Biology-driven discovery.
Life-changing medicines.

Corporate Overview

January 2022

NASDAQ: NGM

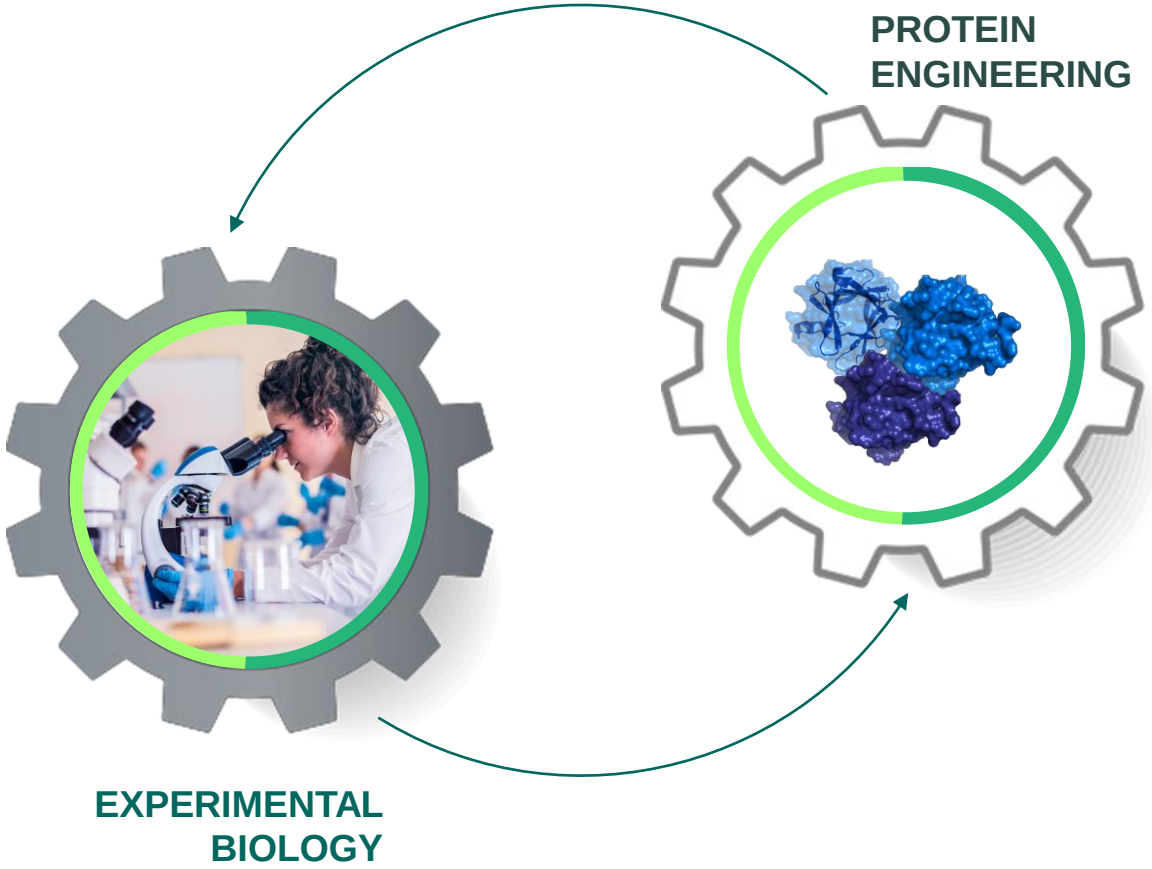
Safe Harbor Statement

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 NGM120, NGM707, NGM831, NGM438, NGM621, MK-3655 and aldafermin; the planned timing of initiation, enrollment, data readouts and results of NGM Bio's clinical trials, including with respect to topline data for NGM621; potential future late-stage development of product candidates in NGM Bio's pipeline, including NGM621 and aldafermin; the potential activity, complementarity, safety, tolerability and efficacy of NGM's product candidates, including the potential of NGM Bio's oncology product candidates to become next-generation treatment options; NGM Bio's belief that myeloid cell reprogramming can be an important additional approach to augment anti-tumor immunity and that its portfolio of product candidates provide multiple opportunities to harness that biology; the design of NGM Bio's and Merck's clinical trials of NGM's product candidates; the preliminary findings in the Phase 1a/1b study of NGM120 providing encouraging initial signals of anti-cancer activity; the preliminary findings in the Phase 1a study of NGM707 and anticipated timing thereof; the availability and anticipated timing of topline data from Phase 2 CATALINA study of NGM621 in patients with geographic atrophy; the continuation of the Phase 2b ALPINE 4 trial of aldafermin; potential activities under NGM's amended collaboration with Merck and the potential receipt of milestone and royalty payments by NGM under the amended collaboration with Merck; the potential roles of regulating the GDF15/GFRAL pathway and ILT2, ILT4, ILT3 and LAIR1 in cancer, the powerful biology of the GDF15 pathway, the potential consequences of ILT2, ILT4, ILT3 and LAIR1 blockade and the opportunity for next generation myeloid checkpoint inhibitors to address limitations of existing immunotherapies; and the potential for NGM621 to have an opportunity for differentiation for the treatment of geographic atrophy and opportunities to achieve category leadership, and its potential for every 8-week dosing; potential option exercises by Merck under NGM Bio's amended collaboration with Merck; NGM Bio's opportunities for value creation and its ability to deliver powerful treatments; NGM Bio's strategy, including its myeloid reprogramming strategy, and potential impact of its portfolio prioritization; NGM Bio's potential near-term catalysts; and any other statements other than statements of historical facts. Because such statements deal with future events and are based on NGM Bio's current plans, objectives, estimates and expectations, they are subject to various significant risks and uncertainties and actual results, performance and achievements and the timing of events could differ materially from those described in or implied by the statements herein. Such risks and uncertainties include, without limitation, those associated with the costly and time-consuming pharmaceutical product development process and the uncertainty of clinical success, including risks related to failure or delays in successfully initiating, enrolling or completing clinical studies, the risk that NGM Bio's ongoing or future clinical studies in humans may show that NGM Bio's product candidates are not tolerable or effective treatments, the risk that preclinical studies or modeling may not be indicative of results in future human clinical trials, the risk that preliminary results from clinical studies may not be predictive of the final results of such studies, the risk that success in earlier-stage clinical studies does not ensure that later clinical trials evaluating NGM Bio's product candidates will generate the same results or otherwise provide adequate data to demonstrate the effectiveness and safety of such product candidates, and the risk that others may discover, develop or commercialize products before or more successfully than NGM Bio, including in NASH and/or geographic atrophy (GA); the ongoing COVID-19 pandemic which has adversely affected, and could materially and adversely affect in the future, NGM Bio's business and operations, including NGM Bio's ability to timely supply, initiate, enroll and complete its ongoing and future clinical trials; the time-consuming and uncertain regulatory approval process, including the risk that NGM Bio or Merck, as applicable, may not receive marketing approvals for any of NGM Bio's product candidates in a timely manner, or at all; seeking and maintaining protection of intellectual property; NGM Bio's reliance on third party manufacturers and delays or problems in the manufacture or testing of product candidates; NGM Bio's dependence on its amended collaboration with Merck for the development and potential commercialization of product candidates falling within the scope of the amended collaboration and its ability to maintain the amended collaboration, including the risk that if Merck were to breach or terminate the amended collaboration or Merck's development funding obligations thereunder, NGM Bio would not obtain all of the anticipated financial and other benefits of the amended collaboration, and the development and/or commercialization of NGM Bio's product candidates falling within the scope of the amended collaboration could be delayed, perhaps substantially; the sufficiency of NGM Bio's cash resources, including to fund development programs that fall outside of the narrower scope of NGM Bio's amended collaboration with Merck, and need for additional capital; and other risks and uncertainties affecting NGM Bio and its research and development programs, including those described under the caption "Risk Factors" and elsewhere in NGM Bio's quarterly report on Form 10-Q for the quarter ended September 30, 2021 filed with the United States Securities and Exchange Commission (SEC) on November 4, 2021 and future filings and reports of NGM Bio with the SEC. The forward-looking statements contained in the following presentation are made only as of the date hereof or as of the dates indicated in the forward-looking statements, even if they are subsequently made available by NGM Bio on its website or otherwise. NGM Bio undertakes no obligation to update or supplement any forward-looking statements after the date hereof, or to update the reasons why actual results may differ or differ materially from those anticipated in the forward-looking statements.

Looking Forward to Multiple Program Milestones in 2022

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

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

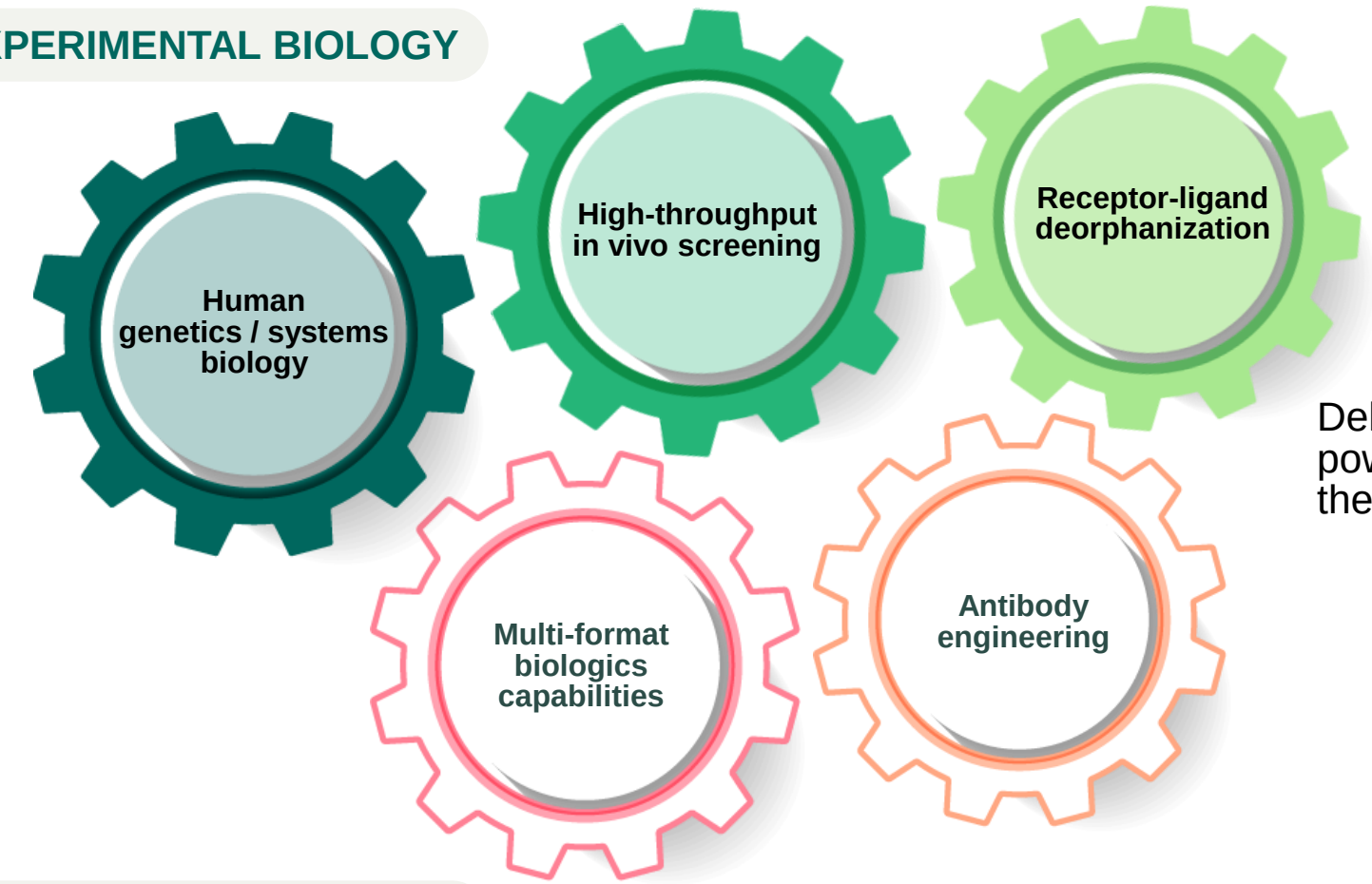


Delivering powerful therapies

EXPANSIVE PIPELINE

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

Reproducible Drug Discovery Process Has Been Applied Successfully Across Biological Frontiers



Delivering powerful therapies

7 Disclosed Programs

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

Our Expansive Pipeline

			Preclinical / Phase 1 / Phase 2 / Phase 3		Rights
ONCOLOGY					
NGM707	ILT2/ILT4 Dual Antagonist Antibody	Advanced Solid Tumors	PHASE 1/2	Initial Ph1a Data Expected in 2H22	Global ngmBIO
NGM831	ILT3 Antagonist Antibody	Advanced Solid Tumors	IND-ENABLING STUDIES	Ph1 Initiation Expected 1Q22	Global ngmBIO
NGM438	LAIR1 Antagonist Antibody	Advanced Solid Tumors	IND-ENABLING STUDIES	Ph1 Initiation Expected 2Q22	Global ngmBIO
NGM120	GFRAL Antagonist Antibody	Cancer & Cancer-related Cachexia	PHASE 1A/1B[1]	Additional Ph1a/1b Data Expected 2H22	Global ngmBIO
		Metastatic Pancreatic Cancer & Cancer-related Cachexia	PHASE 2	Placebo-controlled Expansion Enrolling	Global ngmBIO
RETINAL					
NGM621	Anti-Complement C3 Antibody	Geographic Atrophy	PHASE 2	Topline Data Expected 4Q22	Merck option at PoC; if optioned, NGM to receive milestones + double-digit royalties or up to 50% profit/cost share[2]
LIVER & METABOLIC					
MK-3655 (NGM313)	FGFR1c/KLB Agonist 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[2]
Aldafermin	FGF19 Analog	NASH F4	PHASE 2B	Topline ALPINE 4 Data Expected in 1H23	Global ngmBIO

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

NGM621 in Geographic Atrophy

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

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

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

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

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

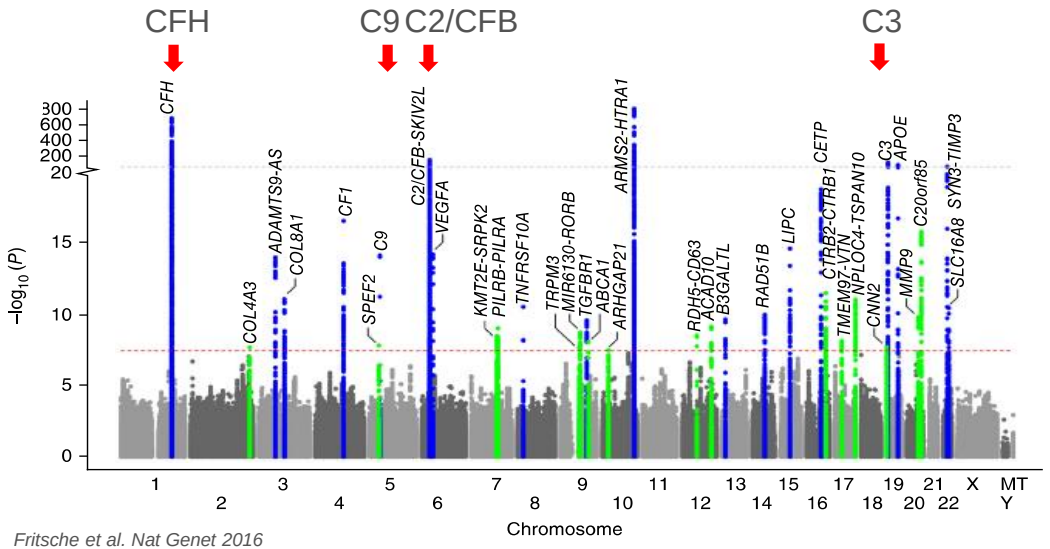
Neurodegenerative Disease of the Retina

No FDA-approved treatments

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

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

Genetic Evidence

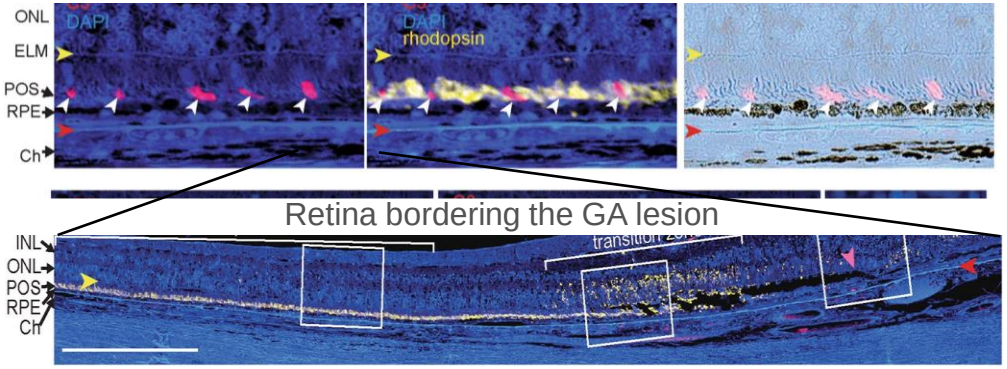


Fritsche et al. Nat Genet 2016

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

Histopathological Evidence

C3 Deposition on Photoreceptors Precedes their Degeneration in Human GA Eyes

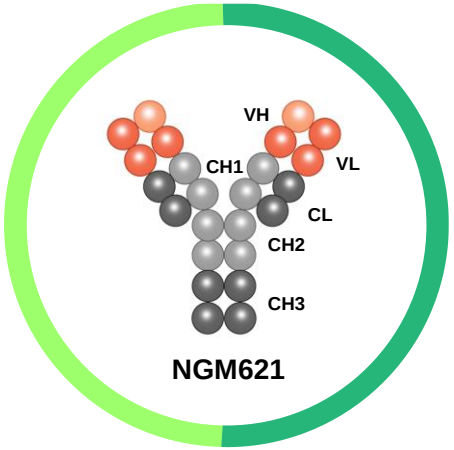


Katschke et al. Sci. Reports 2018

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

AMD = Age-related macular degeneration

NGM621: A Potent Anti-Complement C3 Antibody



NGM621 MOLECULE ATTRIBUTES

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

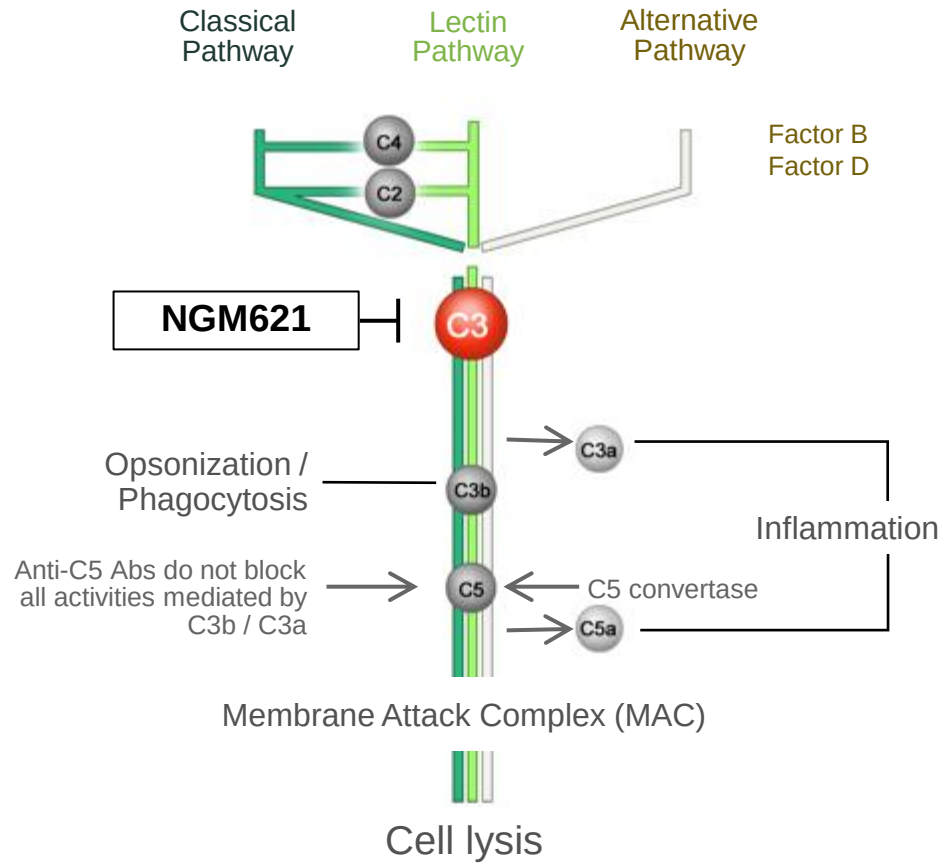
SCIENTIFIC RATIONALE: **NGM621 FOR GEOGRAPHIC ATROPHY**

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

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

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

COMPLEMENT CASCADE



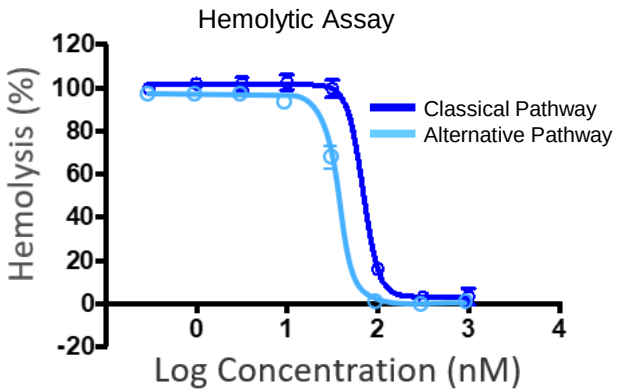
IgG1 = immune globulin G1

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

Potential NGM621 Differentiation in Geographic Atrophy (GA)

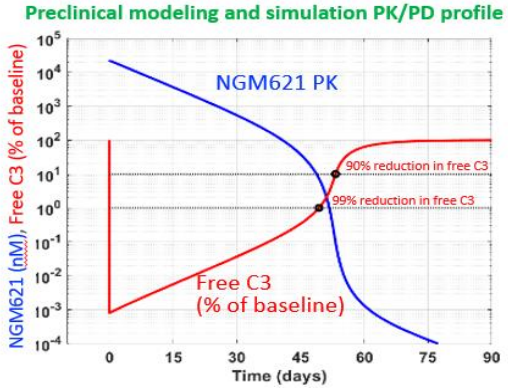
1 Efficacy.

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



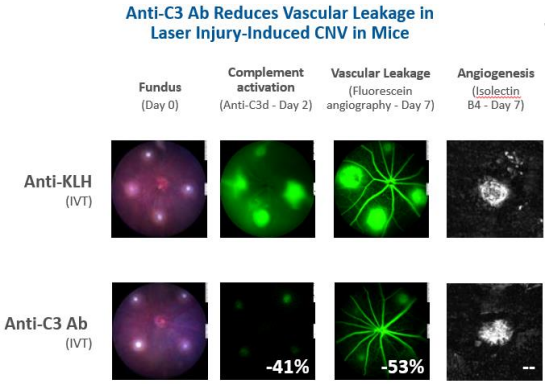
2 Dosing frequency.

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



3 Safety.

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



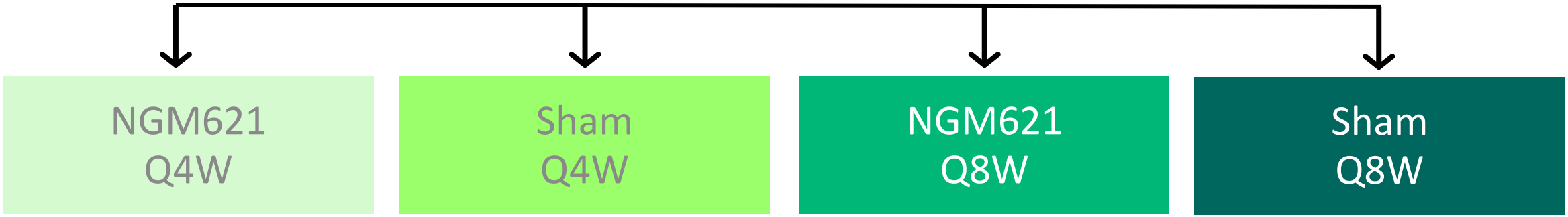
Multiple areas of opportunity for NGM621 to achieve category leadership

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

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

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

Randomly assigned 2:1:2:1



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

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

NGM Bio's Myeloid Reprogramming Strategy to Treat Solid Tumors

NGM707, NGM831, NGM438

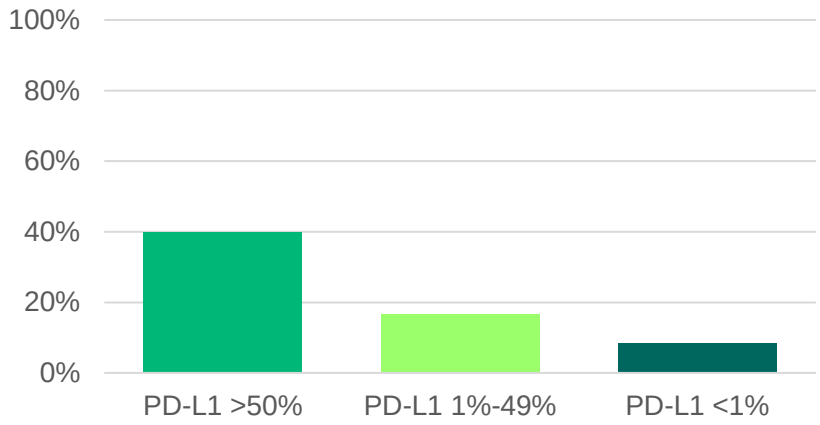
While T Cell Checkpoint Inhibitors Have Advanced the Treatment of Cancer, There is Opportunity to Improve Breadth / Depth of Response

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

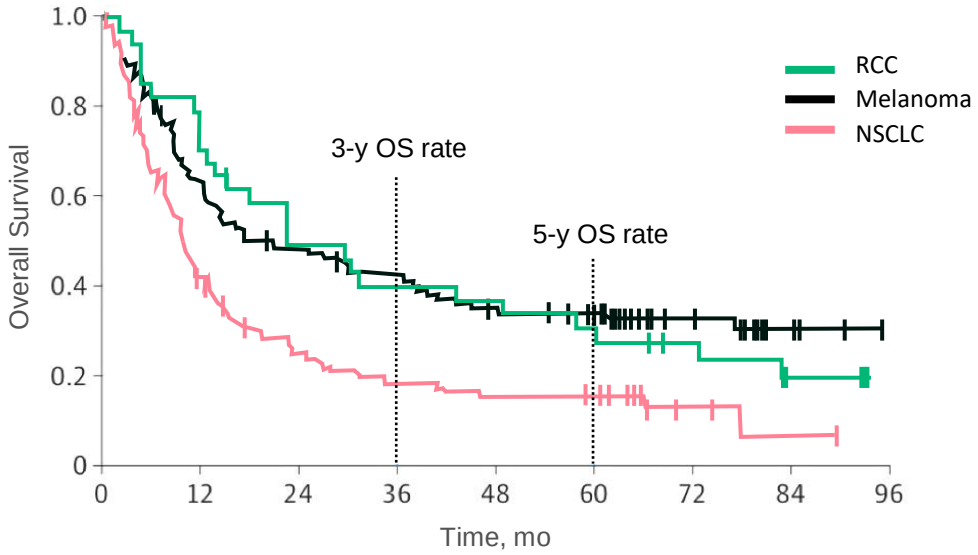
Depth of response: Amongst responders there is opportunity to increase duration of response

PD-1 Response Rate Dependent on PD-L1 Expression

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



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

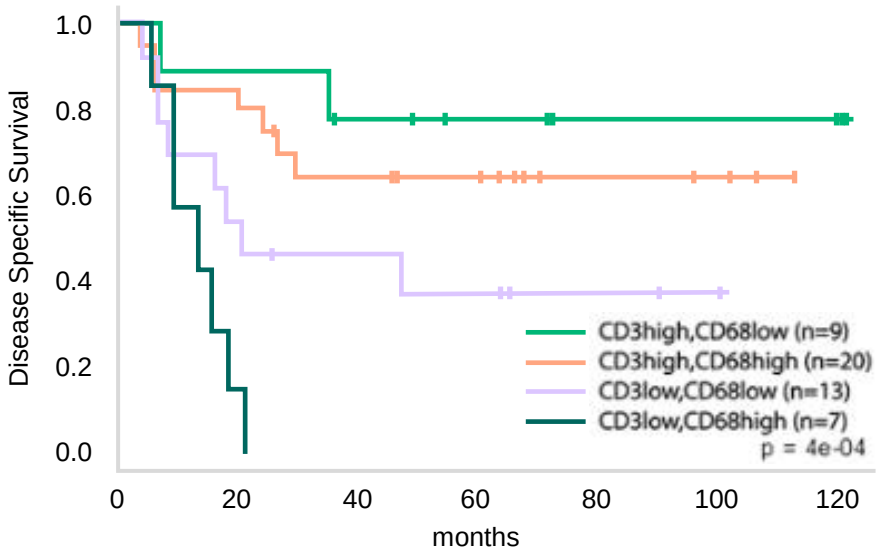


Man et al. *JNCI Spectrum*, 2021; Topalian, Sznol *et al.* - JAMA Oncol 2019
PD-1 = programmed cell death protein 1; PD-L1 = programmed death-ligand 1; ORR = overall response rate; NSCLC = non-small cell lung cancer; RCC = renal cell carcinoma

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

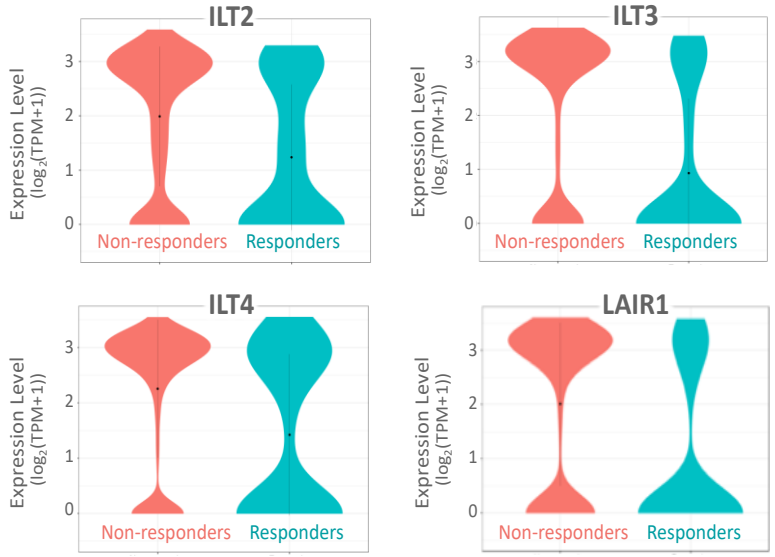
Myeloid-enriched tumors have poor prognosis

High CD68:CD3 ratio is associated with poor survival



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

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

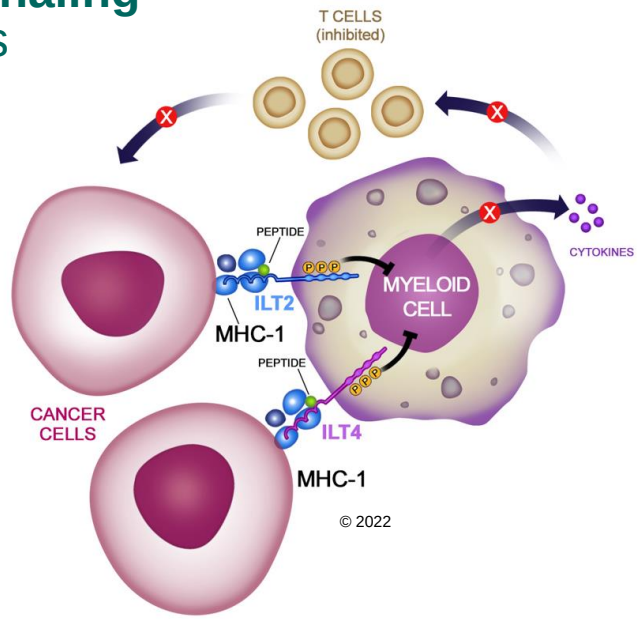


Significant opportunity for next generation myeloid checkpoint inhibitors to address limitations of existing immunotherapies

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

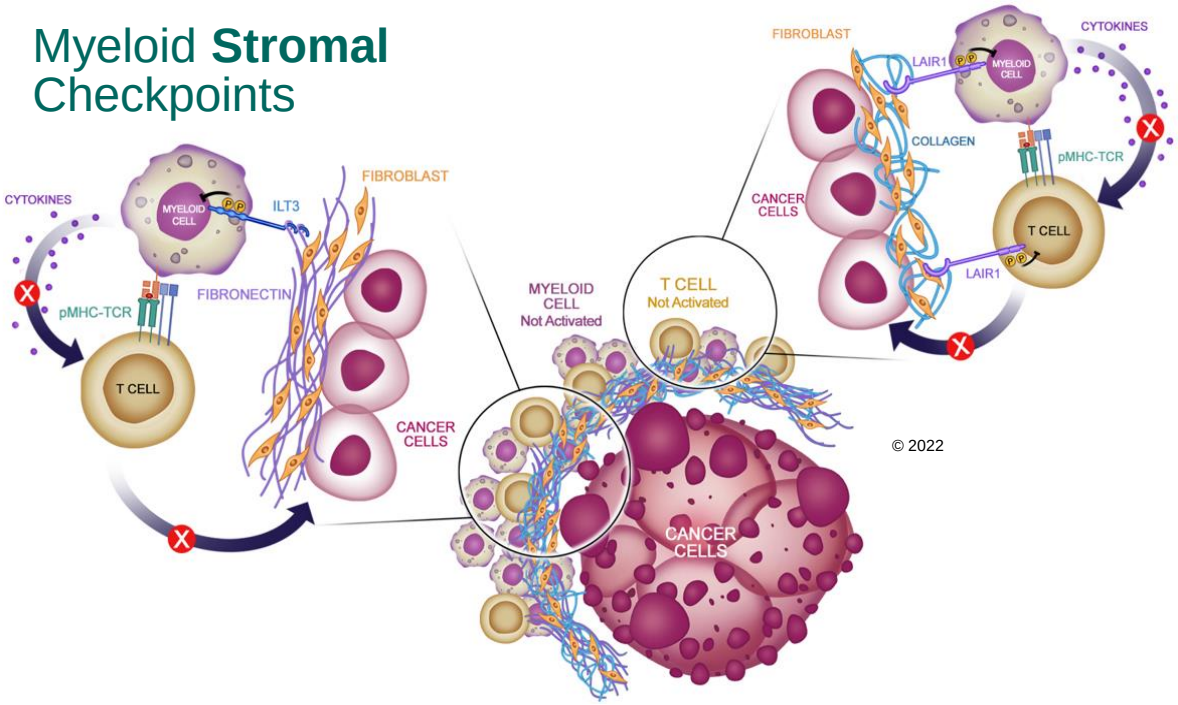
NGM Bio is Targeting Inhibitory Receptors on Myeloid Cells to Attempt to Restore Immune Response Against Tumors

Myeloid **Signaling** Checkpoints



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

Myeloid **Stromal** Checkpoints



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

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

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

Upregulated in certain cancer types[1-5]

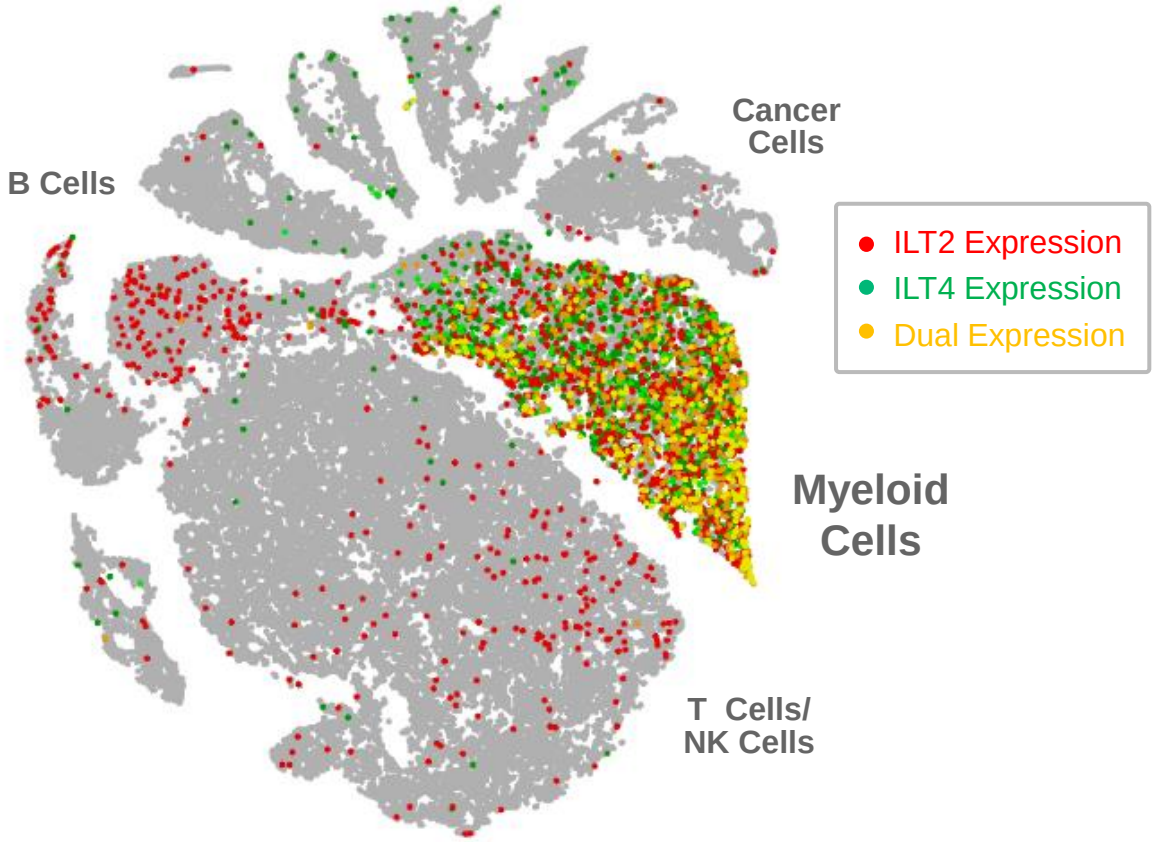
- ILT2 and ILT4 receptors are expressed on myeloid cells (APCs, MDSCs, macrophages, granulocytes) in the tumor microenvironment
- ILT2 additionally exhibits expression on natural killer (NK) cells, B cells and a subset of highly cytolytic T cells

Restrict anti-tumor immunity and promote a tolerogenic state

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

Contribute to T cell checkpoint inhibitor resistance[6]

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



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

Blocking both ILT2 and ILT4 may provide further benefit

1. Tirosh et al., Science, 2016 2. Li et al., Nat Genet, 2017 3. Puram et al., Cell, 2017 4. Azizi et al., Cell, 2018 5. Lambrechts et al., Nat Med, 2018 6. Sade-Feldman et al., Cell, 2018

APC = Antigen-presenting Cell; MDSC = Myeloid-derived Suppressor Cell

NGM707 is a Dual Antagonist Antibody Designed to Inhibit ILT2 and ILT4 that Entered the Clinic in 2021

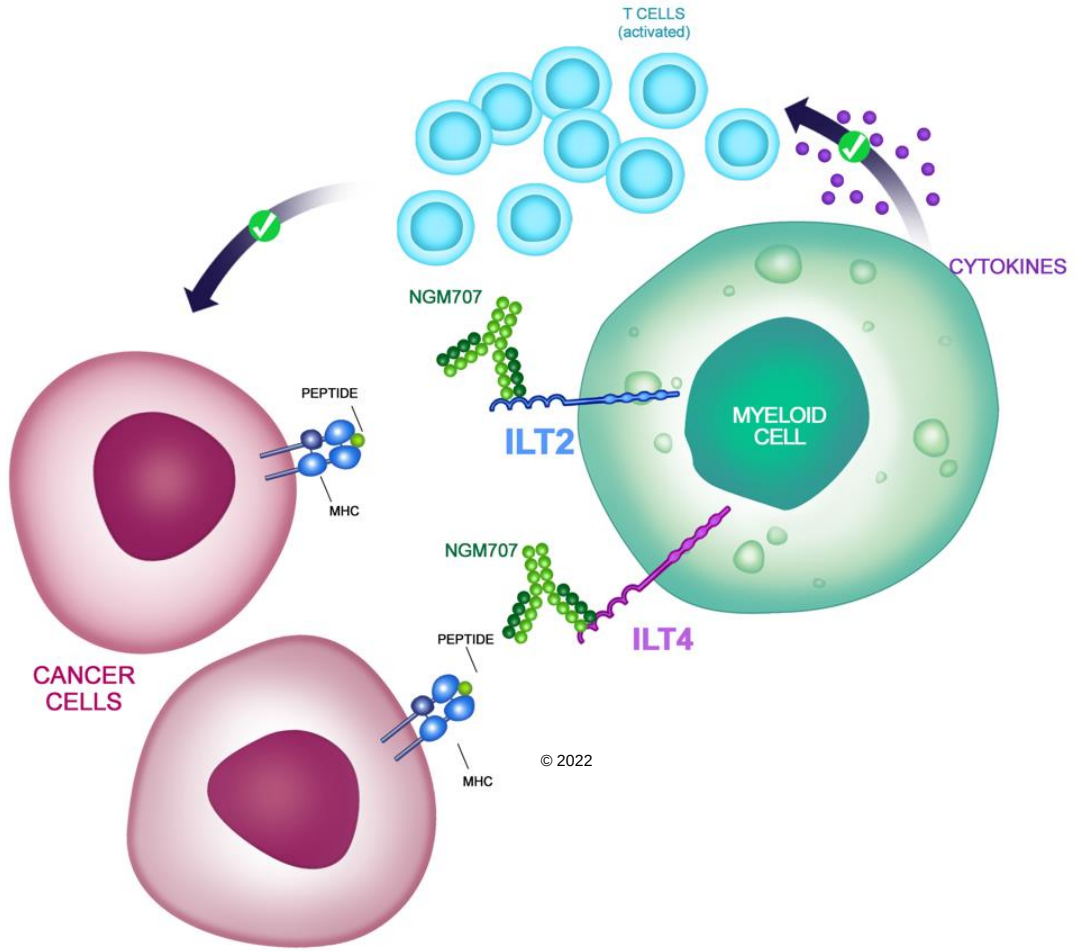
Potent, first-in-class antibody targeting the myeloid-enriched inhibitory receptors ILT2 (LILRB1) and ILT4 (LILRB2)

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

Preclinical studies of NGM707 suggest that:

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

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



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

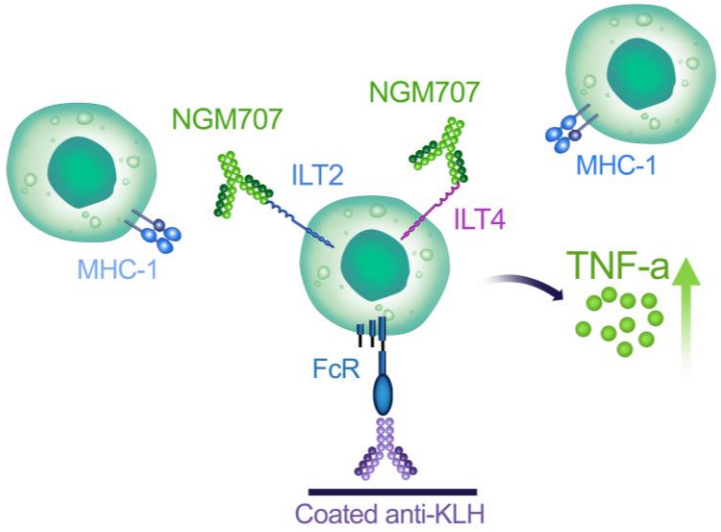
Preclinical Models Suggest That ILT2 and ILT4 Blockade May Act Additively to Enhance Myeloid Cell Activation

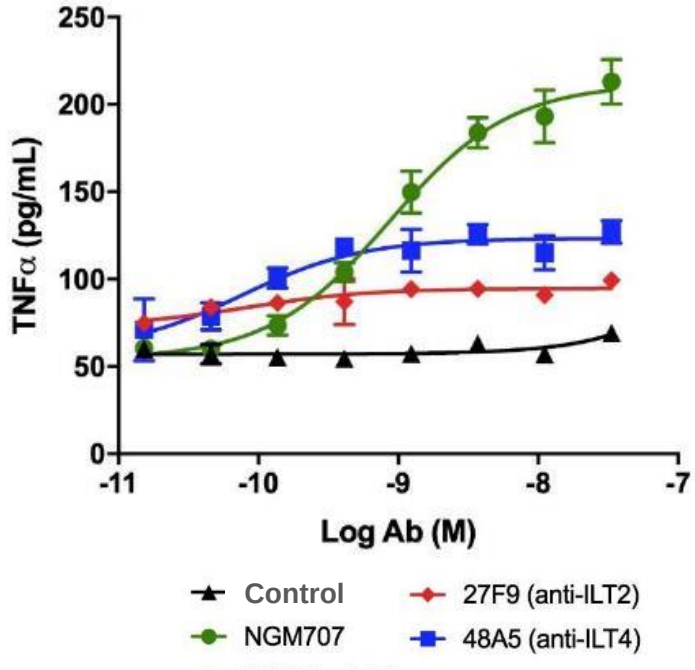
Fc receptors represent key stimulatory receptors on myeloid cells

- 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

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

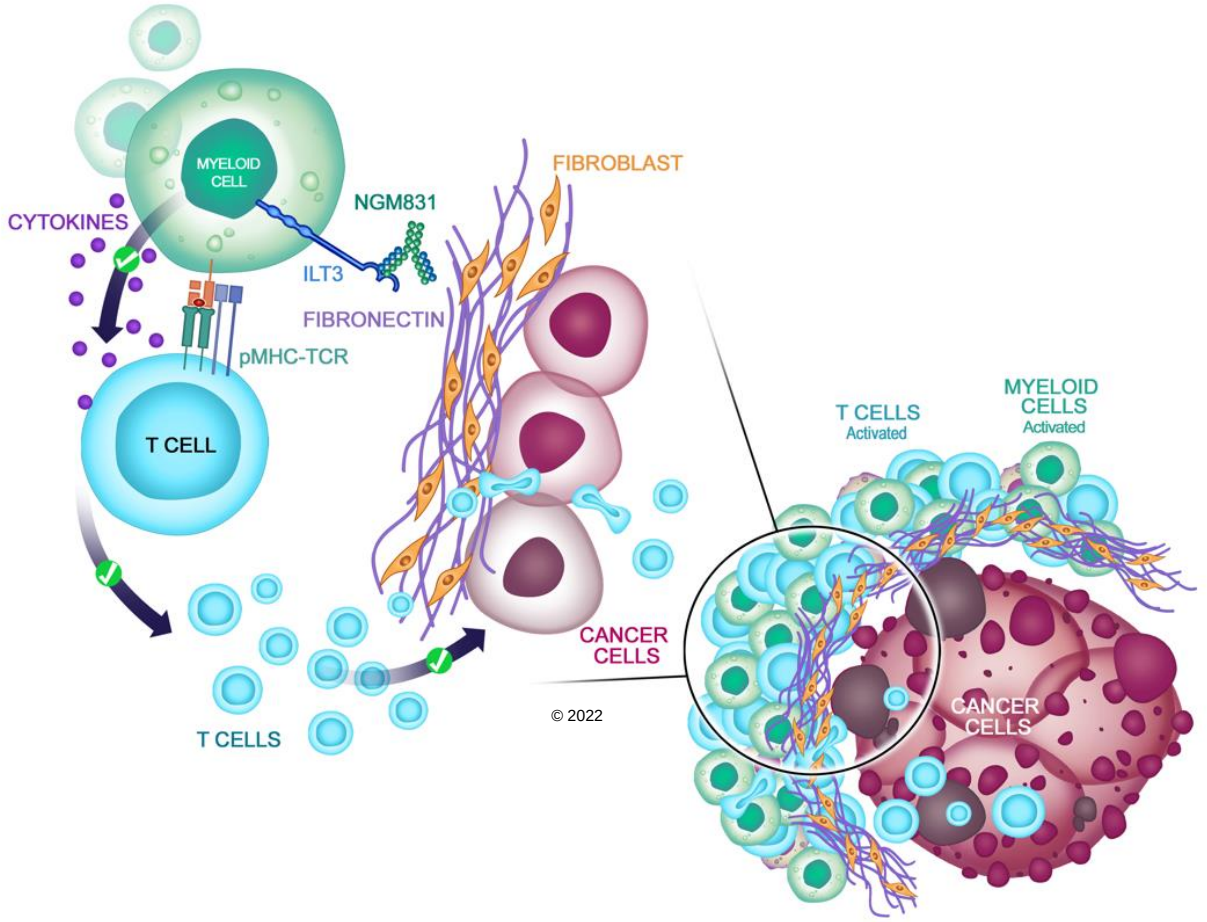
Potent antibody targeting the myeloid-enriched inhibitory receptor ILT3 (LILRB4)

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

Preclinical studies suggest that NGM831 may:

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

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



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

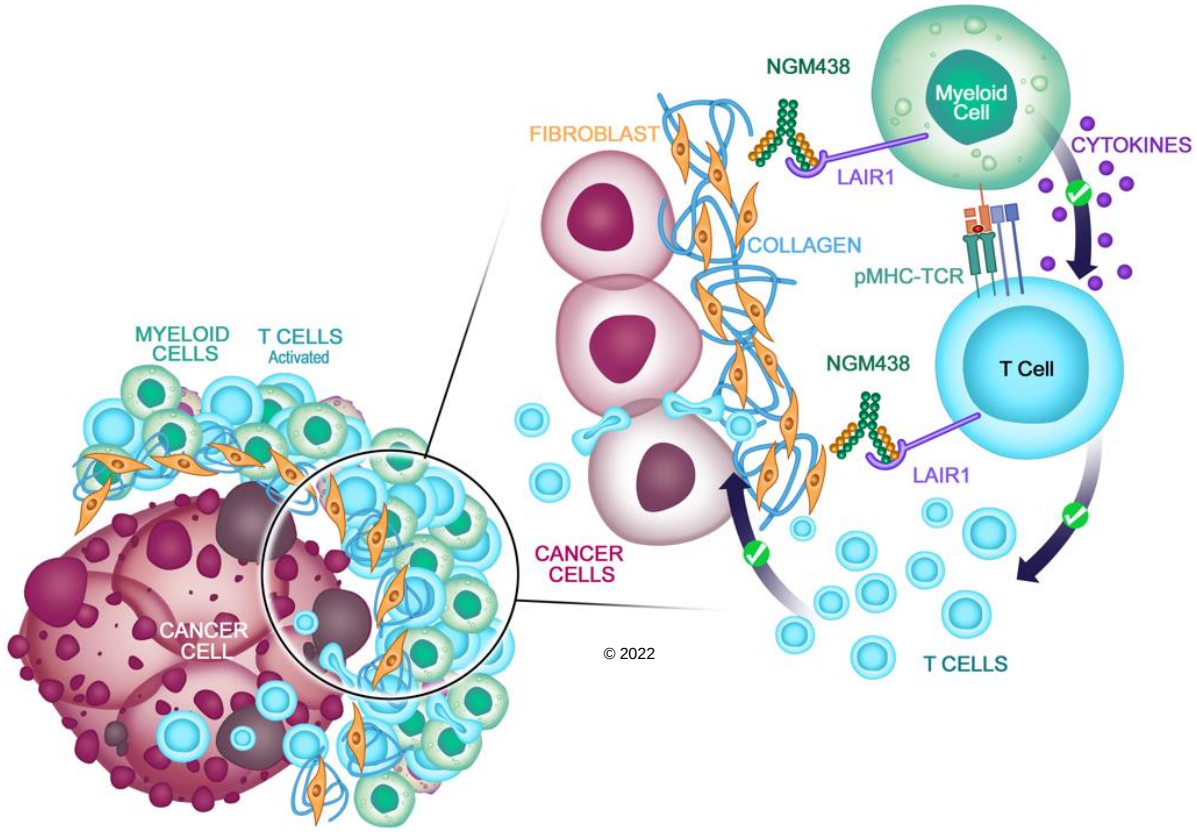
Potent, first-in-class antibody targeting the myeloid-enriched inhibitory receptor LAIR1

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

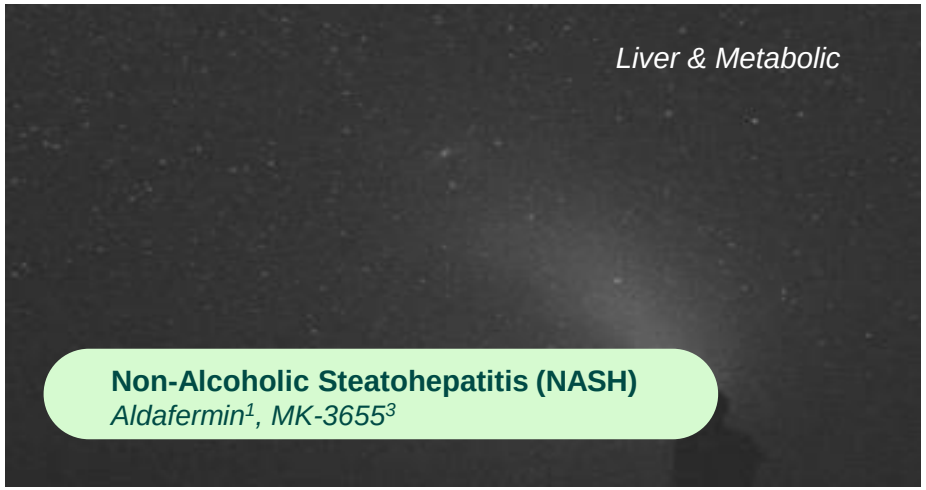
Preclinical studies suggest that NGM438 may:

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

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



NGM Bio: Explorers on the Frontier of Life-Changing Science



All our product candidates have been generated by our in-house discovery engine

~50% Financing to Date from Business Development

[1] NGM Bio has global rights
[2] Merck right to option at proof-of-concept
[3] In 2019, Merck exercised its option to license NGM313 (now MK-3655)

3Q21 and FY20 Financial Results

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

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

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

Looking Forward to Multiple Program Milestones in 2022

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