Biology-driven discovery.
Life-changing medicines.

Corporate Overview

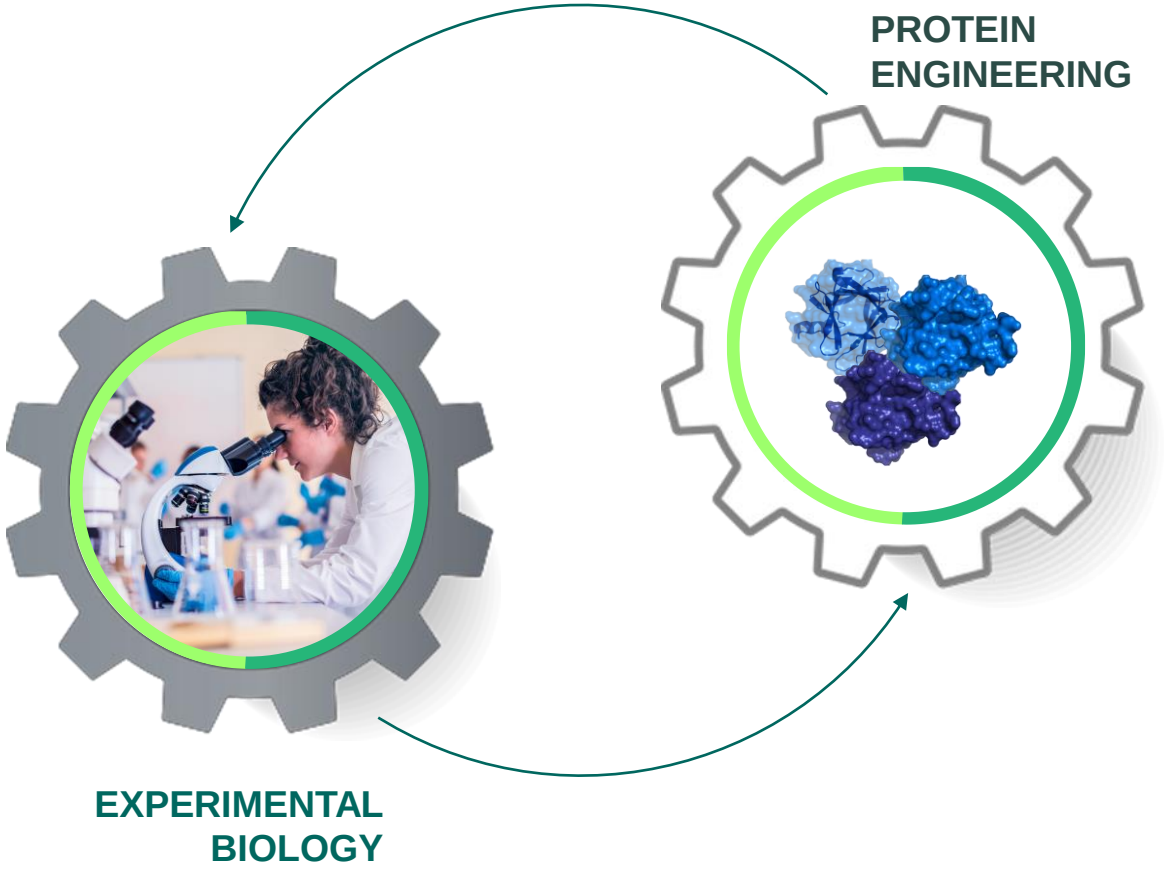
December 2022

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 Bio's pipeline, including NGM707, NGM831, NGM438, NGM120, NGM621, MK-3655 and aldafermin; the planned timing of initiation, enrollment, data readouts and results of NGM Bio's clinical trials, including topline data readout from the Phase 2b ALPINE 4 study of aldafermin and a clinical data readout from the Phase 1a study of NGM707; results of NGM Bio's preclinical studies; the design of NGM Bio's and Merck's clinical trials of NGM Bio's product candidates; the potential receipt of milestone and royalty payments by NGM Bio under the amended collaboration with Merck, including Merck's decision whether or not to option NGM621 either together or bundled with other ophthalmology compounds; the sufficiency of NGM Bio's cash resources to fund planned activities through data readouts in 2023 across oncology portfolio; NGM Bio's belief that modulation of myeloid checkpoint inhibitors has the potential to be a next wave in immune-oncology treatment; the potential roles of regulating the GDF15/GFRAL pathway and ILT2, ILT4, ILT3 and LAIR1 in cancer, 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; development plan and design of the NGM707 Phase 1/2 study; the potential of NGM438 to inhibit LAIR1; the potential role of the GDF15/GFRAL pathway in promoting tumor-associated immune regulation, metabolic regulation and appetite suppression; and other statements that are not historical fact. Because such statements deal with future events and are based on NGM Bio's current expectations, they are subject to various risks and uncertainties, and actual results, performance or achievements of NGM Bio could differ materially from those described in or implied by the statements in this presentation. These forward-looking statements are subject to risks and uncertainties, including, without limitation, risks and uncertainties associated with: the costly and time-consuming pharmaceutical product development process and the uncertainty of clinical success, including whether NGM621 may be able to demonstrate future clinical benefit in patients with geographic atrophy (GA), particularly in light of the failure to achieve the primary endpoint in CATALINA, and that NGM Bio's other product candidates may not be tolerable and effective treatments in their planned indications; failure or delays in successfully initiating, enrolling, reporting data from or completing clinical studies, as well as risks that results obtained in preclinical or clinical trials to date (including preliminary results of the Phase 1 Part 1 trial of NGM707) may not be indicative of results obtained in future trials and that post-hoc analyses performed after unmasking trial results can result in the introduction of bias, have other limitations and may not be predictive of results obtained in future trials; NGM Bio's reliance on its amended collaboration with Merck, including the risks that if Merck fails to exercise its option to license NGM621, NGM Bio would need to partner the NGM621 program in order to further clinical development of NGM621, if any; 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; NGM Bio's reliance on third-party manufacturers for its product candidates and the risks inherent in manufacturing and testing pharmaceutical products; the sufficiency of NGM Bio's cash resources, including to fund its wholly-owned programs, and NGM Bio's need for additional capital; and other risks and uncertainties affecting NGM Bio and its development programs, including those discussed in the section titled "Risk Factors" in NGM Bio's quarterly report on Form 10-Q for the quarter ended September 30, 2022 filed with the Securities and Exchange Commission (SEC) on November 3, 2022 and future filings and reports that NGM Bio makes from time to time 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

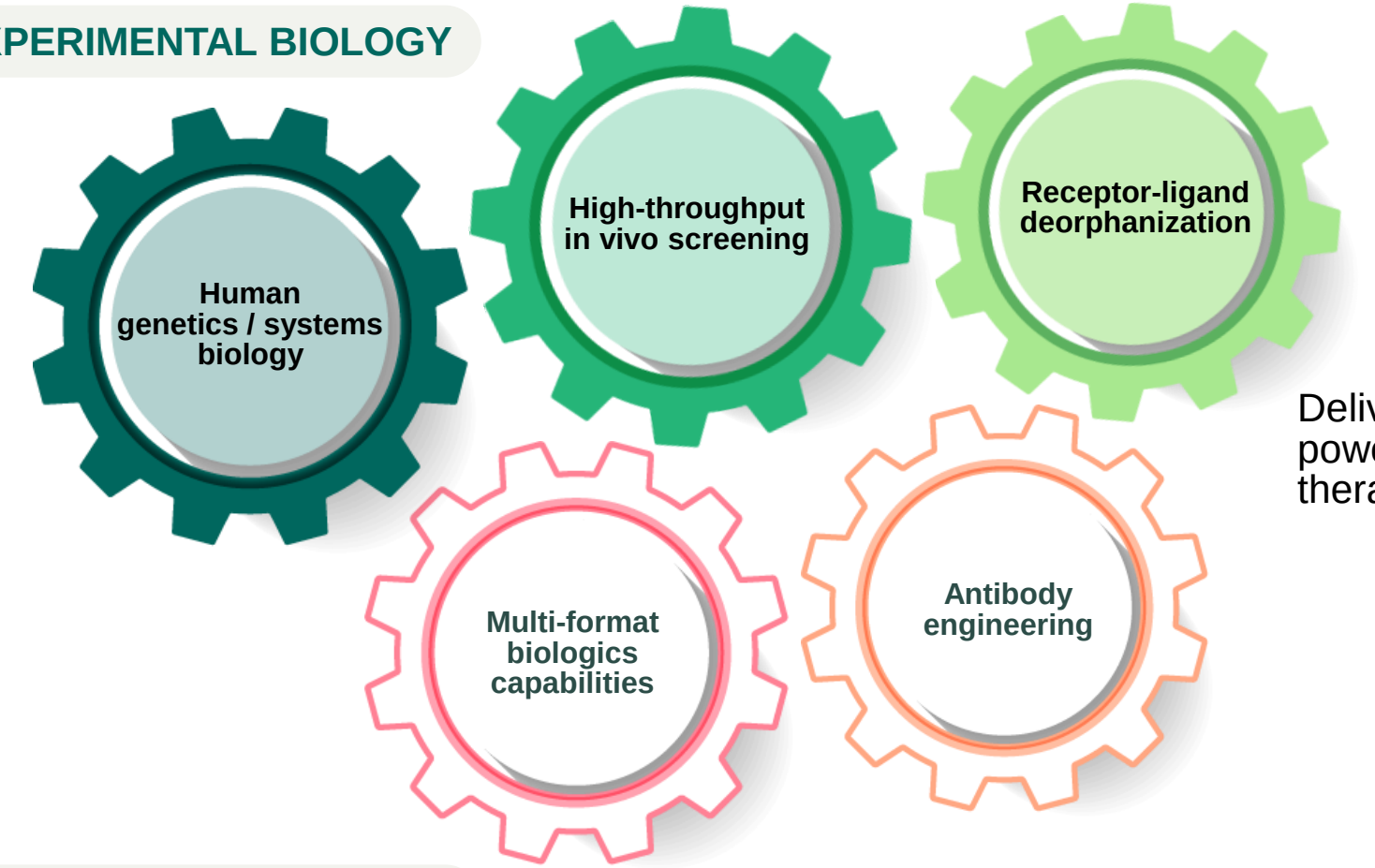
EXPANSIVE PIPELINE

- **3** Therapeutic Areas
- **7** Disclosed Programs
- **7** Programs in Clinical Development
- **4** Ph2/Ph2b Studies Ongoing or Completed[1]

[1]Including the recently completed NGM621 CATALINA trial

Reproducible Drug Discovery Process Has Been Applied Successfully Across Biological Frontiers

EXPERIMENTAL BIOLOGY



PROTEIN ENGINEERING

Delivering powerful therapies

7 Disclosed Programs

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

Our Expansive Pipeline

			Preclinical / Phase 1 / Phase 2 / Phase 3	Status	Rights
ONCOLOGY					
NGM707	ILT2/ILT4 Dual Antagonist Antibody	Advanced Solid Tumors	PHASE 1/2	Enrolling	Global ngmBIO
NGM831	ILT3 Antagonist Antibody	Advanced Solid Tumors	PHASE 1	Enrolling	Global ngmBIO
NGM438	LAIR1 Antagonist Antibody	Advanced Solid Tumors	PHASE 1	Enrolling	Global ngmBIO
NGM120	GFRAL Antagonist Antibody	Cancer & Cancer-related Cachexia	PHASE 1	Ongoing	Global ngmBIO
		Metastatic Pancreatic Cancer & Cancer-related Cachexia	PHASE 2	Ongoing	Global ngmBIO
RETINAL					
NGM621	Anti-Complement C3 Antibody	Geographic Atrophy	PHASE 2	CATALINA Trial Completed	Merck option exercise point triggered; if optioned by Merck, NGM to receive milestones + double-digit royalties or up to 50% profit/cost share[1]
LIVER & METABOLIC					
MK-3655 (NGM313)	FGFR1c/KLB Agonist Antibody	NASH F2/F3	PHASE 2	Enrolling	Merck optioned; NGM to receive milestones + double-digit royalties or up to 50% profit/cost share[1]
Aldafermin	FGF19 Analog	NASH F4	PHASE 2	Topline ALPINE 4 Data Expected in 1H23	Global ngmBIO

[1]At NGM's option at potential Phase 3

NASH = non-alcoholic steatohepatitis; FGF = fibroblast growth factor; KLB = beta-klotho; 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

Multiple Program Milestones in 2022

Program	Mechanism	Status	Milestones	
NGM621 Geographic Atrophy	Anti-Complement C3 Antibody	CATALINA trial completed	**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 4Q22**	✓
NGM831 Advanced Solid Tumors	ILT3 Antagonist Antibody	Enrolling	**Initiation of Ph1 trial in 1Q22**	✓
NGM438 Advanced Solid Tumors	LAIR1 Antagonist Antibody	Enrolling	**Initiation of Ph1 trial in 2Q22**	✓
NGM120 Cancer and Cachexia	GFRAL Antagonist Antibody	Ph1a/1b trial ongoing Ph2 trial enrolling	**Additional Ph1a/1b clinical data readouts in 3Q22**	✓
Aldafermin Cirrhotic NASH	FGF19 Analog	Ph2b ALPINE 4 trial fully enrolled	**Last Patient In (LPI) in 1Q22**	✓
MK-3655 Non-cirrhotic NASH	FGFR1c/KLB Agonist Antibody	Merck-led global Ph2b trial enrolling	**Ongoing enrollment**	

3Q22 $300M cash balance[1] expected to be sufficient to fund operations into 4Q24

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[1]Cash, cash equivalents and short-term marketable securities as of September 30, 2022

NGM Bio's Myeloid Reprogramming Strategy to Treat Solid Tumors

NGM707, NGM831, NGM438

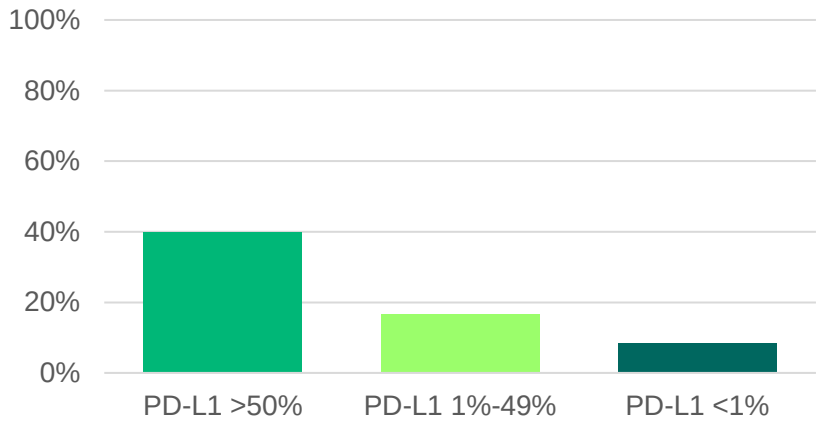
While T Cell Checkpoint Inhibitors Have Advanced the Treatment of Cancer, There is Opportunity to Improve Breadth / Depth of Response

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

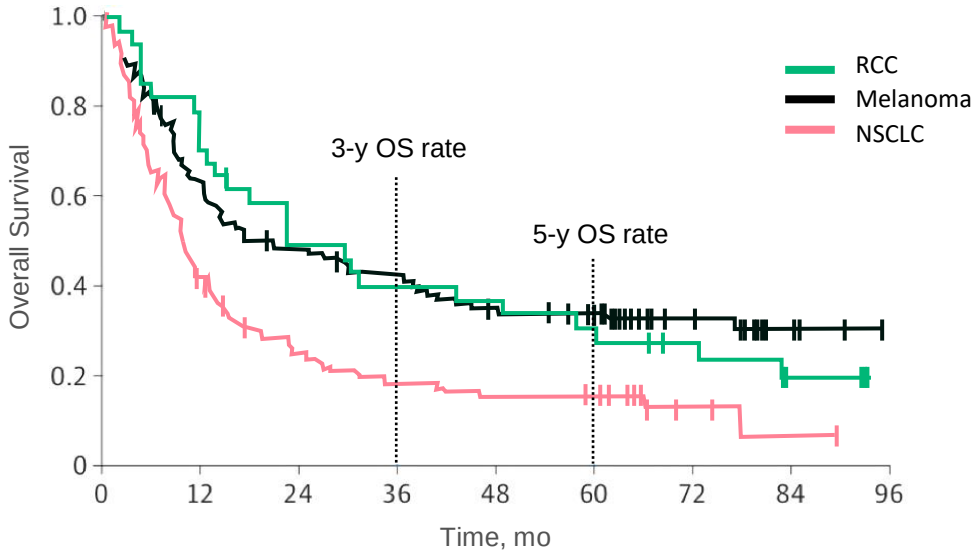
Depth of response: Amongst responders there is opportunity to increase duration of response

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

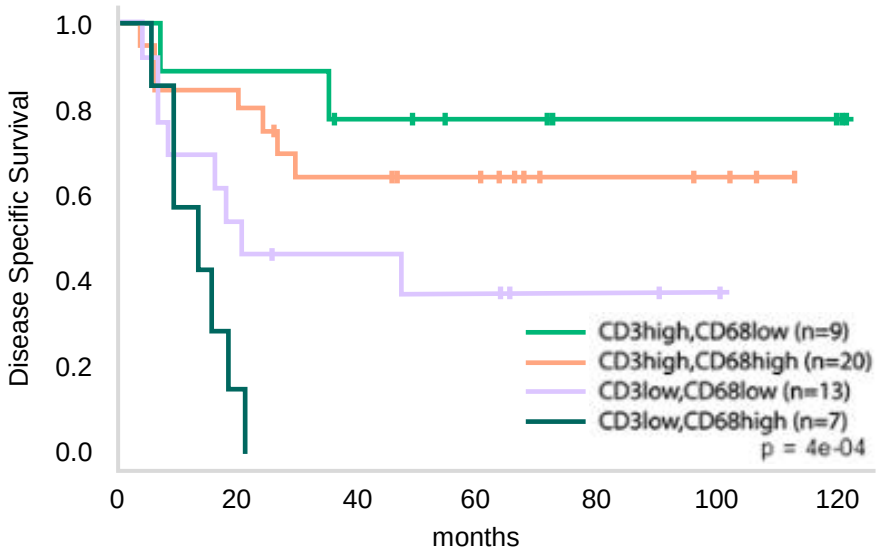


Man et al. *JNCI Spectrum*, 2021; Topalian, Sznol *et al.* - JAMA Oncol 2019
PD-1 = programmed cell death protein 1; PD-L1 = programmed death-ligand 1; ORR = overall response rate; NSCLC = non-small cell lung cancer; RCC = renal cell carcinoma

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

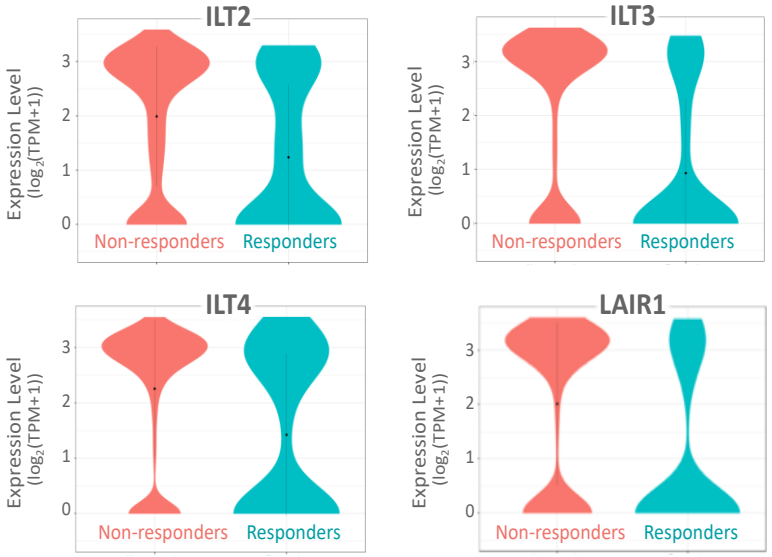
Myeloid-enriched tumors have poor prognosis

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

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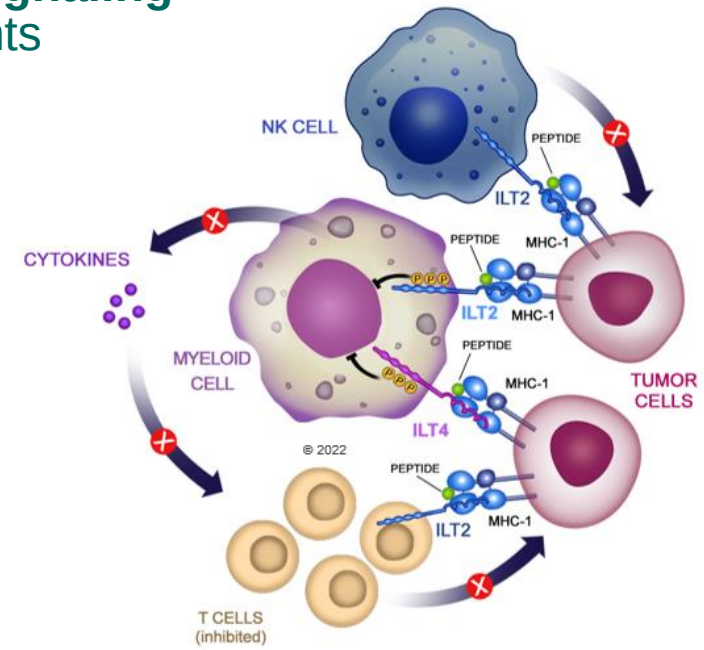
Sjodahl, 2014; Wang *et al.,* 2018, Sade-Feldman, *et al.*, 2019, GSE120575 – scRNAseq analysis
CD68 = Cluster of Differentiation 68; CD3 = Cluster or Differentiation 3; CPI = check-point inhibitors; R/R = relapsed/refractory

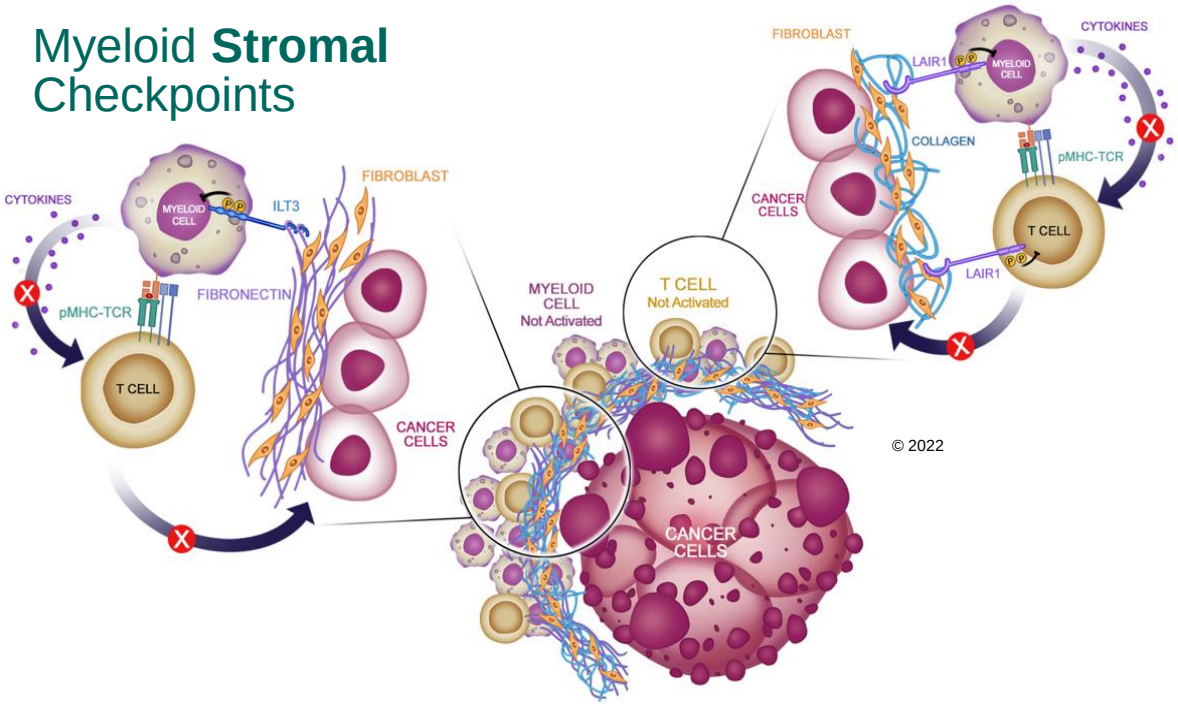
NGM Bio is Targeting Inhibitory Receptors on Myeloid Cells to Attempt to Restore Immune Response Against Tumors

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]

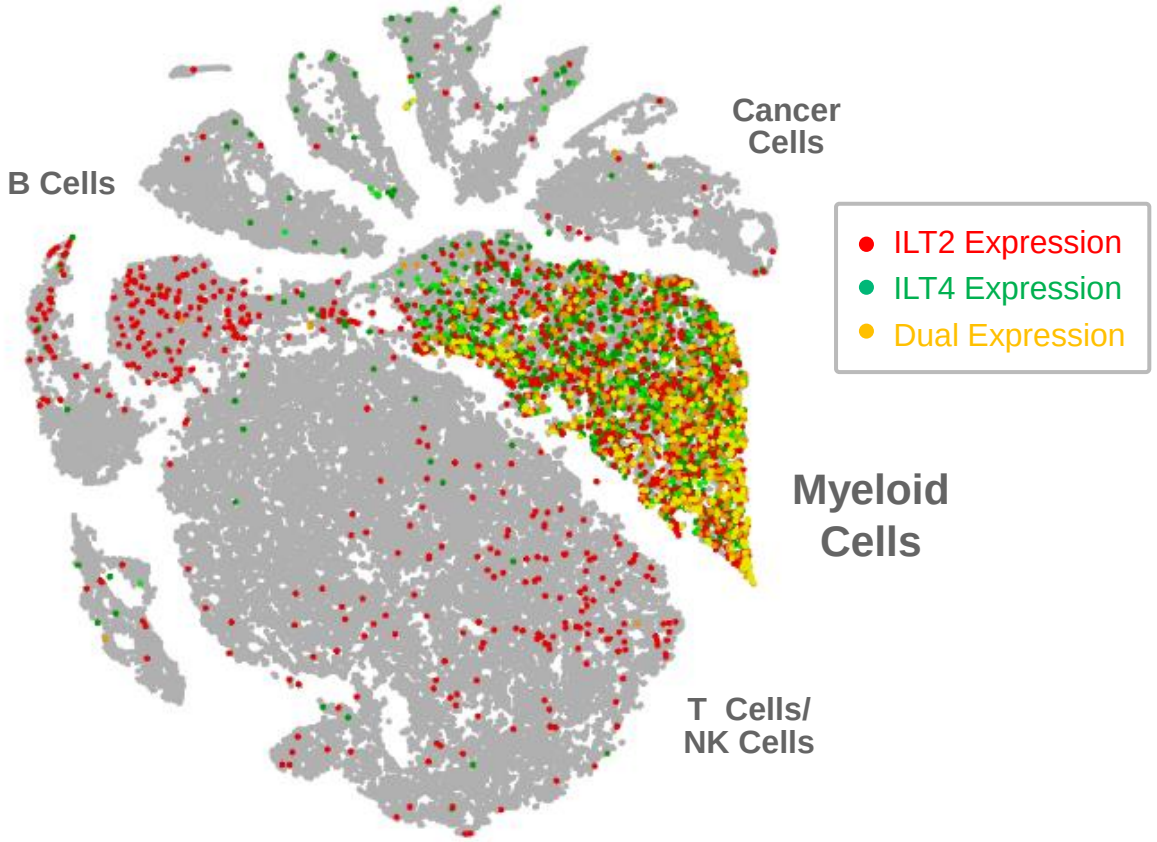
- ILT2 and ILT4 receptors are expressed on myeloid cells (APCs, MDSCs, macrophages, granulocytes) in the tumor microenvironment
- ILT2 additionally exhibits expression on natural killer (NK) cells, B cells and a subset of highly cytolytic T cells

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

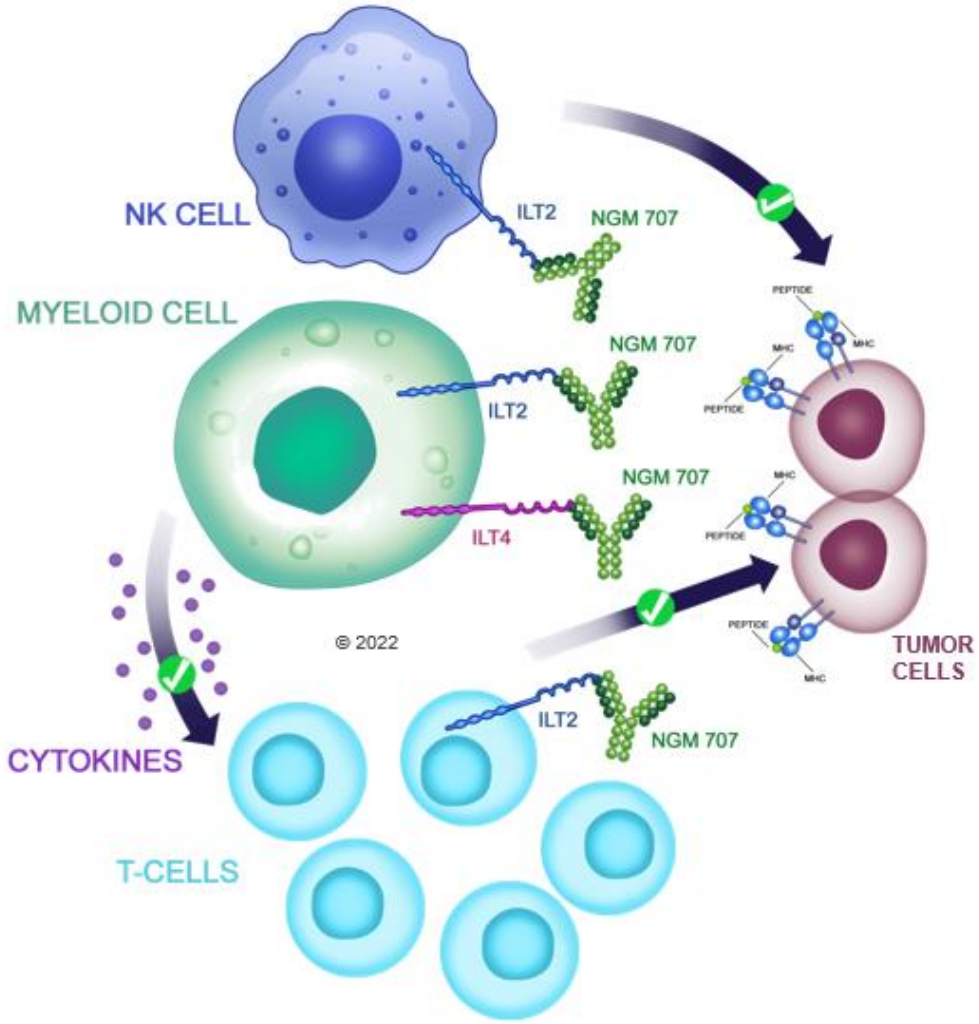
Potent, potential first-in-class antibody targeting the myeloid-enriched inhibitory receptors ILT2 (LILRB1) and ILT4 (LILRB2)

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

Preclinical studies of NGM707 suggest that:

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

Ph1/2 first-in-human trial of NGM707 initiated in Q3 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

Preliminary Data from Phase 1 Part 1 Trial of NGM707 as Presented at ESMO-IO 2022

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

NGM707 was generally well tolerated[1]:

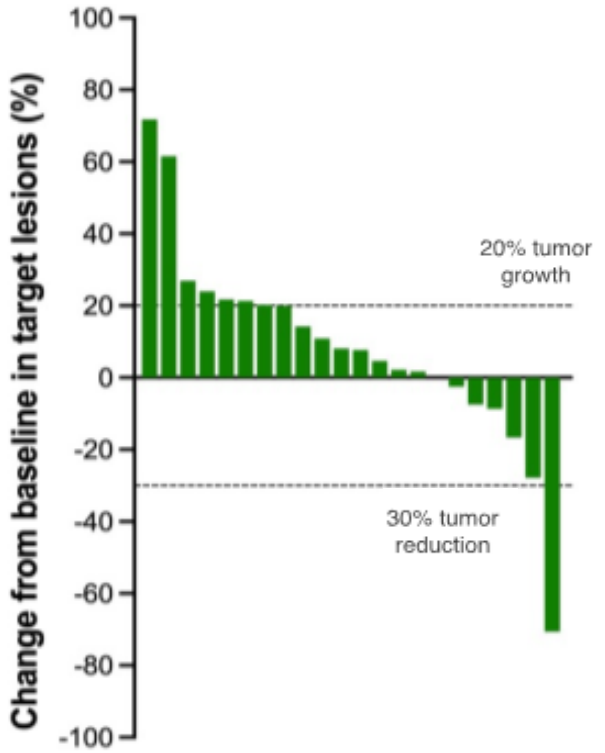
- Treatment (Tx)-related adverse events (any grade/grade ≥3) occurred in 47%/9% of pts.
- One dose-limiting toxicity of pneumonitis (G5) in a pt with pulmonary metastasis was observed at 600 mg.
- A maximum tolerated dose was not reached and the maximum administered dose was 1800 mg.

Encouraging early signals observed:

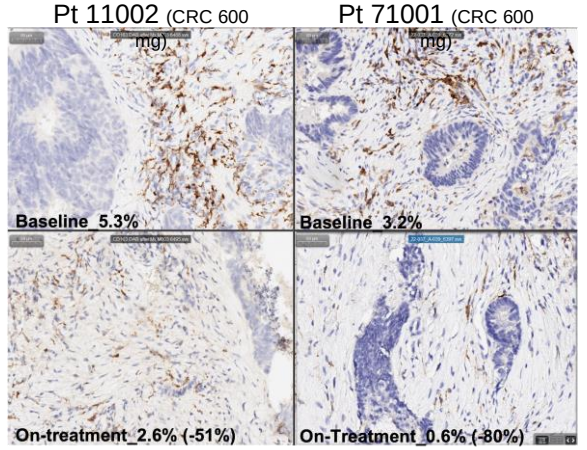
- Of 24 response-evaluable pts[2], best overall responses[1] are partial response in 1 pt, stable disease in 6 pts and non-complete response/non-progressive disease in 1 pt. Six pts had reduced target lesion size incl. a maximum decrease in 1 pt of 70%.
- Preliminary evidence of myeloid reprogramming was seen in peripheral blood and in tumor biopsies.

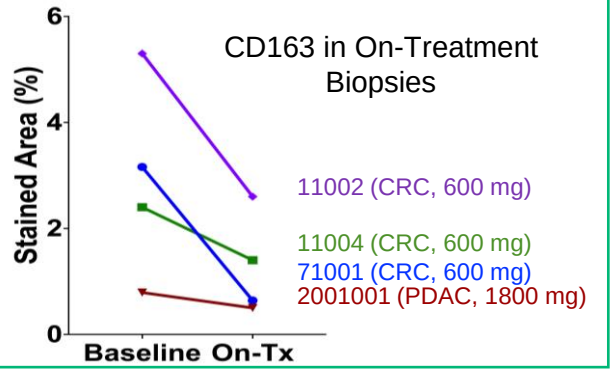
- Ph1a monotherapy dose escalation trial in patients with advanced solid tumors ongoing
- Ph1b dose finding trial in patients with advanced solid tumors in combination with pembrolizumab enrolling

NGM707 Ph1a - Target Lesion Assessment



Preliminary Evidence of Myeloid Reprogramming



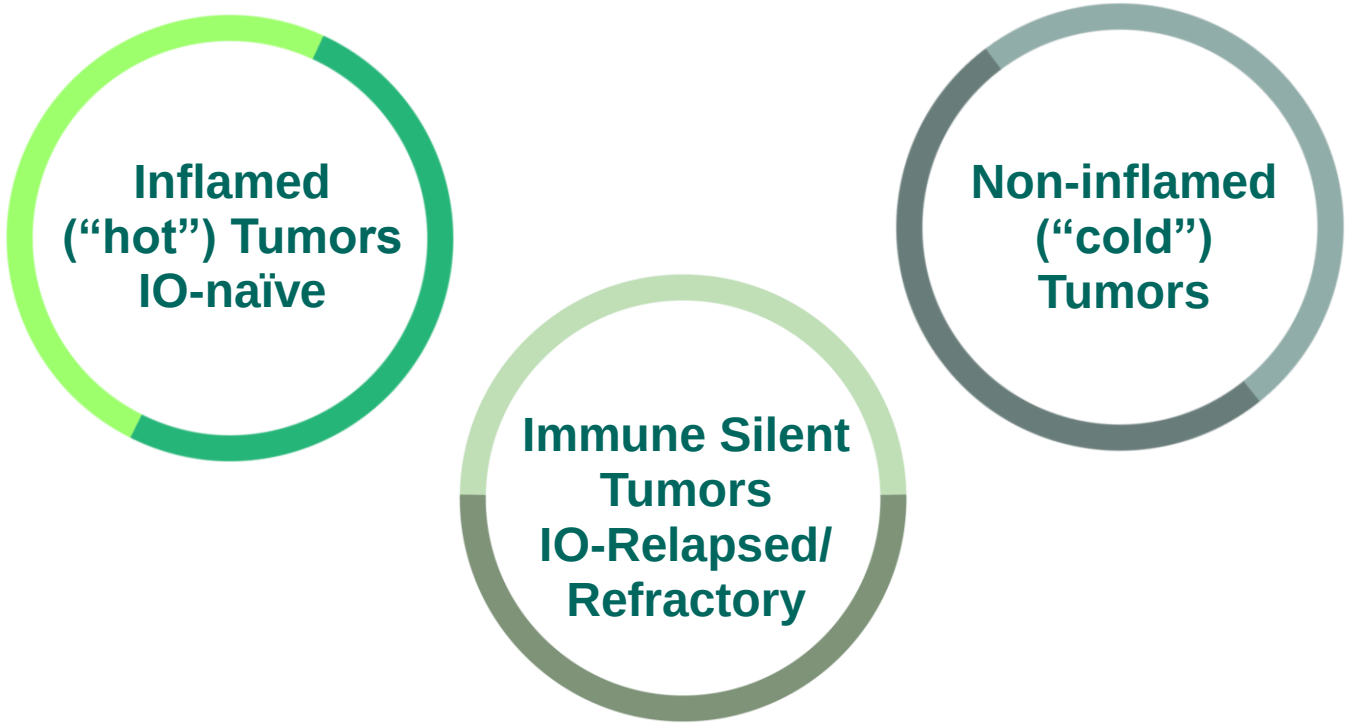


G5=grade 5; SD=stable disease; PD=progressive disease; PT(S)=patient(s) [1] As of November 23, 2022 data cut-off date [2] Those completing at least one on-treatment scan

NGM707 Phase 1/2 Study Designed to Reach Rapid Proof-of-Concept, While Testing Three Therapeutic Hypotheses

Study objectives: (a) Determine safety, PK, PD, and efficacy of NGM707 alone and in combination with anti-PD-1
(b) Test three therapeutic hypotheses in various cancer types

1 NGM707 in combination with anti-PD-1 may **deepen and broaden responses** in inflamed tumor microenvironments



3 NGM707 may **reprogram tumor-associated macrophages** to an inflamed phenotype, enabling responses in immunologically cold tumors

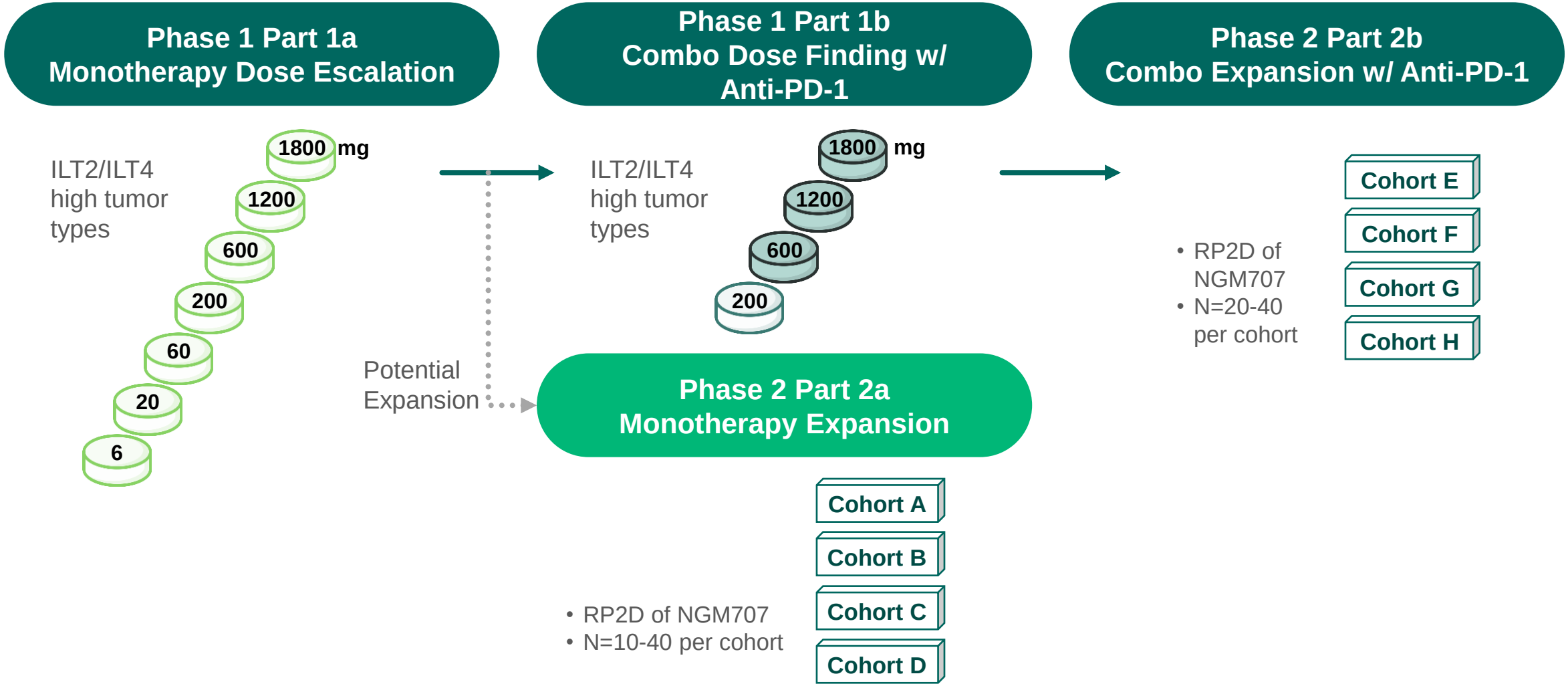
2 NGM707 in combination with anti-PD-1 may **rescue responses in** PD-(L)1 relapsed or refractory tumors

Many Primary Cancer Types Express High ILT2 or ILT4, Suggesting NGM707 May Have Broad Applications

Tumor type	Myeloid cell content	ILT2 expression	ILT4 expression	PD-1/L1 response
NSCLC	Very High	Very High	Very High	High
RCC	Very High	Very High	Very High	High
Mesothelioma	Very High	Very High	Very High	Medium
GBM	Very High	Very High	Medium	Low
Breast Cancer	Very High	High	Medium	Medium
Gastric, GEJ	High	High	High	High
SCCHN	High	Medium	High	Medium
PDAC	High	Medium	High	Low
Ovarian	High	Medium	Medium	Low
CRC	Medium	Medium	Medium	Low
Esophageal	Medium	Medium-Low	Medium	Medium
HCC	Medium	Medium-Low	Medium	Medium
Bladder	Medium	Medium-Low	Medium-Low	High
Thyroid	Medium	Medium-Low	Medium-Low	Low
Uterine	Medium-Low	Medium-Low	Medium-Low	High
Uveal Melanoma	Medium-Low	Low	Low	Medium
Prostate	Medium-Low	Low	Low	Low

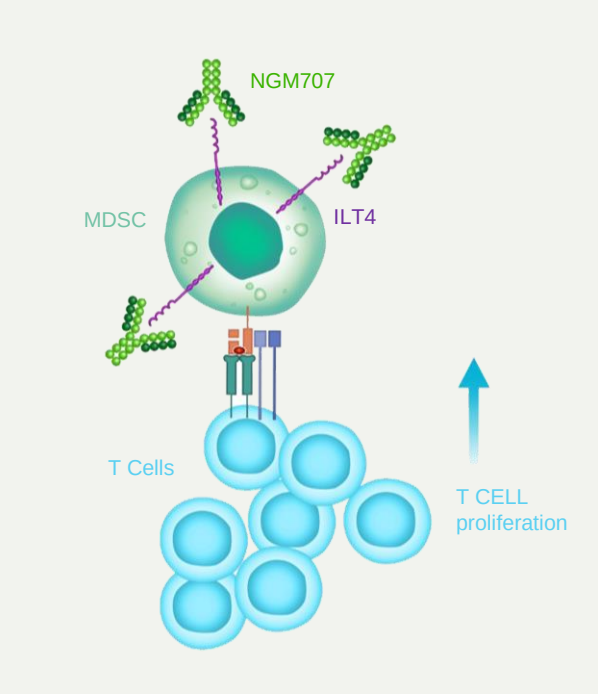
Gene expression data: The Cancer Genome Atlas https://www.genome.gov/Funded-Programs-Projects/Cancer-Genome-Atlas; PD(L)-1 responsiveness: See prescribing information for Keytruda https://www.merck.com/product/usa/pi_circulars/k/keytruda/keytruda_pi.pdf; Opdivo https://packageinserts.bms.com/pi/pi_opdivo.pdf, Tecentriq https://www.gene.com/download/pdf/tecentriq_prescribing.pdf

NGM707 Clinical Development Plan

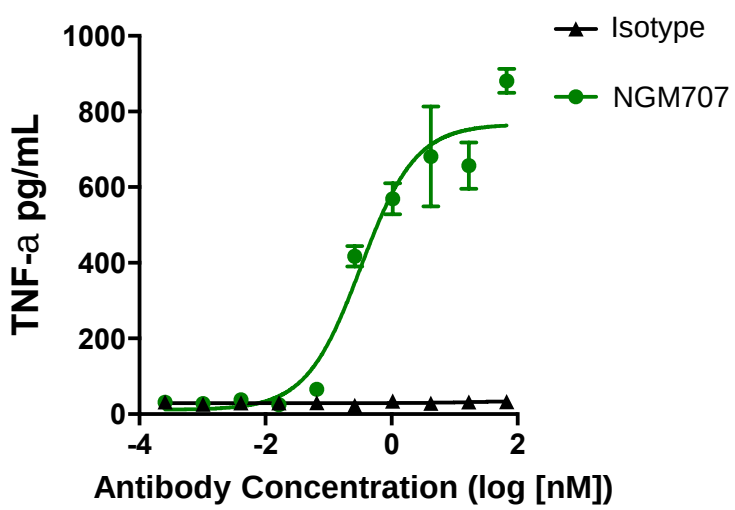


ILT4 Blockade Reprograms Tumor-conditioned Myeloid-derived Suppressor Cells (MDSC)

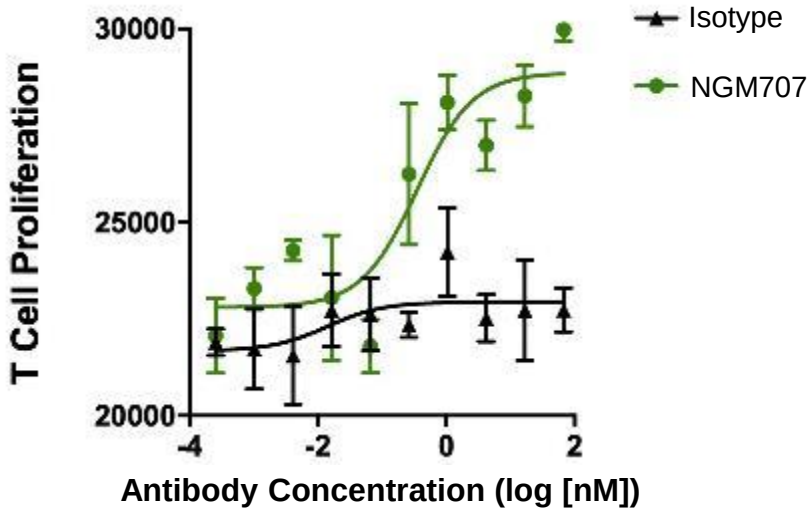
ILT4 antagonism enhances T cell activity and proliferation



T Cell Activation



T Cell Proliferation

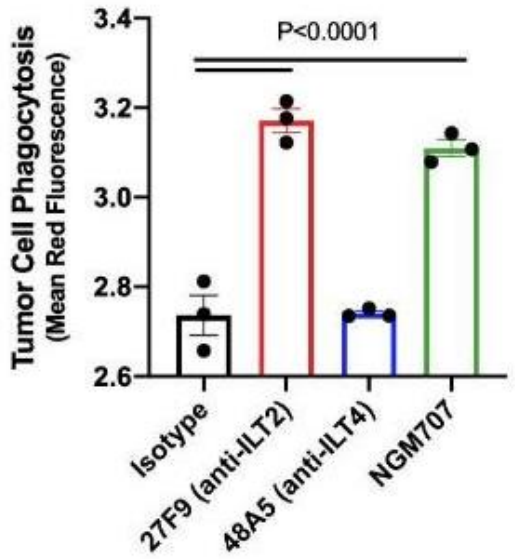


MDSC generated from monocytes using cancer cell-conditioned media (OVISE cells)
MLR performed by mixing MDSC with allogeneic T cells

OVISE = Ovarian Clear Cell Carcinoma; MLR = Mixed Lymphocyte Reaction; TNF = Tumor Necrosis Factor

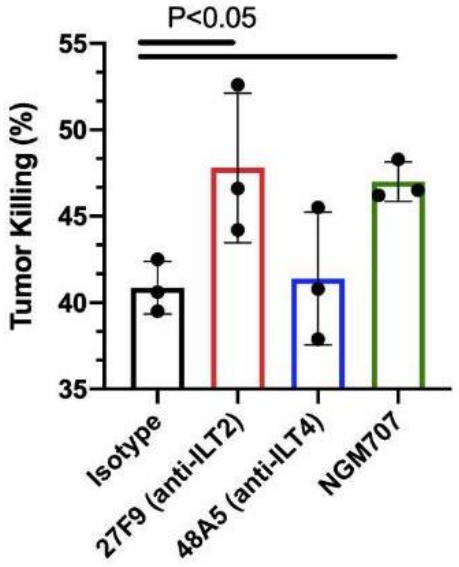
ILT2 Blockade Enhances Macrophage Phagocytosis of Tumor Cells, CD8+ T Cell Cytolytic Activity and Primary NK Cell Killing Activity

Tumor Cell Phagocytosis



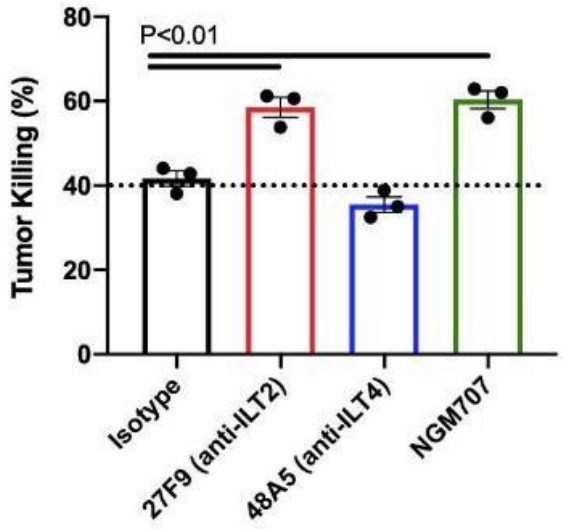
- Macrophage phagocytosis may increase tumor killing and potentially drive antigen spread
- Activity is specific to ILT2/MHC-I interaction despite ILT4 co-expression on macrophages

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

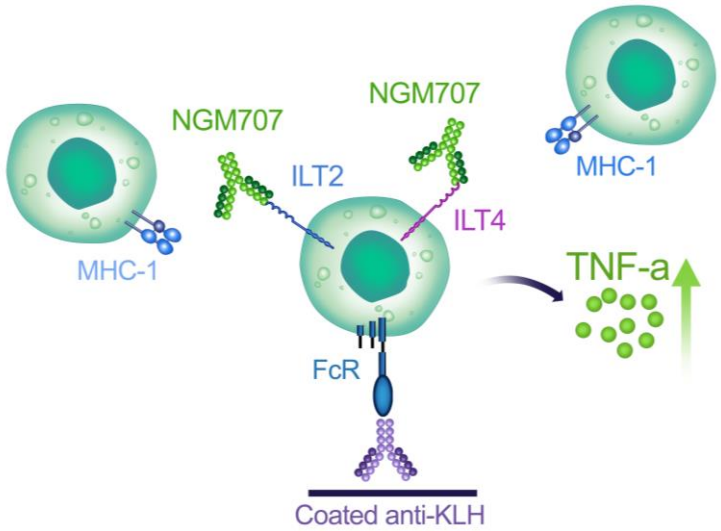
Preclinical Models Suggest That ILT2 and ILT4 Blockade May Act Additively to Enhance Myeloid Cell Activation

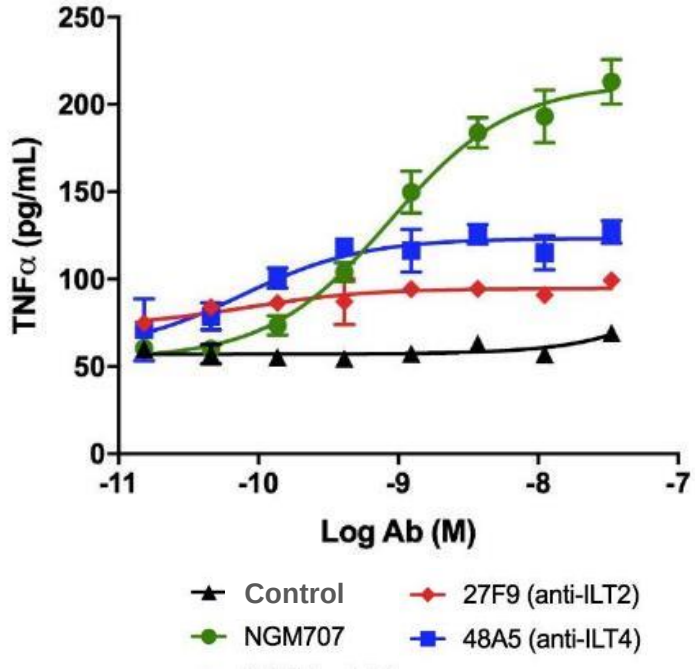
Fc receptors represent key stimulatory receptors on myeloid cells

- 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

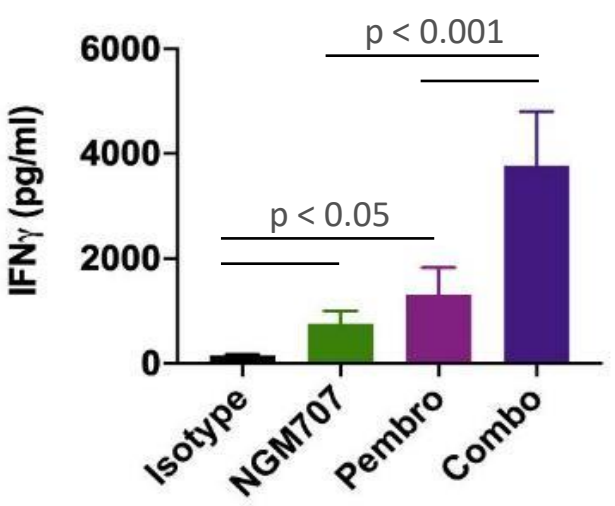
NGM707 and Pembrolizumab Acted Additively to Enhance Immune Stimulating Cytokine Release in a Mixed Lymphocyte Reaction

NGM707 or pembrolizumab alone **modestly** enhanced cytokine secretion (IFNɣ, IL-2, TNFa, GM-CSF)

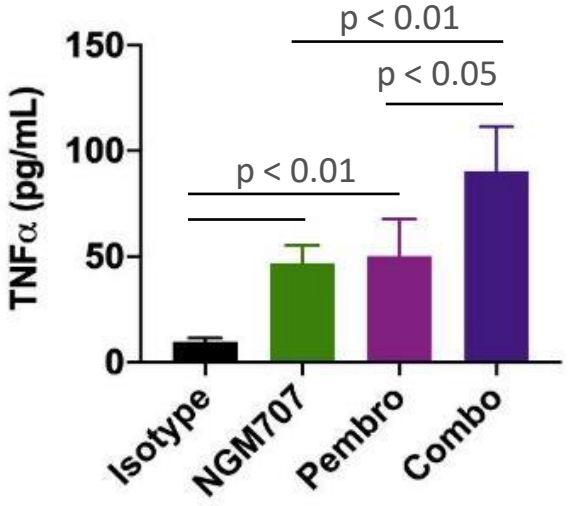
Combination of NGM707 and pembrolizumab led to an **additive** increase in cytokine secretion

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

IFNɣ



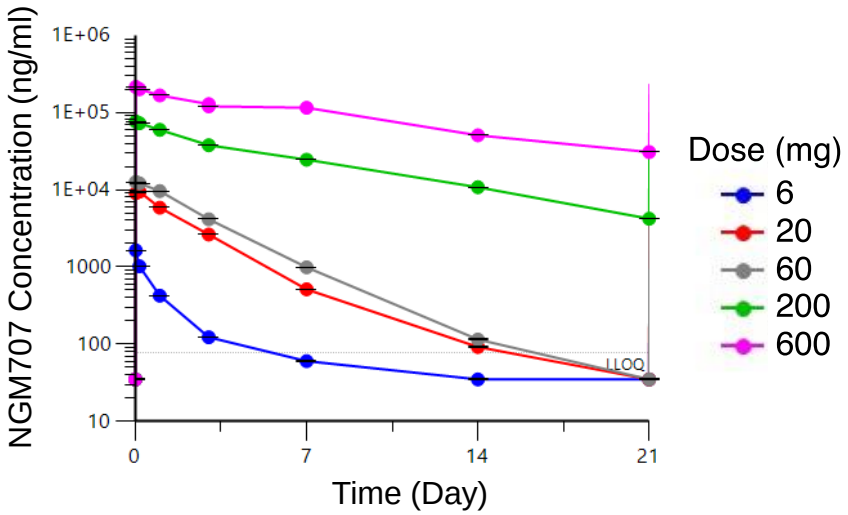
TNFα



Preliminary Clinical Data Show Linear PK and Full Receptor Occupancy for NGM707 at Doses of 200 mg and Above

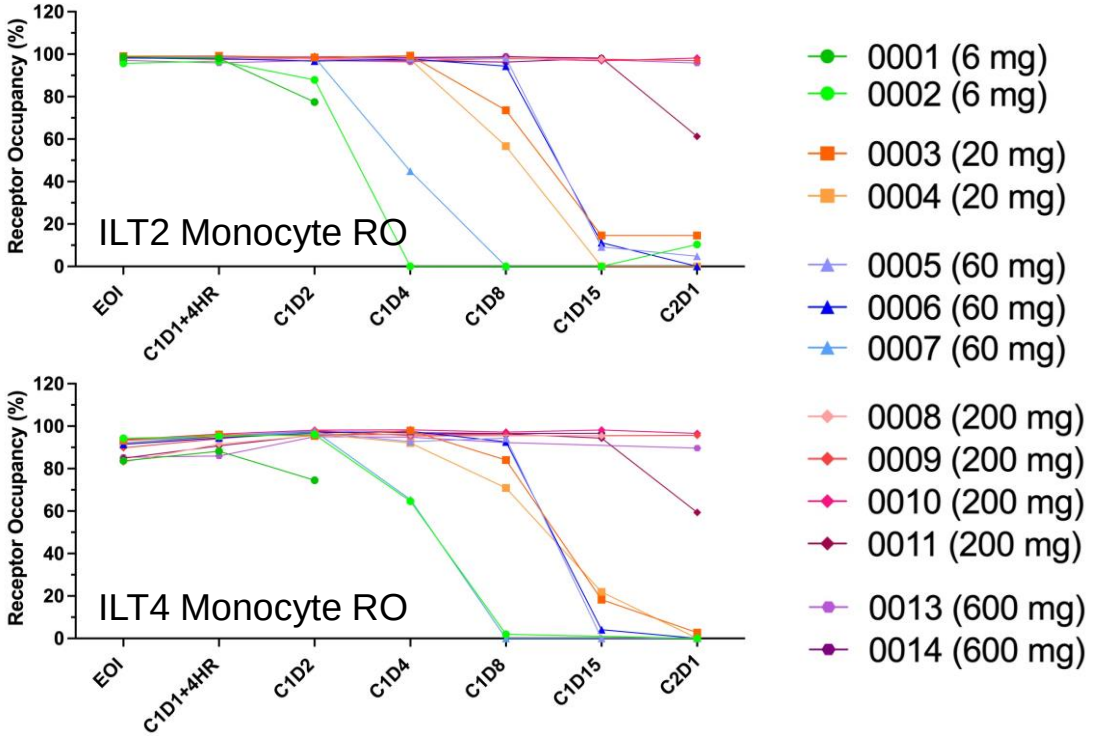
NGM707 Cycle 1 PK Profiles

- Target-mediated drug disposition (TMDD) is apparent at lower dose levels of 6 – 60 mg
- Approximately linear PK (target saturation) observed at doses of 200 mg and above



NGM707 Receptor Occupancy

- Full receptor occupancy of ILT2 and ILT4 observed at doses of 200 mg and above



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

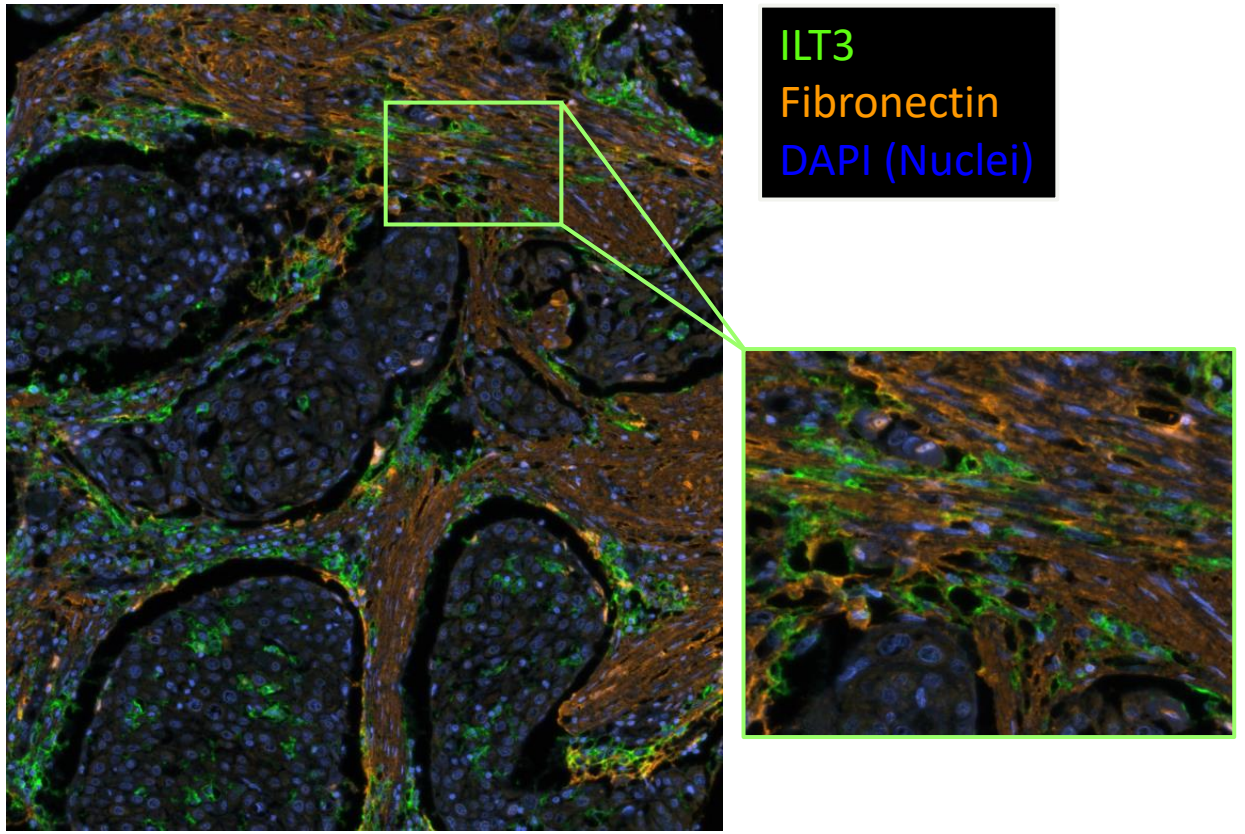
ILT3 is upregulated in several tumor types and is associated with poor survival[1,2]

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+ Myeloid Cells are Localized Within the Fibronectin-Rich Tumor Stroma in Ovarian Cancer



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

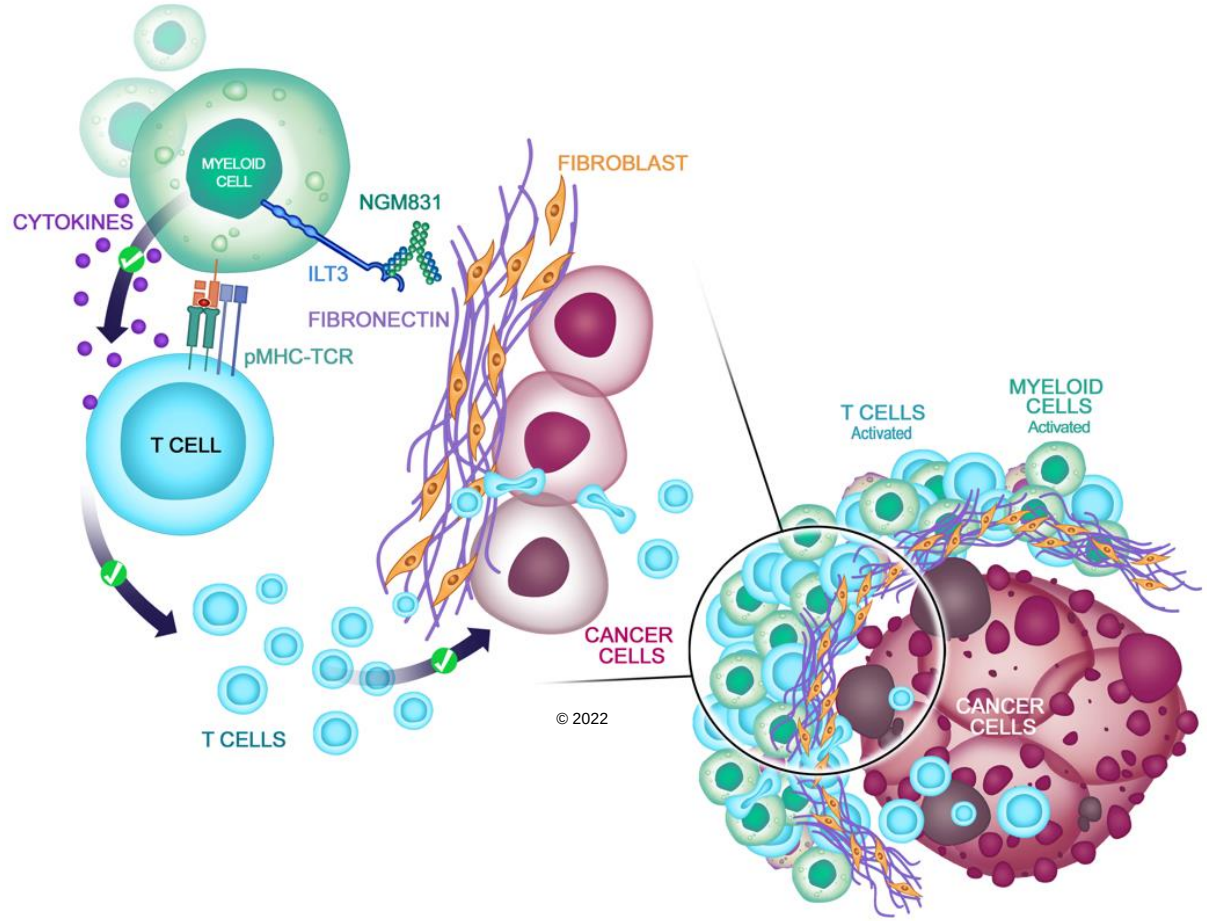
NGM831 is an Antagonist Antibody Designed to Inhibit ILT3 That Entered the Clinic in 2022

Potential to reprogram ILT3-expressing suppressive myeloid cells by blocking signal from the extracellular matrix that promotes myeloid cell suppression

Preclinical studies suggest that NGM831 may:

- Reprogram tolerogenic dendritic cells into stimulatory cells
- Enhance activity through stimulatory receptors such as Fc receptors
- Enhance T cell activation and infiltration of tumors

Ph1/1b first-in-human trial of NGM831 initiated in Q1 2022



ILT3 Blockade Reverses Fibronectin-mediated Inhibition of Fc Receptor Activity

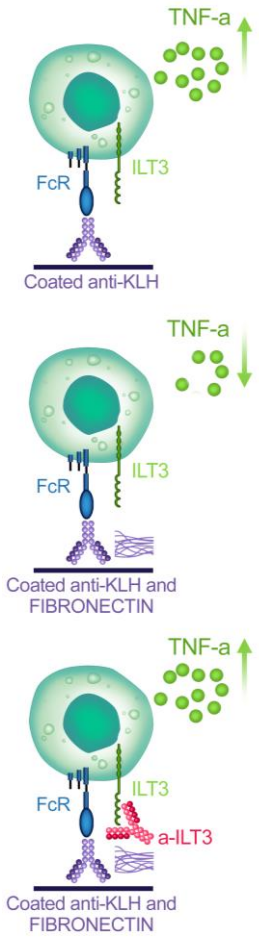
ILT3 blockade restores the ability of dendritic cells to respond to Fc Receptor activation in the presence of fibronectin

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

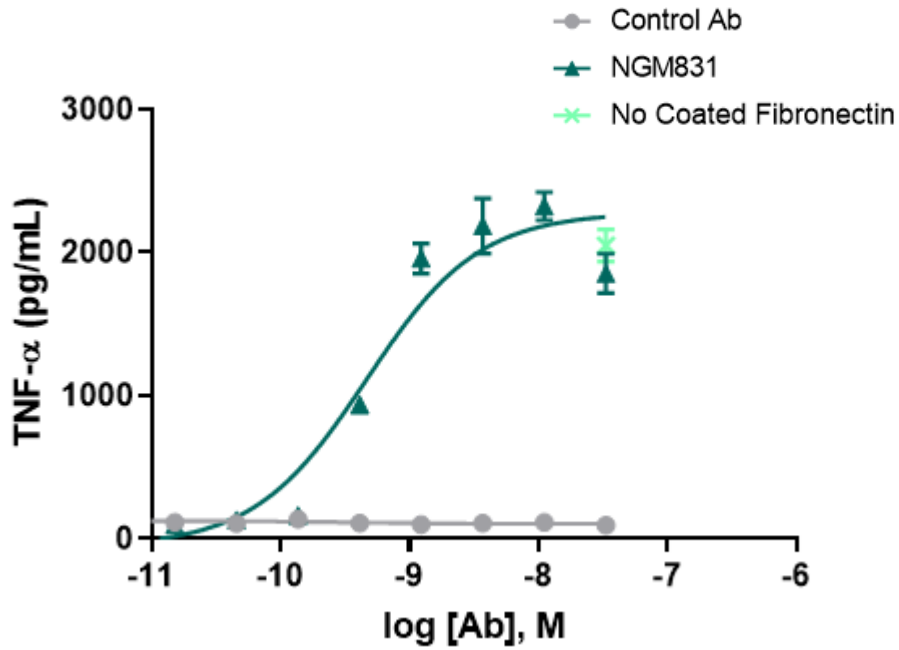
1 DCs stimulated with anti-KLH secrete TNF-α

2 TNF-α secretion is inhibited by platebound fibronectin

3 Inhibition of FcR signaling by fibronectin is blocked by ILT3



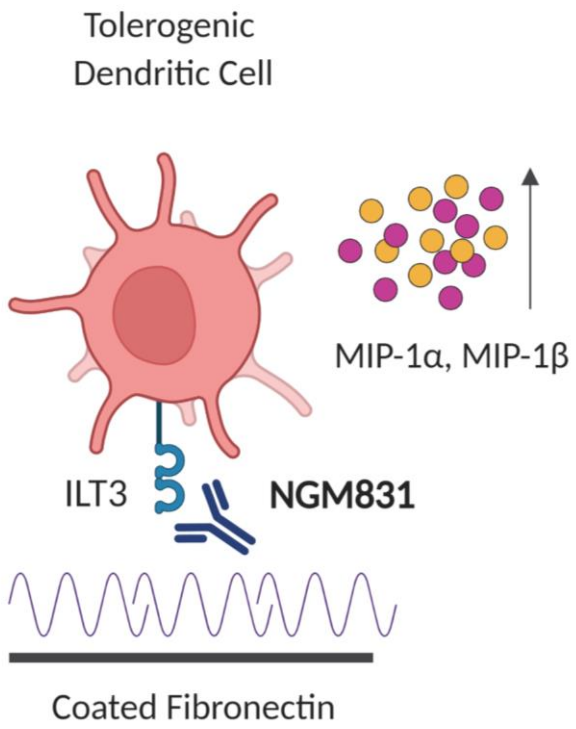
The Inhibitory Effect of Fibronectin on Dendritic Cell Cytokine Production is Reversed by NGM831



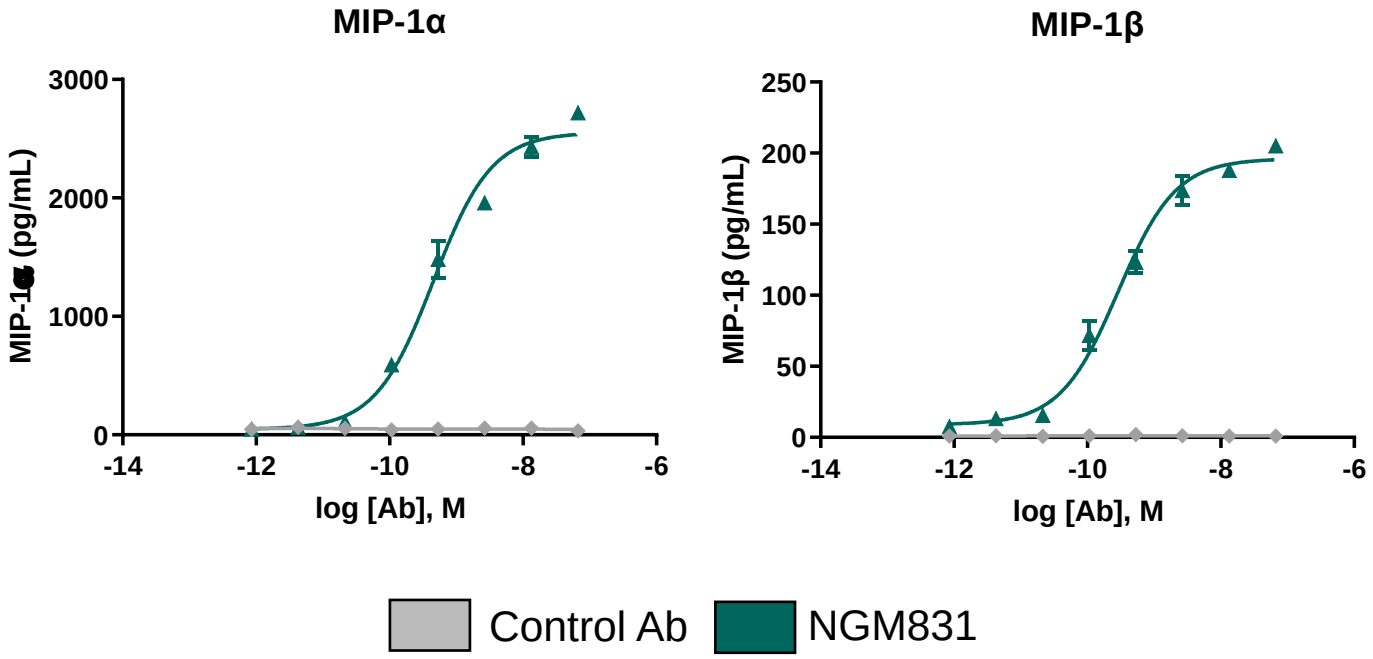
AACR Poster LB216, 2022

Paavola et al, *Cancer Immunology Research,* 2021

NGM831 Increases Chemokine Production by Tolerogenic DCs

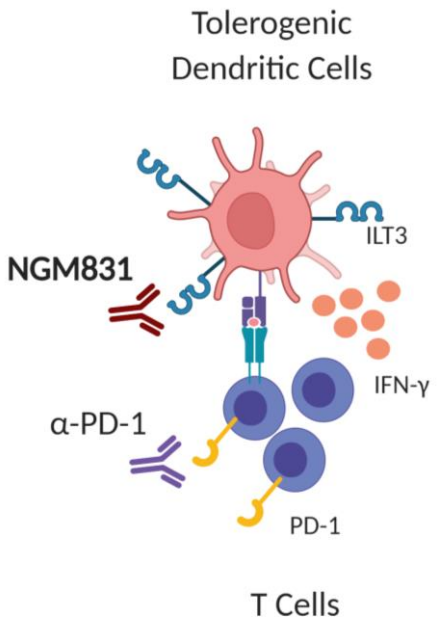


NGM831 Increases Dendritic Cell Chemokine Production

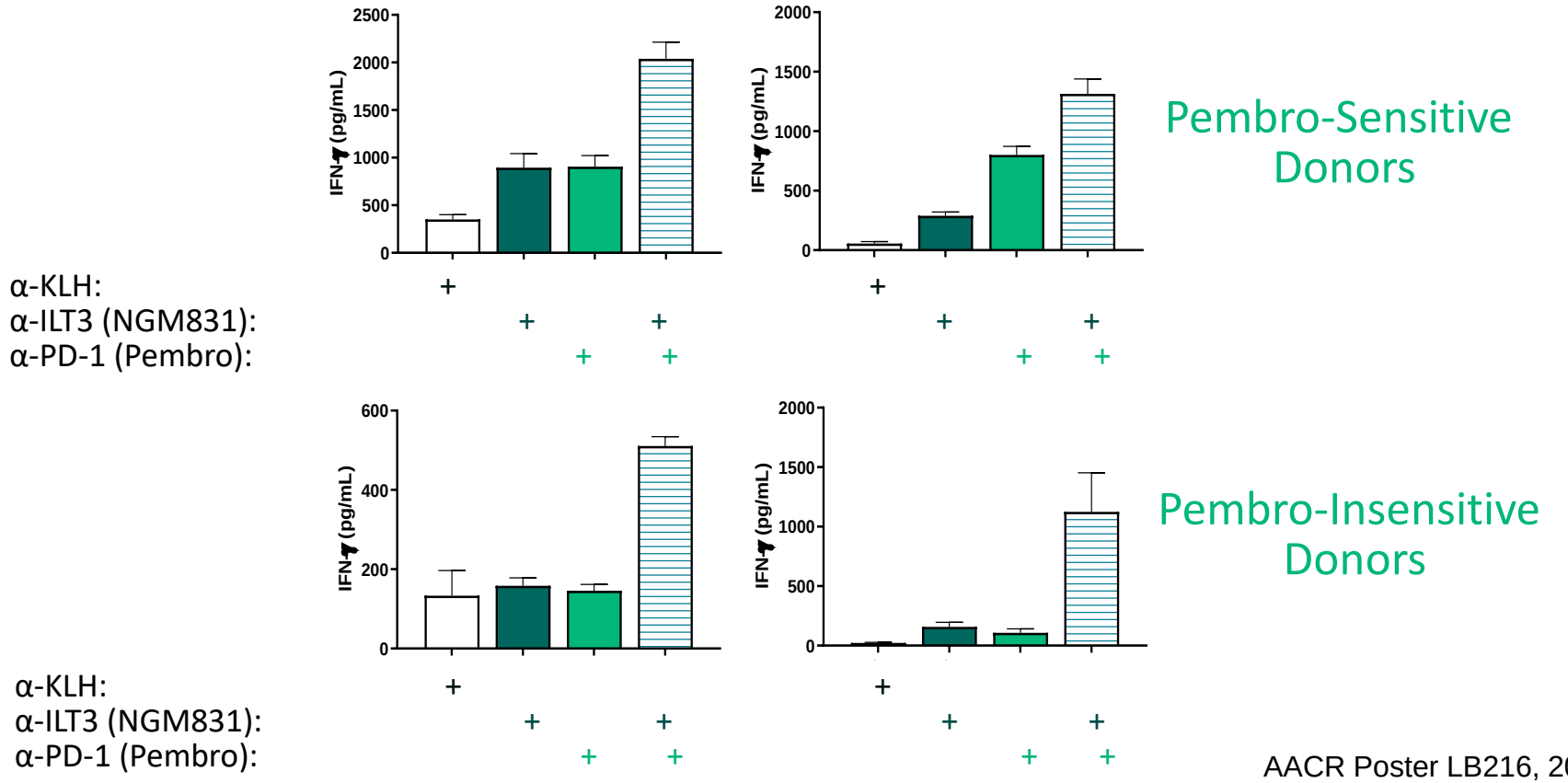


AACR Poster LB216, 2022

NGM831 Increased the Ability of Dendritic Cells to Stimulate T Cells in Combination with Pembrolizumab



T Cell Activation by NGM831 and Pembrolizumab



AACR Poster LB216, 2022

NGM438 in Advanced Solid Tumors

LAIR1: Key Stromal Checkpoint and its Potential Roles in Cancer

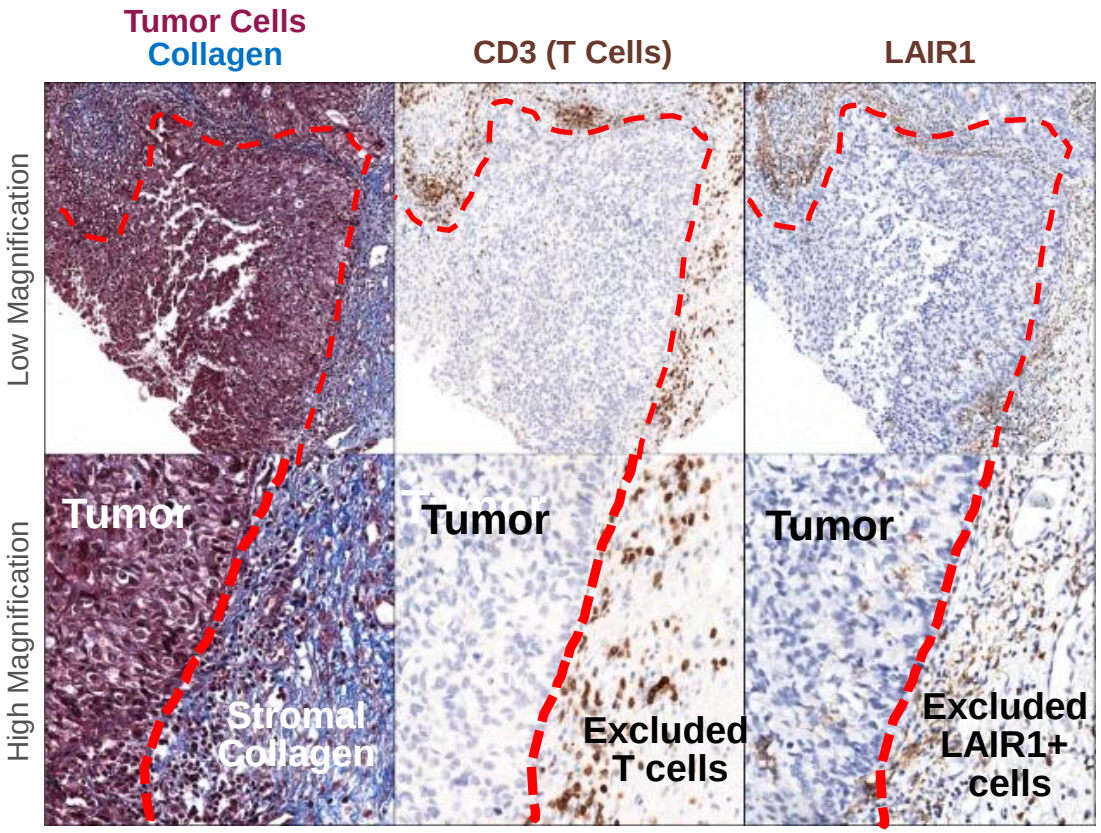
LAIR1 is a collagen-binding inhibitory signaling receptor expressed on immune cells: T cells, B cells, NK cells and myeloid cells[1-2]

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 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

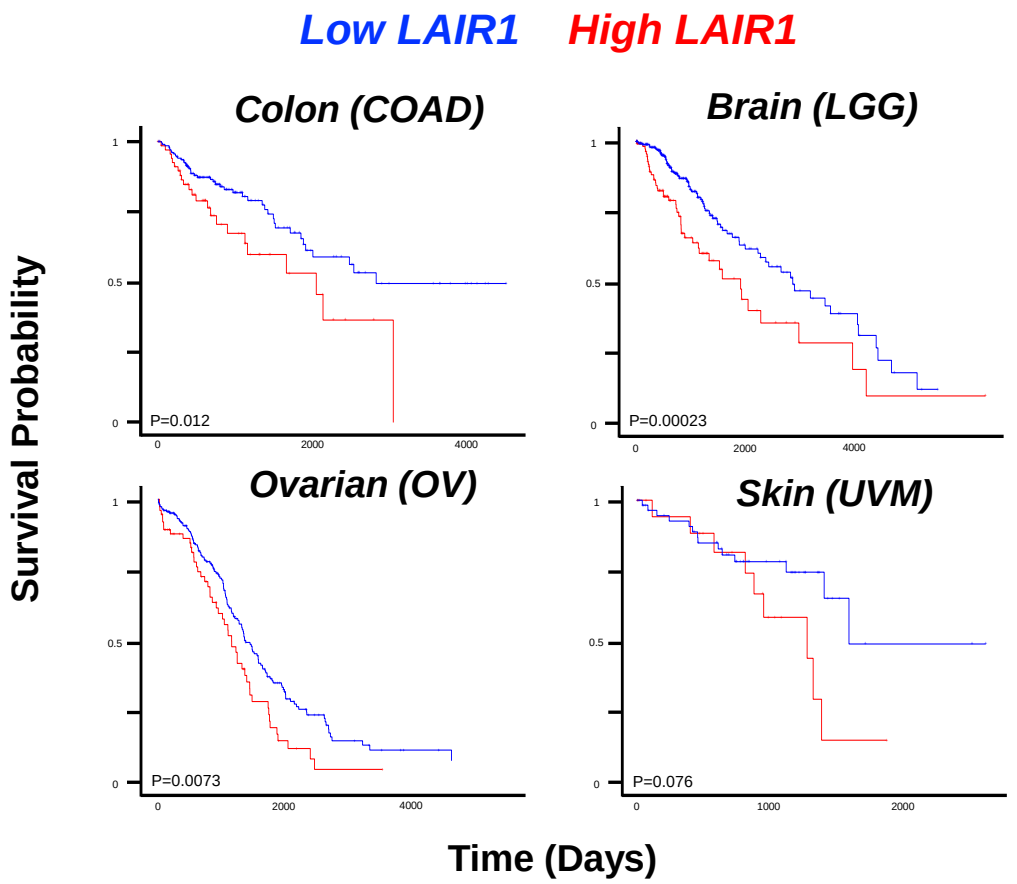


Pancreatic ductal adenocarcinoma tumor section

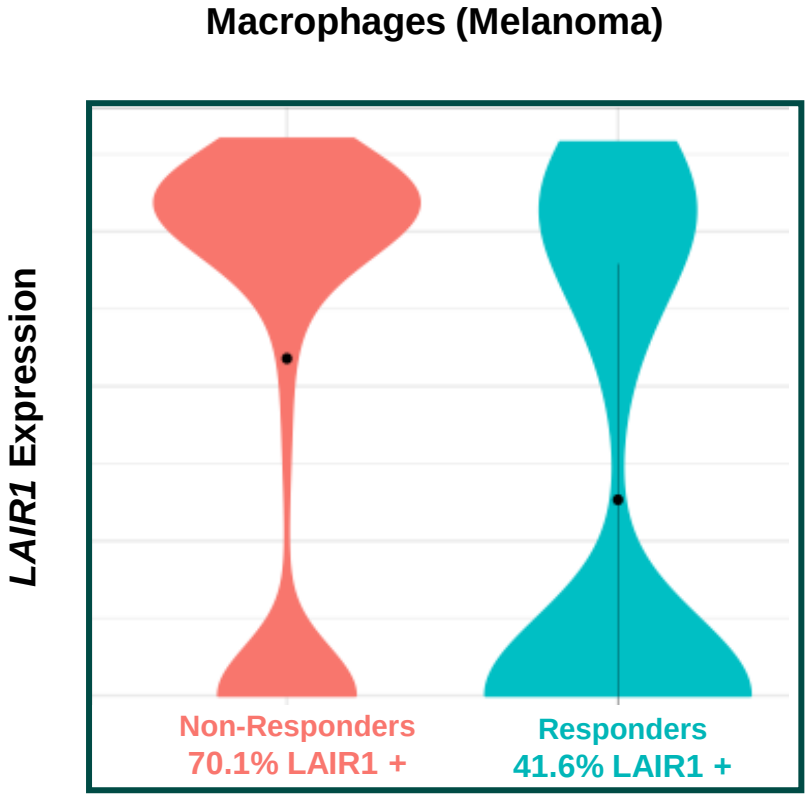
1. Meyaard, Immunity, 1997; 2. Guo, Trans Med, 2020, 3. Cao, 2015, Biochem Biophys Res Commun; 4. Wang, Exp Ther Med, 2016; 5. Wu, CP Cancer, 2018; 6. Yang, Head & Neck, 2018 7. Jingushi, Onc. Reports, 2018; 8. Peng, Nat Comm, 2020; 9. Lijun, Oncoimmunology, 2020

LAIR1 Expression in Tumors Correlates with Poor Survival and Resistance to Checkpoint Inhibitor Therapies

High LAIR1 expression is associated with worse survival outcomes[1]



LAIR1 expression in tumor macrophages correlates with checkpoint inhibitor non-response in melanoma[2]



[1] NGM Bio analysis of TCGA database using UALCAN (http://ualcan.path.uab.edu/). Highest vs. lowest quartile of gene expression. p-value calculated using long-rank test.
[2] NGM Bio analysis of data from Sade-Feldman, et. al., 2019.

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

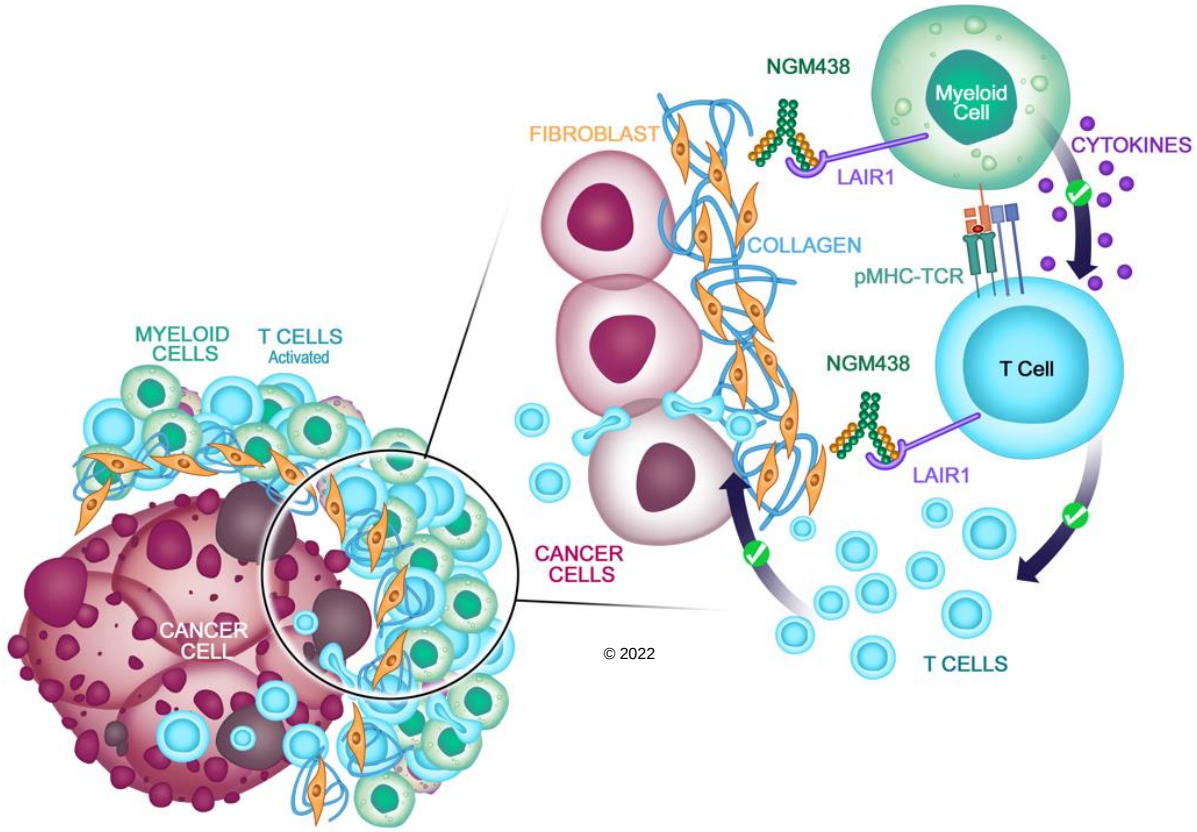
Potent, potential first-in-class antibody targeting the myeloid-enriched inhibitory receptor LAIR1

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:

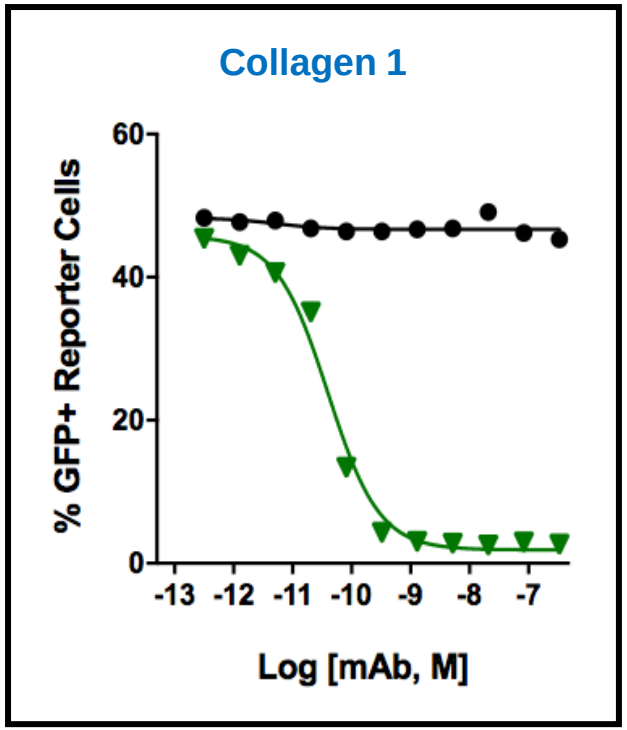
- Reverse collagen mediated suppression of myeloid cells to a stimulatory phenotype
- Stimulate inflammatory cytokine production in myeloid and T cells
- Reprogram collagen suppressed myeloid cells to stimulate T cell activation
- Enhance cellular proliferation of collagen suppressed T cells

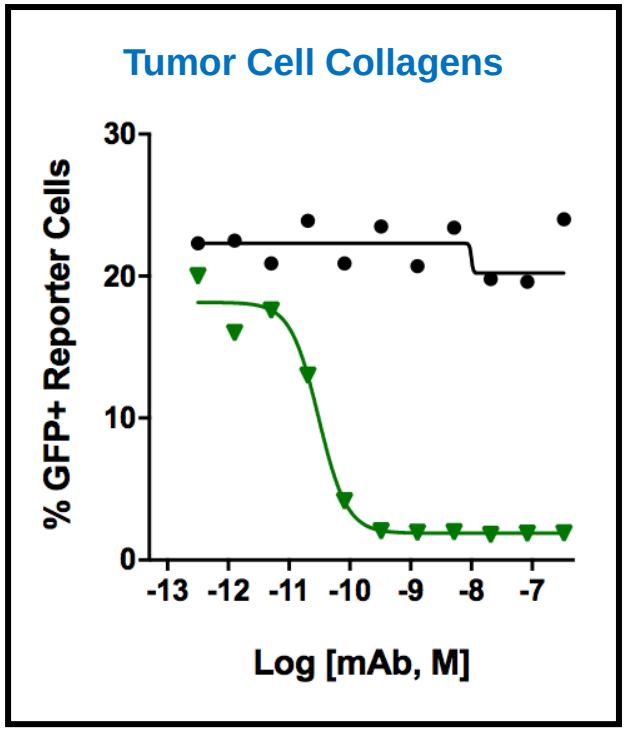
Ph1/1b first-in-human trial of NGM438 initiated in Q2 2022

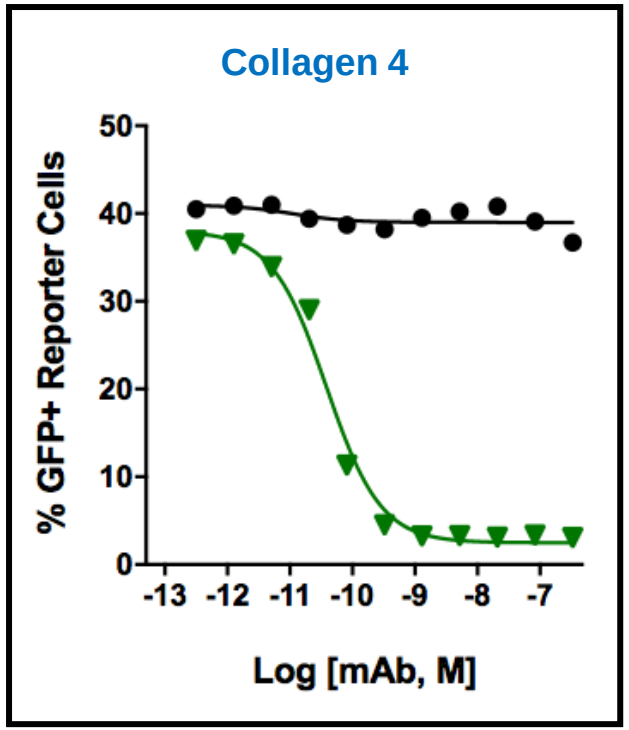


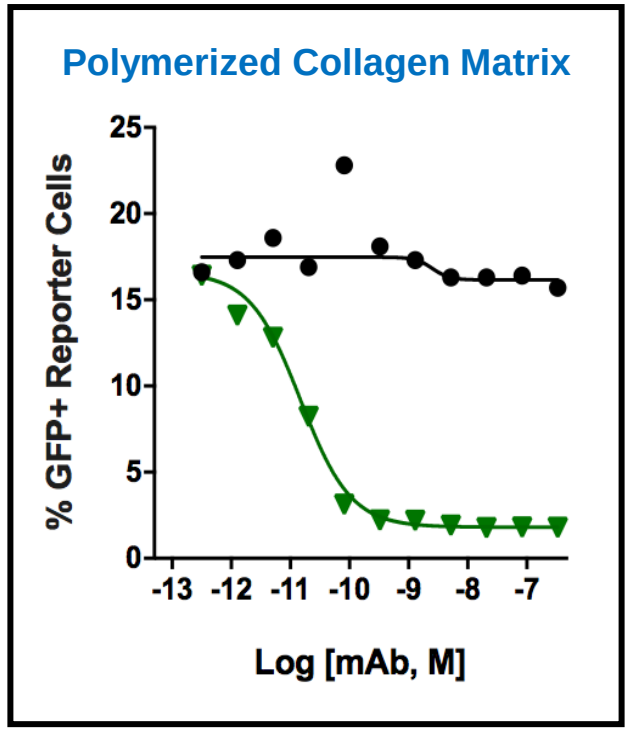
NGM438 Potently Antagonizes LAIR1 Signaling From Many Forms of Collagens

NGM438 ▼ Isotype Control ●





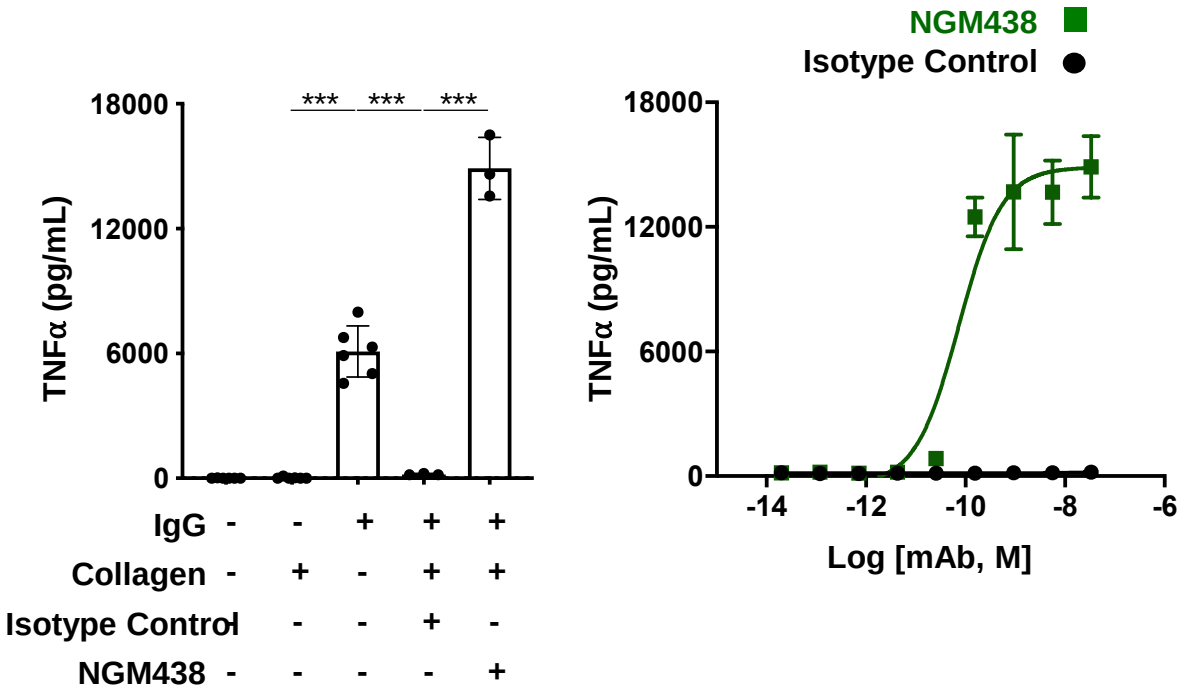




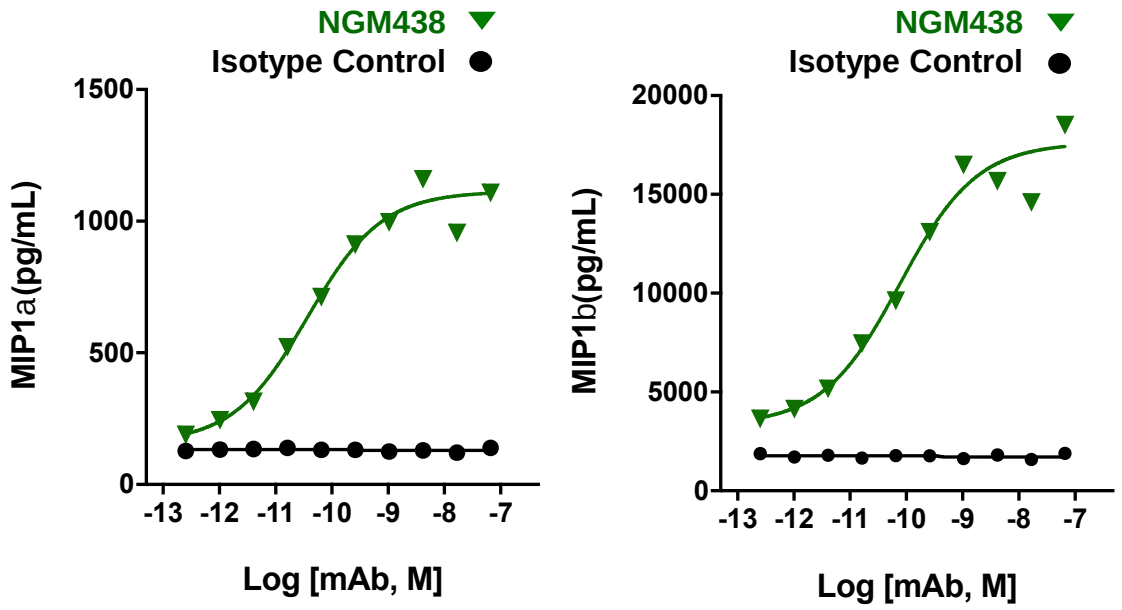
NGM Bio unpublished data
Flow cytometry of LAIR1 GFP responsive reporter cells incubated with NGM438 on collagen-coated plates or co-cultured with collagen-expressing cells

NGM438 Reversed Collagen Suppression and Reprogrammed Macrophages and Dendritic Cells to Induce Inflammation

NGM438 Reversed Collagen-Suppressed Macrophage Fc Receptor Activation

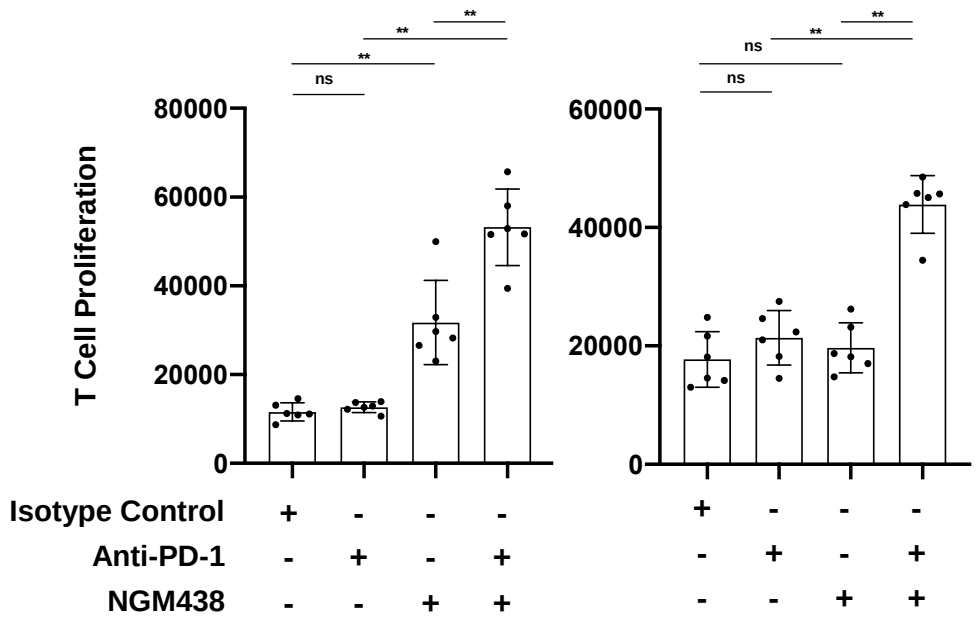


NGM438 Reprogrammed Dendritic Cells to Produce Inflammatory Chemokines

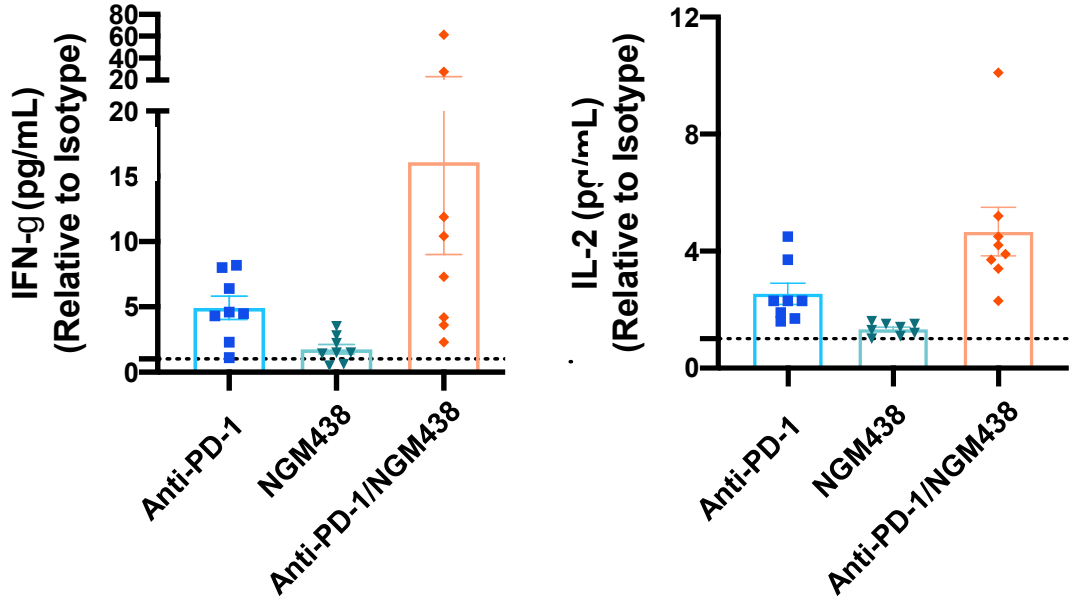


NGM438 Activated T cells and Synergized with Anti-PD-1 mAbs to Induce Robust Proliferation and Cytokine Production

NGM438 Induced T Cell Proliferation



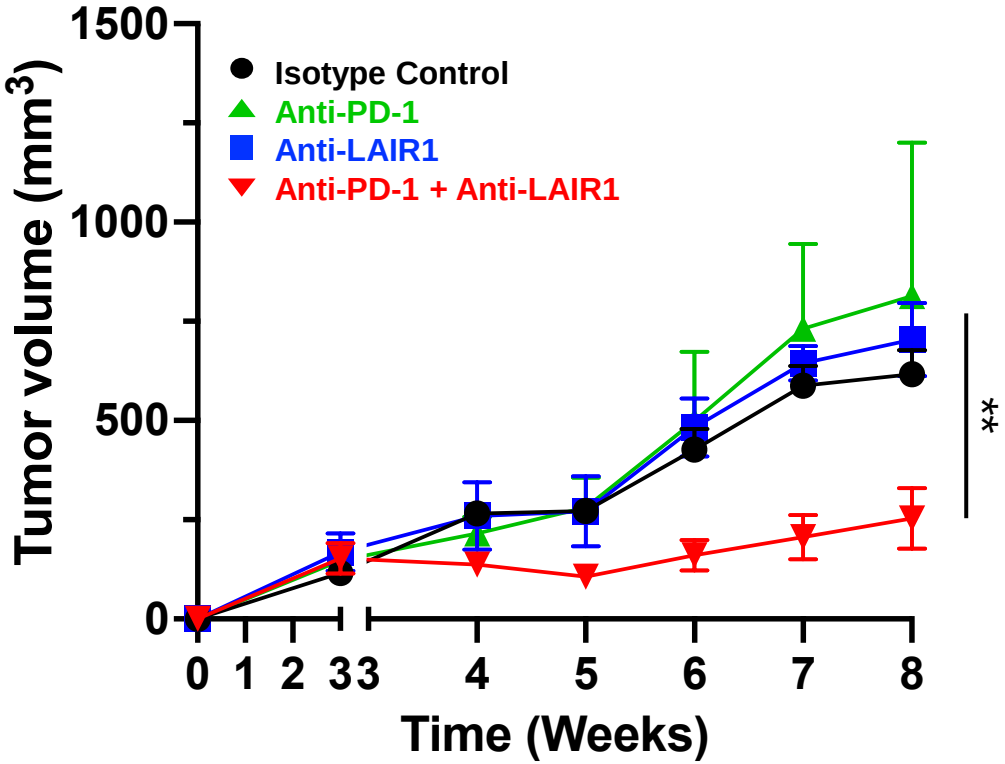
NGM438 Induced Inflammatory Cytokine Production



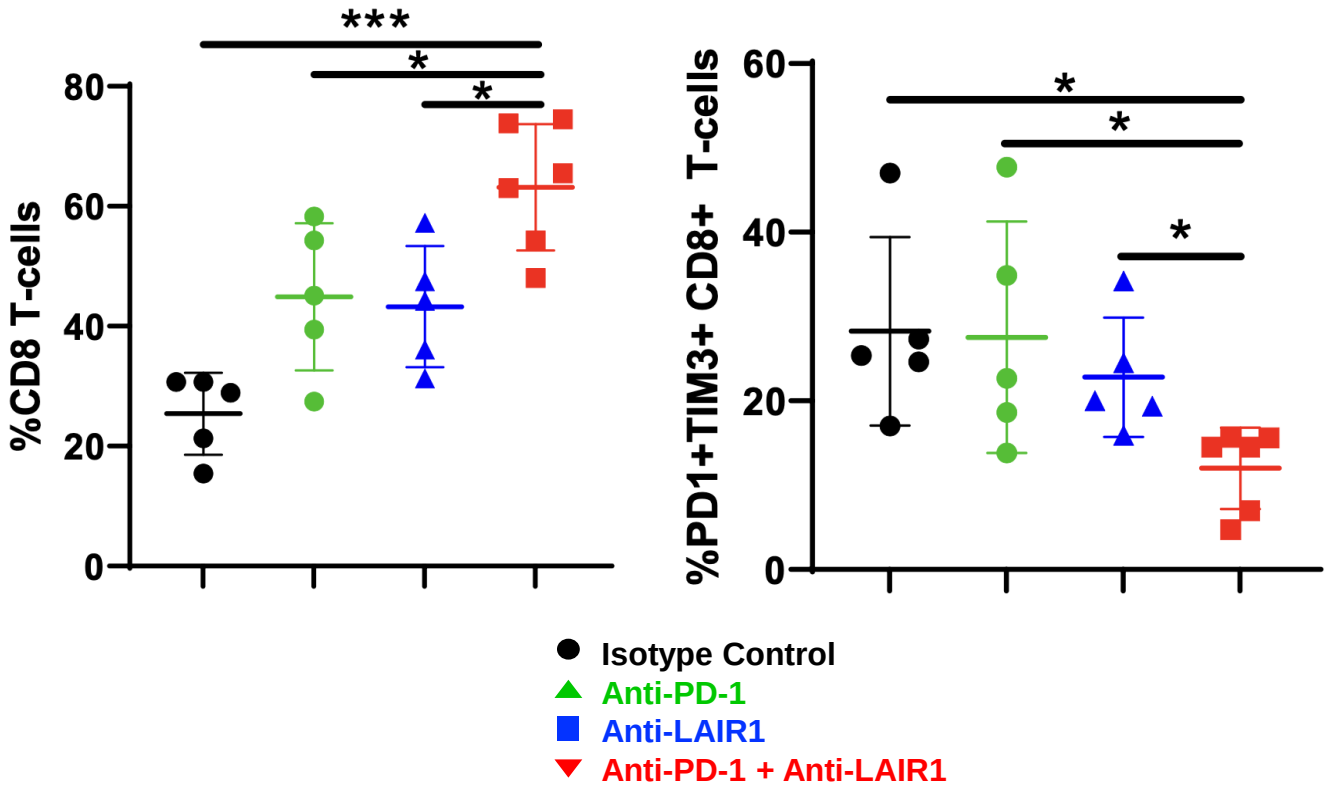
NGM Bio research presented at AACR 2022 Annual Meeting
Proliferation (thymidine incorporation) and cytokine (Luminex) analysis of mixed lymphocyte reaction. Allogeneic T cells and dendritic cells incubated with NGM438 and/or Anti-PD-1 mAbs on collagen-coated plates. Cytokine analysis done on responders from proliferation studies

LAIR1 Antagonism Promoted Significant Tumor Growth Inhibition in a Resistant Mouse Model when Combined with Anti-PD-1 mAbs

A Mouse Surrogate LAIR1 Antagonist mAb Synergized with Anti-PD-1 mAbs to Induce Preclinical Tumor Growth Inhibition



Dual Antagonism Increased Tumor CD8 T Cells and Decreased Exhausted T cell Phenotype

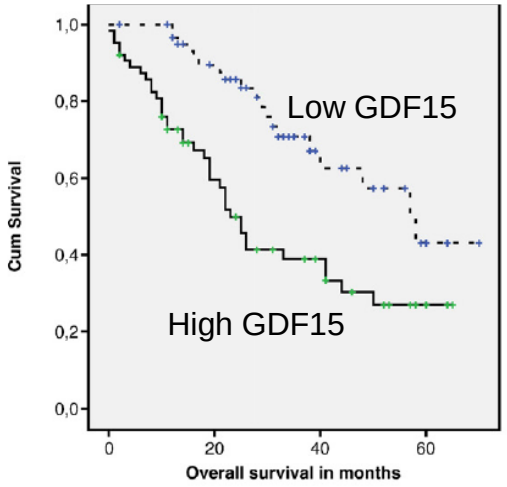


NGM Bio research presented at AACR 2022 Annual Meeting
A surrogate anti-mouse LAIR1 antagonist mAb was developed at NGM Bio with similar pharmacological properties as NGM438 for preclinical mouse studies.
Female 129/Sv mice with subcutaneous KP 344SQ tumors (Peng, 2020) were treated weekly with anti-LAIR1 mAbs +/- anti-PD-1 mAbs (RMP1-14) and tumor growth was measured.

NGM120 for the Treatment of Cancer

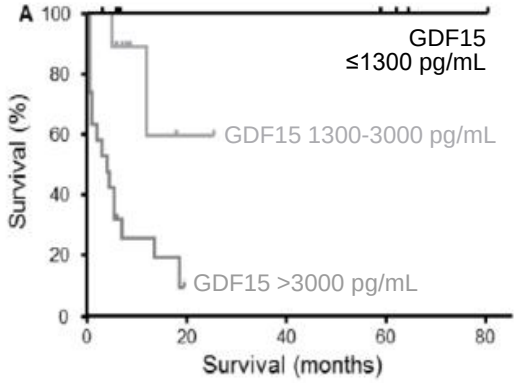
Elevated GDF15 Levels are Linked to Shorter Patient Survival in Multiple Cancer Types

Ovarian Cancer



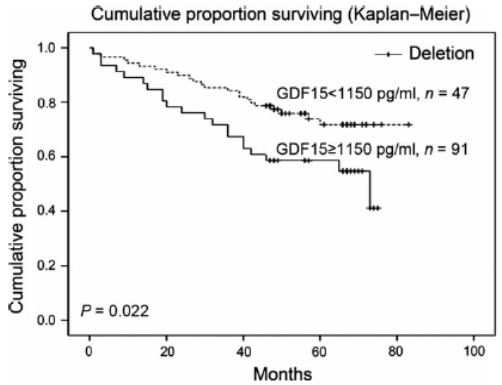
Staff *et al*., *Gynecologic Oncology* 2010

Prostate Cancer



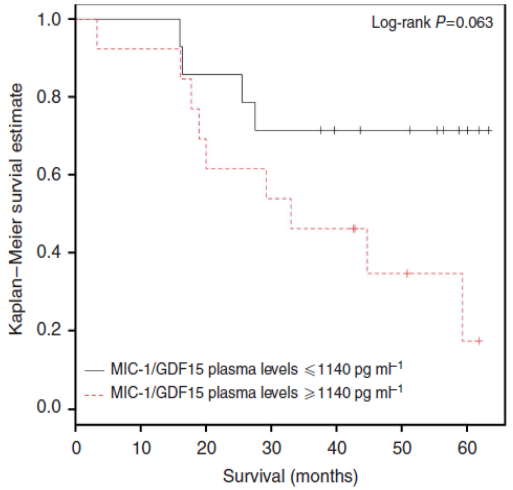
Winand *et al.*, *J Mol Biomark Diagn* 2014

Colorectal Cancer (CRC)



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

Esophageal Cancer



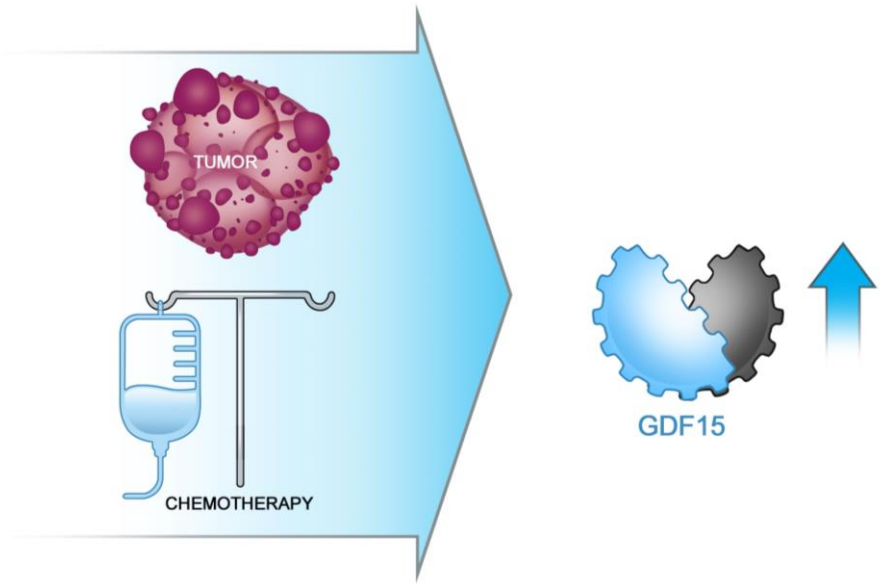
Fisher *et al., BJC* 2015

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

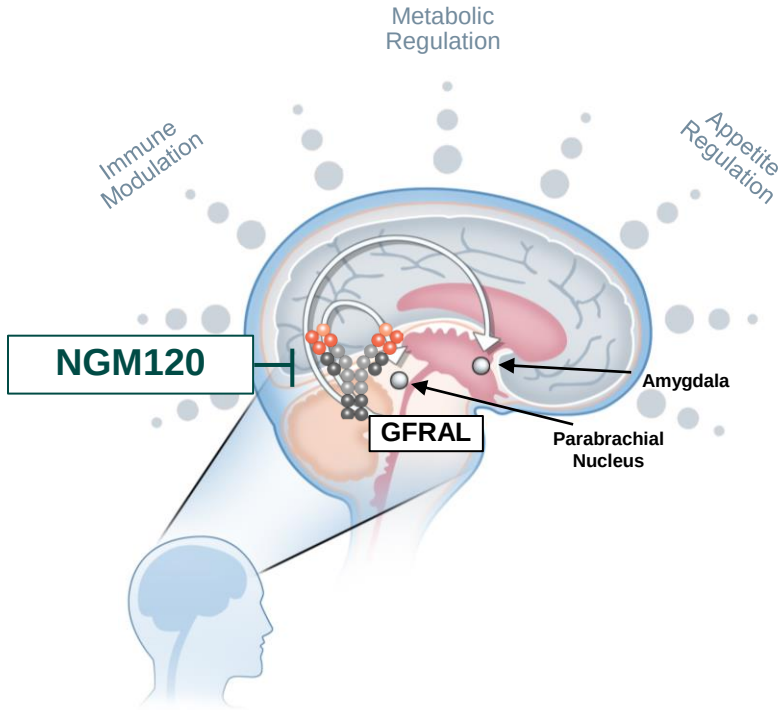
There are Multiple Mechanisms by Which the GDF15-GFRAL Pathway May Impact Tumor Growth

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

Updated Results of Phase 1a/1b trial of NGM120 as Presented at ESMO 2022 and AACR: Pancreatic Cancer 2022

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

In the Ph1a trial a subgroup of patients with advanced prostate cancer:

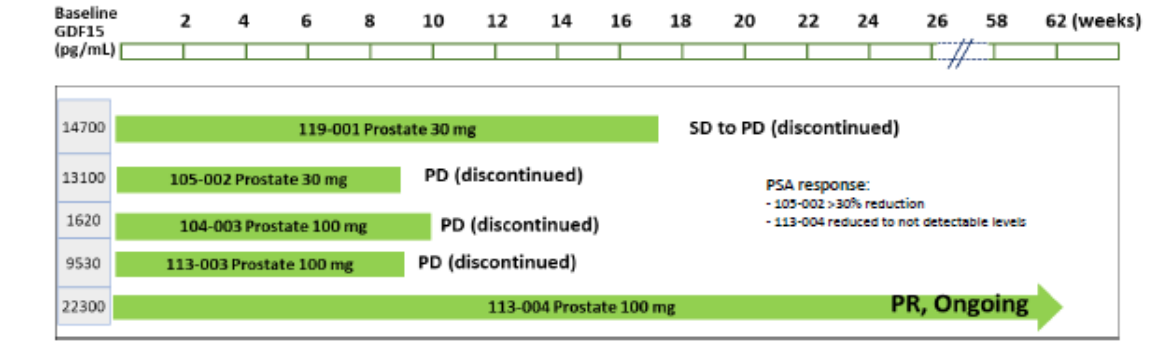
- Two of five prostate cancer patients demonstrated disease control with one partial response (PR) ongoing at 62 weeks[3]
- Two patients experienced reductions in prostate-specific antigen (PSA) levels, one with a >30% reduction and one patient with reduction of PSA to undetectable levels

In the Ph1b trial in patients with mPDAC[2] in combination with Gem/Nab-P:

- Among the six evaluable patients in the combination cohort, a disease control rate of 100% was observed, mPFS had not been reached and the 12-month survival rate was 83.3%[4]
- Three of the six evaluable patients experienced PR extending more than 32 weeks including one patient with a PR that was ongoing at 90 weeks[5]

- Ph1a/1b NGM120 dose escalation study in patients with cancer
- Ph1b NGM120 in combination with one or more lines of hormone therapy in patients with mCRPC
- Ph2 NGM120 expansion trial in patients with metastatic pancreatic cancer

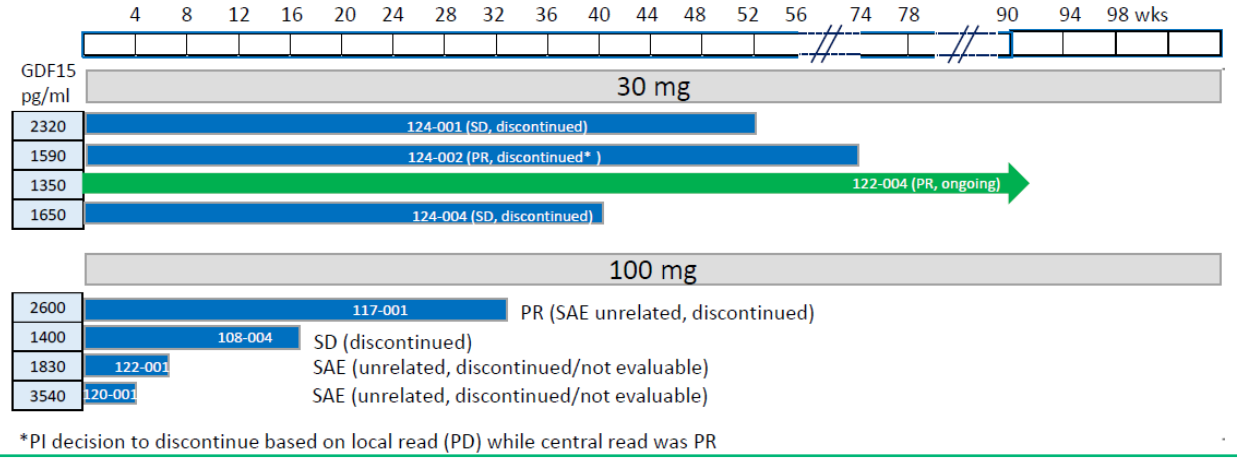
Phase 1a Prostate Cancer Cohort – ESMO 2022



Patient 113-004 Demonstrating Durable Responses with NGM120 Monotherapy

- 70-year-old man with a history of multiple lines of treatment refractory prostate cancer (Gleason 4+4)
- RECIST measurable target lesions in the pelvic area and non-target lesions in mediastinum, pelvic and bone

Phase 1b in 1L mPDAC[2] – AACR: Pancreatic Cancer 2022



*PI decision to discontinue based on local read (PD) while central read was PR

Updated results presented at European Society for Medical Oncology (ESMO) Congress 2022 and American Association for Cancer Research (AACR): Pancreatic Cancer 2022

[1] NGM120 30 mg and 100 mg; [2] mPDAC – metastatic pancreatic ductal adenocarcinoma

[3]As of the 7/30/22 data cutoff [4]As of the 3/15/22 [5]As of 8/15/22

SD = Stable Disease; PR = Partial Response; NE = Nonevaluable; mPFS = median Progresion Free Survival; SAE = Serious Adverse Event

NGM120 in Clinical Development

ADVANCING CLINICAL DEVELOPMENT

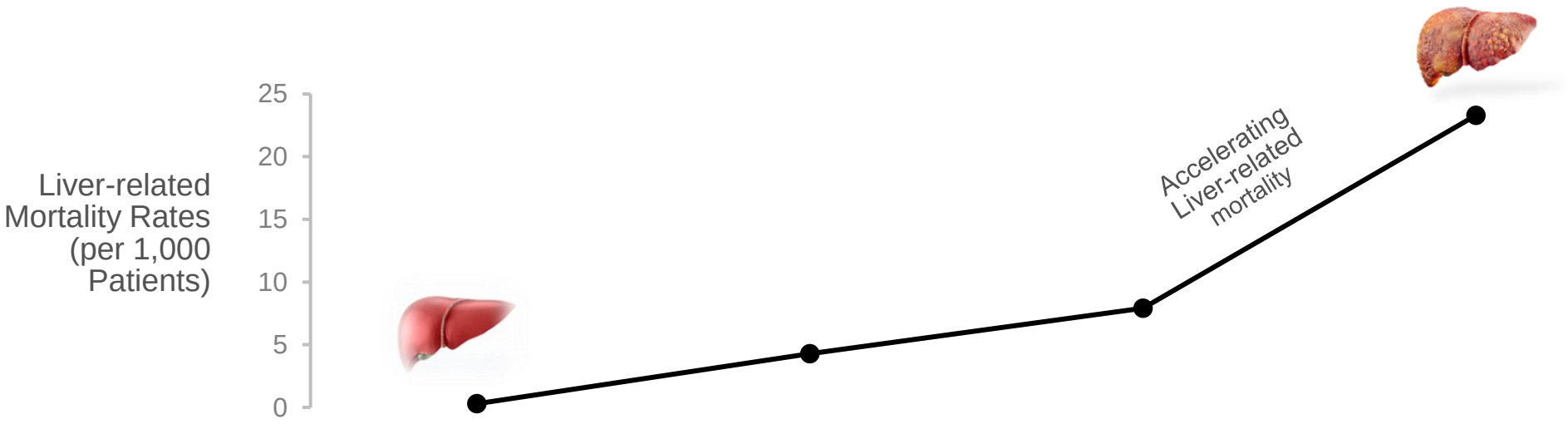
- ✓ **Completed** single (n=48) and multiple (n=44) ascending dose cohorts in first-in-human healthy volunteer studies

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

- ✓ **Phase 2** expansion trial initiated in metastatic pancreatic cancer patients (PINNACLES)
  - Randomized, single-blind (investigator-blinded), multi-center expansion study
  - Patients have background standard of care treatment (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

MK-3655 and aldafermin in NASH

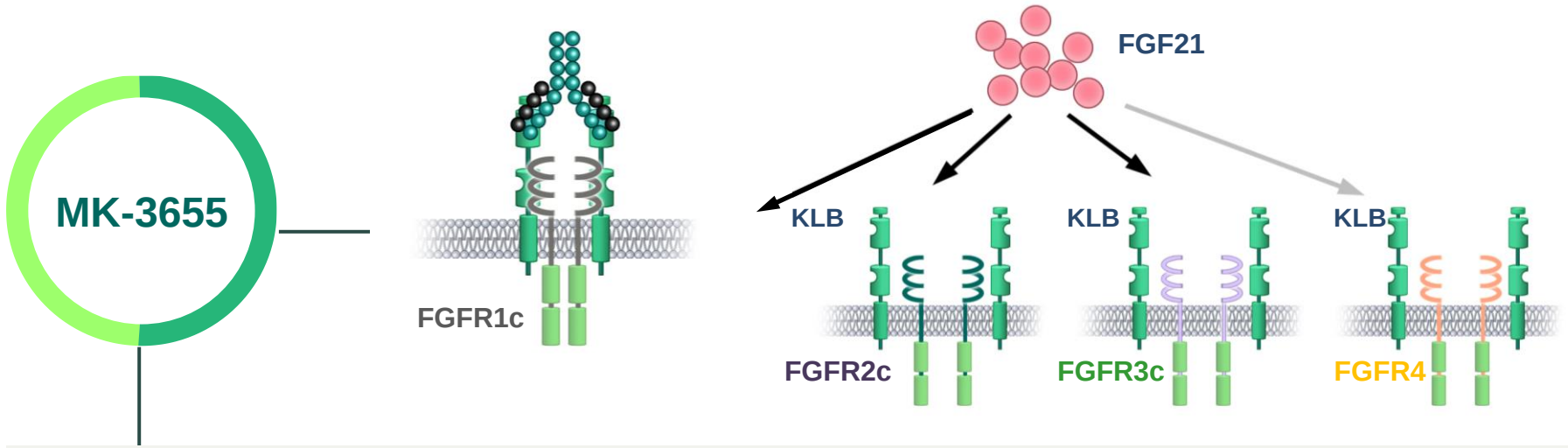
NASH: A Serious and Growing Disease, with Negative Outcomes Linked to Severity of Fibrosis



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
- **Favorable safety profile** demonstrated in Ph1b trial: all AEs mild in severity, no SAEs or Grade 3/4 AEs, no pattern or organ system AEs of note, no significant change in blood pressure
- 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

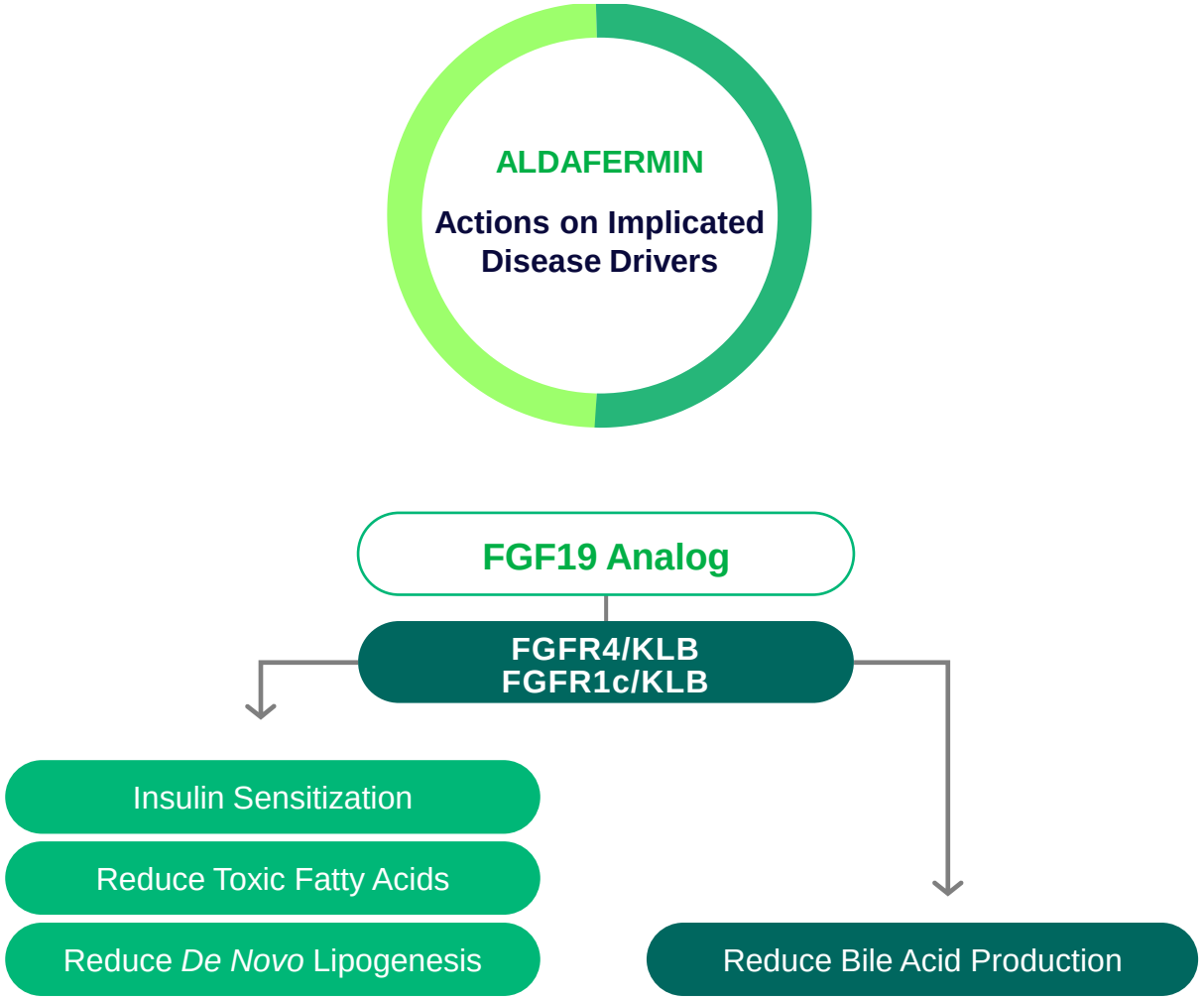
Aldafermin for the Treatment of F4 NASH

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]

Last patient enrolled in January 2022 in our ongoing 48-week Phase 2b ALPINE 4 trial with readout expected in 1H23

Enhanced liver fibrosis (ELF) test will be used as primary endpoint in ALPINE 4 study to understand the profile of aldafermin in patients with F4 NASH and compensated cirrhosis



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

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

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

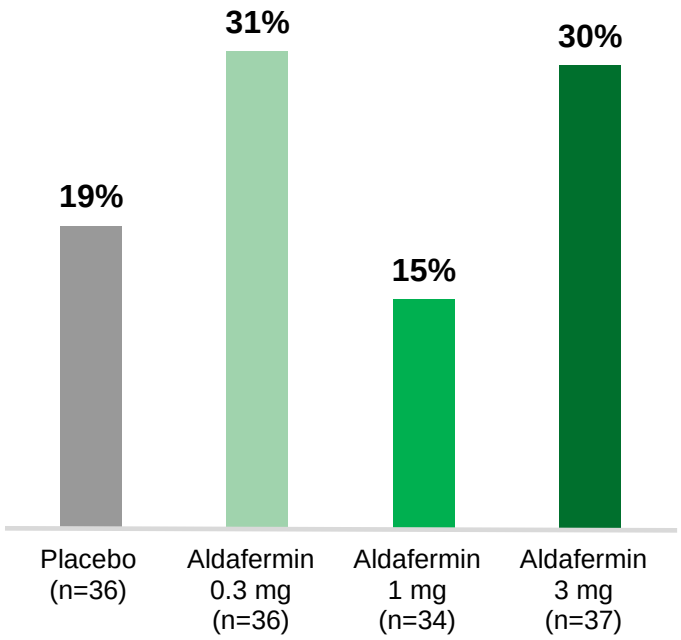
EASL 2019, Oral Abstract, DePaoli et.al.

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)

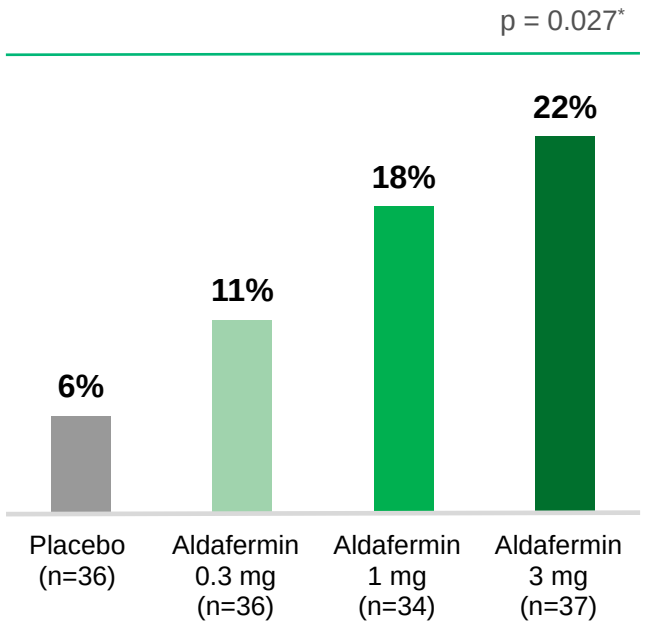


[1] Defined as patients who have an improvement in liver fibrosis by ≥1 stage with no worsening of NASH (no worsening of steatosis, lobular inflammation or hepatocyte ballooning grade) from baseline to W24

NASH Resolution

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

(% of Patients)

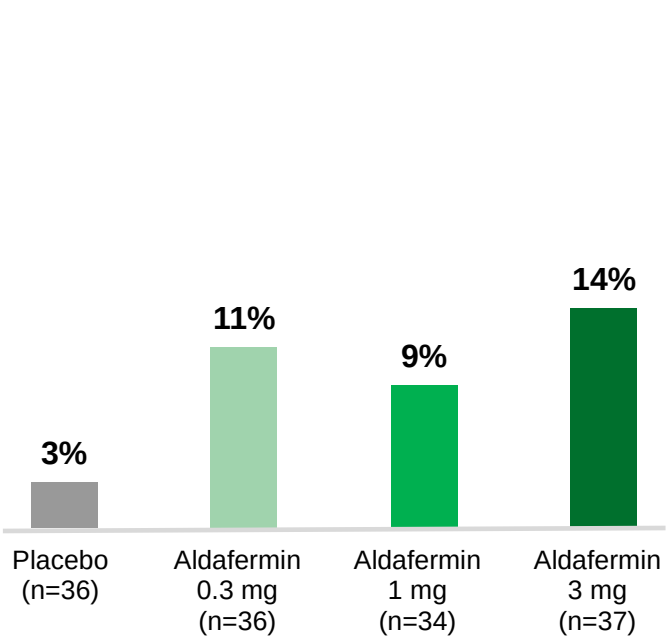


[2] Defined as patients having a NAS score of 0 or 1 for inflammation and 0 for ballooning, with no worsening of fibrosis (no progression of NASH CRN fibrosis stage) from baseline to W24

Fibrosis Improvement and NASH Resolution

Fibrosis Improvement and NASH Resolution[3] at W24

(% of Patients)



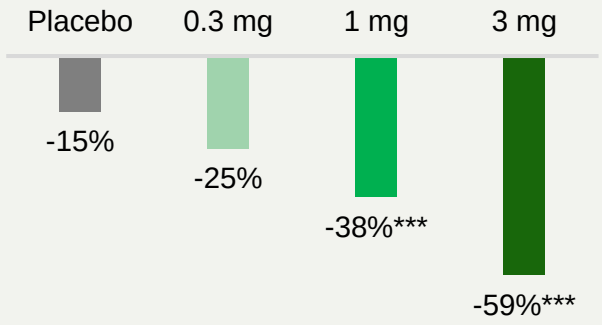
[3] Defined as patients who have an improvement in liver fibrosis by ≥1 stage AND have a NAS score of 0 or 1 for inflammation and 0 for ballooning and no worsening of steatosis at W24

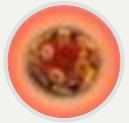
Population with paired biopsies. *Only the 3 mg dose met statistical significance in NASH resolution.

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

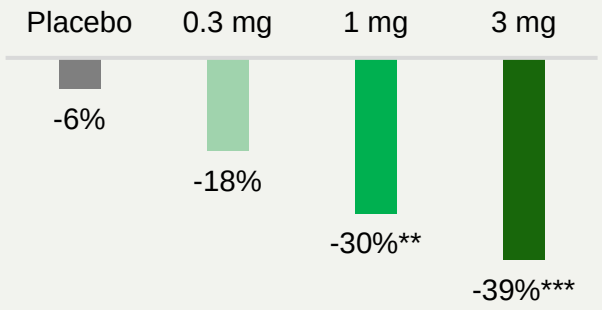
ALPINE 2/3 Topline: Consistent Results Across Non-Invasive Measures

Change in Relative LFC by MRI-PDFF from Baseline

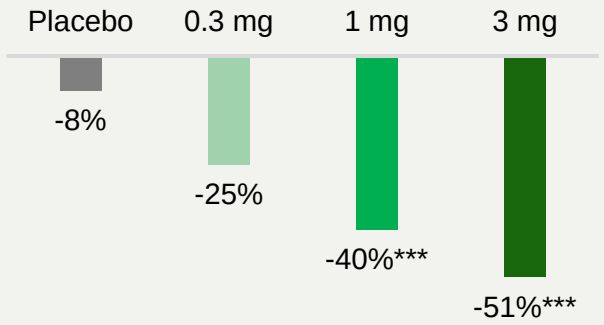


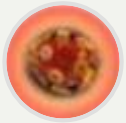


Change in Relative AST from Baseline

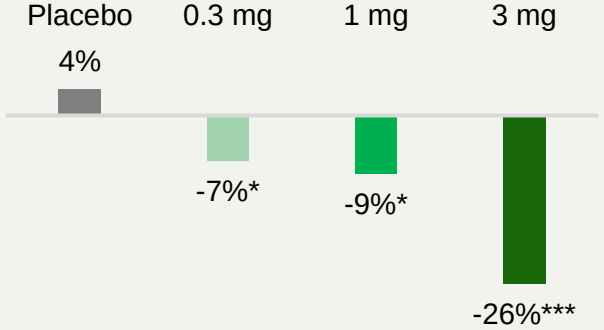


STATISTICALLY SIGNIFICANT
Differences vs Placebo with 1 mg and 3 mg Aldafermin





Change in Relative ALT 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

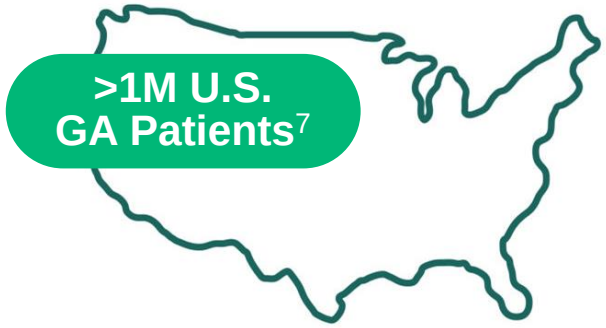
- Aldafermin-induced LDL-C elevations safely and effectively managed by background statin regimen

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

NGM621 in Geographic Atrophy

Geographic Atrophy (GA): The Next Frontier for Potentially Life-Changing Ophthalmology Treatments

- GA is an age-related, progressive retinal degenerative disease associated with **irreversible loss of vision**
- Over time, GA robs patients of their central vision, which can lead to a **loss of independence**, **social isolation**, **depression** and an **increased risk of falls and fractures**[1]
- GA has **no FDA-approved treatments**[2] and is a **leading cause of blindness** in the developed world[3,4]
- GA prevalence is similar to wet AMD in the U.S.; both rise exponentially with age[5,6]



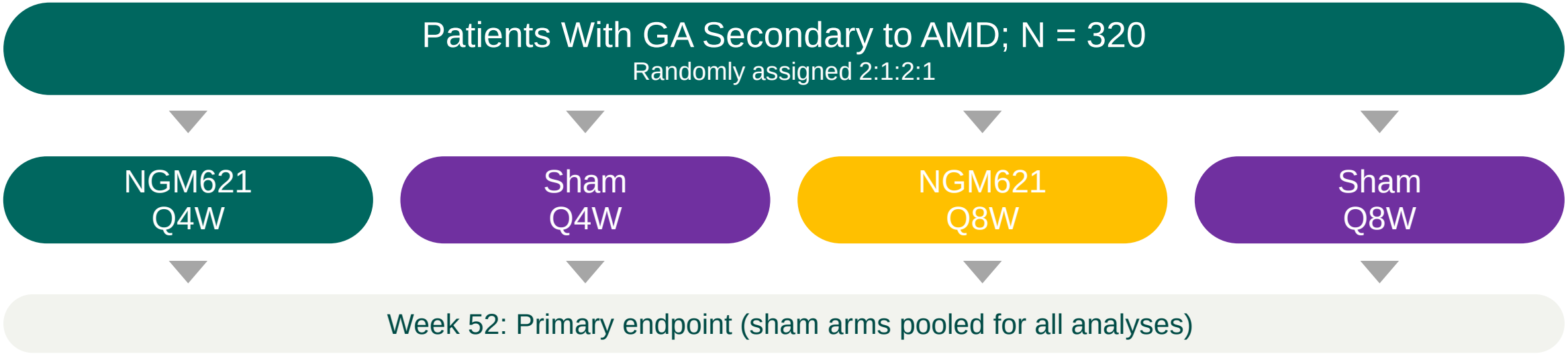
Neurodegenerative Disease of the Retina

No FDA-approved treatments for GA[2]

[1]Chakravarty U et al. Ophthalmology. 2018 Jun;125(6):842-849; [2] 7/19/22 Apellis announced FDA acceptance and priority review of NDA for pegcetacoplan for the treatment of GA
[3]Eye Vis (Lond). 2016; 3: 34; [4]Wong et al. Lancet 2014.;
[5]Fleckenstein, 2018; [6]Friedman, 2004; BrightFocus® Foundation; [7] Boyer DS et al., Retina 2017.

AMD = Advanced Macular Degeneration

Phase 2 CATALINA Study Design



Primary Endpoint	The rate of change in GA lesion area (slope) as measured by fundus autofluorescence over 52 weeks of treatment
Design	Multicenter, randomized, double-masked, sham-controlled, overseen by an independent data safety monitoring board

IVT = intravitreal; Q4 = every 4 weeks; Q8 = every 8 weeks

Summary of Phase 2 CATALINA Trial Topline Findings

1. **Trial did not meet primary endpoint of statistically significant rate of change in GA lesion area using slope analysis over 52 weeks *vs.* sham**

2. **In a pre-specified secondary MMRM analysis, NGM621 Q4W showed a treatment effect at 24 weeks (nominal $p<0.05$) that diminished by 52 weeks**

3. **Additional post-hoc analysis that adjusted for large lesion variability showed potentially encouraging findings warranting further evaluation**

4. **NGM621 showed no evidence of increased CNV conversion *vs.* sham and was well-tolerated with no treatment-related SAEs**

GA = geographic atrophy; CNV = choroidal neovascularization; MMRM = mixed effects model for repeated measures; SAE = serious adverse event

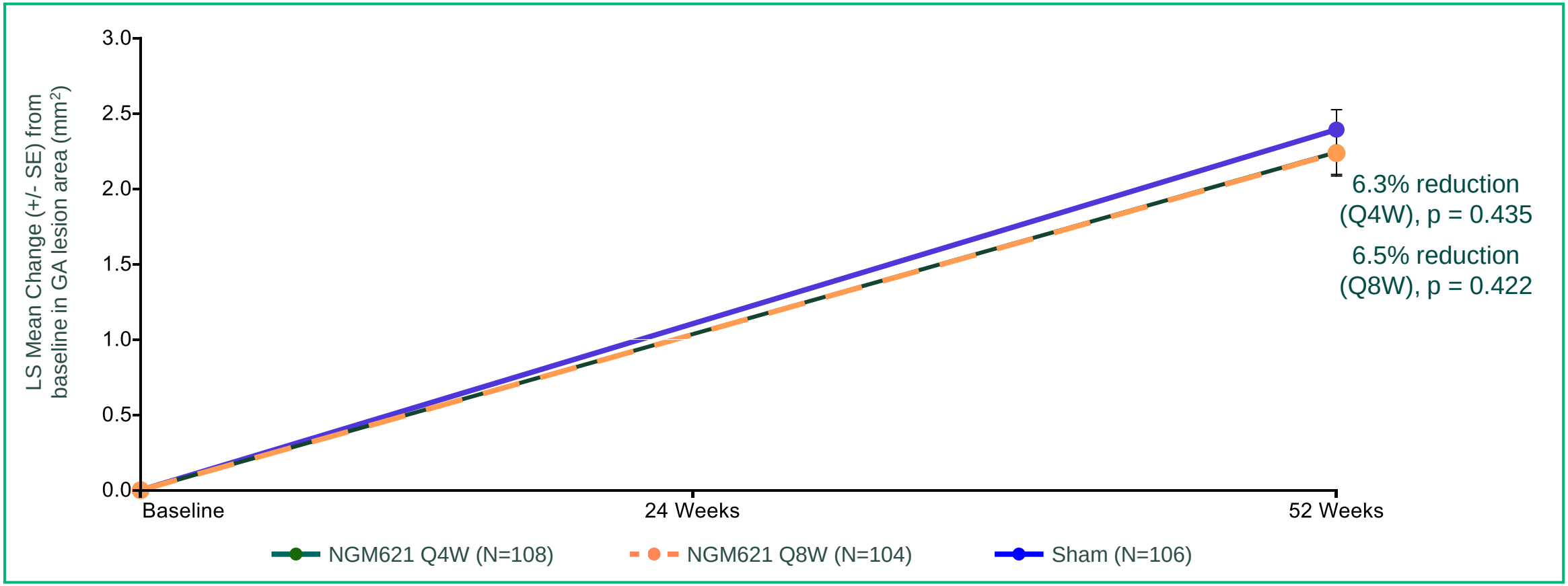
Patient Demographics and Baseline Ocular Characteristics

	Q4 weeks NGM621 15 mg (N = 108)	Q8 weeks NGM621 15 mg (N = 104)	Sham Pooled (N = 106)	Total (N = 318[2])
Age, mean (SD)	78.5 (8.17)	79.1 (7.51)	77.6 (8.42)	78.4 (8.04)
Female, n (%)	67 (62.0)	63 (60.6)	68 (64.2)	198 (62.3)
White, n (%)	107 (99.1)	102 (98.1)	101 (95.3)	310 (97.5)
GA area, mean (SD) mm^2	7.02 (3.964)	7.62 (3.968)	7.75 (4.007)	7.46 (3.980)
Square Root GA area, mean (SD) mm^2	2.56 (0.699)	2.67 (0.708)	2.69 (0.710)	2.64 (0.706)
Foveal Involved GA (%)	62 (57.4%)	65 (62.5%)	66 (62.3%)	193 (60.7%)
Multifocal lesions (%)	58 (53.7%)	56 (53.8%)	51 (48.1%)	165 (51.9%)
BCVA, mean (SD) ETDRS letters	62.8 (14.73)	58.4 (15.33)	60.6 (14.20)	60.6 (14.82)
Snellen Equivalent	20/63	20/80	20/63	20/63
LLD (BCVA - LLVA), mean ETDRS letters	29.9 (16.82)	29.4 (16.60)	27.1 (16.10)	28.8 (16.50)
Bilateral GA, n (%)	99 (91.7)	88 (84.6)	95 (89.6)	282 (88.7)
CNV in Fellow Eye, n (%)[1]	22 (20.4)	17 (16.3)	20 (18.9)	59 (18.6)

[1]Fellow Eye CNV is defined as a history of CNV or neovascular AMD
[2]The mITT analysis set includes all randomized and treated (with at least one study treatment) patients
LLD = low luminance deficit; LLVA = low luminance visual acuity

Primary Endpoint Analysis

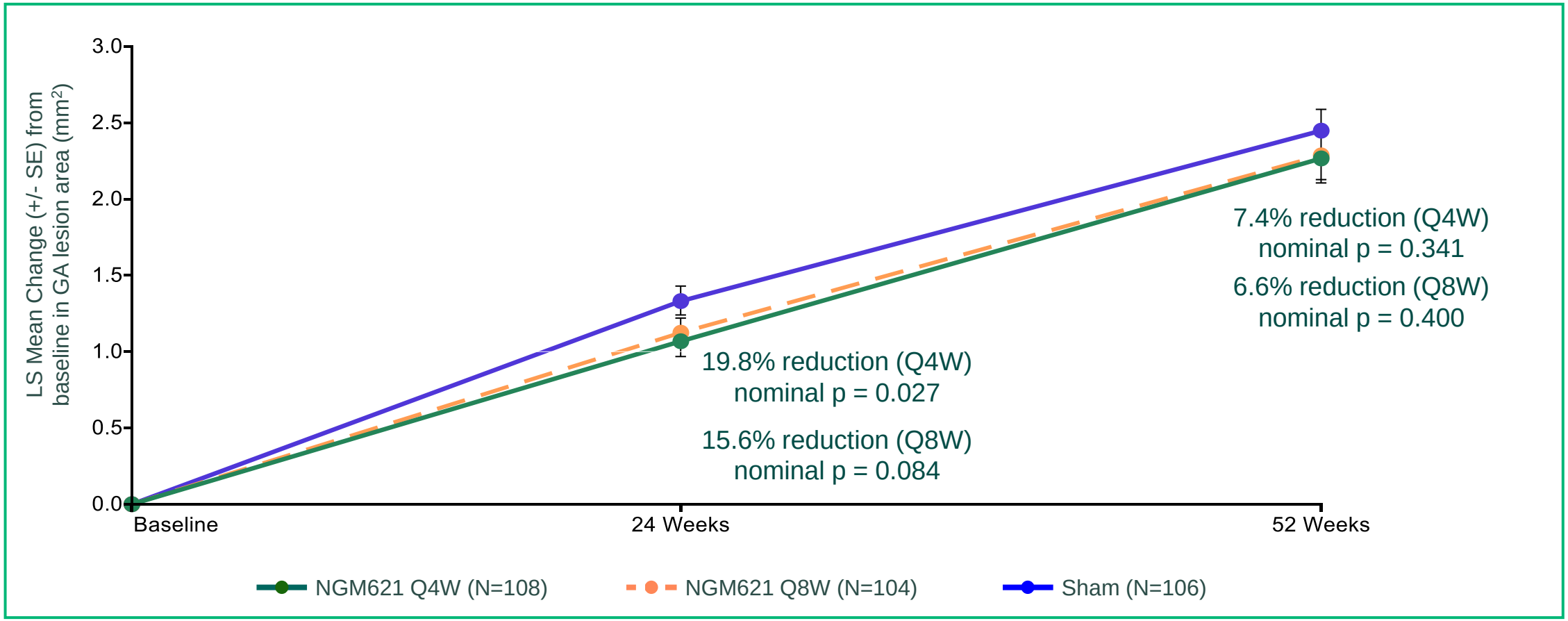
Rate of Change (Slope Analysis) in GA Lesion Area over 52 Weeks



Slope is generated from all available timepoints (Baseline, 24 weeks, 52 weeks)
The Least Square (LS) mean is estimated from a random coefficients linear growth model
The mITT analysis set includes all randomized and treated (with at least one study treatment) patients
SE = standard error

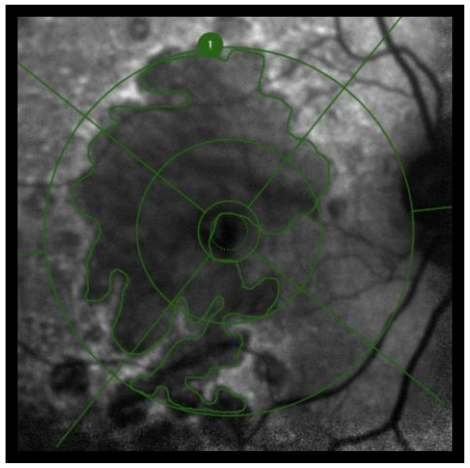
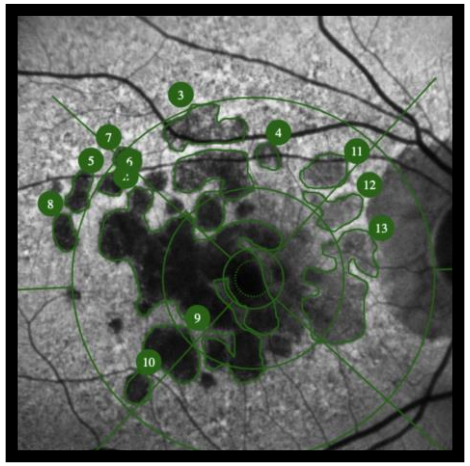
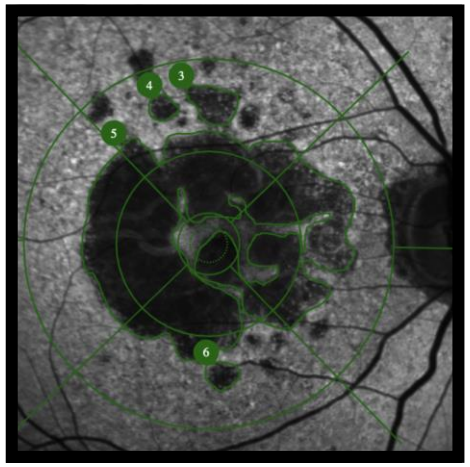
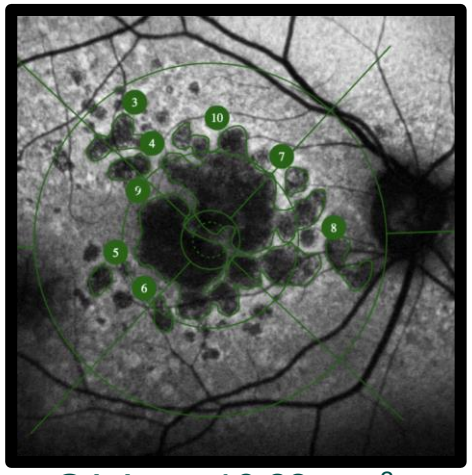
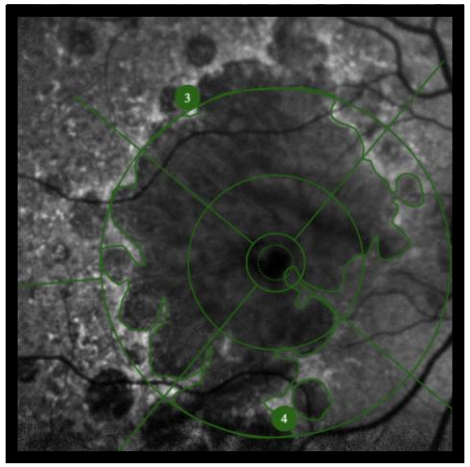
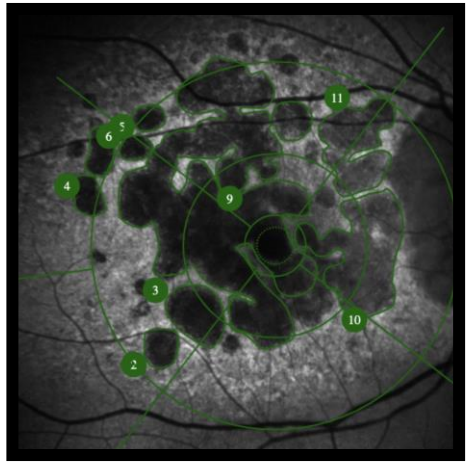
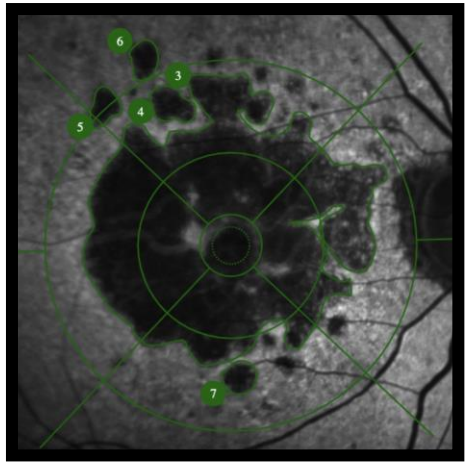
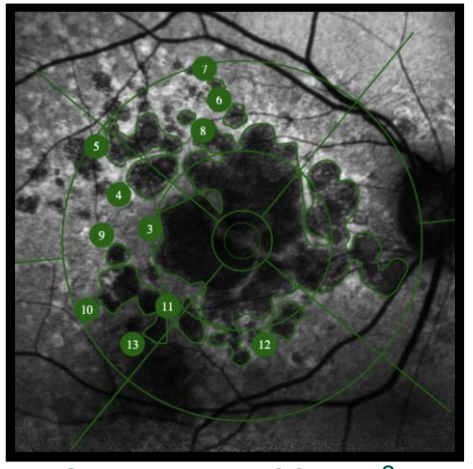
Pre-specified Secondary Analysis (MMRM)

Change from Baseline in GA Lesion Area (MMRM) over 52 Weeks



The Least Square (LS) means is estimated from a mixed model for repeated measures (MMRM)
The mITT analysis set includes all randomized and treated (with at least one study treatment) patients

Representative Baseline Large GA Lesion Cases

	Case 1[1]	Case 2	Case 3	Case 4
Baseline	 GA Area: 19.18 mm^2	 GA Area: 14.50 mm^2	 GA Area: 16.40 mm^2	 GA Area: 10.38 mm^2
Week 52	 GA Area: 28.59 mm^2	 GA Area: 19.27 mm^2	 GA Area: 22.36 mm^2	 GA Area: 15.22 mm^2

[1]Patient is a protocol violation as it did not meet eligibility criteria at baseline with GA >17.5 mm^2

CNV Conversions in the CATALINA Trial

Study Eye CNV Conversions Over 56 Weeks[1]

	NGM621 Q4W (N = 108)	NGM621 Q8W (N = 104)	Sham Pooled (N = 106)
Study Eye CNV Conversions	3 (2.8%)	2 (1.9%)	4 (3.8%)[2]
Reading Center Confirmed CNV Conversions	3 (2.8%)	2 (1.9%)	4 (3.8%)

Fellow eye CNV conversion rate was 4.2% (N = 11)[3] over 56 weeks

[1]Events include preferred terms of CNV and neovascular AMD
[2]There was a coding error for 1 additional CNV patient in the sham arm which was removed from this table
[3]259 = number of patients that did not have medical history of CNV or neovascular AMD in fellow eye at baseline

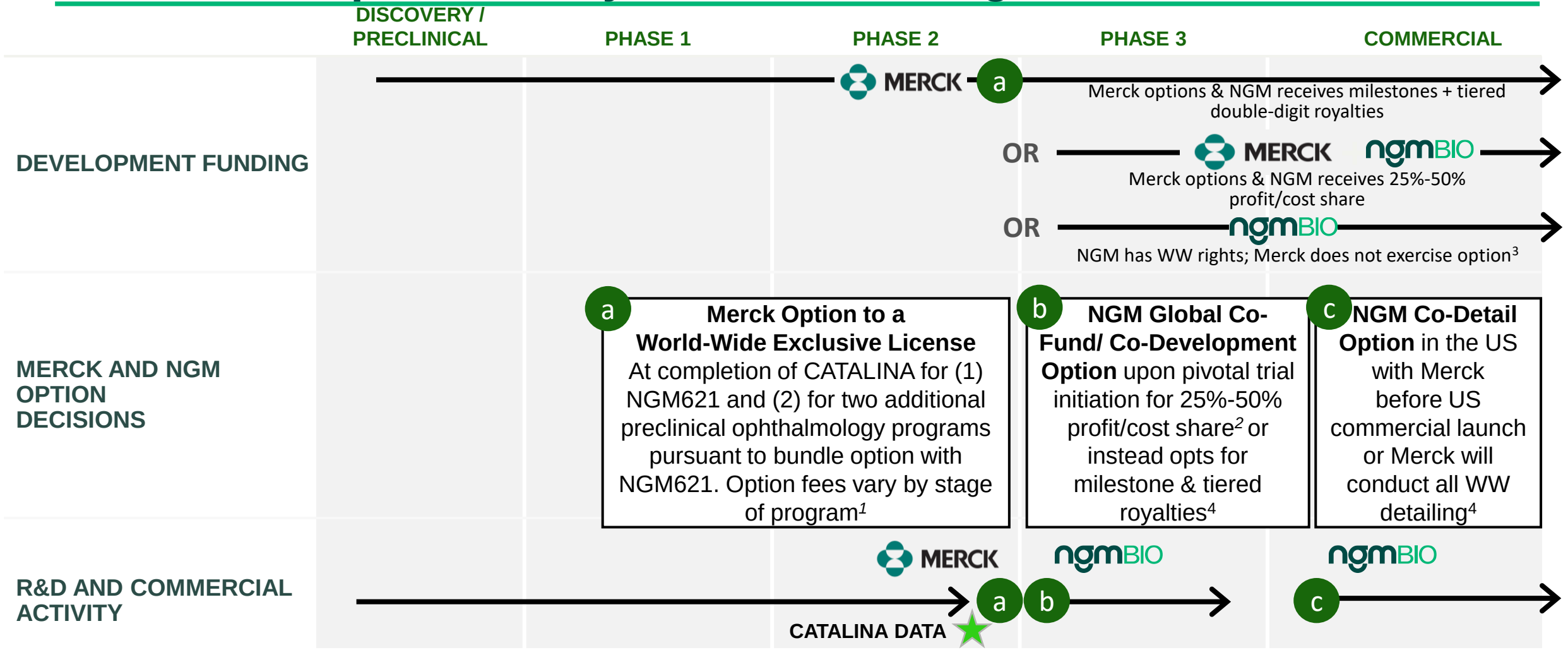
Ocular SAEs in the Study Eye

	NGM621 Q4W (N = 108)	NGM621 Q8W (N = 104)	Sham Pooled (N = 106)
Number of subjects with ≥1 Ocular SAE[1]	8 (7.4%)	8 (7.7%)	3 (2.8%)
Eye Disorders	8 (7.4%)	8 (7.7%)	3 (2.8%)
Visual Acuity Reduced	2 (1.9%)	3 (2.9%)	0
Dry AMD	4 (3.7%)	3 (2.9%)	1 (0.9%)
Visual Impairment	1 (0.9%)	2 (1.9%)	1 (0.9%)
Neovascular AMD	1 (0.9%)	0	1 (0.9%)
Retinal Artery Occlusion	1 (0.9%)	1 (1.0%)	0

No SAEs were deemed related to NGM621 by the Investigator

[1]Protocol defined sight threatening events (a decrease of visual acuity of >30 letters in any post-baseline visit; severe intraocular inflammation; adverse events that require surgical or medical intervention to prevent permanent loss of sight; any decrease to light perception or worse lasting more than an hour) were reported as serious adverse events

NGM621 is Optionable by Merck Following CATALINA Data



[1] *If Merck does not exercise the bundle option at completion of CATALINA, but exercises its option for NGM621 at that time, the option fee will be $20.0M. If Merck does exercise the bundle option at that time, option fee will depend upon the stage of development of one of the two additional ophthalmology programs included in the bundle option.*

[2] *NGM may opt to receive an advance from Merck of up to 25% of development costs per pivotal trial to be paid back after commercialization.*

[3] *If Merck chooses not to option NGM621, NGM will have the world-wide rights to NGM621, will be responsible at its own cost for all further R&D and commercial activity, and will owe low single-digit royalties to Merck.*

[4] *NGM options to global co-fund/co-development and NGM co-detail in the US are only applicable if Merck exercises its option to NGM621 or the ophthalmology bundle.*

For additional information, see Note 5 to NGM's condensed consolidated financial statements included in Part I, Item 1, "Financial Statements" of NGM's Quarterly Report on Form 10-Q for the quarter ended June 30, 2022.

ngmBIO

Financial Overview

3Q22 Financial Results

STATEMENT OF OPERATIONS (In thousands)	QUARTER ENDED September 30, 2022[1] (unaudited)	QUARTER ENDED June 30, 2022 (unaudited)
RELATED PARTY REVENUE	$7,911	$8,293
RESEARCH AND DEVELOPMENT EXPENSES	$46,106	$45,433
GENERAL AND ADMINISTRATIVE EXPENSES	$10,109	$9,927
TOTAL OPERATING EXPENSES	$56,215	$55,360
NET LOSS	**($47,261)**	**($46,519)**

BALANCE SHEET	September 30, 2022 (unaudited)	June 30, 2022 (unaudited)
CASH, CASH EQUIVALENTS AND SHORT-TERM MARKETABLE SECURITIES	**$300.2M**	**$297.8M**

[1] See NGM Bio's quarterly report on Form 10-Q for the quarter ended September 30, 2022 filed with the SEC on November 3, 2022 for more complete financial information