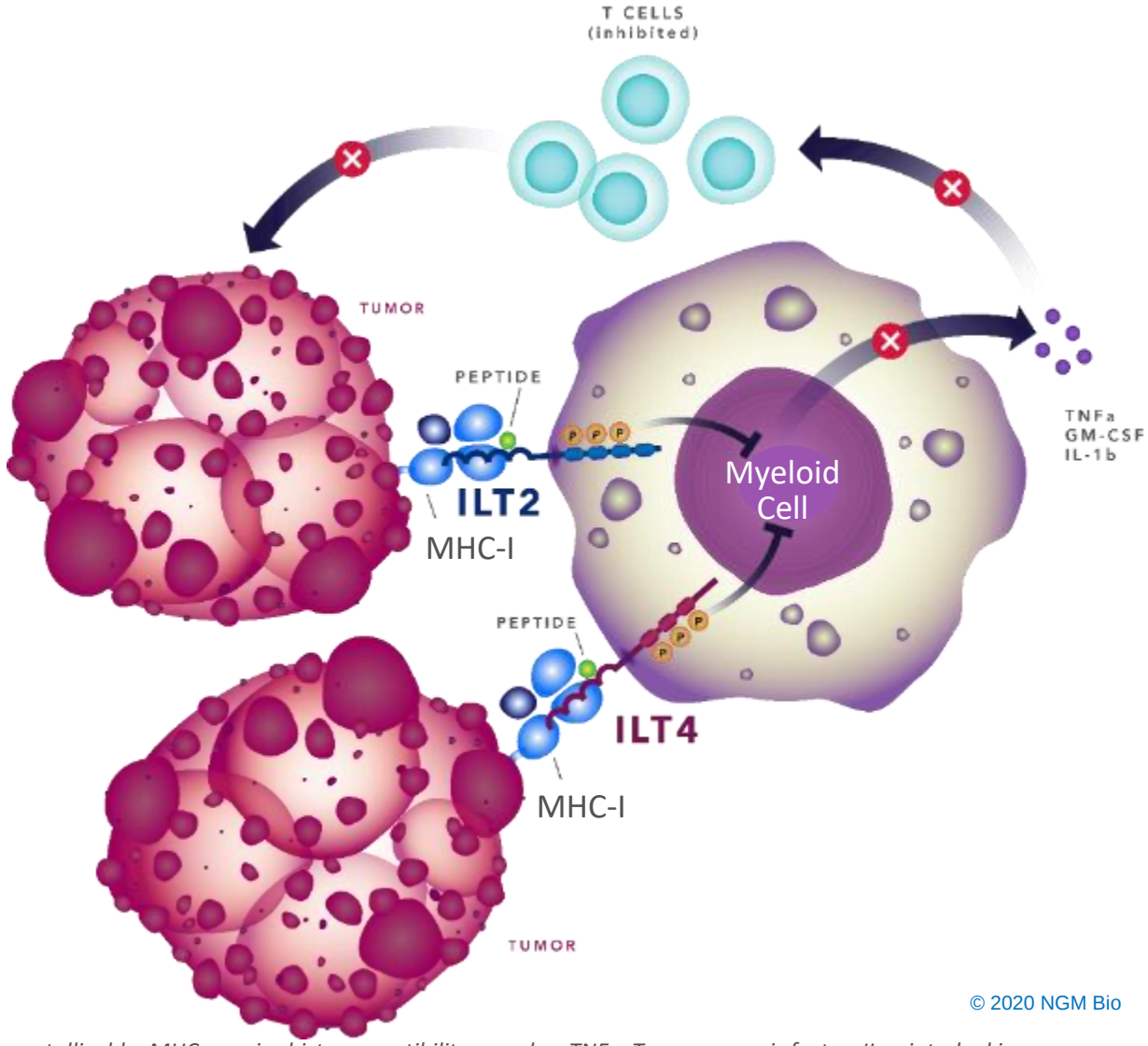
Potent, first-in-class antibody targeting the myeloid-enriched inhibitory receptors ILT2 (LILRB1) and ILT4 (LILRB2)

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 acts synergistically to reverse suppression of Fc receptor signaling

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



ILT2 = Immunoglobulin-like transcript 2; ILT4 = Immunoglobulin-like transcript 4; NK = Natural killer; FC = fragment crystallizable; MHC = major histocompatibility complex; TNF = Tumor necrosis factor; IL = interleukin, GM-CSF = Granulocyte-macrophage colony-stimulating factor

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

Upregulated in certain cancer types[1-5]

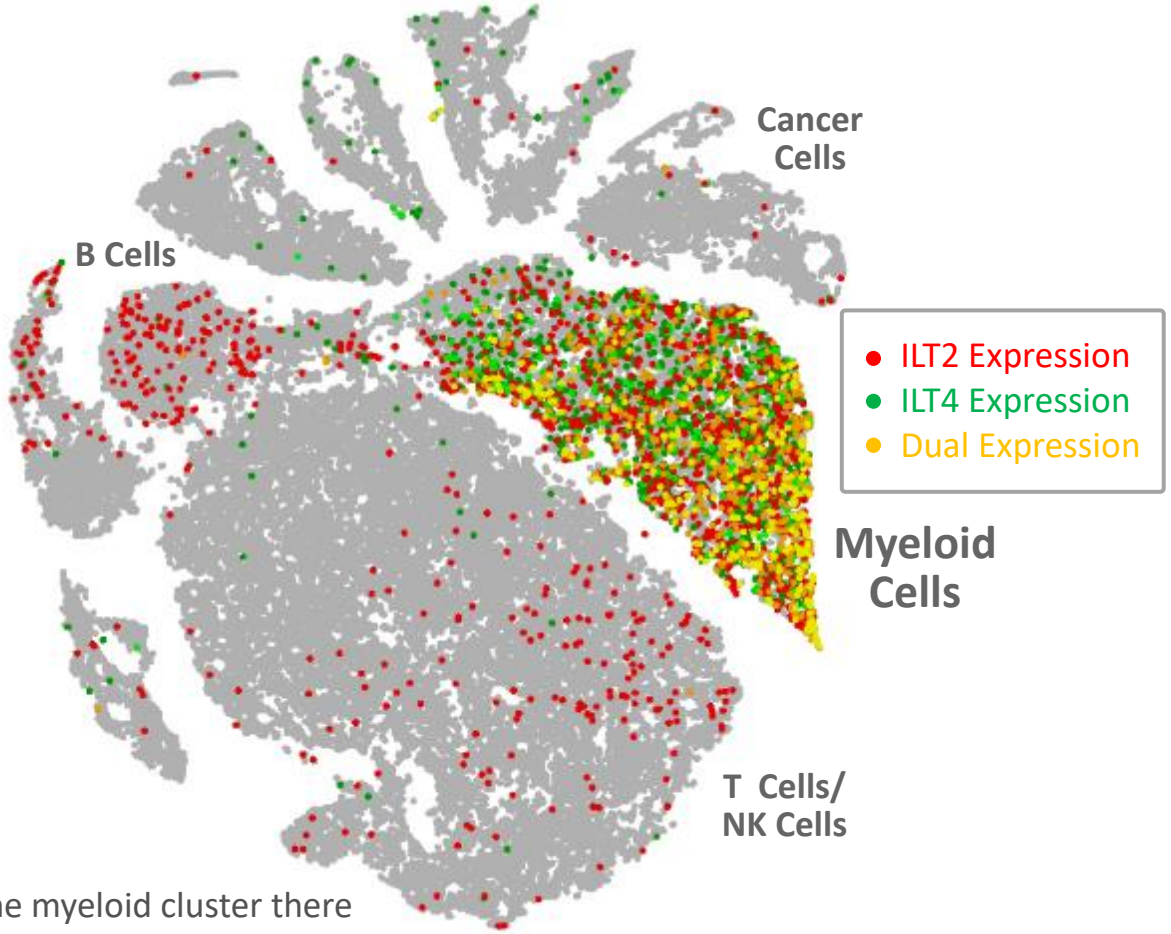
- ILT2 and ILT4 receptors are expressed on myeloid cells (APCs, MDSCs, macrophages, granulocytes) in the tumor microenvironment
- ILT2 additionally exhibits expression on natural killer (NK) cells, B cells and a subset of highly cytolytic T cells

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:

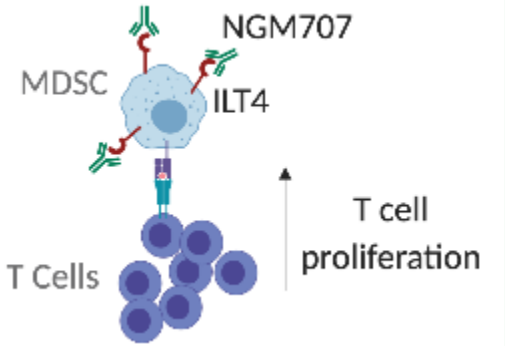
ILT2+, ILT4-
ILT2-, ILT4+
ILT2+, ILT4+

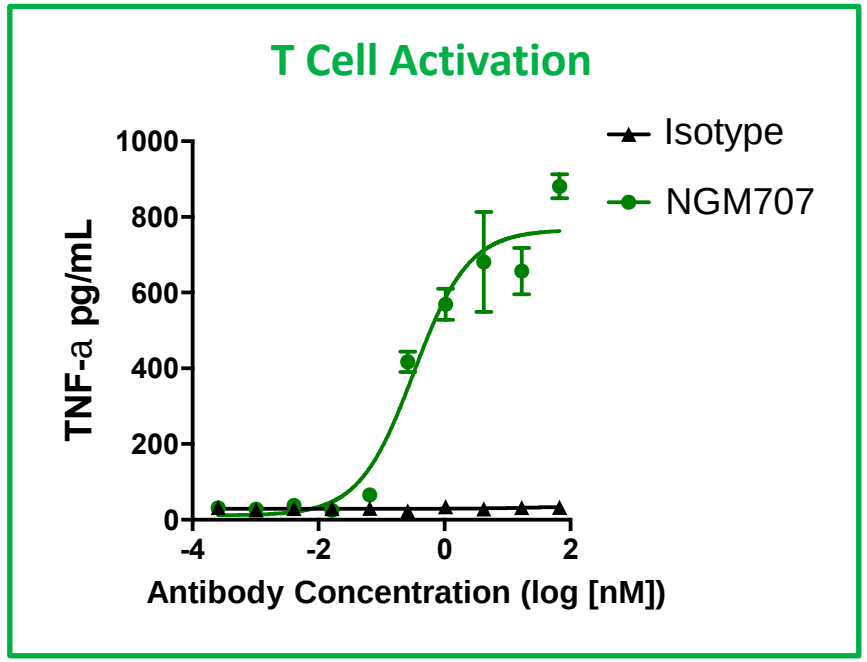
Blocking both ILT2 and ILT4 may provide further benefit

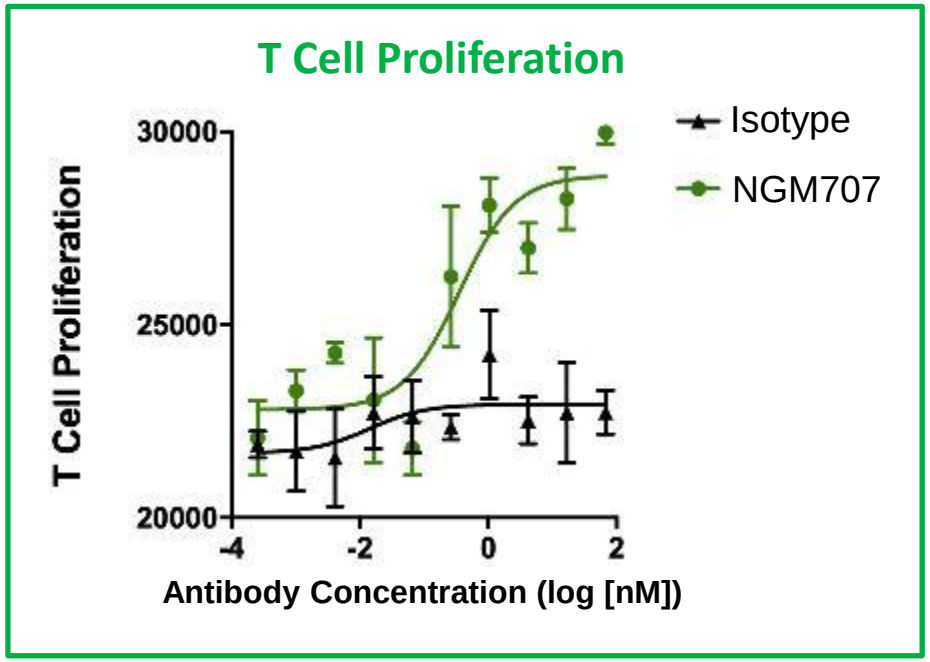
1. Tirosh et al., Science, 2016 2. Li et al., Nat Genet, 2017 3. Puram et al., Cell, 2017 4. Azizi et al., Cell, 2018 5. Lambrechts et al., Nat Med, 2018 6. Sade-Feldman et al., Cell, 2018
TAM = Tumor-associated Macrophage; APC = Antigen-presenting Cell; MDSC = Myeloid-derived Suppressor Cell

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

ILT4 antagonism enhances T cell activity and proliferation







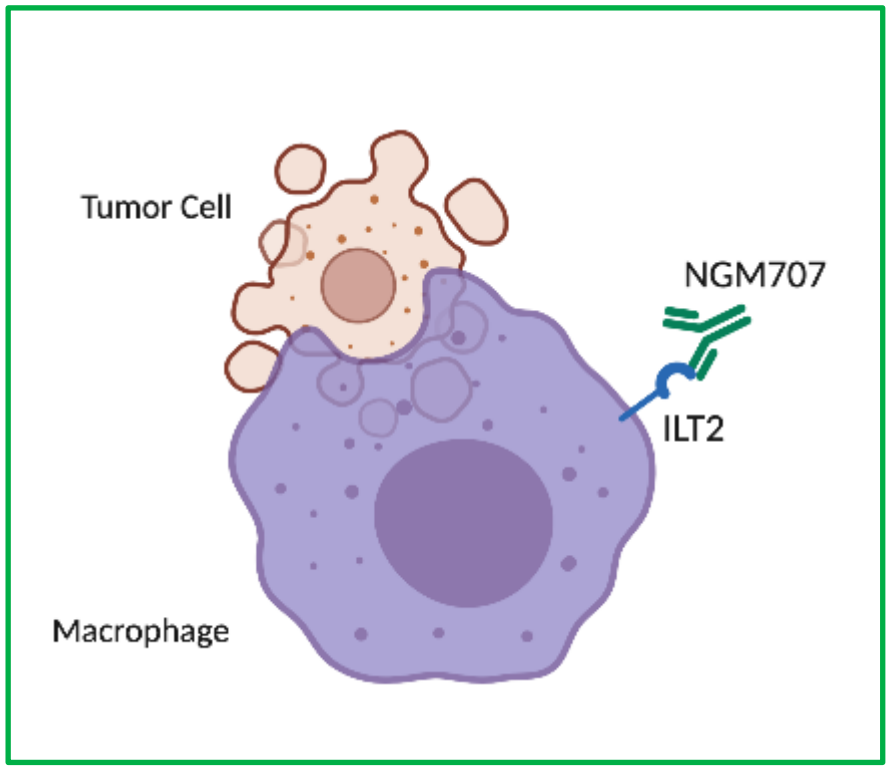
MDSC generated from monocytes using cancer cell-conditioned media (OVISE cells)
MLR performed by mixing MDSC with allogeneic T cells

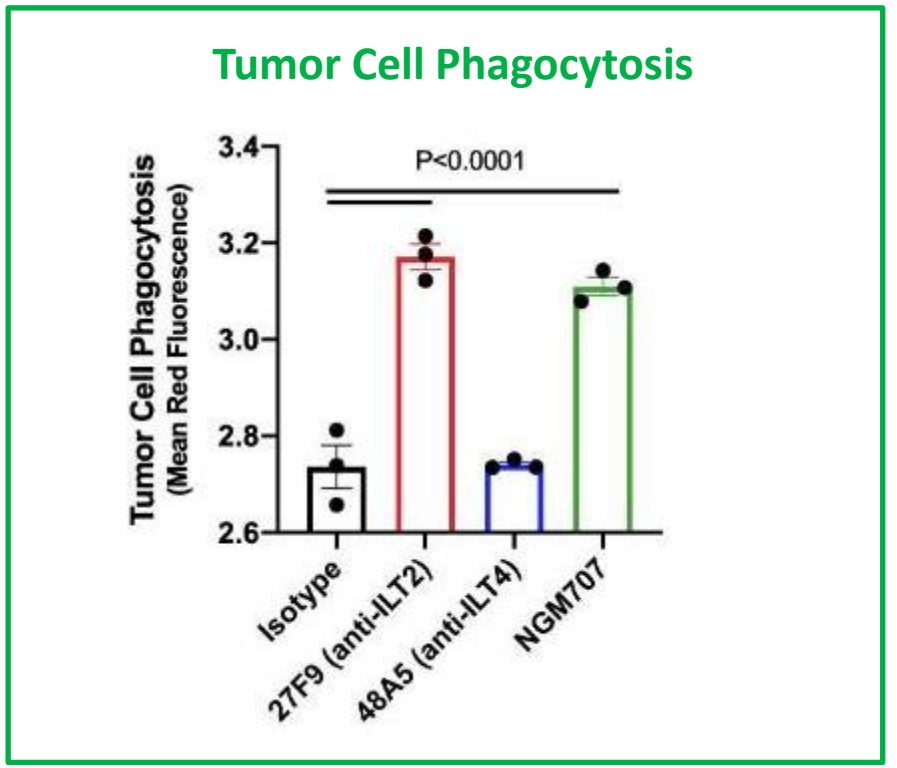
MDSC = Myeloid-derived Suppressor Cells; OVISE = Ovarian Clear Cell Carcinoma; MLR = Mixed Lymphocyte Reaction; TNF = Tumor Necrosis Factor; CPM = 3H-thymidine Counts Per Minute

ILT2 Blockade Enhances Macrophage Phagocytosis of Tumor Cells

- **Increased macrophage phagocytosis of cancer cells with ILT2 blockade**
  - 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

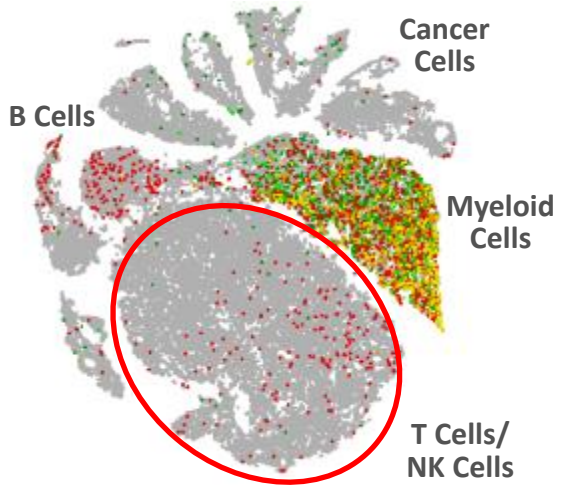
Macrophage Phagocytosis of Tumor Cells





ILT2 Blockade Enhances CD8+ T Cell Cytolytic Activity

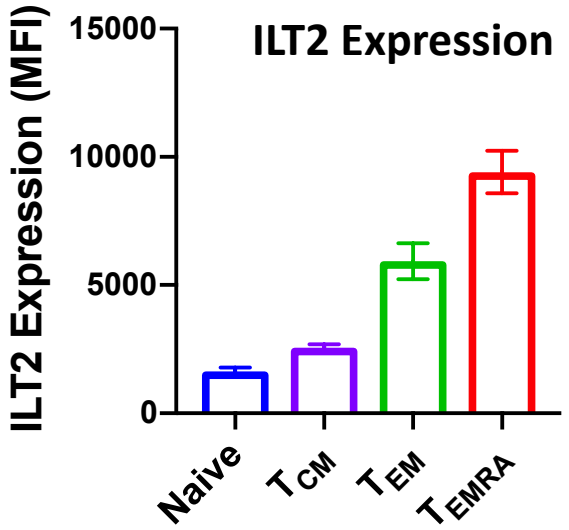
Intra-tumoral ILT2 Expression on CD8+ T cells and NK cells (NSCLC)



ILT2 Expression
ILT4 Expression
Dual Expression

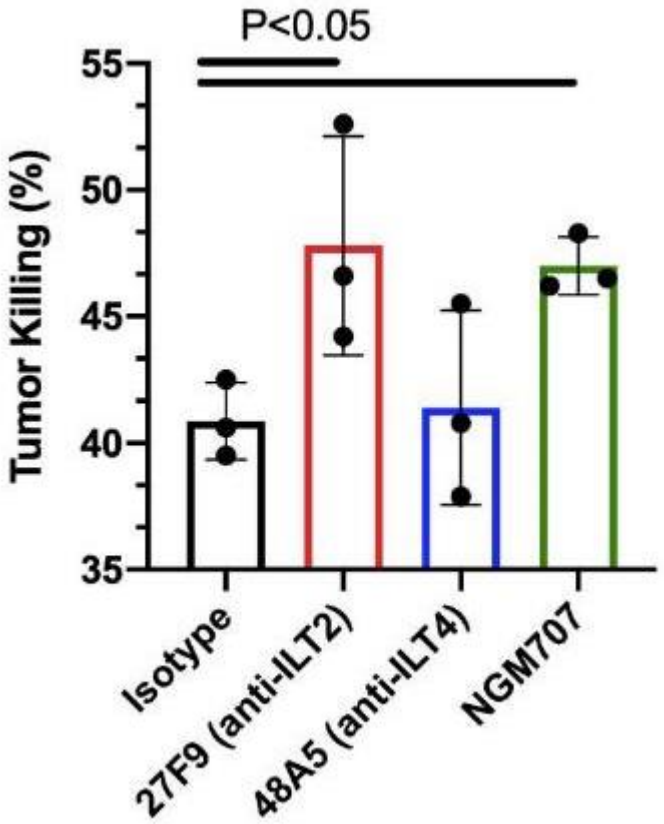
NGM Analysis of Lambrechts et al., Nat Med, 2018

ILT2+ expression on CD8+ TEMRA T-cells



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

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

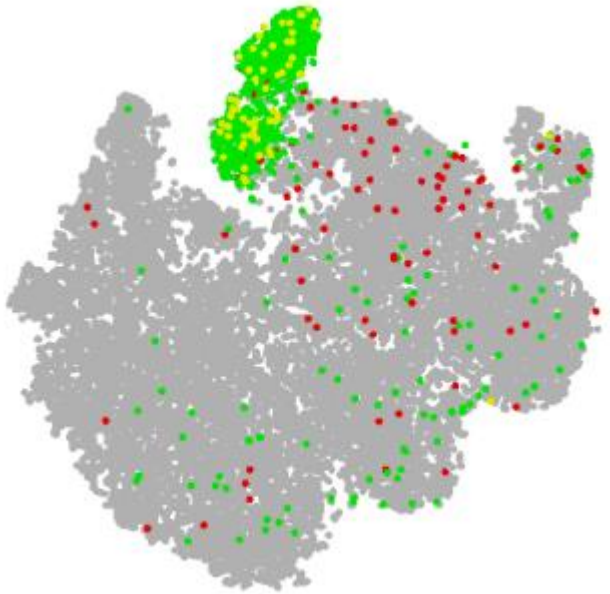


HLA-G = human leukocyte antigen G; TIM3 = T cell immunoglobulin and mucin domain- containing protein 3; LAG3 = Lymphocyte-activation protein 3

ILT2 Blockade Enhances Primary NK Cell Killing Activity

ILT2 expression on T and NK cells in NSCLC

Gated on T and NK cells



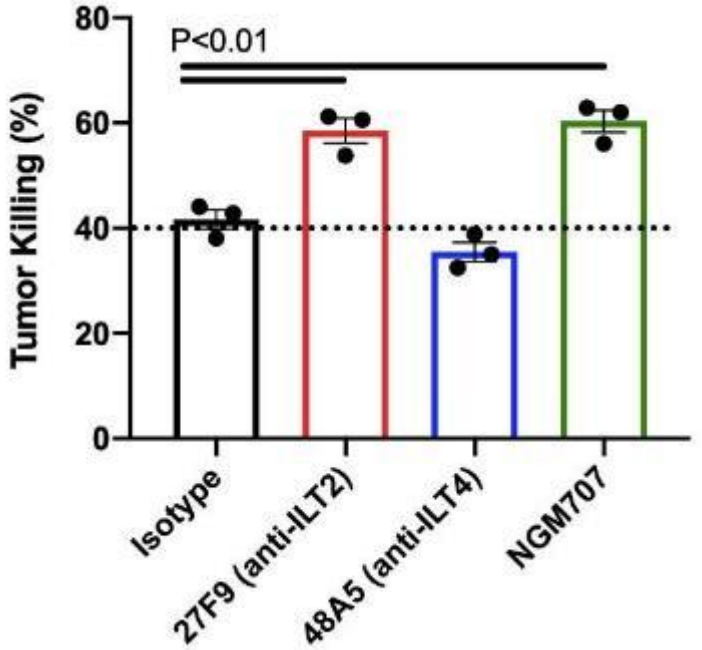
ILT2+ T cells

FGFBP2 (NK cell marker)

ILT2+ NK cells

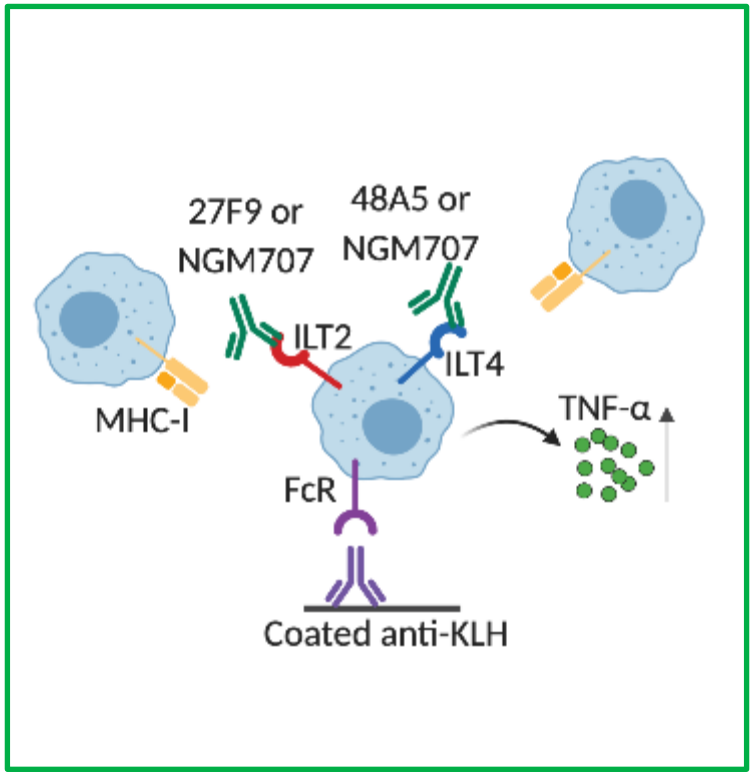
NGM Analysis of Lambrechts et al., Nat Med, 2018

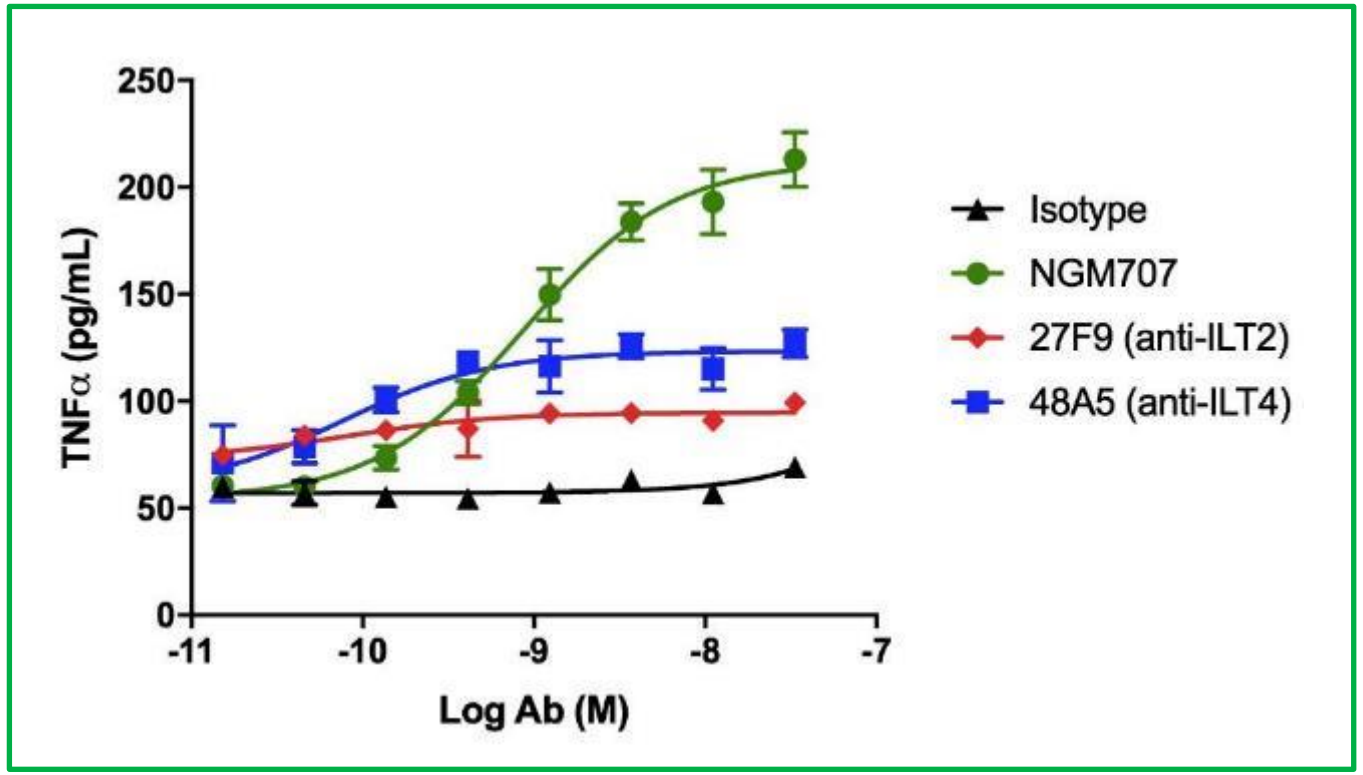
NGM707 enhances primary NK cell tumor killing activity



ILT2 and ILT4 Blockade Act Additively to Enhance Myeloid Cell Activation

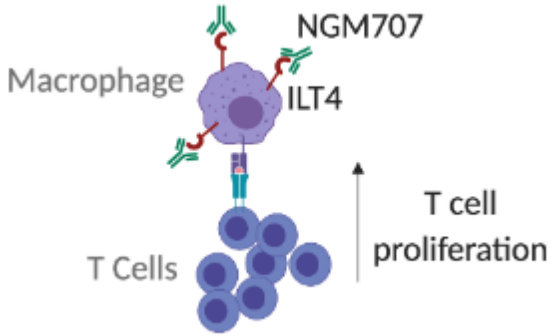
- **Fc receptors represent key stimulatory receptors on myeloid cells**
  - Inhibition of Fc receptor signaling by ILT2 and ILT4 promotes a suppressive myeloid cell phenotype
- **Dual blockade of ILT2 and ILT4 strongly potentiates Fc receptor signaling**
  - Blockade of ILT2 or ILT4 alone leads to a modest increase in Fc receptor signaling

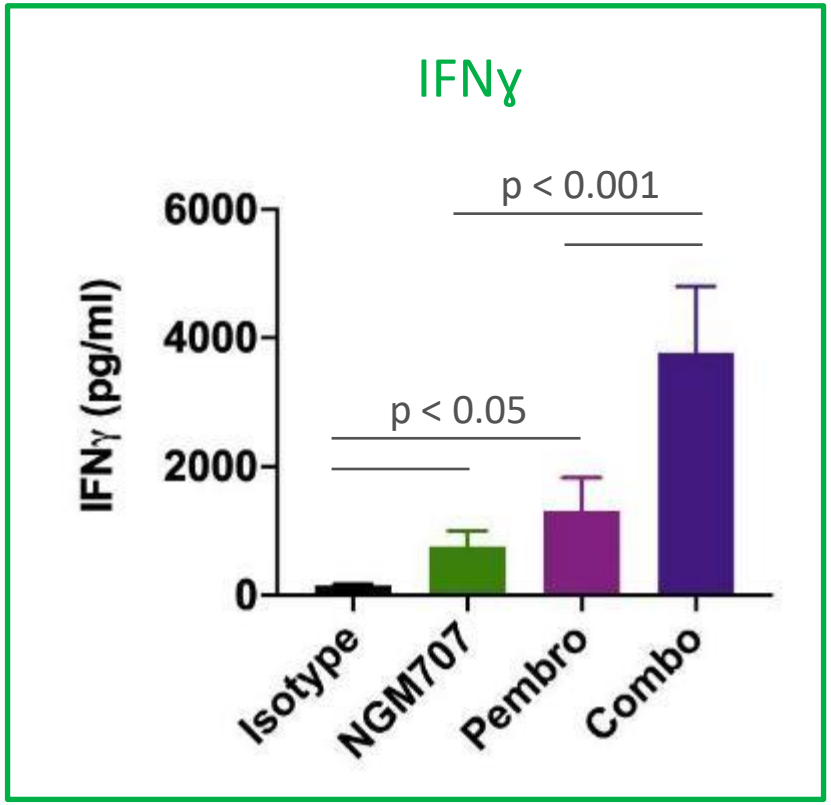


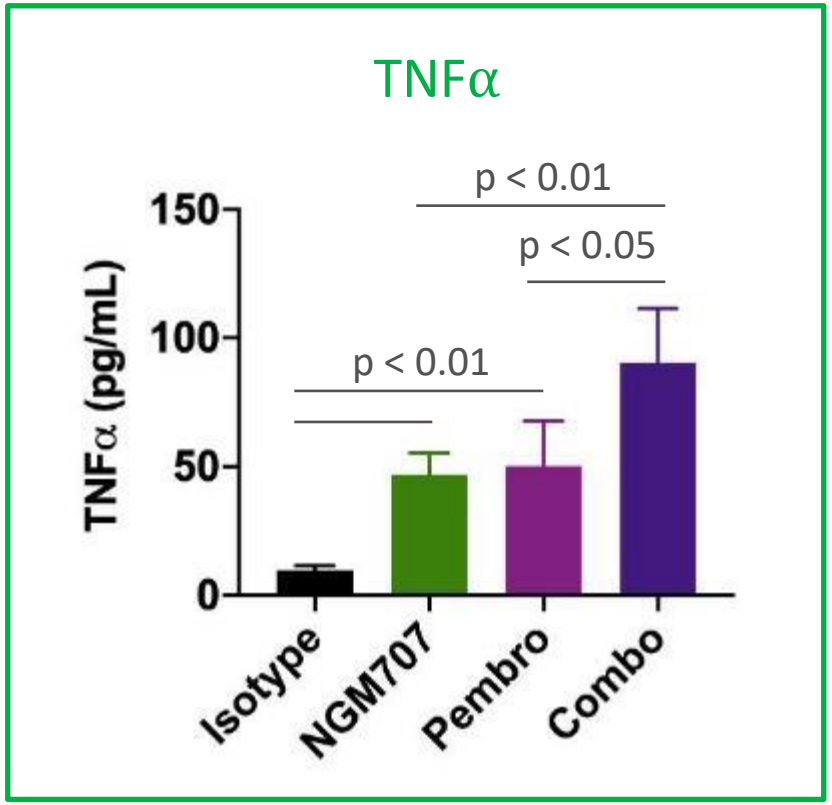


NGM707 and Pembrolizumab Acted Additively to Enhance T Cell Activation in a Mixed Lymphocyte Reaction

- NGM707 or pembrolizumab alone **modestly** enhanced T cell activation and increase in cytokine secretion (IFNg, 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







IFNγ = Interferon Gamma; TNFα = Tumor Necrosis Factor alpha, Pembro= pembrolizumab, Combo = pembrolizumab and NGM707

NGM831 in Advanced Solid Tumors

NGM831 is an Antagonist Antibody Inhibiting ILT3

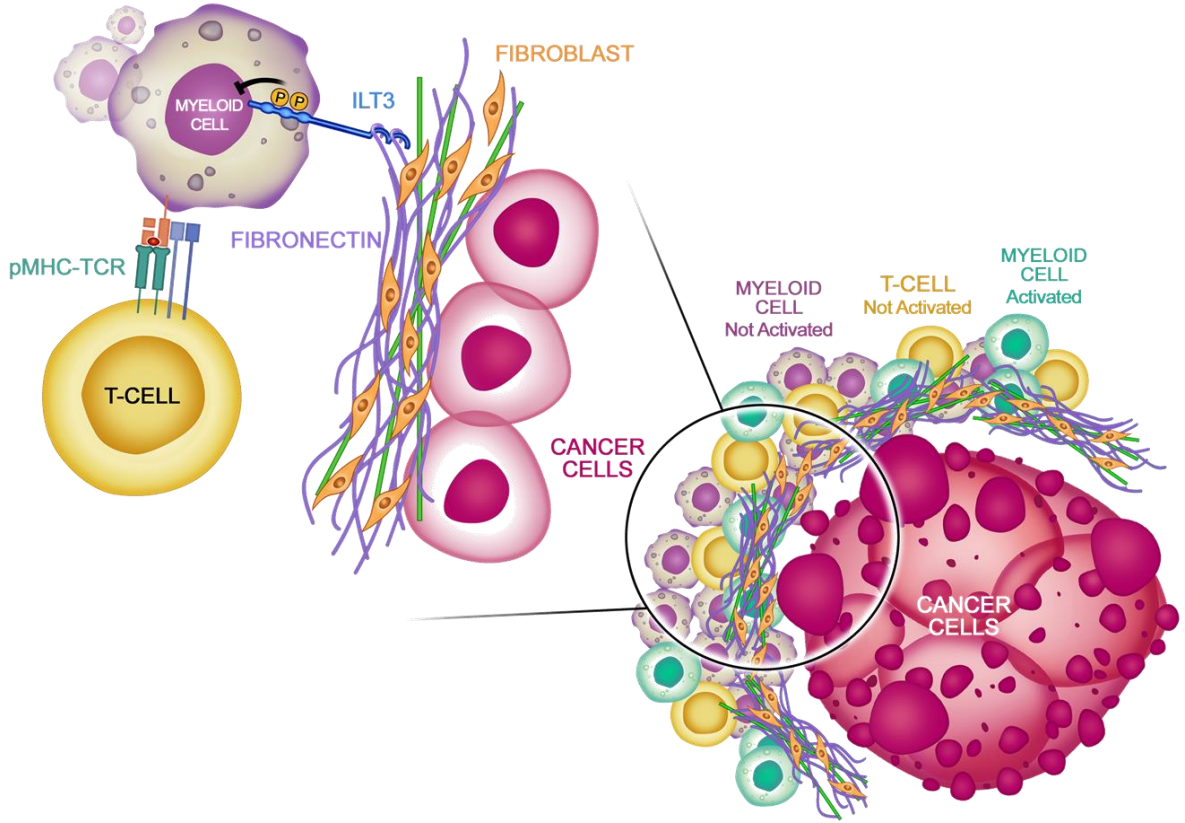
Potent antibody targeting the myeloid-enriched inhibitory receptor ILT3 (LILRB4)

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 infiltration and activation

Plan to initiate first-in-human study of NGM831 in 1H22



ILT3 = Immunoglobulin-like transcript 3; LILRB = Leukocyte immunoglobulin-like receptor subfamily B

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

- with 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]

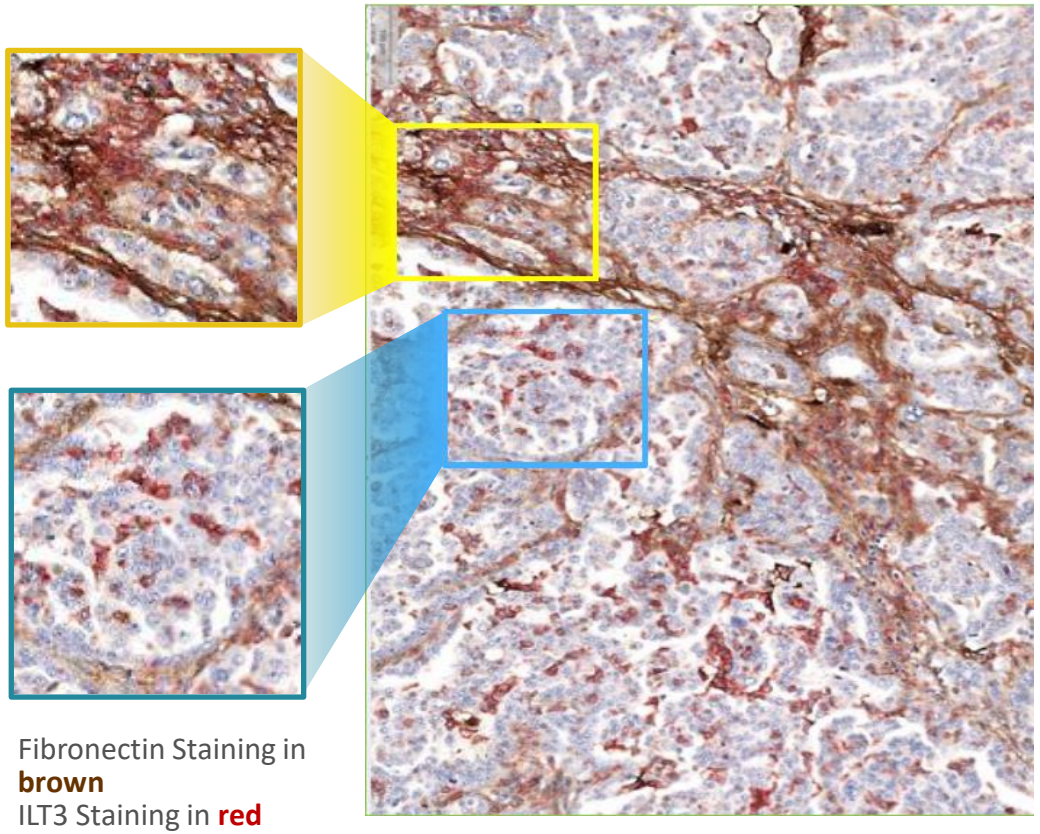
ILT3-fibronectin interactions may form a stromal checkpoint within the tumor microenvironment

- 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)



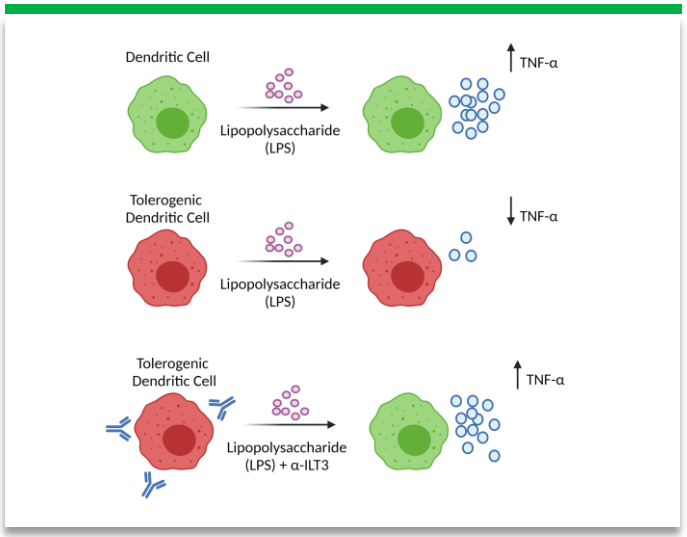
Fibronectin Staining in **brown**
ILT3 Staining in **red**

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

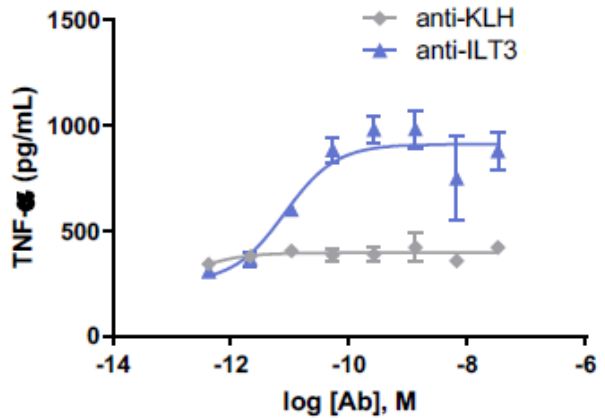
ILT3 Blockade Reprogrammed Tolerized Dendritic Cells into Stimulatory Cells

Dendritic cells that have been tolerized with a wide range of immunosuppression may be reprogrammed into stimulatory cells with ILT3 blockade

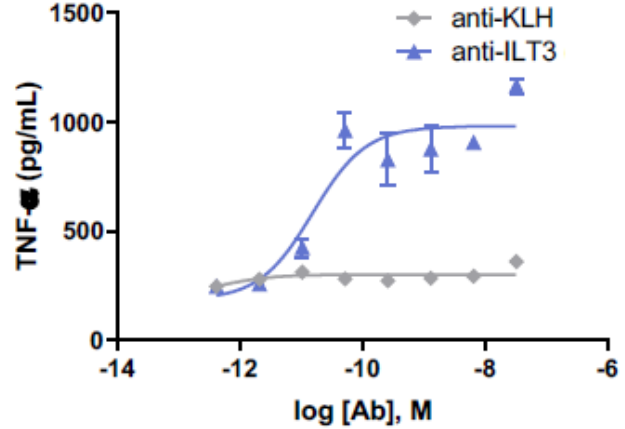
- Dendritic cells were tolerized by culturing them with several different immunosuppressants (cyclosporine, IL-6, VD3/Dex, and PGE_2)
- Tolerogenic dendritic cells were then stimulated with a bacterial product (LPS)
- Only activated dendritic cell will respond to LPS stimulation by secreting pro-inflammatory cytokines such as TNF-a



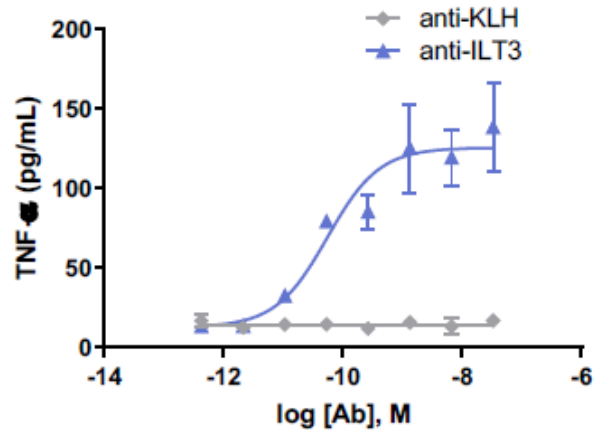
Cyclosporine + LPS



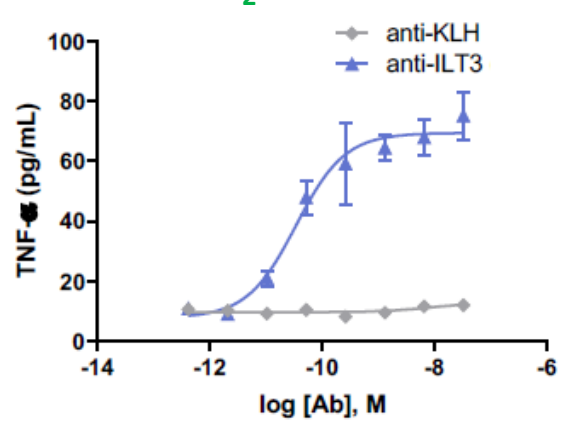
IL-6 + LPS



VD3/Dex + LPS



PGE_2 + LPS



LPS = lipopolysaccharide; VD3 = vitamin D3; Dex = dexamethasone; PGE_2 = prostaglandin E2; TNF-alpha = tumor necrosis factor-alpha

ILT3 Blockade Reverses Fibronectin-mediated Inhibition of Fc Receptor Activity

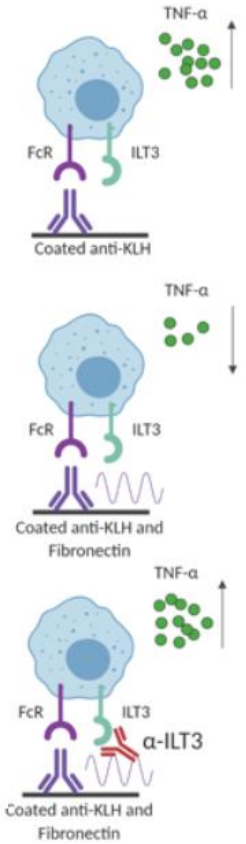
ILT3 blockade restores the ability of dendritic cells to respond to Fc Receptor activation in the presence of fibronectin

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

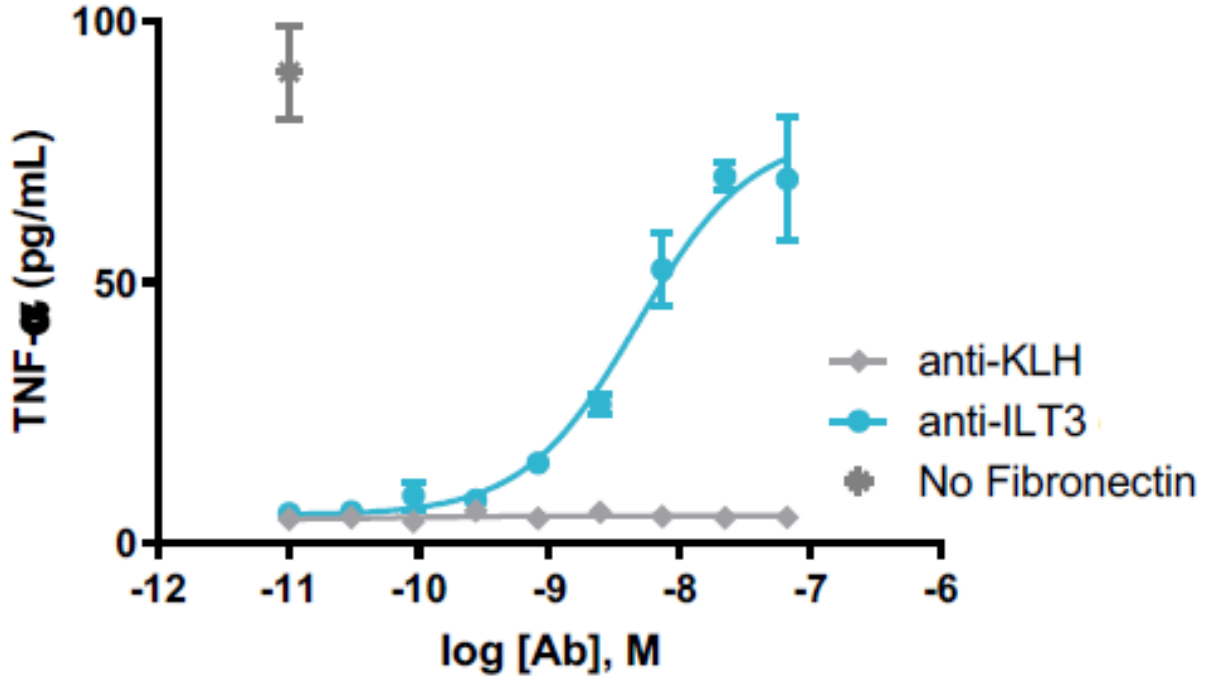
1. DCs stimulated with anti-KLH secrete TNF-α

2. TNF-α secretion is inhibited by platebound fibronectin

3. Inhibition of FcR signaling by fibronectin is blocked by ILT3

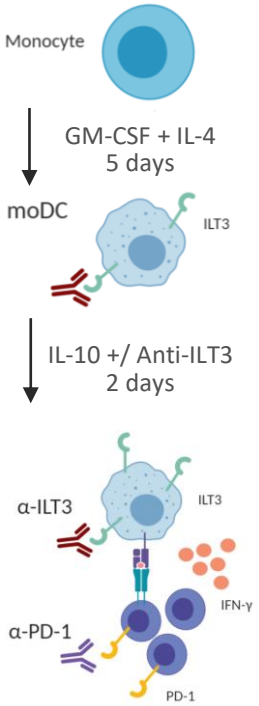


TNF-α Secretion by Monocyte-Derived Dendritic Cells

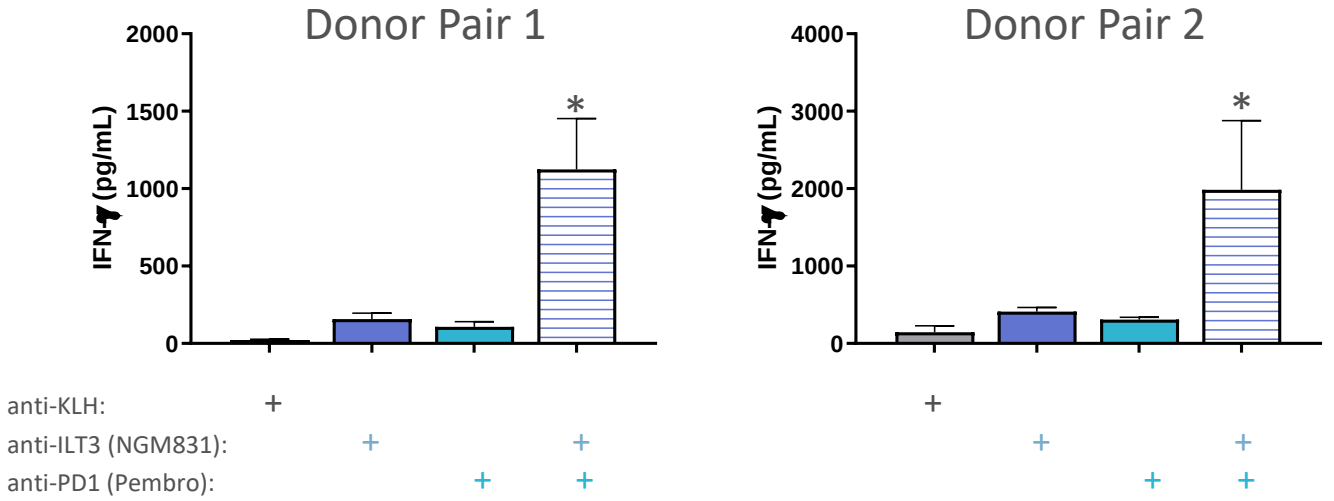


NGM831 and Pembrolizumab Act Synergistically to Enhance T Cell Activation in Preclinical Study

- 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

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

NGM438 in Advanced Solid Tumors

NGM438 is an Antagonist Antibody Inhibiting LAIR1

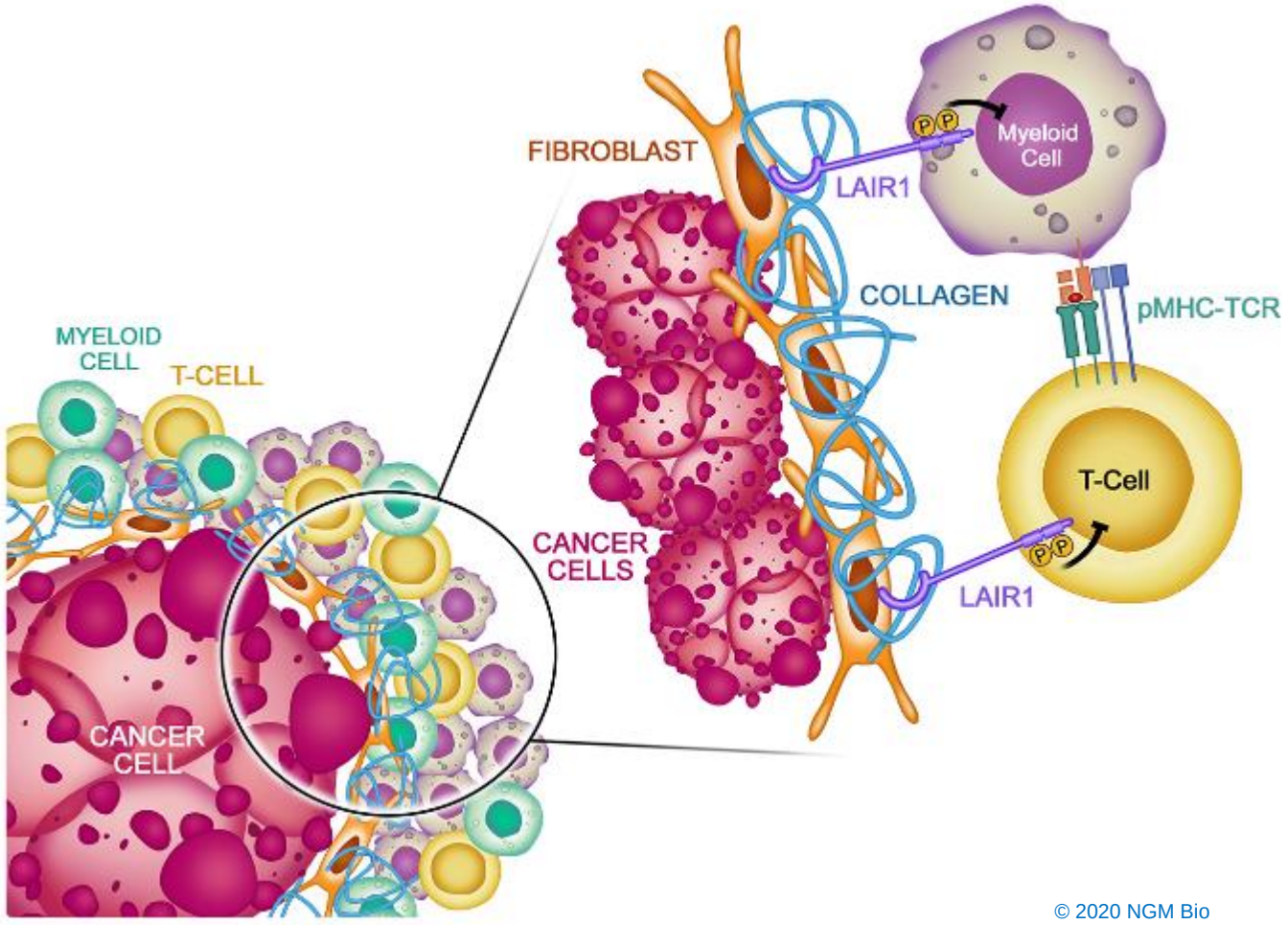
Potent, first-in-class antibody targeting the myeloid-enriched inhibitory receptor LAIR1

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:

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

Plan to initiate first-in-human study of NGM438 in 1H22



LAIR1 = Leukocyte-associated immunoglobulin-like receptor 1

LAIR1: Key Stromal Checkpoint and its Potential Roles in Cancer

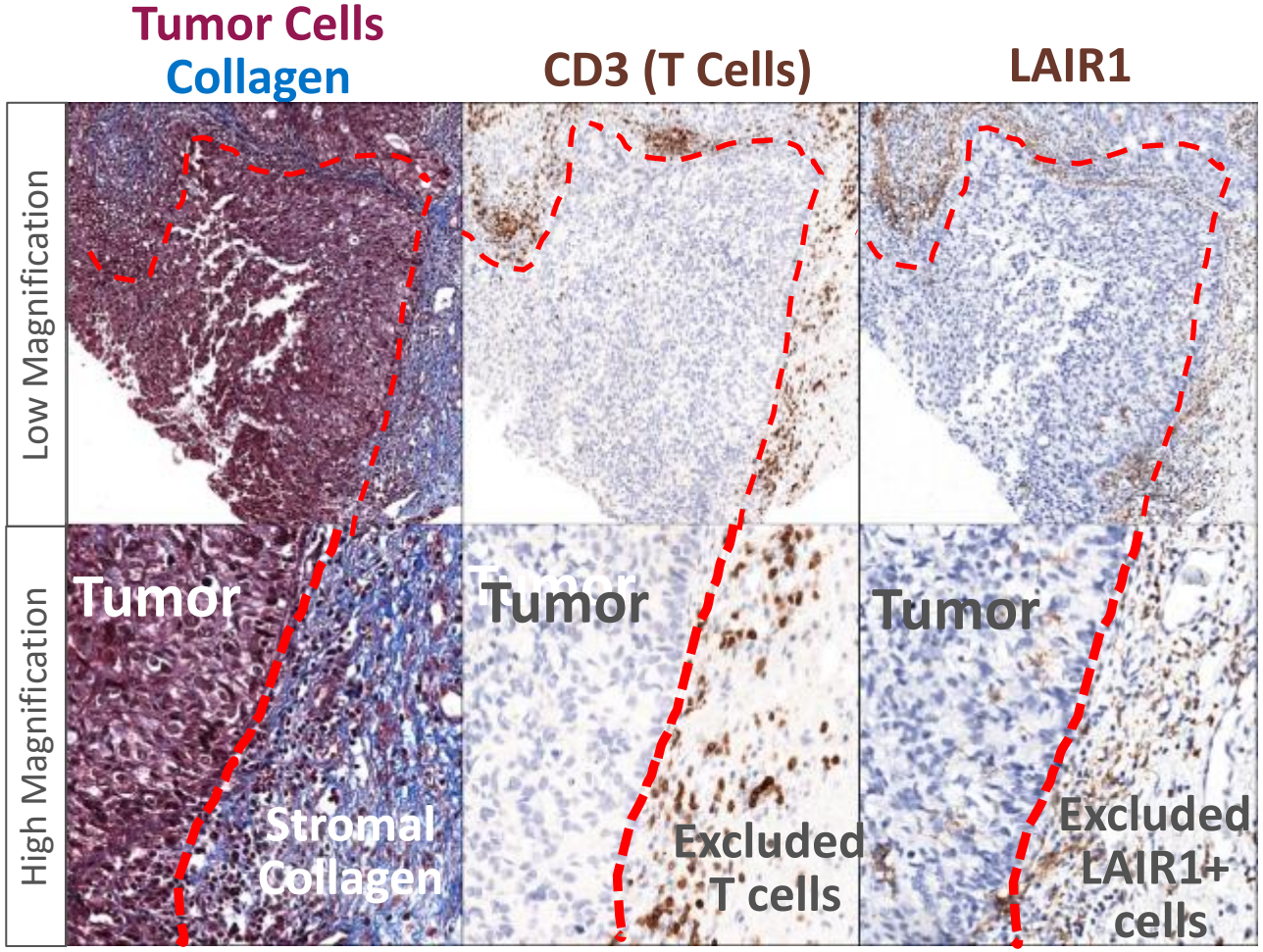
LAIR1 is a collagen-binding inhibitory signaling receptor expressed on immune cells: T cells, B cells, NK cells and myeloid cells[1-2]

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

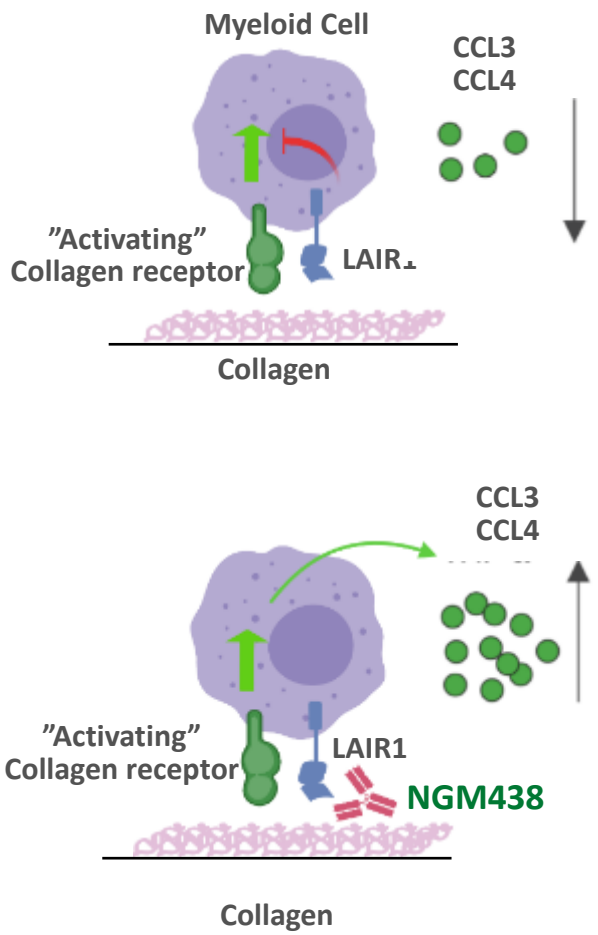
LAIR1-Expressing Immune Cells in Pancreatic Tumor



Pancreatic ductal adenocarcinoma tumor section

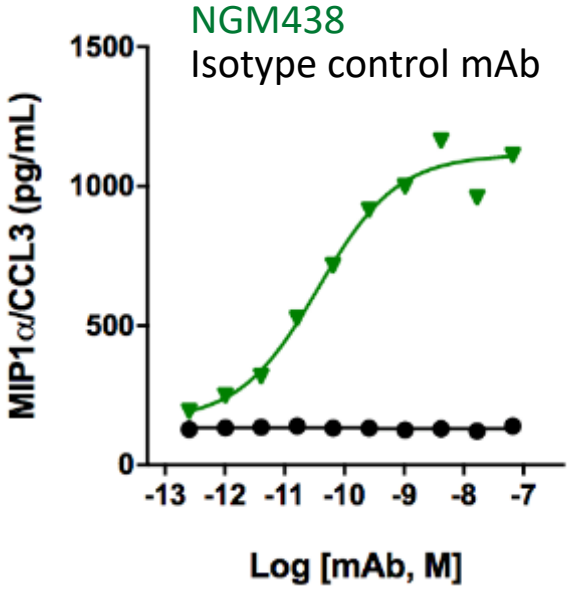
1. Meyaard, Immunity, 1997; 2. Guo, Trans Med, 2020, 3. Cao, 2015, Biochem Biophys Res Commun; 4. Wang, Exp Ther Med, 2016; 5. Wu, CP Cancer, 2018; 6. Yang, Head & Neck, 2018 7. Jingushi, Onc. Reports, 2018; 8. Peng, Nat Comm, 2020; 9. Lijun, Oncoimmunology, 2020

NGM438 Reversed Collagen-Mediated Suppression and Induced Reprogramming in Myeloid Antigen Presenting Cells

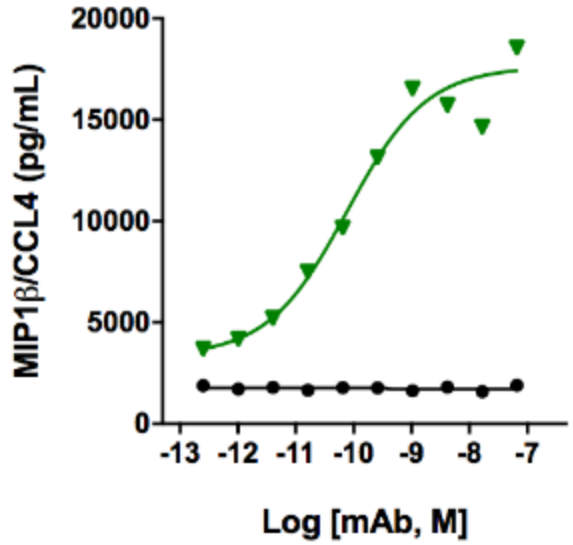


- **Collagen receptors, such as integrins, represent key stimulatory receptors on myeloid cells**
  - Inhibition of activating receptors via Collagen-LAIR1 signaling promotes a suppressive myeloid cell phenotype
- **NGM438 blockade of LAIR1-collagen binding reprograms myeloid cells to be pro-inflammatory**
  - Blockade of LAIR1-collagen leads to a potent increase in inflammatory cytokines, including CCL3 and CCL4 that are involved in recruiting lymphocytes to areas of inflammation

CCL3 production

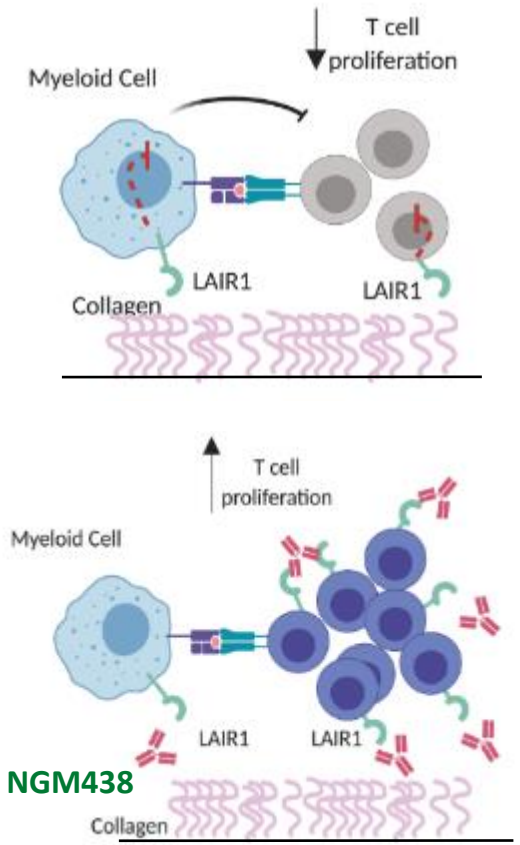


CCL4 production

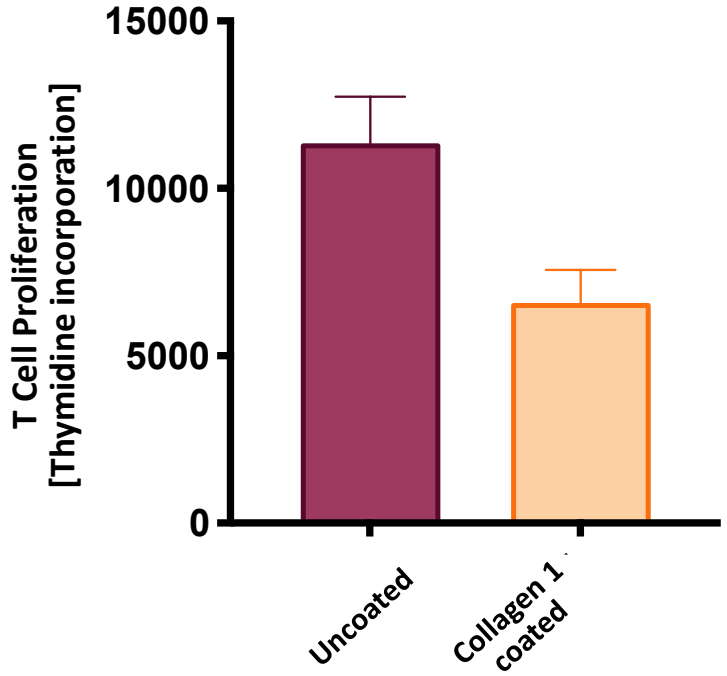


CCL = CC chemokine; CCL3 = macrophage inflammatory protein-1 α; CCL4 = macrophage inflammatory protein-1 β

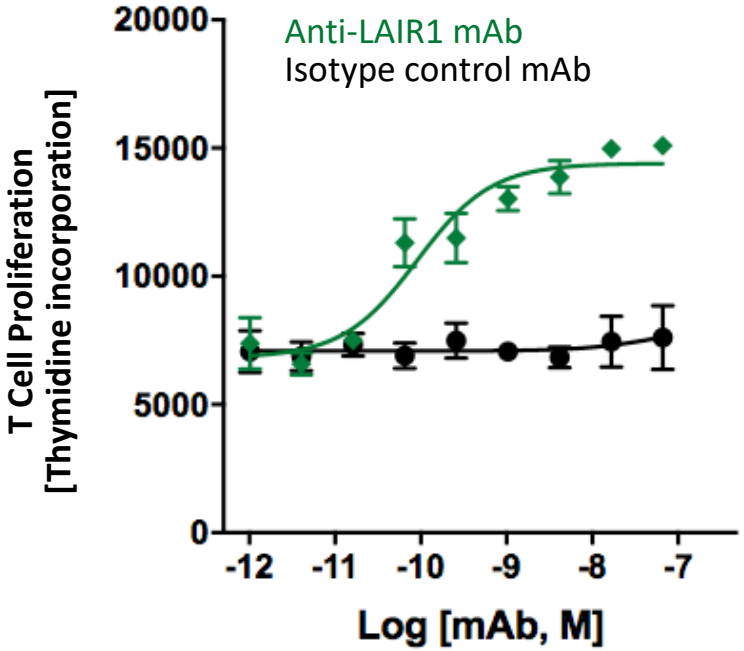
LAIR1 Blockade Reverses Suppression of Myeloid Cells by Collagen Leading to Enhanced T Cell Proliferation



T Cell Proliferation (MLR) is Suppressed by Collagen



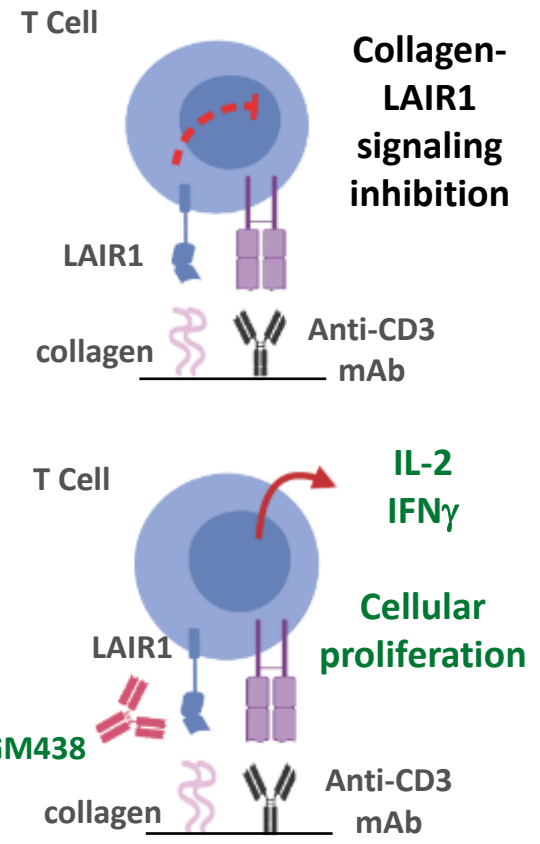
NGM438 Reverses T Cell Proliferation Suppression in a Dose-Dependent Manner



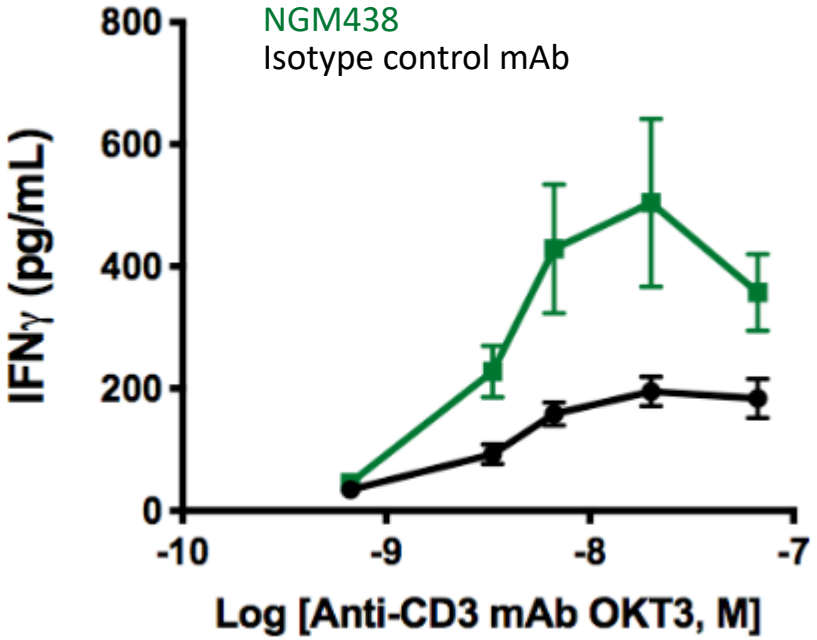
donor-mismatched monocyte-derived dendritic cells and T cells were incubated on plates coated with human collagen type 1 with soluble antagonists in the media
MLR = Mixed Lymphocyte Reaction; mAb = monoclonal antibodies

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

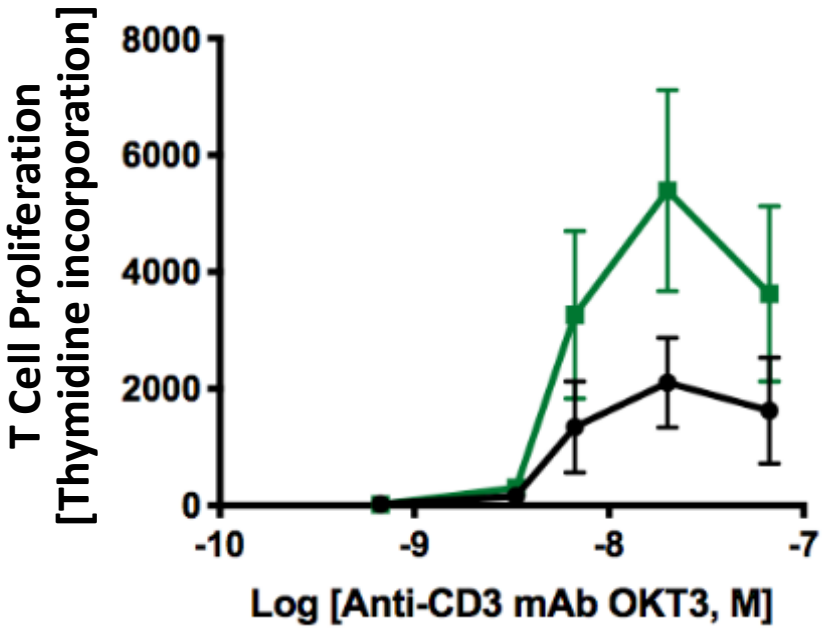
Collagen-Suppressed T cell Assay



NGM438 Promotes Th1 Cytokine Production



NGM438 Enhances T Cell Proliferation *in vitro*



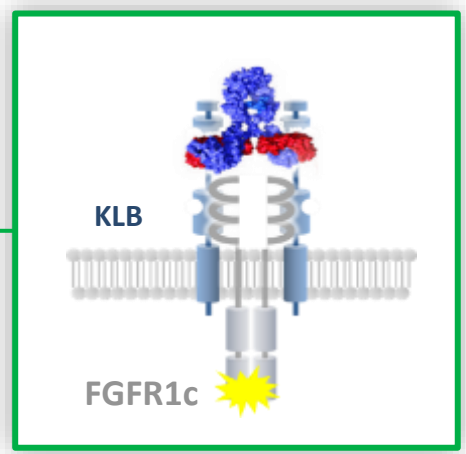
Human T cells incubated on plates coated with collagen and range of anti-CD3 stimulating mAb with single concentration of soluble antagonist mAbs in the media

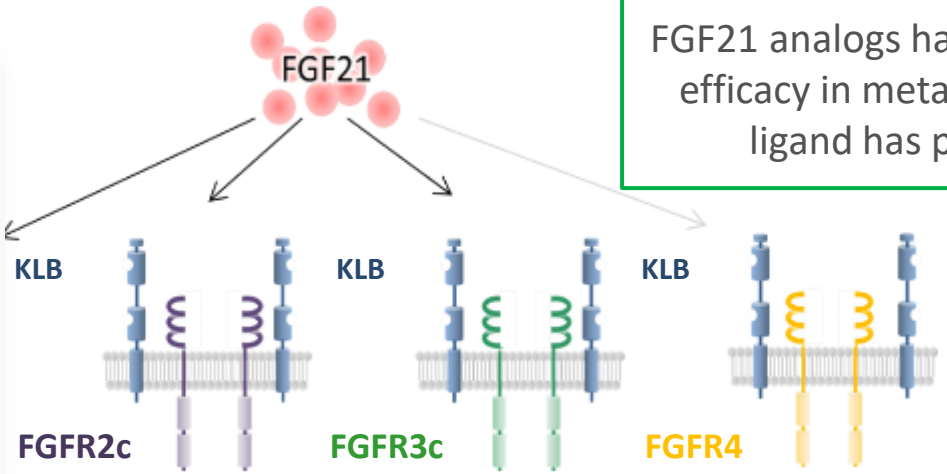
MK-3655 in NASH

A Monthly FGFR1c/KLB-specific Agonist Antibody Distinct from FGF21

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

MK-3655





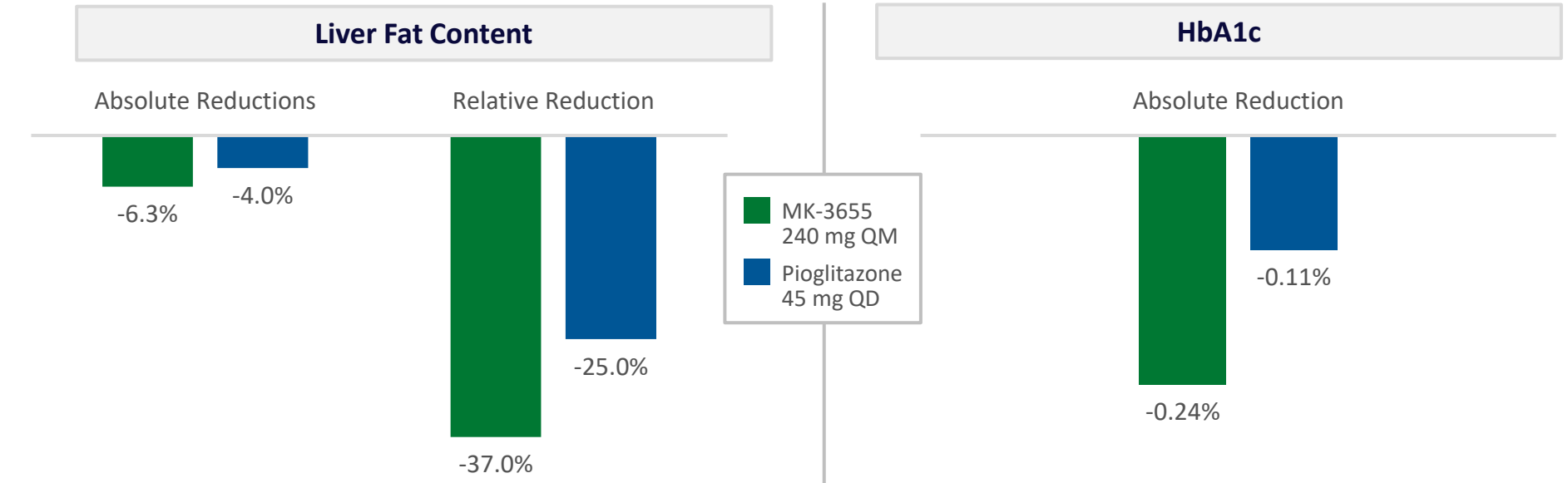
FGF21 analogs have demonstrated variable clinical efficacy in metabolic syndrome patients; native ligand has potential for safety liabilities

- 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 (NGM313) and initiated a Ph2b study during 4Q20**

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



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)

EASL 2019, Oral Abstract, DePaoli et.al.

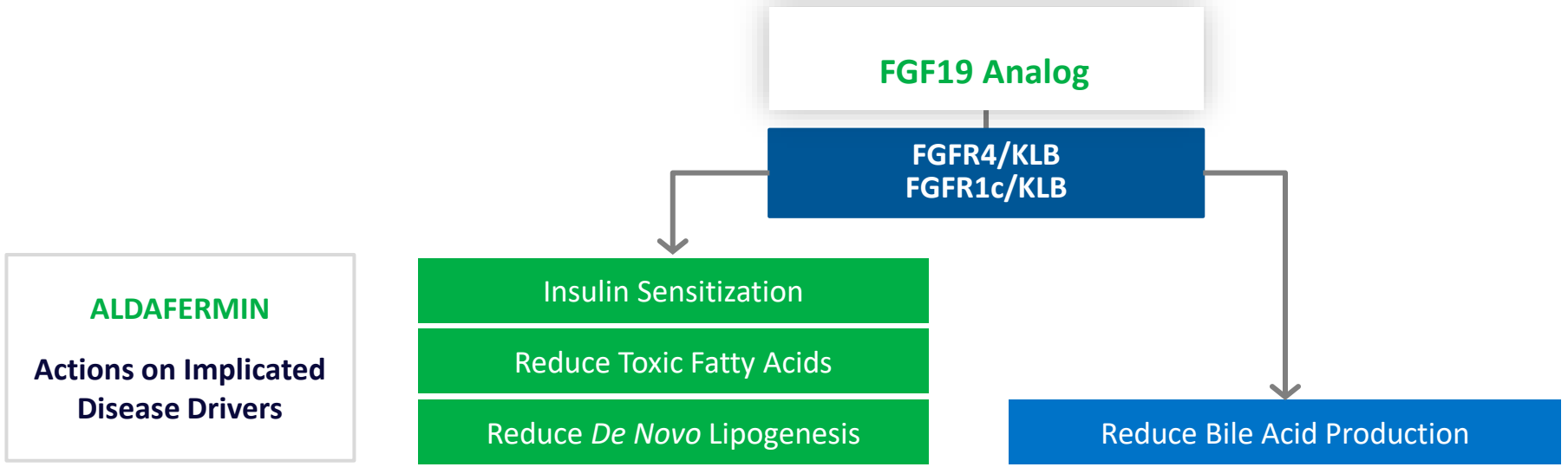
Phase 1b Safety: MK-3655 was Well Tolerated and AEs Generally Comparable to Placebo

MK-3655 Safety Results

- Favorable safety and tolerability profile consistent with other MK-3655 studies
- All AEs were mild in severity
- No 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

Aldafermin in NASH

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
- We plan to continue 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

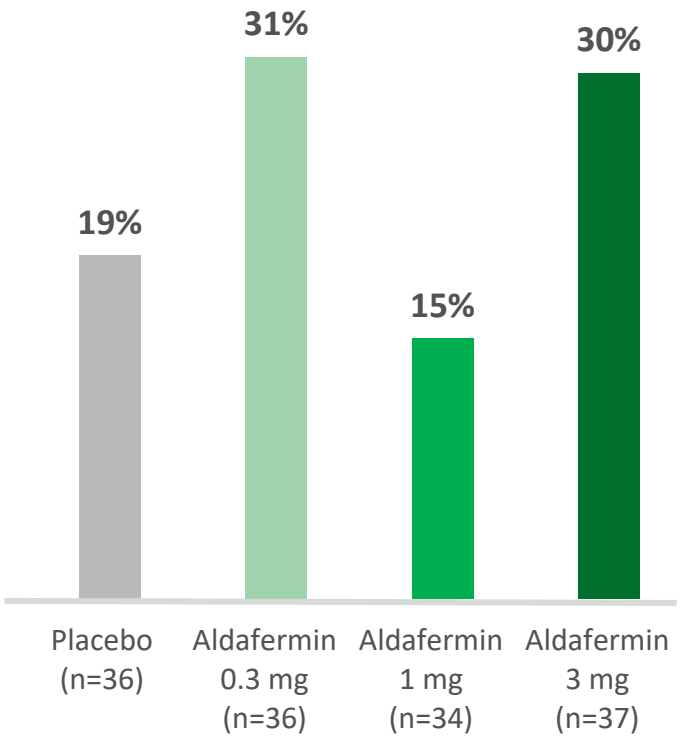
1 = Caussy et al, Aliment Pharmacology 2019

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

Fibrosis Reversal

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

(% of patients)

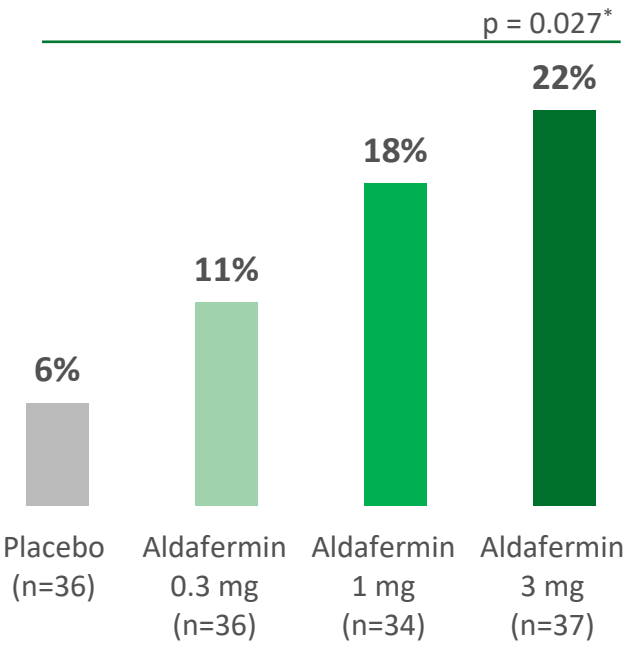


[1] Defined as patients who have an improvement in liver fibrosis by ≥1 stage with no worsening of NASH (no worsening of steatosis, lobular inflammation or hepatocyte ballooning grade) from baseline to W24

NASH Resolution

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

(% of Patients)

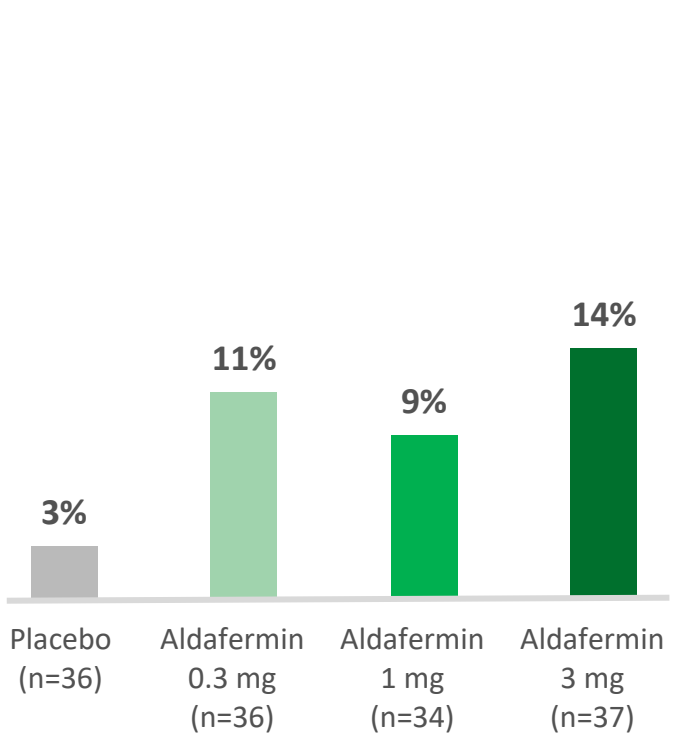


[2]Defined as patients having a NAS score of 0 or 1 for inflammation and 0 for ballooning, with no worsening of fibrosis (no progression of NASH CRN fibrosis stage) from baseline to W24

Fibrosis Improvement and NASH Resolution

Fibrosis Improvement and NASH Resolution[3] at W24

(% of Patients)



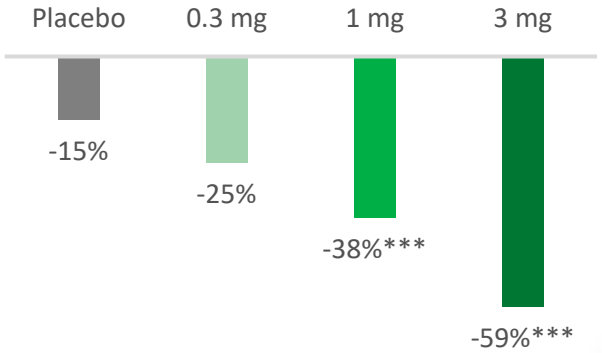
[3]Defined as patients who have an improvement in liver fibrosis by ≥1 stage AND have a NAS score of 0 or 1 for inflammation and 0 for ballooning and no worsening of steatosis at W24

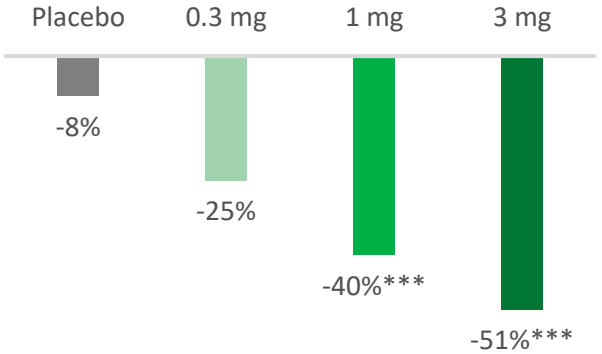
Population with paired biopsies. *Only the 3 mg dose met statistical significance in NASH resolution.

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

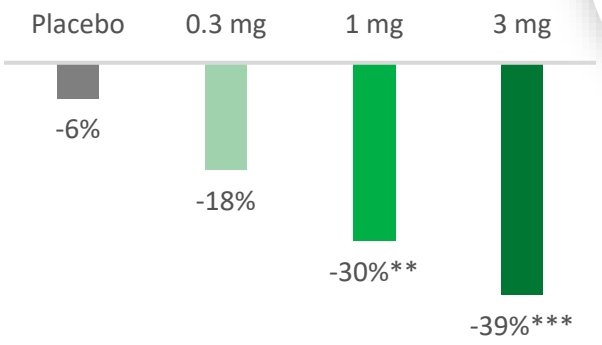
Change in Relative LFC by MRI-PDFF from Baseline



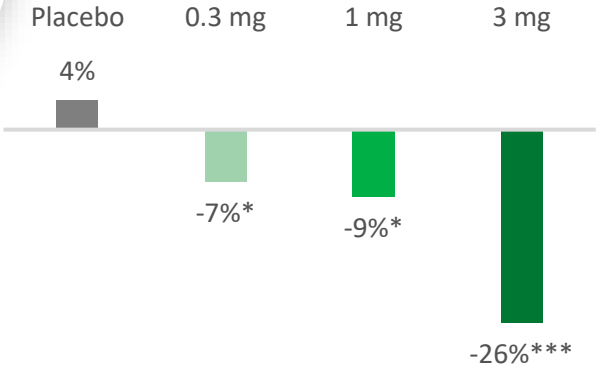


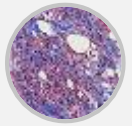
Change in Relative ALT from Baseline

Change in Relative AST from Baseline



STATISTICALLY SIGNIFICANT
Differences vs Placebo with 1 mg and 3 mg Aldafermin





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

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

Financial Overview & Key Milestones

2Q21 and FY20 Financial Results

STATEMENT OF OPERATIONS (In thousands)	THREE MONTHS ENDED June 30, 2021[1] (unaudited)	FULL YEAR ENDED December 31, 2020
RELATED PARTY REVENUE	$16,773	$87,368
RESEARCH AND DEVELOPMENT EXPENSES	$43,570	$163,972
GENERAL AND ADMINISTRATIVE EXPENSES	$9,823	$27,229
TOTAL OPERATING EXPENSES	$53,393	$191,201
NET LOSS	**($36,692)**	**($102,487)**

BALANCE SHEET	June 30, 2021 (unaudited)	December 31, 2020
CASH, CASH EQUIVALENTS AND SHORT-TERM MARKETABLE SECURITIES	**$390.6M**	**$295.2M**

[1] *See the Company's Quarterly Report on Form 10-Q for the quarter and year ended June 30, 2021 for more complete financial information*

Multiple Potential Near-Term Catalysts

Program	Mechanism	Status & Upcoming Events
NGM621 for Geographic Atrophy	Anti-Complement C3 Antibody	• Ph2 CATALINA Topline Data Expected in 2H22
NGM120 for Cancer & Cachexia	GFRAL Antagonistic Antibody	• Ph2 Trial Enrolling • Ph1a/Ph1b Dose-Finding Trial Interim Data Expected at ESMO 2021
NGM707 for Advanced Solid Tumors	ILT2/ILT4 Dual Antagonist Antibody	• Ph1/2 Trial Enrolling
NGM831 for Advanced Solid Tumors	ILT3 Antagonist Antibody	• Ph1 Trial Initiation Expected in 1H22
NGM438 for Advanced Solid Tumors	LAIR1 Antagonist Antibody	• Ph1 Trial Initiation Expected in 1H22
Aldafermin for NASH	FGF19 Analog	• Ph2b ALPINE 4 (NASH F4) Trial Enrolling
MK-3655 for NASH	FGFR1c/KLB Agonistic Antibody	• Merck Global Ph2b Trial Enrolling

Thank You

Novel Biology. Powerful Medicines. Transformative Impact.