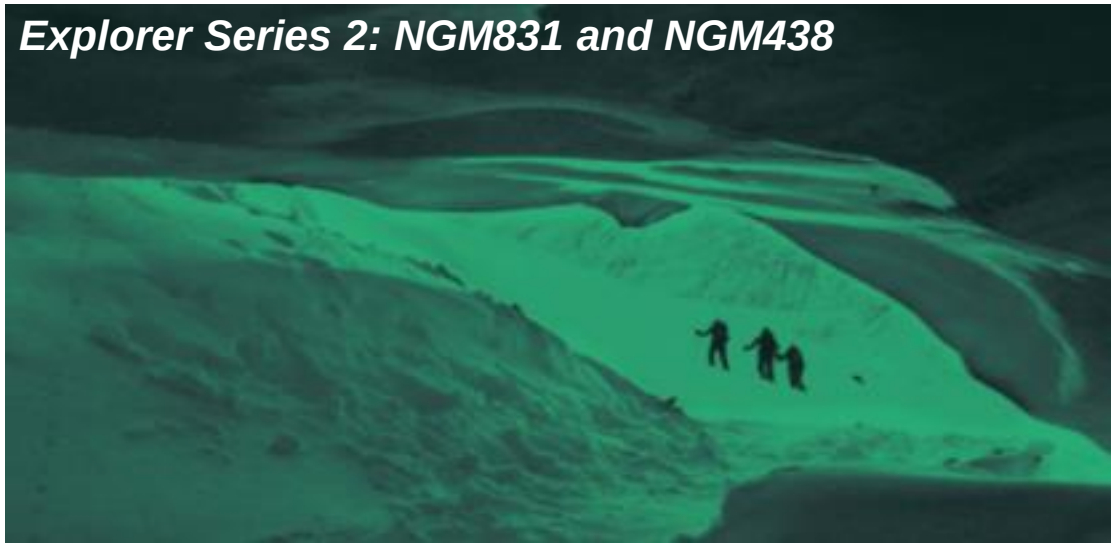
Explorer Series 4: NGM621

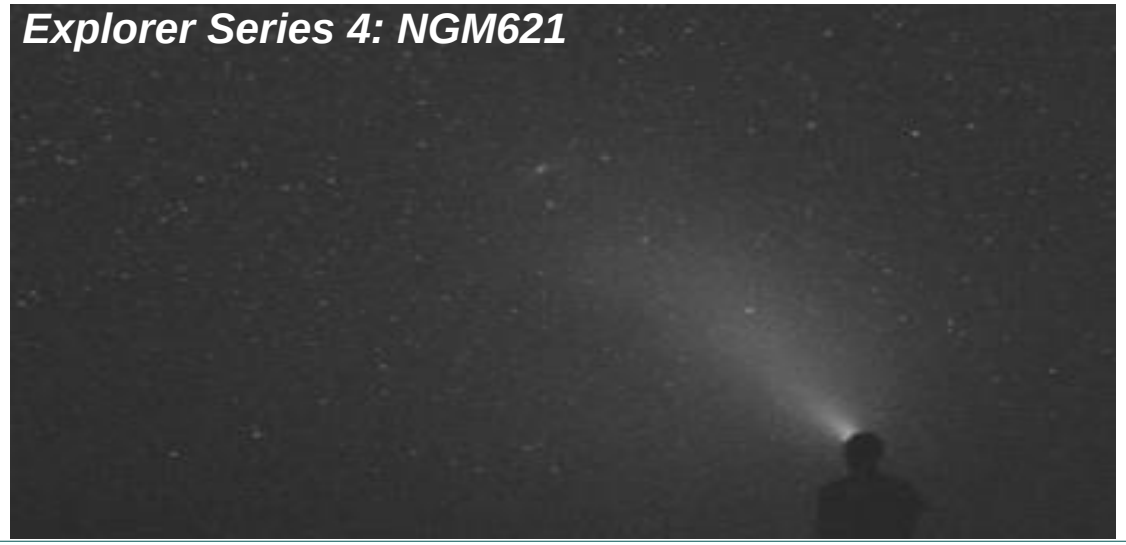
June 29, 2022

Safe Harbor Statement

Statements contained in this presentation and accompanying videos regarding matters that are not historical facts are “forward-looking statements” within the meaning the Private Securities Litigation Reform Act of 1995. Words such as “will,” “may,” “designed for,” “potential,” “plan,” “aim” and similar expressions (as well as other words or expressions referencing future events, conditions or circumstances) are intended to identify forward-looking statements. These statements include, but are not limited to, statements regarding potential indications and applications for, planned and continued development and advancement of, and therapeutic potential of, NGM Bio’s product candidate NGM621; the promise of complement C3 as a target for treating GA; the clinical adoption and patient selection related to the treatment of geographic atrophy (GA); potential differentiation of and opportunities for NGM621 to advance the treatment of GA, including potential dosing of NGM621, potential safety advantages of NGM621 and potential for better efficacy compared to other complement inhibitors; the availability and anticipated timing of the announcement of Phase 2 CATALINA topline results and the possibility that CATALINA may be treated as a pivotal trial; the market potential of NGM621, the opportunity for category leadership and the possible relevance of market experiences for other indications on the GA market; NGM Bio’s collaboration with Merck; expected program milestones in 2022 for product candidates in NGM Bio’s pipeline, including NGM621, NGM707, NGM831, NGM438, NGM120, aldafermin and MK-3566; 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; 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 March 31, 2021 filed with the United States Securities and Exchange Commission (SEC) on May 5, 2022 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.

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





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

Explorer Series 4:

NGM621, a Monoclonal Antibody Product Candidate Engineered to Potently Inhibit Complement C3 for Patients with Geographic Atrophy

1. Introduction to Geographic Atrophy
2. Complement Hypothesis and NGM621 Molecular Attributes
3. Presentation from Dr. Charles Wykoff

Retina Consultants of Texas™

RETINA CONSULTANTS OF AMERICA

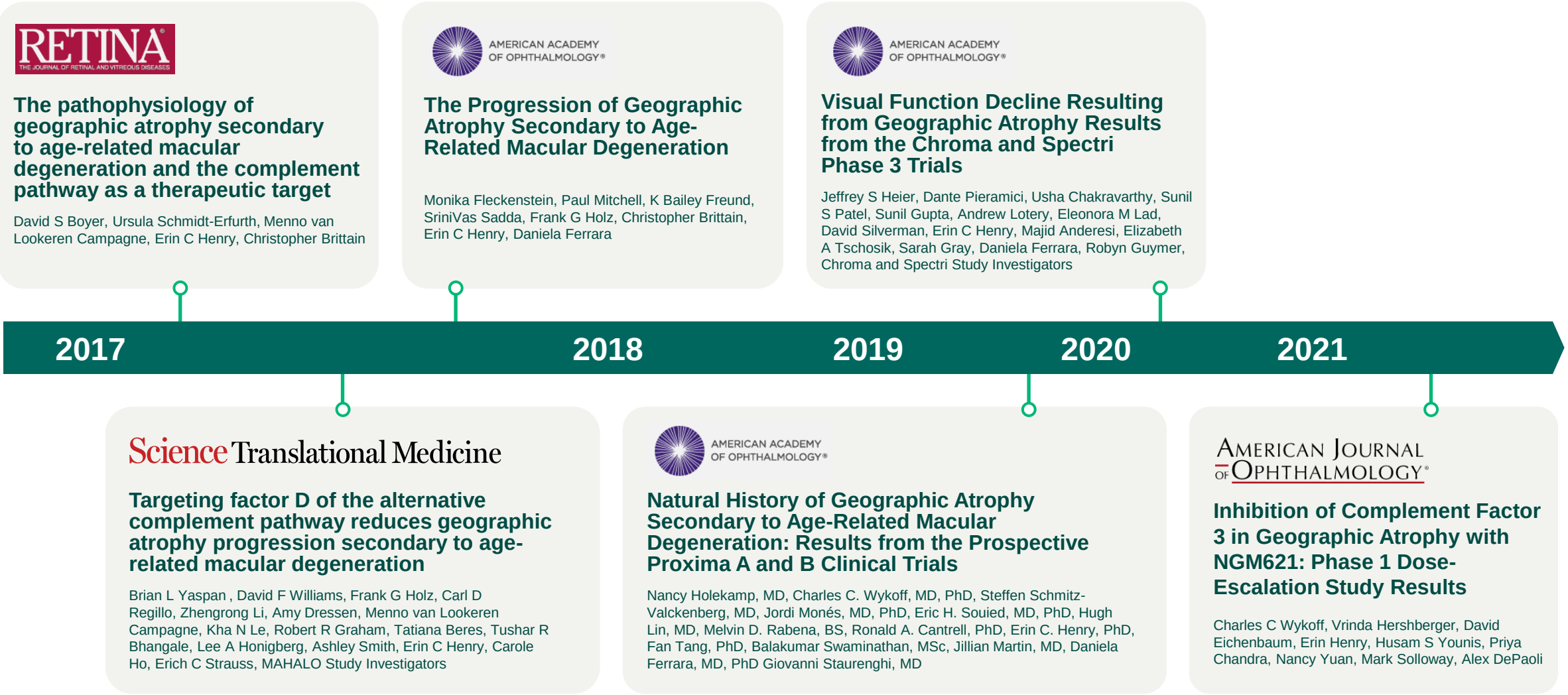
4. NGM621, a Differentiated Complement Inhibitor
5. Concluding Remarks
6. Q&A Session

Introduction To Geographic Atrophy

Erin C. Henry, Ph.D.

Head of Ophthalmology Clinical Development

My Journey With Geographic Atrophy (GA) Research



RETINA
THE JOURNAL OF RETINAL AND VITREOUS DISEASES

The pathophysiology of geographic atrophy secondary to age-related macular degeneration and the complement pathway as a therapeutic target

David S Boyer, Ursula Schmidt-Erfurth, Menno van Lookeren Campagne, Erin C Henry, Christopher Brittain

AMERICAN ACADEMY OF OPHTHALMOLOGY®

The Progression of Geographic Atrophy Secondary to Age-Related Macular Degeneration

Monika Fleckenstein, Paul Mitchell, K Bailey Freund, SriniVas Sadda, Frank G Holz, Christopher Brittain, Erin C Henry, Daniela Ferrara

AMERICAN ACADEMY OF OPHTHALMOLOGY®

Visual Function Decline Resulting from Geographic Atrophy Results from the Chroma and Spectri Phase 3 Trials

Jeffrey S Heier, Dante Pieramici, Usha Chakravarthy, Sunil S Patel, Sunil Gupta, Andrew Lotery, Eleonora M Lad, David Silverman, Erin C Henry, Majid Anderesi, Elizabeth A Tschosik, Sarah Gray, Daniela Ferrara, Robyn Guymer, Chroma and Spectri Study Investigators

Science Translational Medicine

Targeting factor D of the alternative complement pathway reduces geographic atrophy progression secondary to age-related macular degeneration

Brian L Yaspan , David F Williams, Frank G Holz, Carl D Regillo, Zhengrong Li, Amy Dressen, Menno van Lookeren Campagne, Kha N Le, Robert R Graham, Tatiana Beres, Tushar R Bhangale, Lee A Honigberg, Ashley Smith, Erin C Henry, Carole Ho, Erich C Strauss, MAHALO Study Investigators

AMERICAN ACADEMY OF OPHTHALMOLOGY®

Natural History of Geographic Atrophy Secondary to Age-Related Macular Degeneration: Results from the Prospective Proxima A and B Clinical Trials

Nancy Holekamp, MD, Charles C. Wykoff, MD, PhD, Steffen Schmitz-Valckenberg, MD, Jordi Monés, MD, PhD, Eric H. Souied, MD, PhD, Hugh Lin, MD, Melvin D. Rabena, BS, Ronald A. Cantrell, PhD, Erin C. Henry, PhD, Fan Tang, PhD, Balakumar Swaminathan, MSc, Jillian Martin, MD, Daniela Ferrara, MD, PhD Giovanni Staurenghi, MD

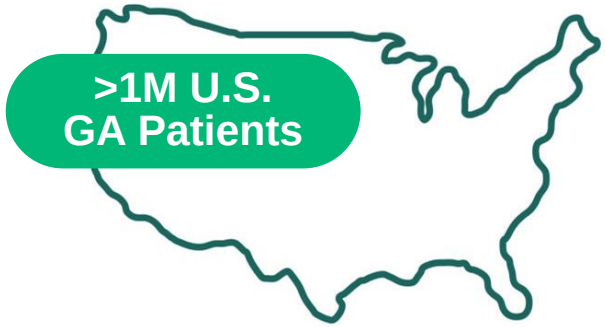
AMERICAN JOURNAL OF OPHTHALMOLOGY®

Inhibition of Complement Factor 3 in Geographic Atrophy with NGM621: Phase 1 Dose-Escalation Study Results

Charles C Wykoff, Vrinda Hershberger, David Eichenbaum, Erin Henry, Husam S Younis, Priya Chandra, Nancy Yuan, Mark Solloway, Alex DePaoli

GA: the Next Frontier for 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 approved treatments** and is a **leading cause of blindness** in the developed world[2,3]
- GA prevalence is similar to wet AMD in the U.S.; both rise exponentially with age[4,5]



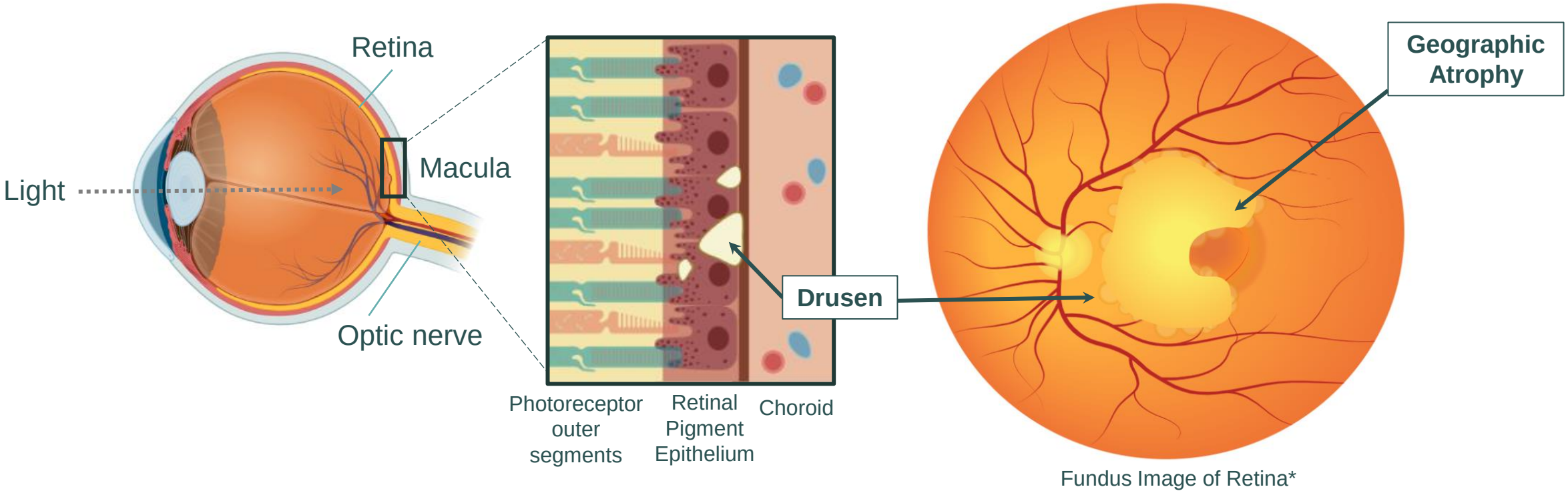
Neurodegenerative Disease of the Retina

No FDA-approved treatments

[1]Chakravarty U et al. Ophthalmology. 2018 Jun;125(6):842-849;
[2]As of 2016. Eye Vis (Lond). 2016; 3: 34; [3]Wong et al. NEJM 2014. https://www.acms.org/2019/03/age-related-macular-degeneration-in-2019;
[4]Fleckenstein, 2018; [5]Friedman, 2004; BrightFocus® Foundation;

Age-Related Macular Degeneration (AMD) Overview

AMD is a disease effecting the macula – the region of the retina responsible for fine, central vision – and can develop into advanced forms including Geographic Atrophy



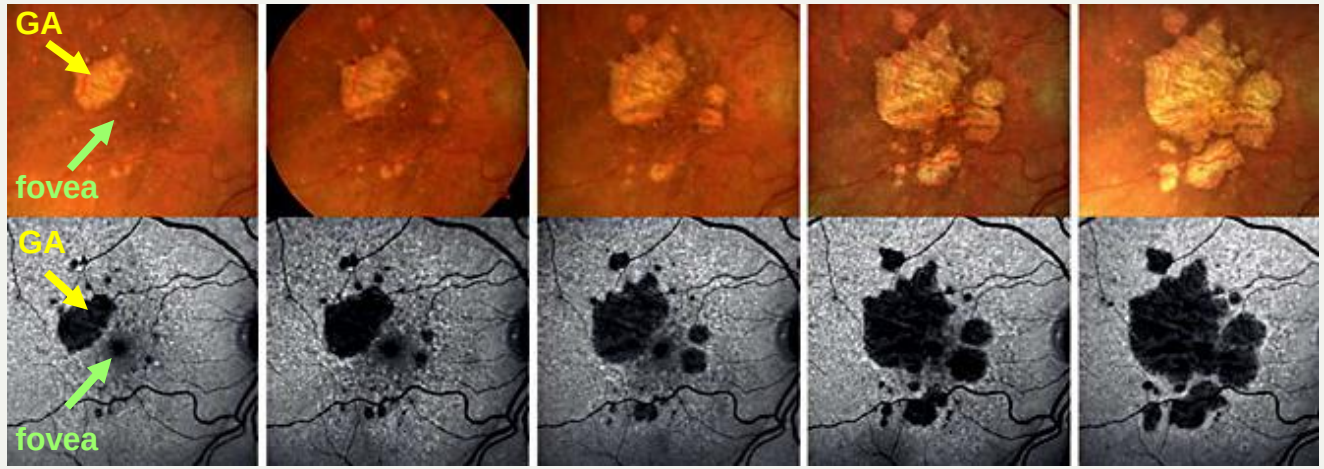
*Renderings created with BioRender.com

GA Lesions Relentlessly Expand Over Time

GA is characterized by the loss of photoreceptors, retinal pigment epithelium, and choriocapillaris, which leads to **irreversible loss of vision**

- Typically impacts both eyes
- Progression is correlated with increasing visual dysfunction
- The aim of most interventional studies is to reduce progression of GA lesion area enlargement

Clinical monitoring of GA over a 4-year period

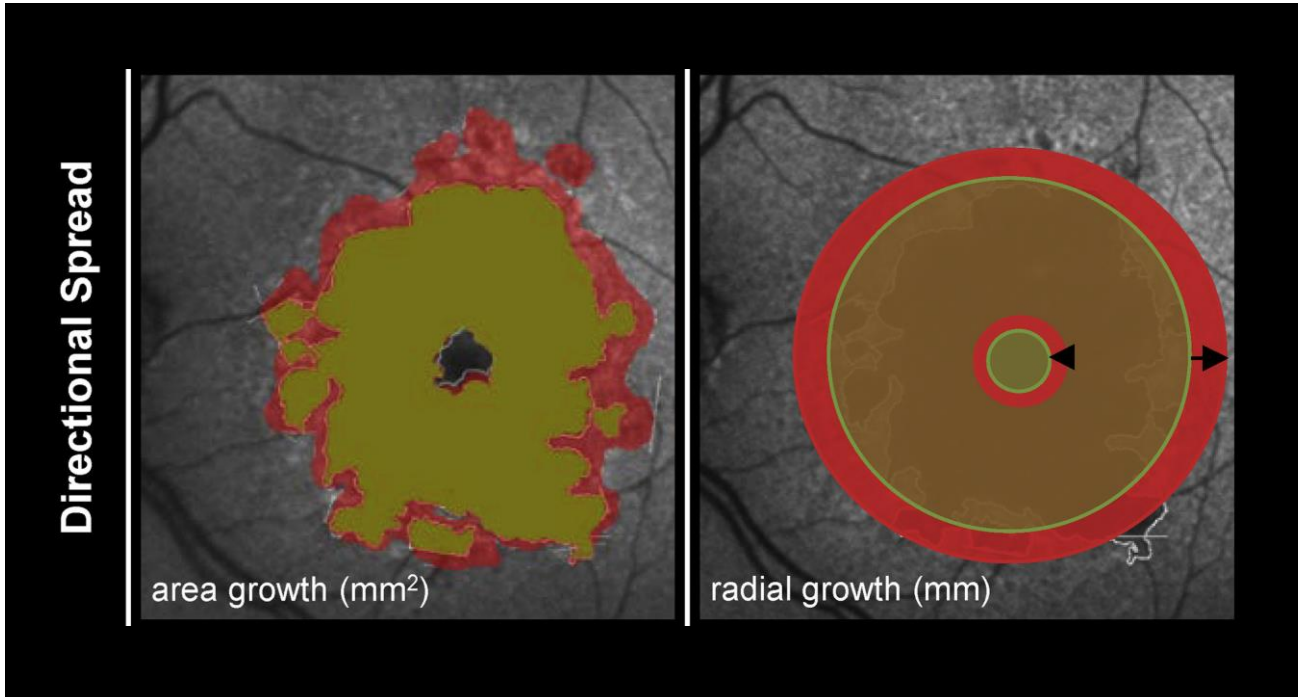


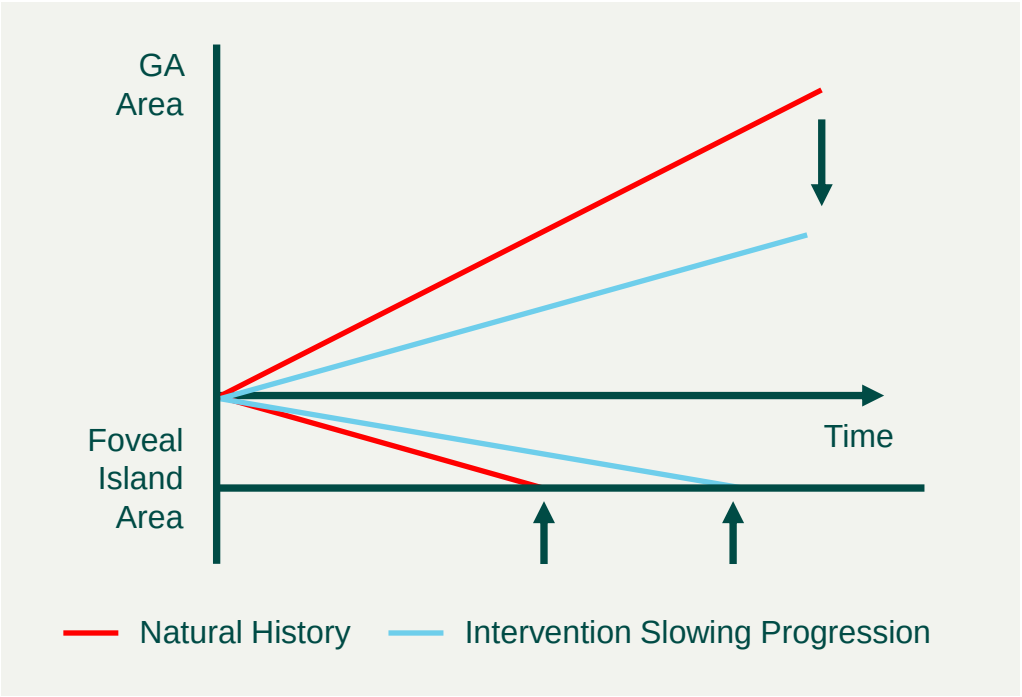
The Journey of "Geographic Atrophy" through Past, Present, and Future; Ophthalmologica 2017

Geographic Atrophy Lesion Growth

- GA lesions grow faster towards the periphery than the fovea
- Slowing growth towards the fovea preserves central vision for a longer period of time
- While visual symptoms are often present prior to foveal involvement, vision can drop dramatically when the fovea is impacted

GA treatments aim to slow lesion growth both outward (periphery) and inward (toward the fovea)





Fleckenstein et al. Ophthalmology 2018 125369-39

Complement Hypothesis and NGM621 Molecular Attributes

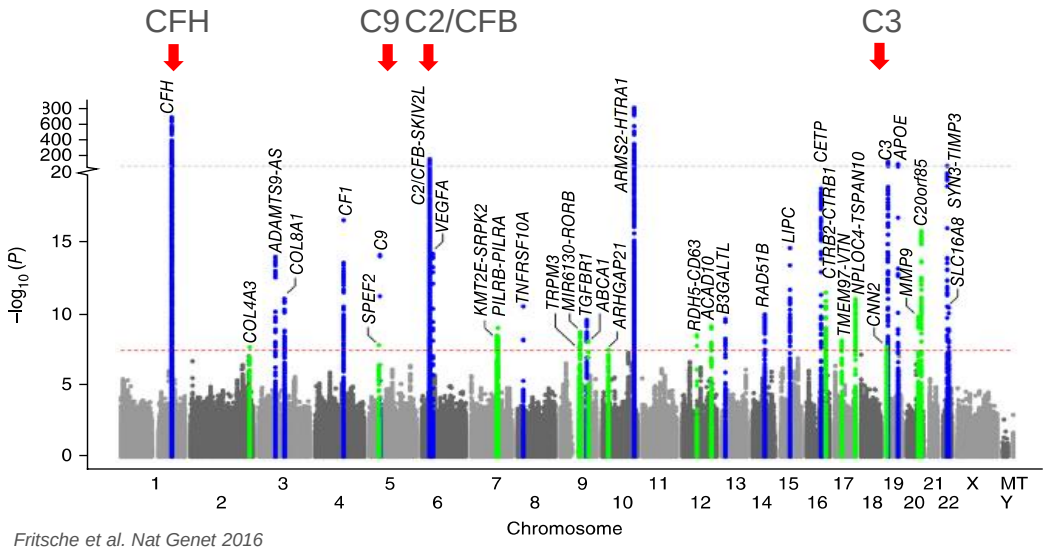
Mark Solloway, Ph.D.

Principal Scientist, Biology

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

Approach: unbiased identification of targets with strong genetic correlation to human disease

Genetic Evidence

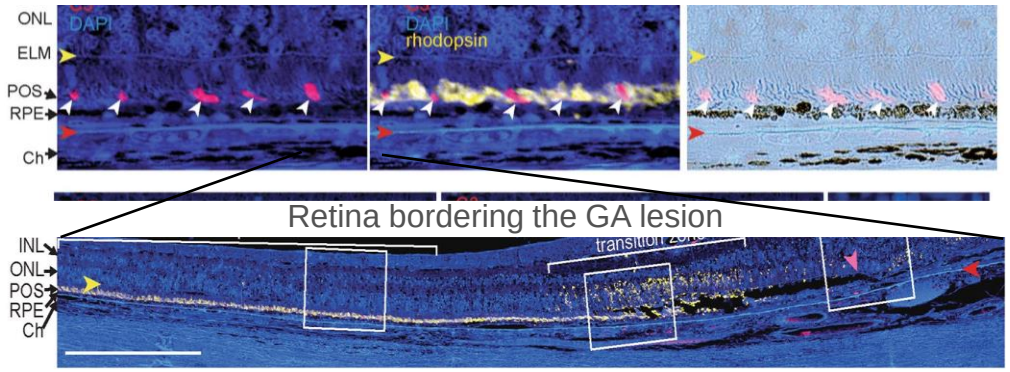


Fritsche et al. Nat Genet 2016

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

Histopathological Evidence

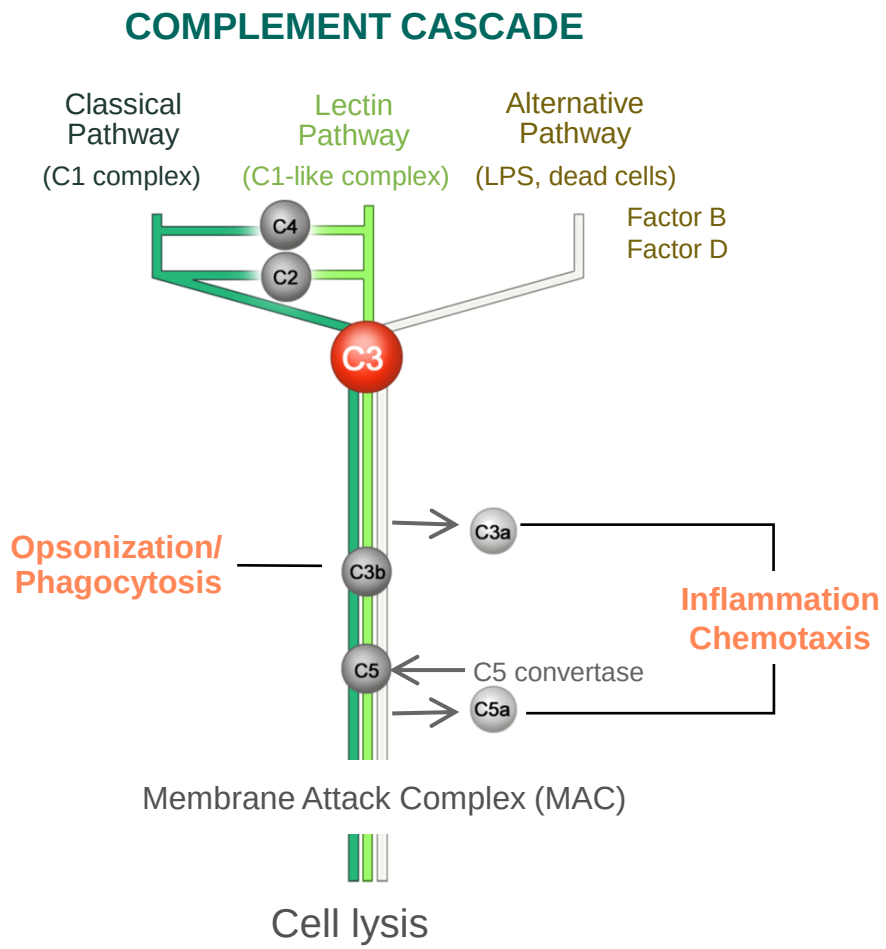
C3 Deposition on Photoreceptors Precedes their Degeneration in Human GA Eyes



Katschke et al. Sci. Reports 2018

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

GA Treatment Strategy: Targeting the Complement Pathway



Overactivation of the complement system has been implicated in the onset and progression of GA

C3 is a central component of the pathway and the first point of convergence for the 3 initiating pathways

Inhibiting C3 blocks all downstream complement signaling

Wykoff et al. AJO 2021

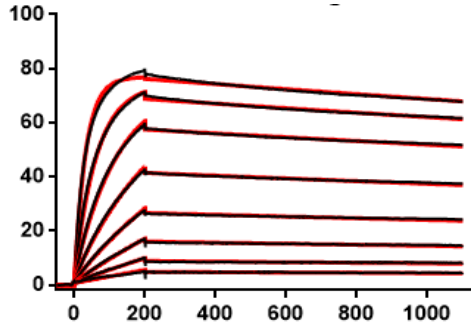
Generation of NGM621: A Potent Anti-C3 Antibody

NGM Hybridoma Antibody Discovery Platform

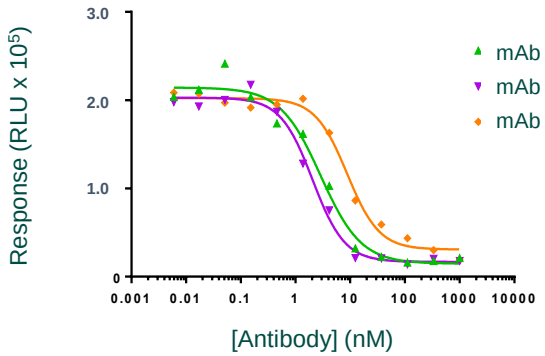
NGM621 is a humanized monoclonal antibody selected for

- High affinity binding to intact C3
- Complete and potent inhibition of C3a release in biochemical assay
- Complete and potent inhibition of complement activation in hemolytic assays

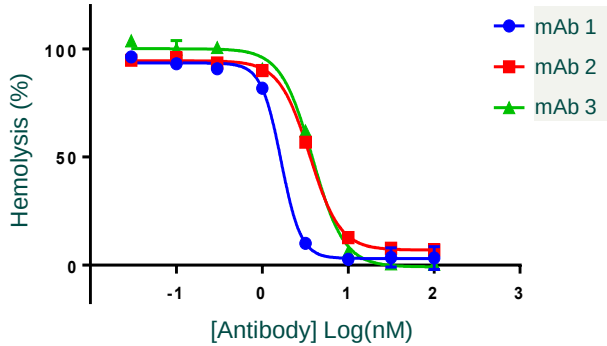
Binding by SPR



C3a Release Assay



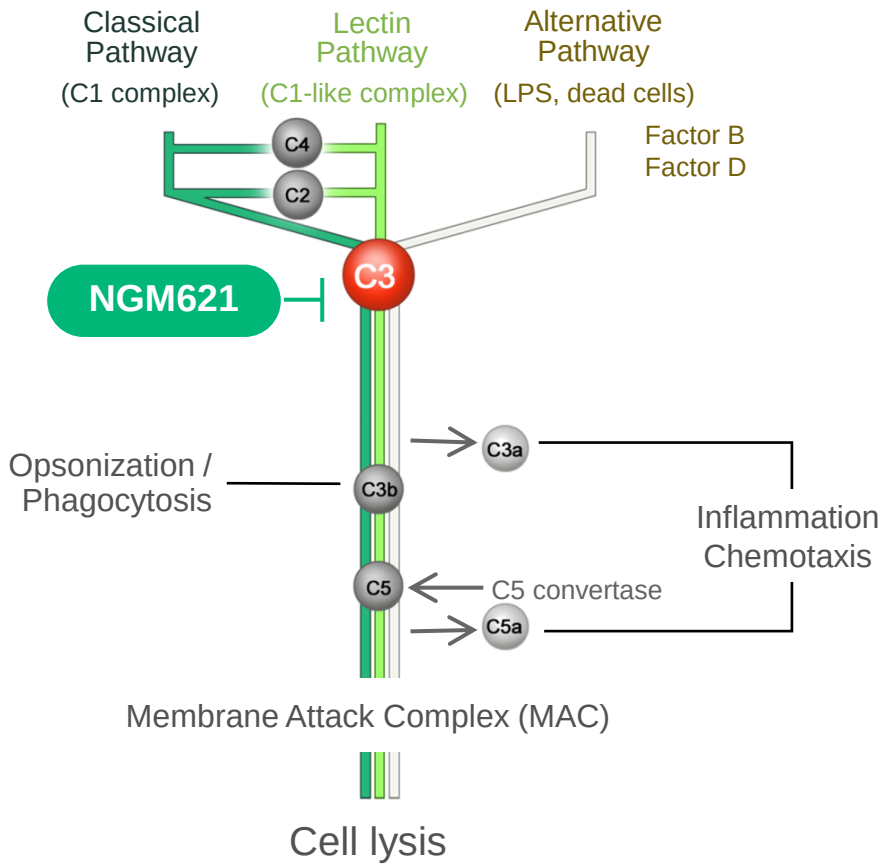
Hemolytic Assays

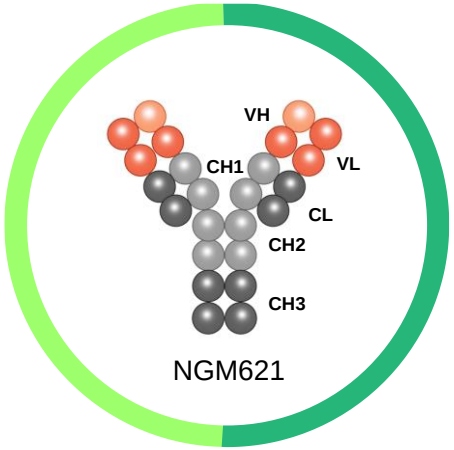


SPR = surface plasmon resonance
Loktev et. al. ARVO 2020

NGM621: A Potent Anti-Complement C3 Antibody

COMPLEMENT CASCADE





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 GA

C3 is a central component of the complement system, and the first point of convergence for all three initiating pathways

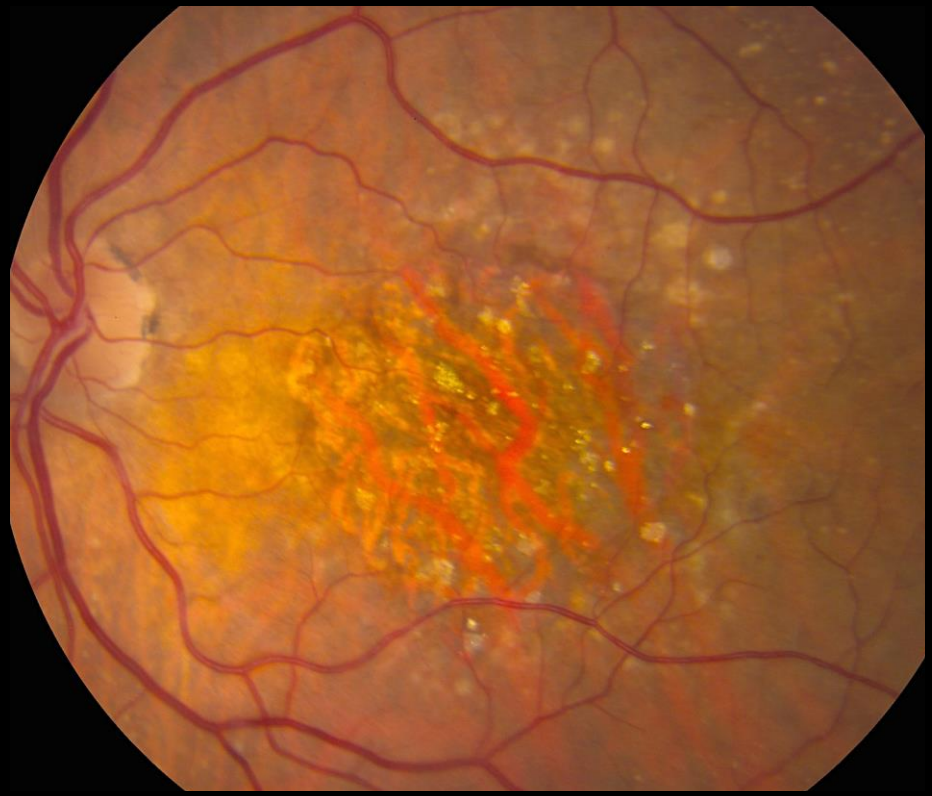
NGM621 is a novel monoclonal antibody that potently inhibits C3, effectively blocking all downstream complement signaling

Treatment of GA
Current State of Play

Charles C. Wykoff, MD, PhD

Retina Consultants of Texas

RETINA Consultants of America



Development in Context

Ongoing Programs

Patient Perspective & Clinical Adoption

Development in Context

JAMA Ophthalmology | Original Investigation

Efficacy and Safety of Lampalizumab for Geographic Atrophy Due to Age-Related Macular Degeneration

Chroma and Spectri Phase 3 Randomized Clinical Trials

Frank G. Holz, MD; Srinivas R. Sadda, MD; Brandon Busbee, MD; Emily Y. Chew, MD; Paul Mitchell, MD, PhD; Adnan Tufail, MD, FRCOphth; Christopher Brittain, MBBS; Daniela Ferrara, MD, PhD; Sarah Gray, PhD; Lee Honigberg, PhD; Jillian Martin, MD; Barbara Tong, PhD; Jason S. Ehrlich, MD, PhD; Neil M. Bressler, MD; for the Chroma and Spectri Study Investigators

2018

Systemic Complement Inhibition with Eculizumab for Geographic Atrophy in Age-Related Macular Degeneration

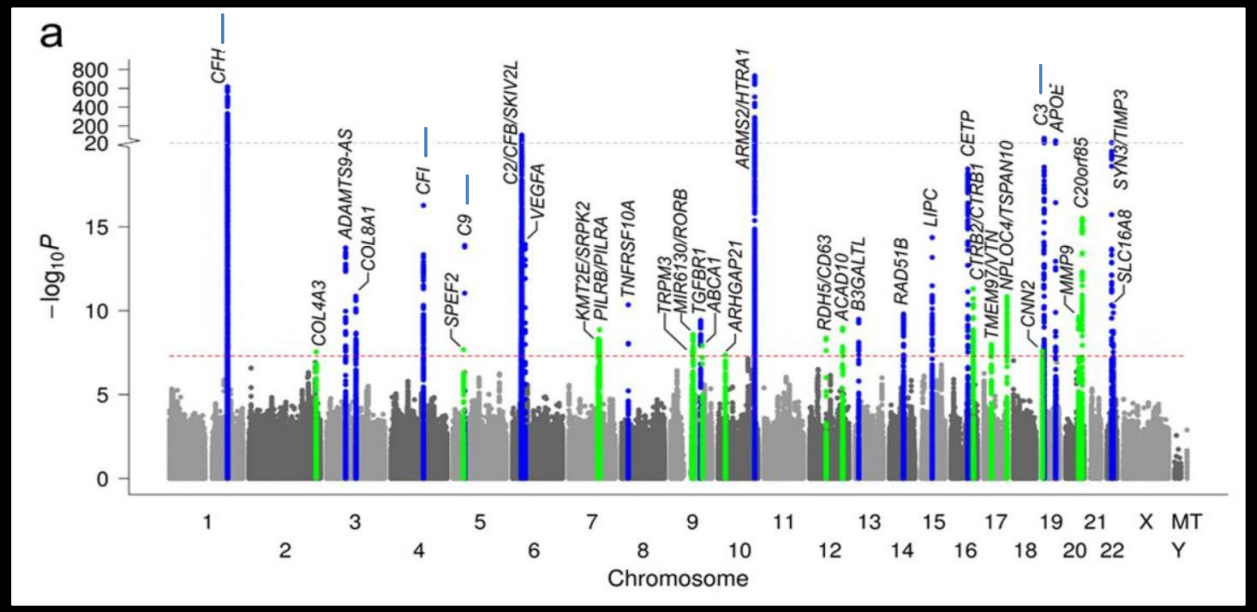
The COMPLETE Study

2014

Zohar Yehoshua, MD, MHA,[1] Carlos Alexandre de Amorim Garcia Filho, MD,[1,2] Renata Portella Nunes, MD,[1] Giovanni Gregori, PhD,[1] Fernando M. Penha, MD, PhD,[1,2] Andrew A. Moshfeghi, MD, MBA,[1] Kang Zhang, MD, PhD,[3] SriniVas Sadda, MD,[4] William Feuer, MS,[1] Philip J. Rosenfeld, MD, PhD[1]

Genetics & AMD

- **> 40 loci implicated**
- **Account for >50% of Risk**
- **Complement components**: CFH, CFI, C3, C9, C2/CFB
  - Variants predicted to increase activation or decrease inactivation of complement cascade → increased inflammatory activation



Complement Factor H Variant Increases the Risk of Age-Related Macular Degeneration

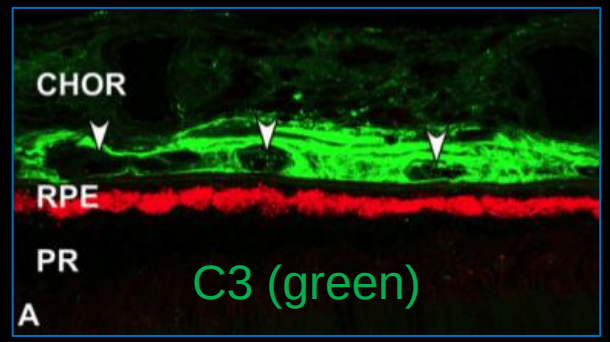
Jonathan L. Haines,[1] Michael A. Hauser,[2] Silke Schmidt,[2] William K. Scott,[2] Lana M. Olson,[1] Paul Gallins,[2] Kylee L. Spencer,[1] Shu Ying Kwan,[2] Maher Noureddine,[2] John R. Gilbert,[2] Nathalie Schnetz-Boutaud,[1] Anita Agarwal,[3] Eric A. Postel,[4] Margaret A. Pericak-Vance[2]*

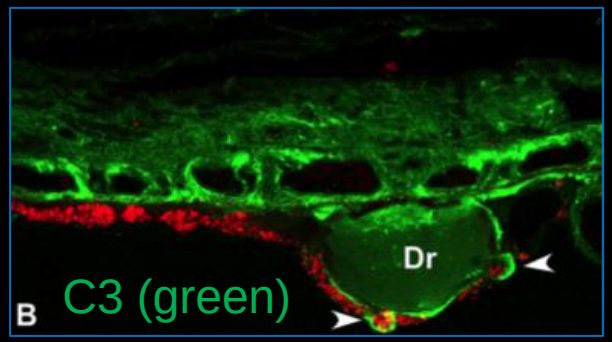
Complement Factor H Polymorphism and Age-Related Macular Degeneration

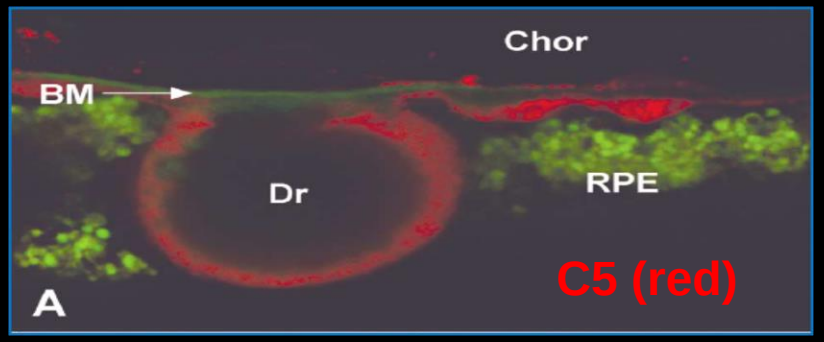
Albert O. Edwards,[1]*† Robert Ritter III,[1] Kenneth J. Abel,[2] Alisa Manning,[3] Carolien Panhuysen,[3,6] Lindsay A. Farrer[3,4,5,6,7]

Complement Activation in AMD Eyes

- Histopathologic studies of AMD eyes
- Confocal immunofluorescence microscopy:
  - C3 & C5 accumulation in drusen and sub-RPE space







Anderson, DH et al.,Progress in Retinal and Eye Research Dec. (2010) 29;2, 95-110

Anderson DH, Mullins RF, Hageman GS, Johnson LV, *Am J Ophthalmol*. 2002 Sep;134(3):411-31

AMERICAN ACADEMY
OF OPHTHALMOLOGY®

Complement C3 Inhibitor Pegcetacoplan for Geographic Atrophy Secondary to Age-Related Macular Degeneration

A Randomized Phase 2 Trial

David S. Liao, MD,[1] Federico V. Grossi, MD, PhD,[2] Delphine El Mehdi, PhD,[2] Monica R. Gerber, MD, PhD,[2] David M. Brown, MD,[3] Jeffrey S. Heier, MD,[4] Charles C. Wykoff, MD, PhD,[5] Lawrence J. Singerman, MD,[6] Prema Abraham, MD,[7] Felix Grassmann, PhD,[8,9] Peter Nuernberg, PhD,[10] Bernhard H.F. Weber, PhD,[8] Pascal Deschatelets, PhD,[2] Robert Y. Kim, MD,[2] Carol Y. Chung, PhD,[2] Ramiro M. Ribeiro, MD, PhD,[2] Mohamed Hamdani, MS,[2] Philip J. Rosenfeld, MD, PhD,[11] David S. Boyer, MD,[12] Jason S. Slakter, MD,[13,14] Cedric G. Francois, MD, PhD[2]

AMERICAN ACADEMY
OF OPHTHALMOLOGY®

C5 Inhibitor Avacincaptad Pegol for Geographic Atrophy Due to Age-Related Macular Degeneration

A Randomized Pivotal Phase 2/3 Trial

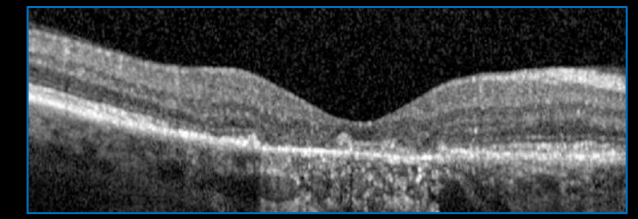
Glenn J. Jaffe, MD,[1] Keith Westby, MBA,[2] Karl G. Csaky, MD, PhD,[3] Jordi Monés, MD, PhD,[4] Joel A. Pearlman, MD, PhD,[5] Sunil S. Patel, MD, PhD,[6] Brian C. Joondeph, MD, MPS,[7] John Randolph, MD,[8] Harvey Masonson, MD,[2] Kourous A. Rezaei, MD[2]

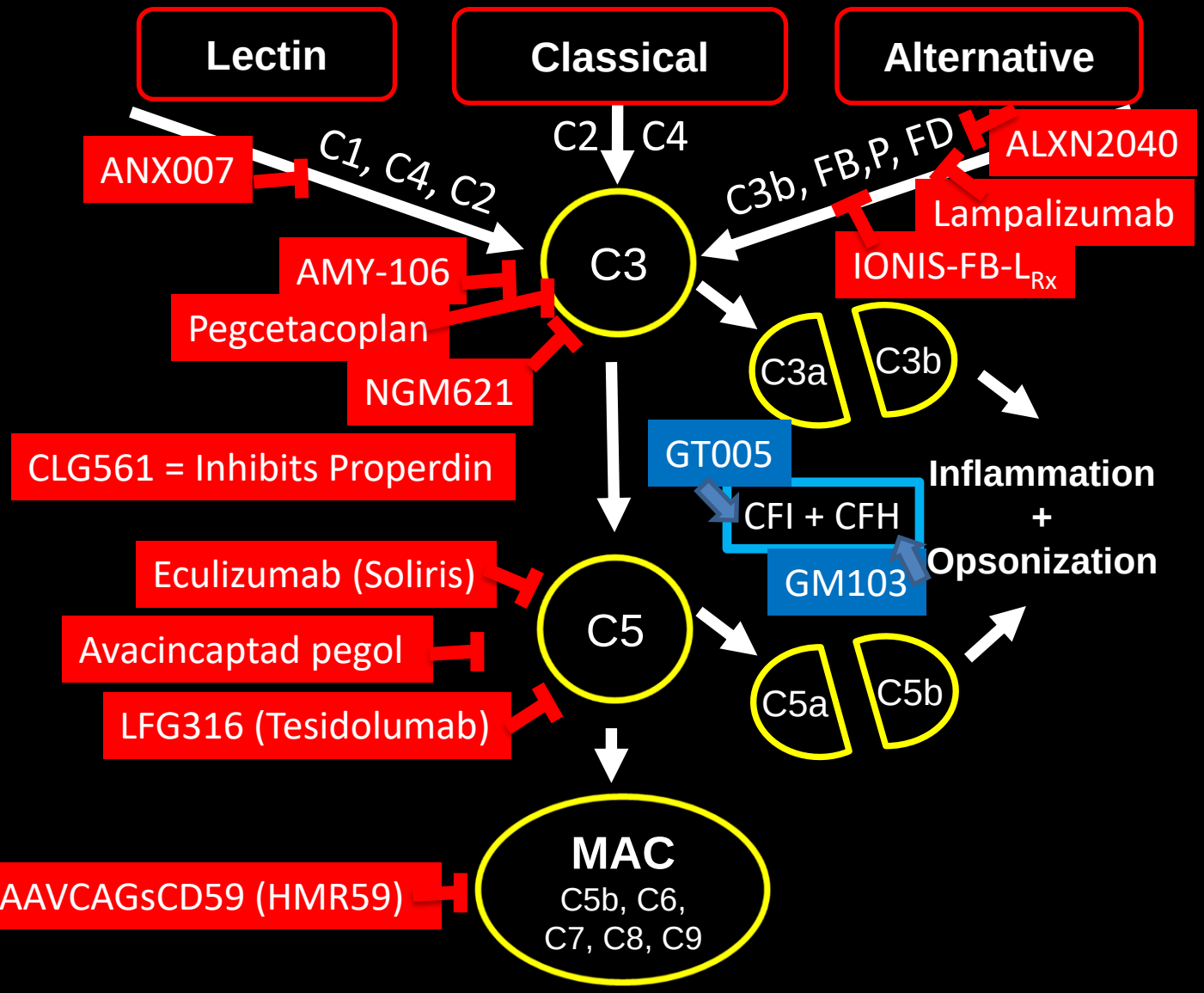
Ongoing Programs

GA: Target Validation & Clinical Introduction

Non-Compliment Targets

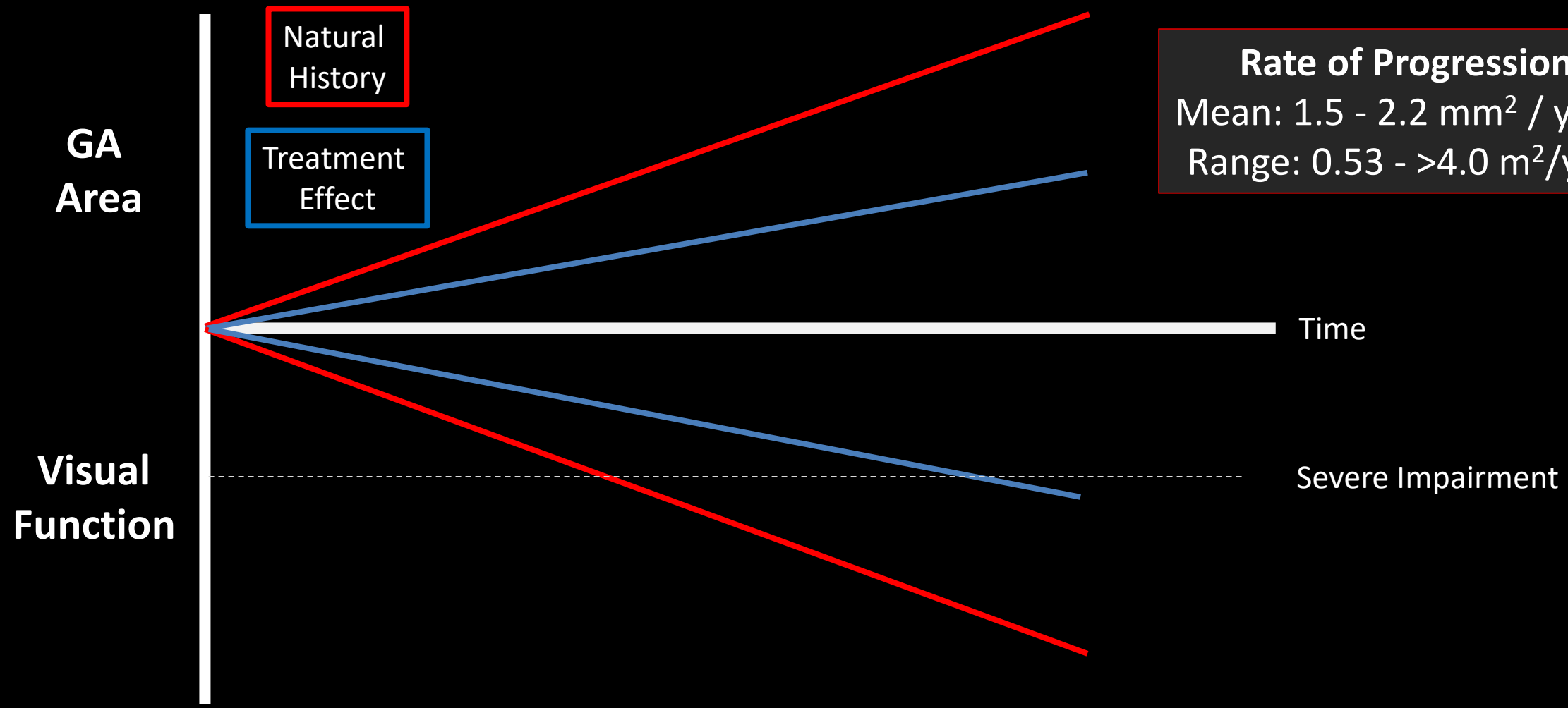
ONL1204 = inhibits FAS signaling
FHTR2163 = inhibits HtrA1 activity
GAL-101 = A-beta aggregation inhibitor
RT001 = Docosahexaenoic acid
ALK-001 = oral deuterated vitamin A
Risuteganib = Integrin regulator
Elamipretide = binds cardiolipin
PBM = Mitochondrial manipulation
Ciliary Neurotrophic Factor (CNTF)
Brimonidine = alpha2A agonist





#1 Improved Efficacy

Goal of Therapy = Slow Rate of Progression



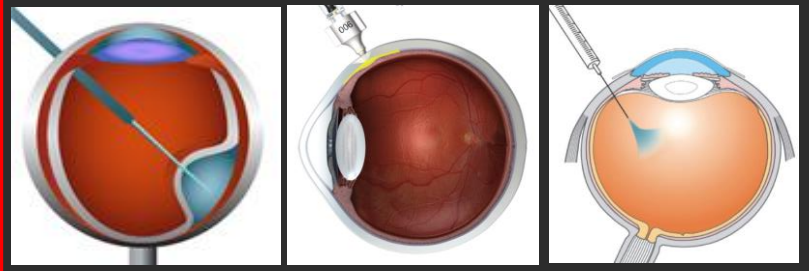
Rate of Progression
Mean: 1.5 - 2.2 mm^2 / year*
Range: 0.53 - >4.0 m^2/year

*1.93 mm^2/year in lampalizumab phase 3 program

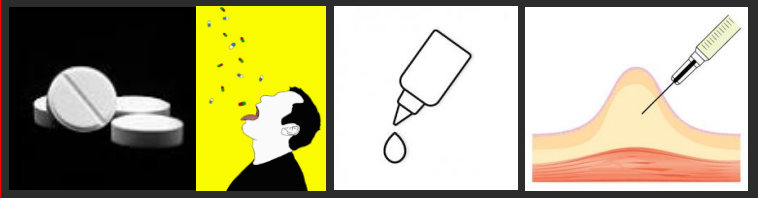
#2 Improved Durability or Delivery

- Reduced injection frequency
  - Q4W → Q8W → Q3M → 6M
  - One & done
- Non-Intravitreal Injection

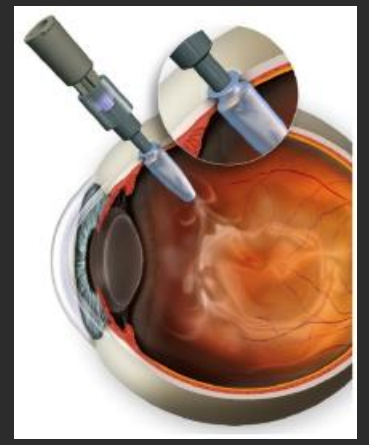
Gene Therapy

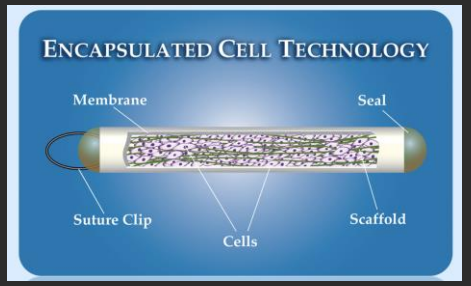


Non-ocular Delivery



Sustained Drug Delivery





#3 Safety

- Better understanding of safety profile
- Exudative AMD development
  - Harnessing
  - Avoiding

#4

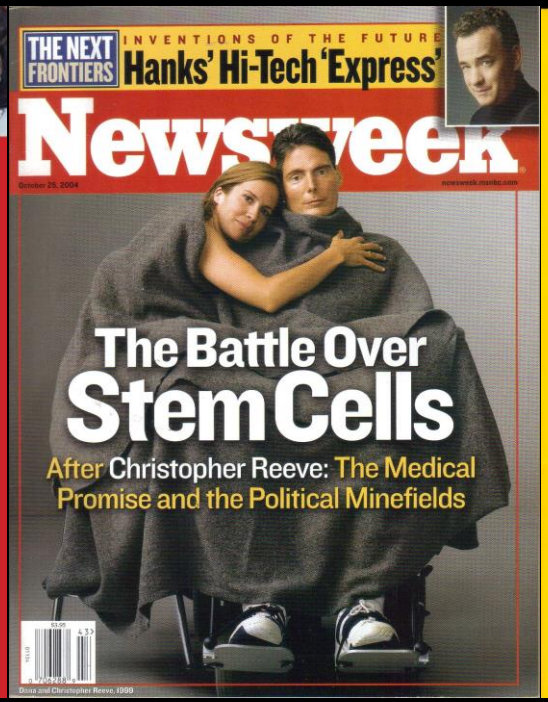
Cell-Based Therapies

Regenerative & Paracrine

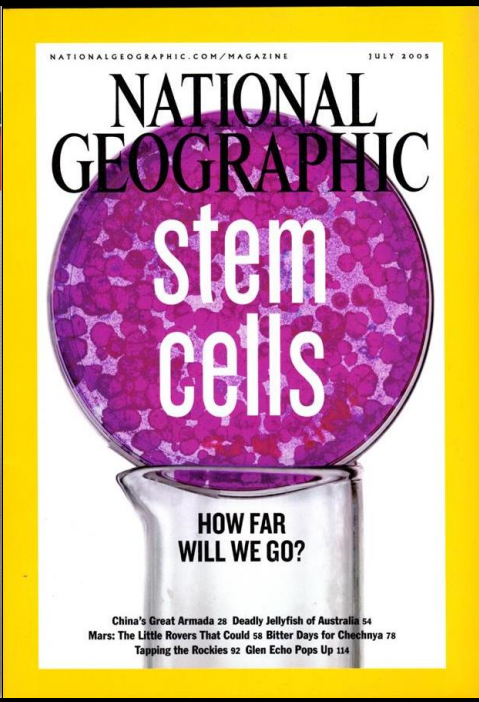
1999

2009

2006



2004



2005

#5 End-point Evolution


Research Opportunities

IOVS 2017

Report From the NEI/FDA Diabetic Retinopathy Clinical Trial Design and Endpoints Worksh

Prasbant Nair,[1] Lloyd Paul Aiello,[2] Thomas W. Gardner,[3] and Frederick L. Ferris III[5]

Research Opportunities

IOVS 2016

Report From the NEI/FDA Endpoints Workshop on Age-Related Macular Degeneration and Inherited Retinal Diseases

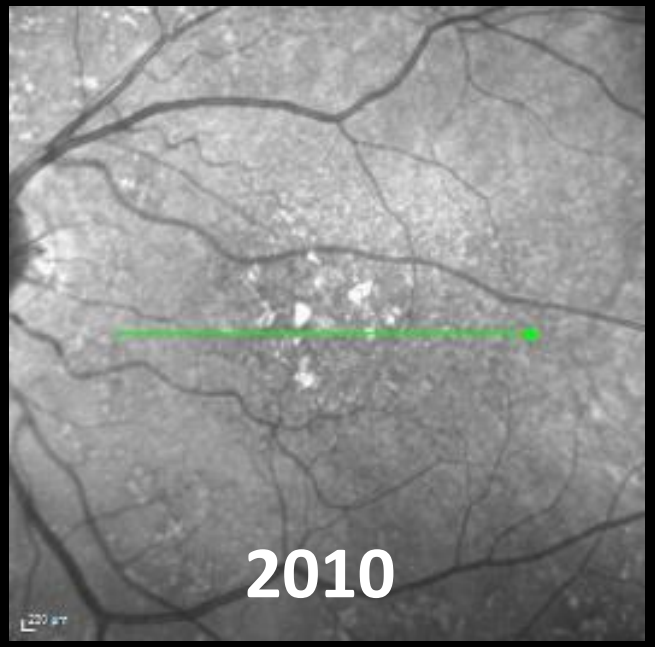
Karl Csaky,[1] Frederick Ferris III,[2] Emily Y. Chew,[2] Prasbant Nair,[3] Janet K. Cheetham,[4] and Jacque L. Duncan[5]

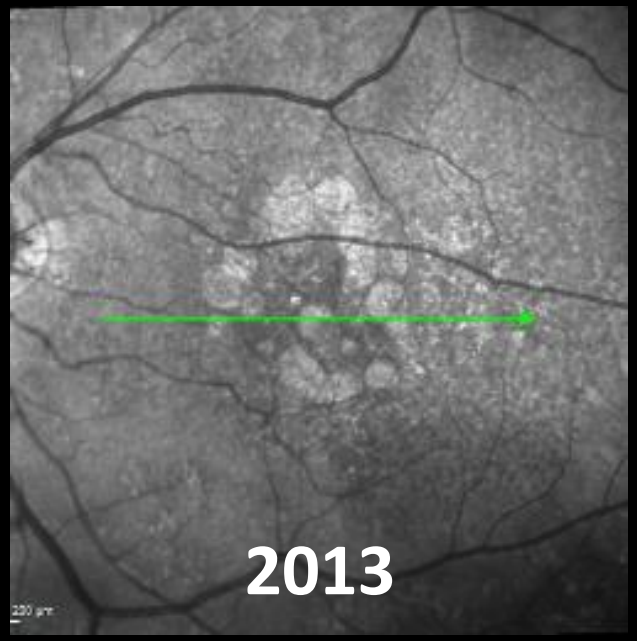
- **Jane Moseley (EMA):**
  - "Area of GA could be acceptable as a primary efficacy variable in principle, but the European regulators would like this use to be supported by positive effect on function."
- **Wiley Chambers (FDA)**
  - "EZ area loss… likely to be acceptable as a surrogate endpoint"
  - "Preventing photoreceptor loss …. would be considered a clinically meaningful endpoint"
  - "Drusen volume changes and step changes on the AMD severity scale… would not be recommended at this present time."
  - Related to GA onset as an endpoint: "Any measurable change should be both statistically significant and clinically significant."

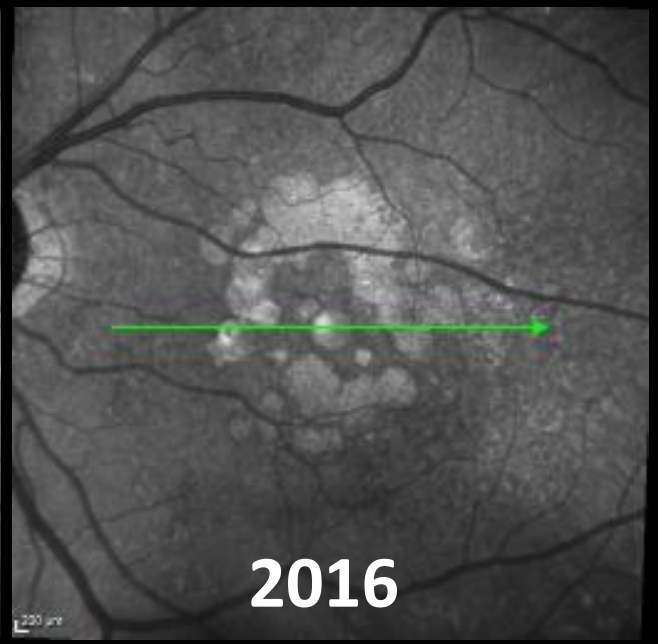
1. **ETDRS BCVA**
   - Maintain = lose < 15
   - Mean Change
   - Gain or Time to Achieve ≥ 15
2. **FP**
   - DRSS Improvement
   - CMV retinitis progression
3. **OCT**
   - Resolution of VMA
4. **FAF**
   - Atrophic lesion / GA area
5. **Physical Exam**
   - Vitreous Haze
   - Rate of recurrence of uveitis
6. **Mobility**
   - Multi-luminance mobility testing (MLMT)

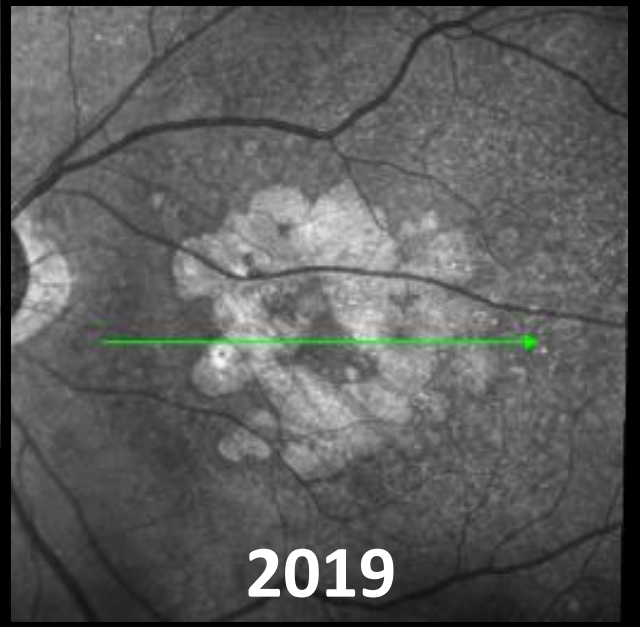
Patient Perspective
Clinical Adoption

GA



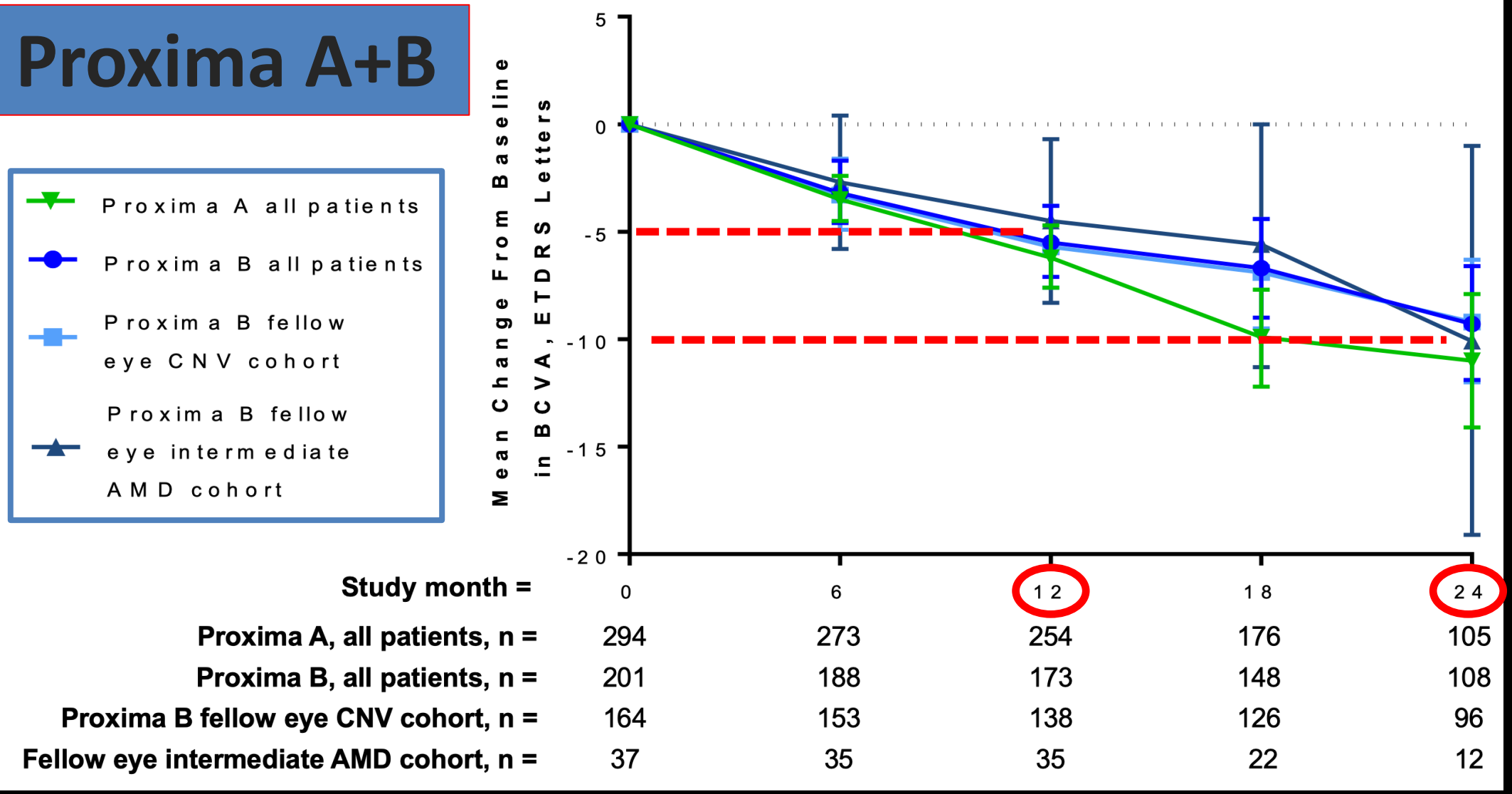






BCVA Mean Change From Baseline

Proxima A+B



Study month =	0	6	12	18	24
Proxima A, all patients, n =	294	273	254	176	105
Proxima B, all patients, n =	201	188	173	148	108
Proxima B fellow eye CNV cohort, n =	164	153	138	126	96
Fellow eye intermediate AMD cohort, n =	37	35	35	22	12

Quantifying the Burden of GA

Characterizing Disease Burden and Progression of Geographic Atrophy Secondary to Age-Related Macular Degeneration

Data from UK EMR systems at 10 clinical sites: 2000 – 2016

- Among patients OU GA
- Loss of 6-10 letters / 2 years
- 2/3rd unable to drive within 2 years

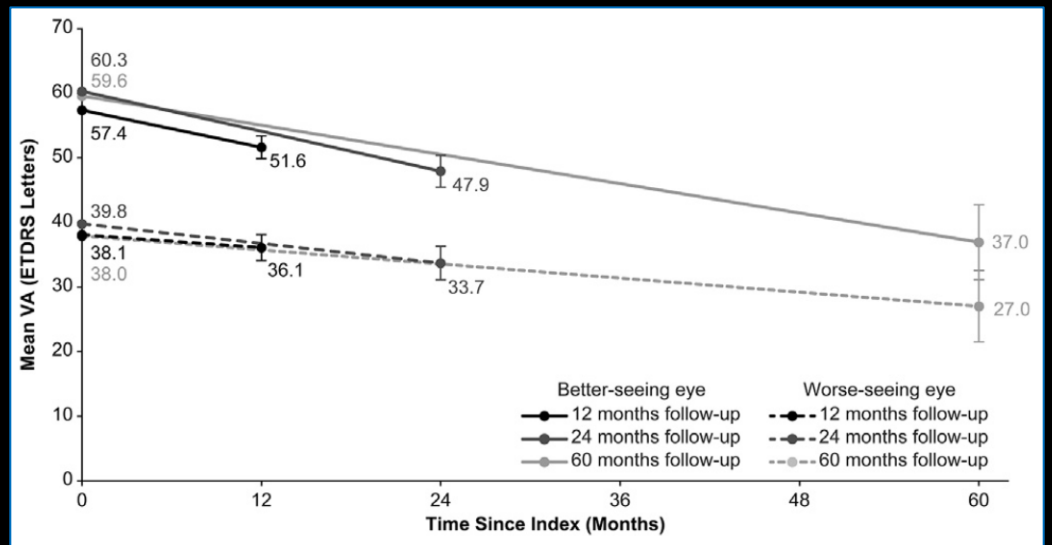
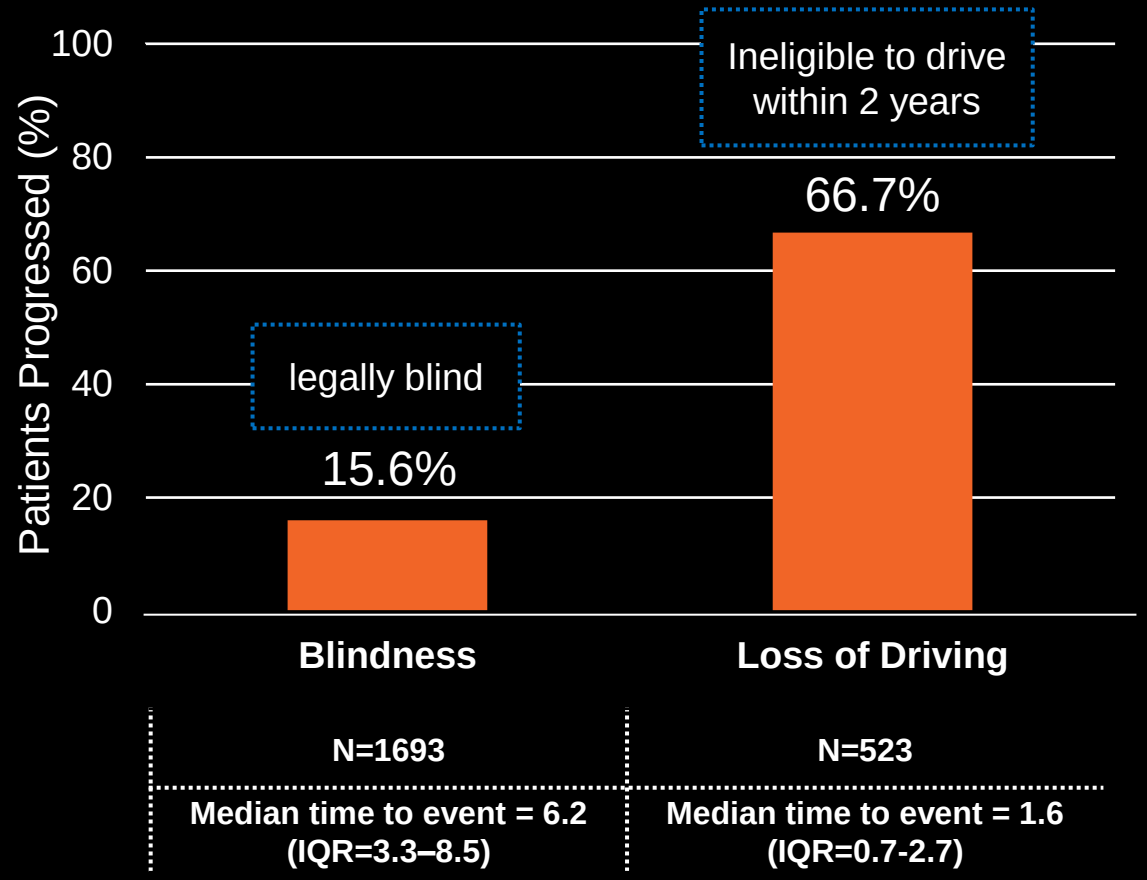


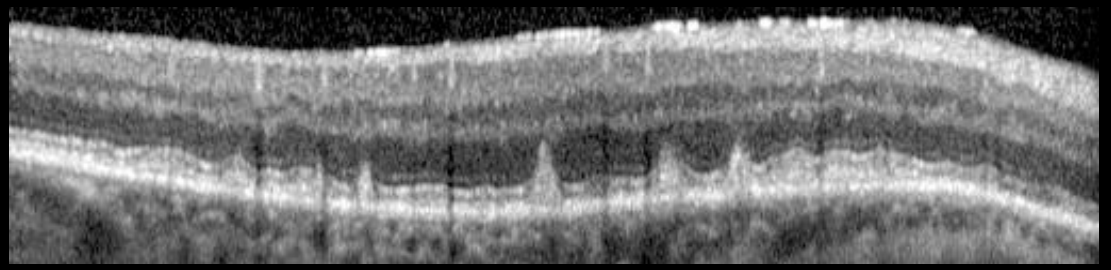
Figure 2. Change in mean visual acuity (VA) from baseline in the worse-seeing (study) eye and better-seeing (fellow) eye through 60 months' follow-up in patients with bilateral geographic atrophy identified in the

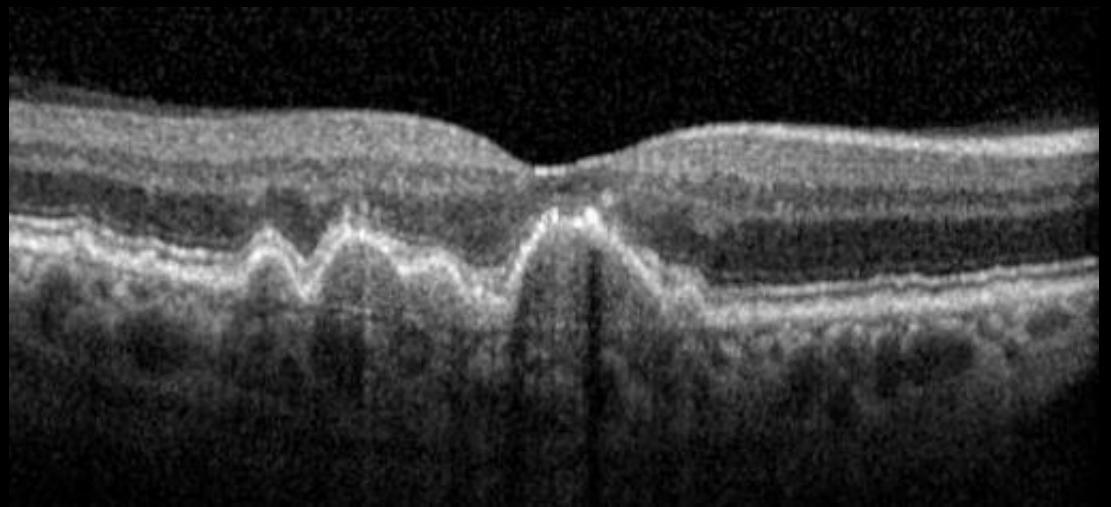


Chakravarty U et al. Ophthalmology. 2018 Jun;125(6):842-849.

Clinical Adoption

- Patients Selection
  - All will be interested & many will want to initiate therapy
  - Foveal vs Non-Foveal
- Challenge = Long-term Adherence
  - Patient-perceived benefit
- Shift in focus to earlier stages
  - iRORA
  - cRORA



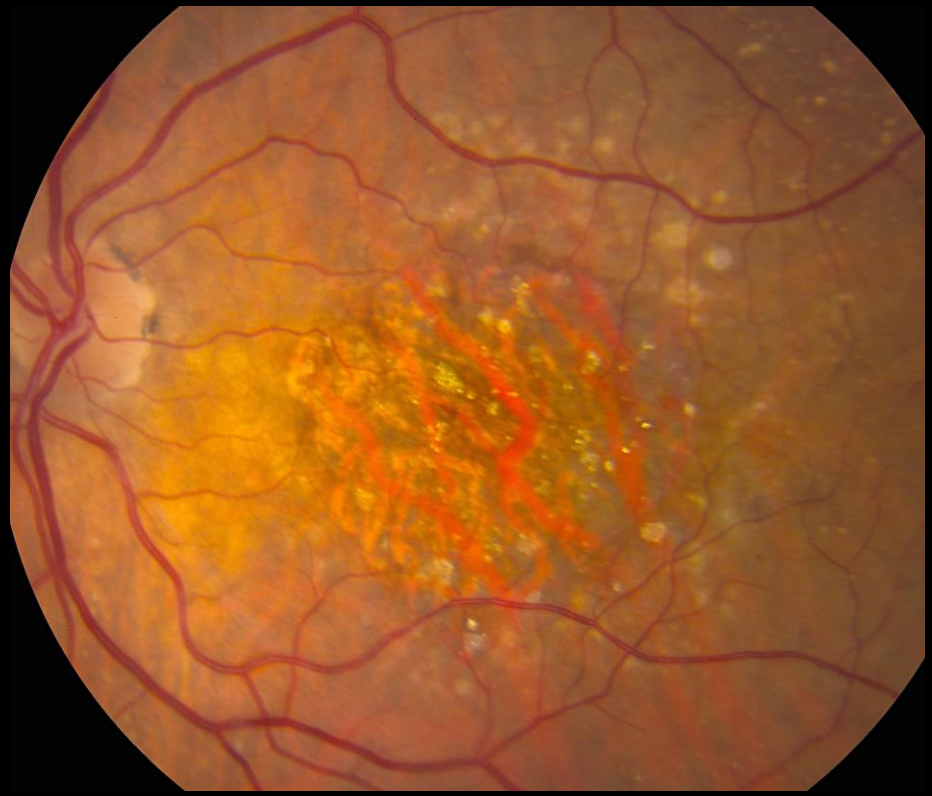


Treatment of GA
Current State of Play

Charles C. Wykoff, MD, PhD

Retina Consultants of Texas

RETINA Consultants of America

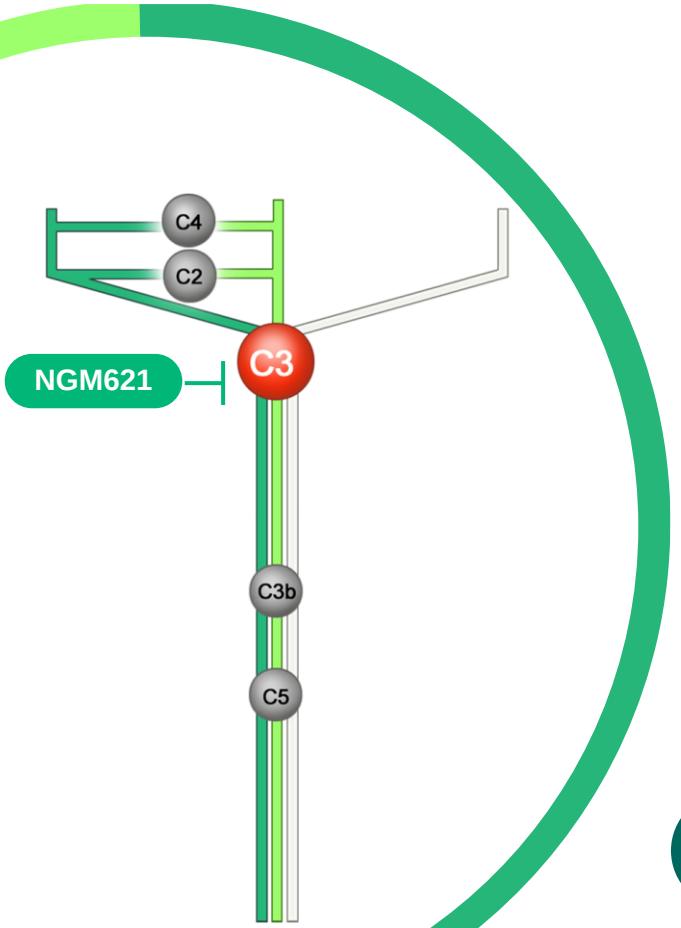


NGM621, a Differentiated Complement Inhibitor

Erin C. Henry, Ph.D.

Head of Ophthalmology Clinical Development

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



Potential NGM621 Differentiation in Geographic Atrophy (GA)

1 Efficacy

NGM621 is a potent inhibitor of complement; this may translate into better clinical outcomes than other approaches

2 Dosing Frequency

Pharmacokinetic modeling supports the potential for extended, every 8-week dosing

3 Safety

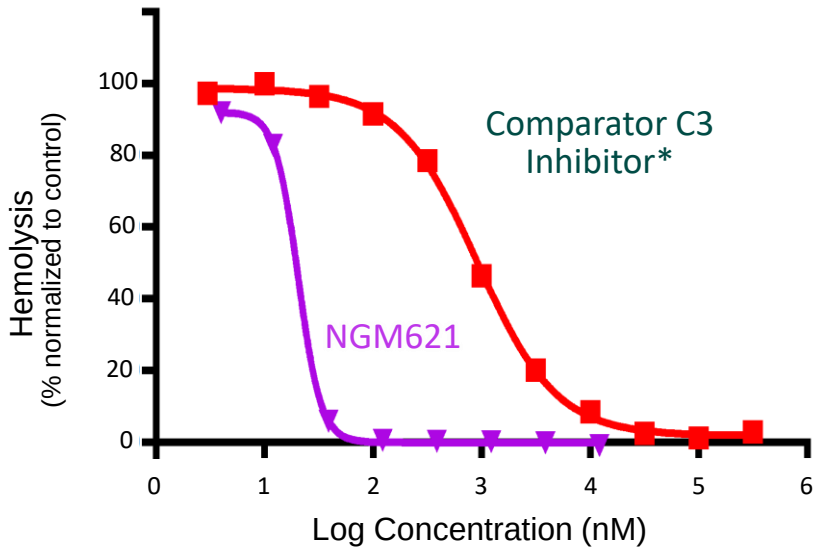
Unique molecular attributes may translate clinically into better safety

Multiple potential opportunities for NGM621 to advance the treatment of GA

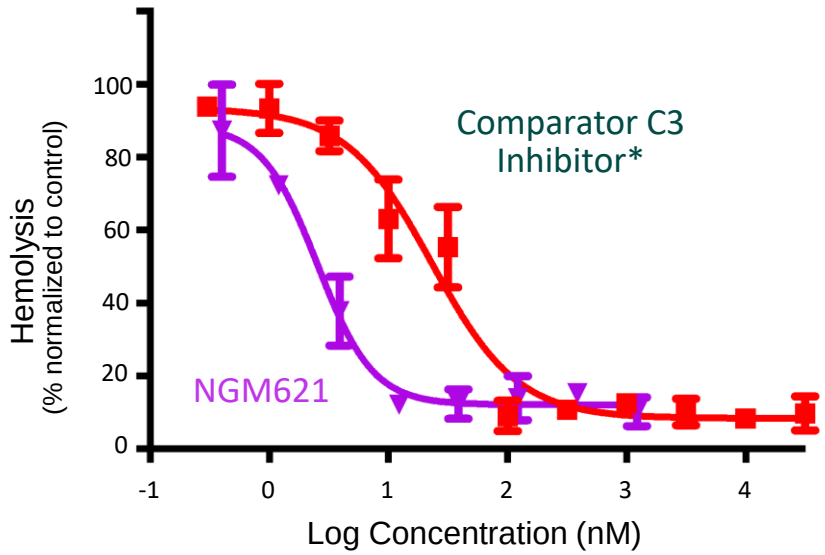
NGM621 Potently Inhibits Complement Activation in Preclinical Studies

1 **In preclinical studies, NGM621 was a potent inhibitor of complement**

Hemolytic Assay
(Classical Pathway)



Hemolytic Assay
(Alternative Pathway)



	CP IC_{50}	AP IC_{50}
NGM621	2 nM	2.5 nM
Comparator C3 inhibitor*	90 nM	23 nM

*APL-1, a compstatin derived, cyclic peptide targeting C3, manufactured by NGM Bio for preclinical comparability purposes.

NGM621 Binds Preferentially to C3 But Also Binds C3b in Preclinical Assays

1 **In preclinical assays, NGM621 demonstrated high binding affinity to both C3 and C3b**

Comparative Binding Assay to C3 & proteolytic products			
Molecule	**Binding Affinity (K_D, nM)**		
	C3	**C3a**	**C3b**
NGM621	0.34	N/A	53
Comparator*	19.1	N/A	~100

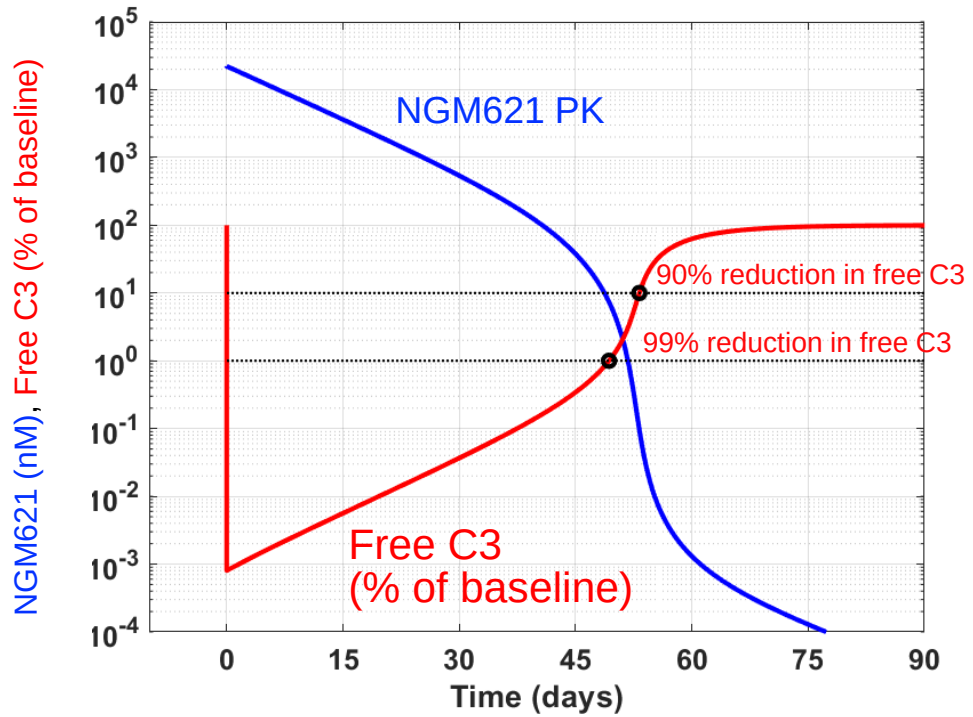
- NGM621 binded to C3 with ~100-fold higher affinity than to any of the C3 proteolytic fragments
- NGM621 affinity to C3b was comparable to comparator*

*APL-1, a compstatin derived, cyclic peptide targeting C3 manufactured by NGM Bio for preclinical comparability purposes.

NGM621 Ocular PK/PD Modeling Supports Q8 Week Dosing

2 PK modeling supports the potential to dose NGM621 every 8 weeks

Predicted Vitreous Humor PK/PD Profile*



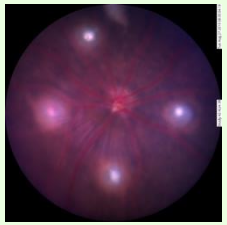
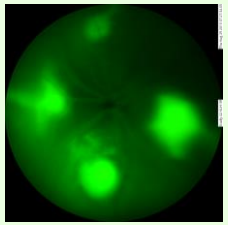
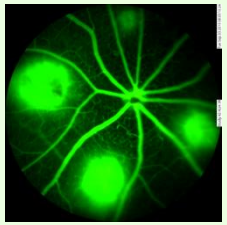
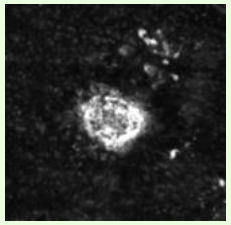
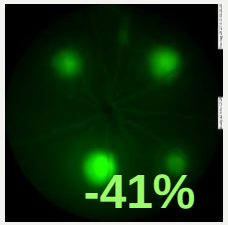
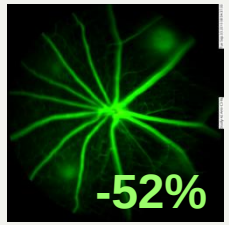
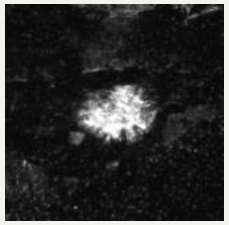
- NGM621 is predicted to achieve >90% C3 target engagement in the eye for 7 weeks following a single IVT dose of 15 mg based on a PK/PD model
- PK/PD modeling and simulation supported testing an every 8 week IVT dosing regimen at the 15-mg dose level in clinical trials

IVT, intravitreal; PD, pharmacodynamic; PK pharmacokinetic; Q8W, every 8 weeks.
*Ocular PK was not collected in phase 1; model based on preclinical ocular PK data.
Thank you to the Merck Clinical Pharmacology team for their support of the PK/PD modeling work.

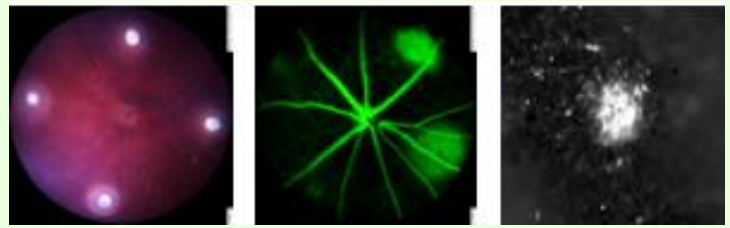
Preclinical data showed anti-C3 Monoclonal Antibody Reduces CNV while PEG exacerbated CNV (*Laser-induced CNV Rodent Model)*

3 **Preclinical data suggests potential for safety advantage**

Anti-C3 Mab Reduces Vascular Leakage

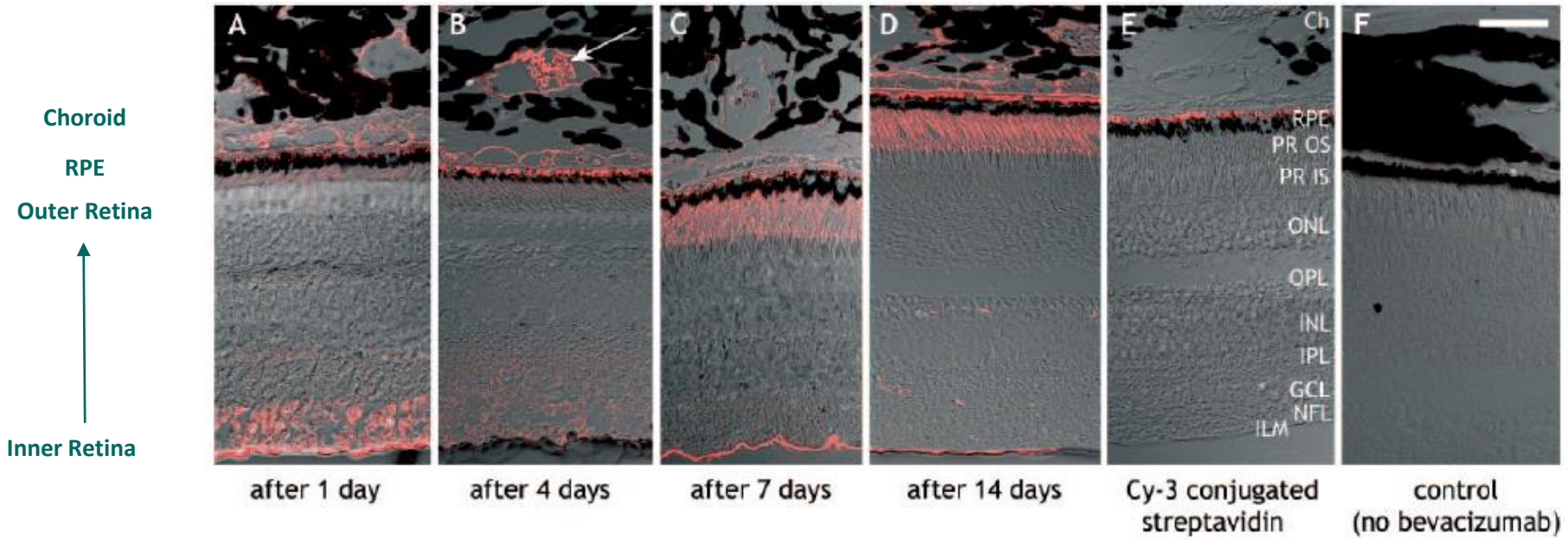
	Fundus (Day 0)	Complement activation (Anti-C3d - Day 2)	Vascular Leakage (Fluorescein angiography - Day 7)	Angiogenesis (Isolectin B4 - Day 7)
Control Anti-KLH (IVT)				
Anti-C3 Mab (IVT)				
		p<0.01	*p<0.05*	

PEG-40KD Exacerbates Vascular Leakage and Angiogenesis

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

Monoclonal antibodies penetrate the retina & reach the choroid

Retinal Penetration of Intravitreally-Injected Bevacizumab[1]



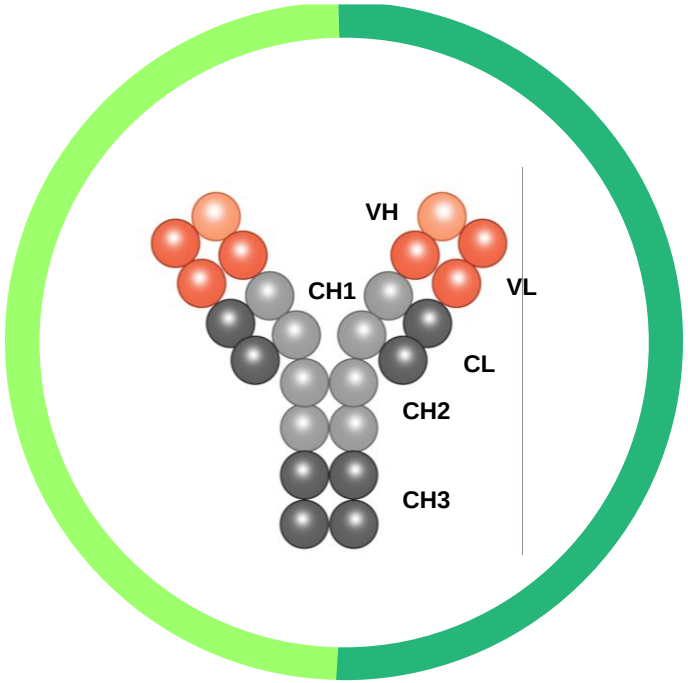
[1]Heiduschka et al. Penetration of Bevacizumab through the Retina after intravitreal injection in the monkey. IOVS 2007

A Different Kind of Complement Inhibitor

NGM621 is designed for optimized inhibition of the pathologic complement activity contributing to Geographic Atrophy

- Selective, high binding affinity for C3 in preclinical models
- Humanized with no Fc receptor effector function
- Favorable biophysical properties
  - High solubility, relatively low viscosity
  - Excellent long-term stability
- Antibodies are generally well tolerated in the eye (low inflammation risk)
- Antibodies can rapidly penetrate the retina

NGM621



Clinical Development of NGM621 Is Rapidly Advancing

Phase 1
Single dose escalation & multi-dose evaluation

15mg Dose

Phase 2 CATALINA
Proof of Concept

Safety & Tolerability (N=15)

- NGM621 was well tolerated at all dose levels evaluated
- No drug-related adverse events or serious adverse events
- No ocular safety signals (No CNV, no endophthalmitis, no inflammation)
- Study results published in AJO (Wykoff et al., 2021)

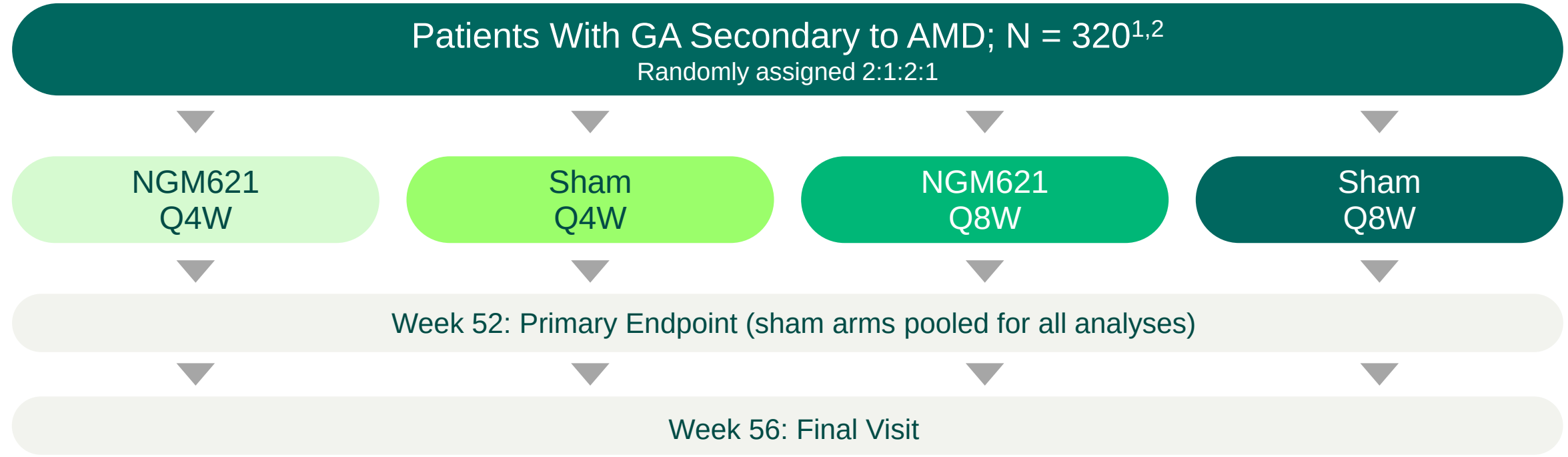
*FDA granted NGM621 Fast Track Designation for treatment of GA secondary to age-related macular degeneration
February 2022

Safety and Efficacy of Intravitreal NGM621 (N=320)

- Enrollment complete
- 65 U.S. sites
- First Patient In July 2020

CATALINA

Phase 2 CATALINA Topline Results Announcement Planned in 4Q22



Primary Objective	To evaluate the rate of change in GA lesion area and safety of NGM621 IVT injections administered Q4W or Q8W compared with sham control (pooled) in patients with GA
Design	Multicenter, randomized, double-masked, sham-controlled, overseen by an independent data safety monitoring board

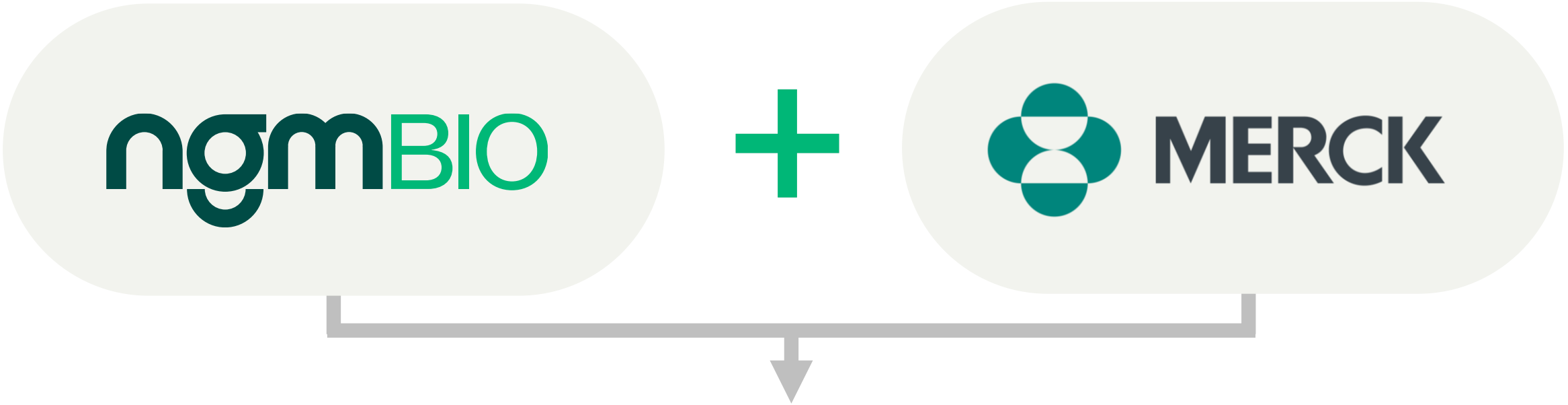
[1]NGM Corporate Press Release July 22, 2021; [2]ClinicalTrials.gov NCT04465955
IVT = intravitreal; Q4 = every 4 weeks; Q8 = every 8 weeks

Concluding Remarks

Siobhan Nolan Mangini

Chief Financial Officer

NGM Bio and Merck Have had a Long and Productive Collaboration



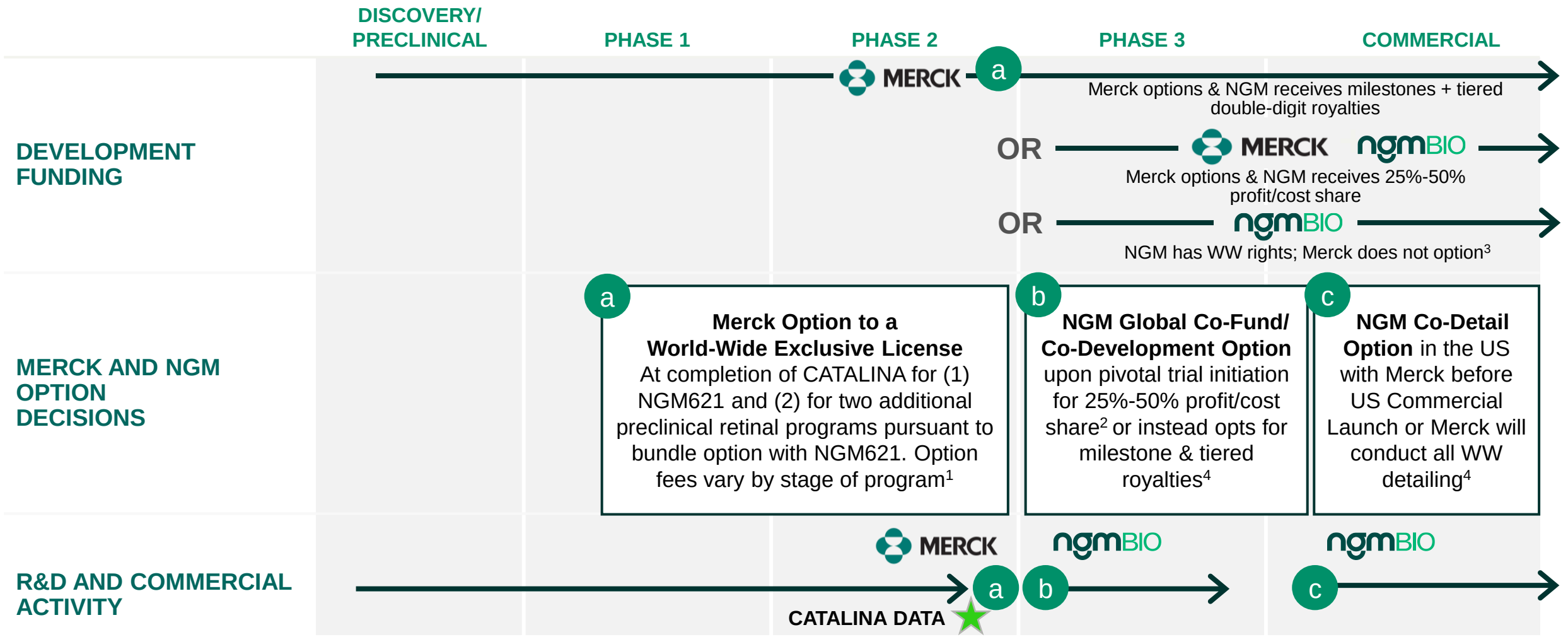
NGM has received over $500M in R&D funding to-date from Merck

MK-3655 was optioned by Merck in 2019, now in a global Phase 2b trial

Collaboration between Merck & NGM was rescoped in July 2021

Ophthalmology is a specific area of focus for the Collaboration

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 retinal 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.*

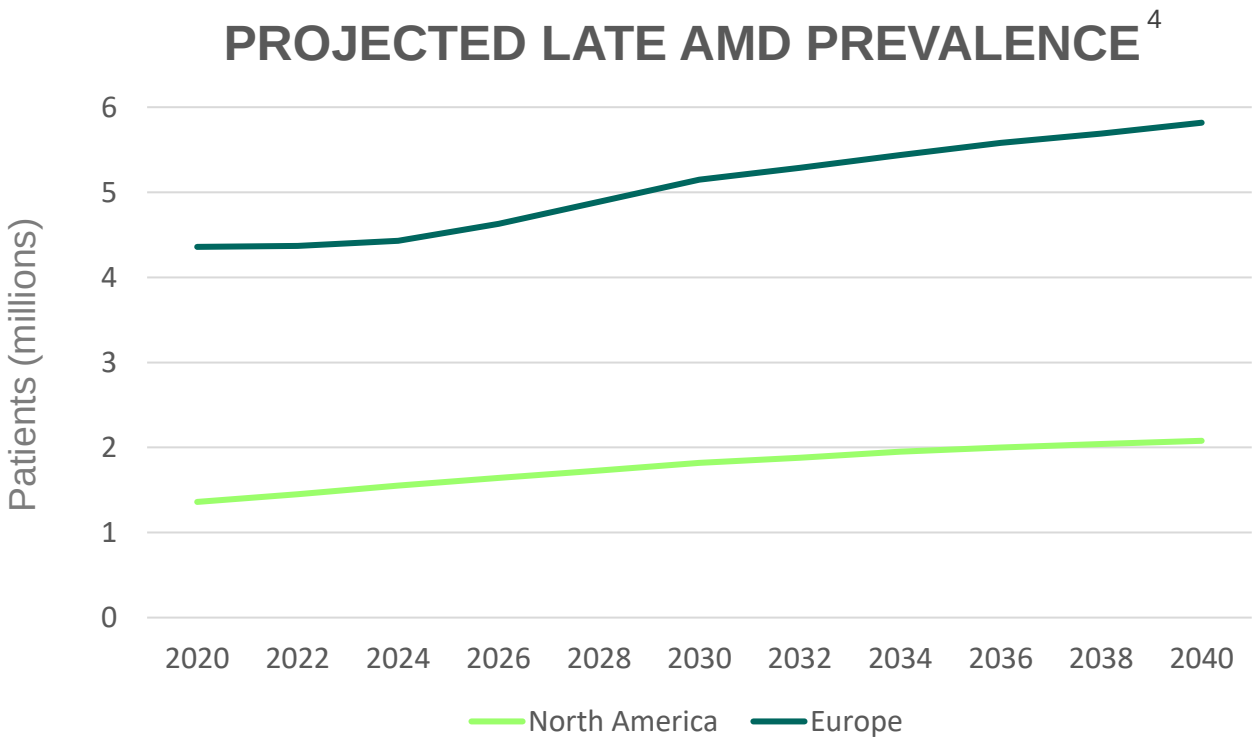
[3] *If Merck chooses not to option NGM621, NGM will have the world-wide rights to NGM621, will be responsible at its own cost for all further R&D and commercial activity, and will owe low single-digit royalties to Merck.*

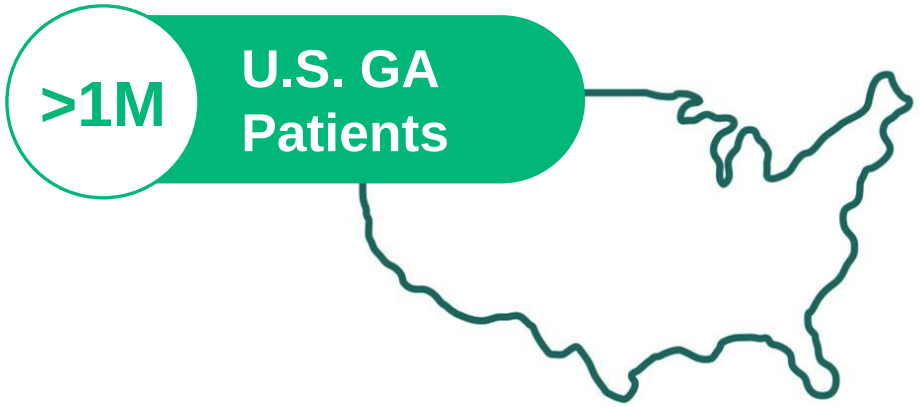
[4] *NGM options to global co-fund/co-development and NGM co-detail in the US are only applicable if Merck exercises its option to NGM621 or the retinal bundle*

For more detail please see Note 5 to NGM's consolidated financial statements included in Part II, Item 8, "Financial Statements and Supplementary Data," of NGM's Annual Report on Form 10-K for the year ended December 31, 2021

The Opportunity for NGM621 is Significant

- GA has **no approved treatments** 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[1,2]





[1]Fleckenstein, 2018; [2]Friedman, 2004; BrightFocus® Foundation; [3]As of 2016. Eye Vis (Lond). 2016; 3: 34.
[4]Wong et al. NEJM 2014. https://www.acms.org/2019/03/age-related-macular-degeneration-in-2019

Four FDA-Approved Therapies Revolutionized the Treatment of Wet AMD

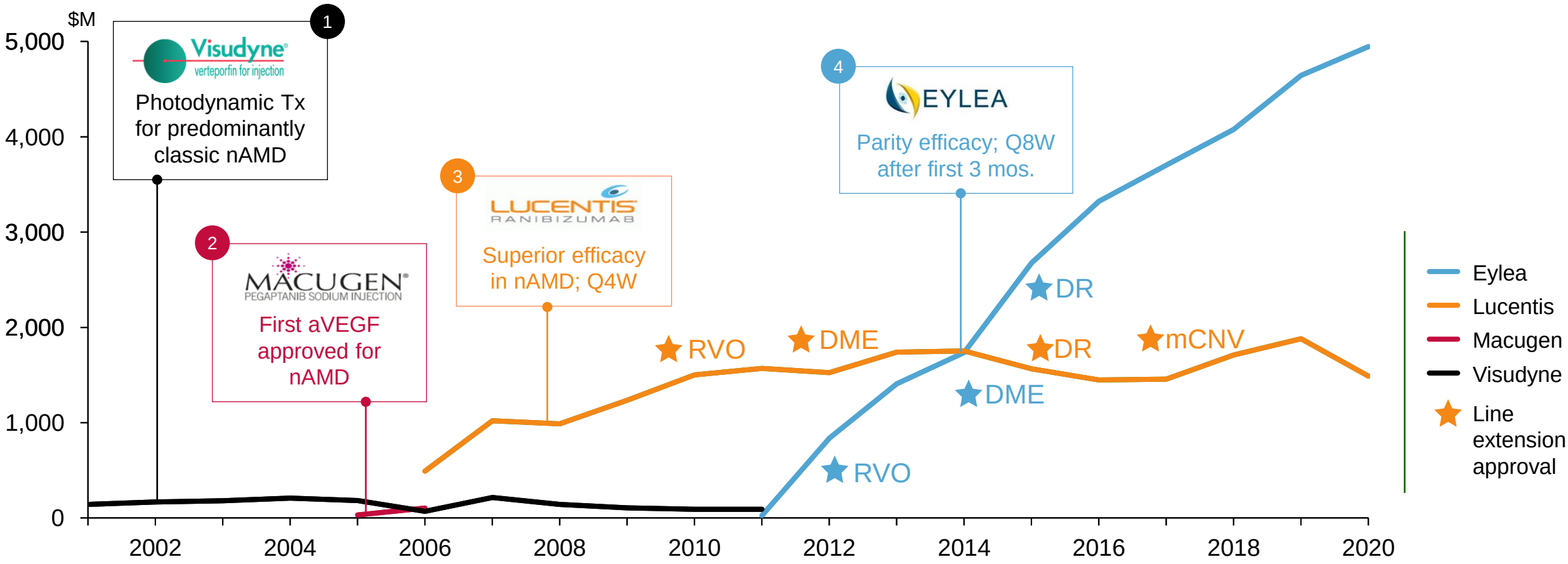
	1 Visudyne® verteporfin for injection	2 MACUGEN® PEGAPTANIB SODIUM INJECTION	3 LUCENTIS® RANIBIZUMAB	4 EYLEA
	Approved April 2000 *Priority Review status*	*Approved Dec 2004* *Fast Track Designation /* *Priority Review status*	*Approved Jun 2006* *Priority Review status*	*Approved Nov 2011* *Priority Review status*
MOA	Infused verteporfin activated by light releases reactive oxygen radicals, resulting in vessel occlusion	Anti-VEGF	Anti-VEGF	Anti-VEGF
Efficacy (Endpoint = Percentage of patients who maintained visual acuity[1] and BCVA letter change over time)	61% PDT vs. 46% sham	70% Macugen vs 55% sham	95% Q4 Lucentis vs. ~60% control[4]; 6.6-10.7 letters gained Q4 Lucentis vs. 14.9-9.8 letters lost control[4]	95% Q4/Q8 Eylea vs. 95% Q4 Lucentis; 7.6-10.9 letters gained Q4/Q8 Eylea vs 8.1-9.4 letters gained Q4 Lucentis
Safety	4.4% of treated patients experienced severe decrease in vision	<1% reported SAEs	<0.1% reported SAEs <2% reported SOAEs[3]	<0.1% reported SAEs
Treatment Frequency	Re-evaluation every 3 months via FA[2], retreatment may be needed	Every 6 weeks	Monthly injections[5]	Bimonthly injections[6]

ngmBIO

Notes. 1. Defined as losing fewer than 15 letters of visual acuity compared from baseline assessment after one year of treatment. 2. FA: fluorescein angiography. 3. SOAEs: serious ocular adverse events. 4. Control arm in one ranibizumab pivotal study (MARINA) was sham and in the other study (ANCHOR) it was PDT. 5. Or PRN (pro re nata)/quarterly injections after 3-4 loading injections (associated with lower efficacy. 6. Bimonthly injections after 3 month loading doses, or monthly injections (similar efficacy), or 12W injections in year 2 (lower efficacy)

Eylea Overtook Lucentis in 3 Years Post-Approval

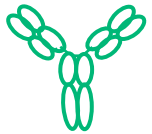
US Sales of Neovascular "Wet" AMD Products* 2001-2020 ($M)



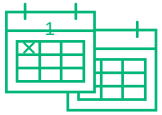
*Sales data inclusive of other approved indications. Source: BioMedTracker

DME = diabetic macular edema; DR = diabetic retinopathy; mCNV = myopic choroidal neovascularization; RVO = macular edema following retinal vein occlusion; OCT = optical coherence tomography

NGM621 has the potential to be the Treatment of Choice in GA



As a mAb, potential for better efficacy compared to other complement inhibitors



Preclinical PK modeling suggests potential for competitive, Q8W IVT dosing

Poised for clean safety/tolerability profile (e.g., less risk of CNV conversion)

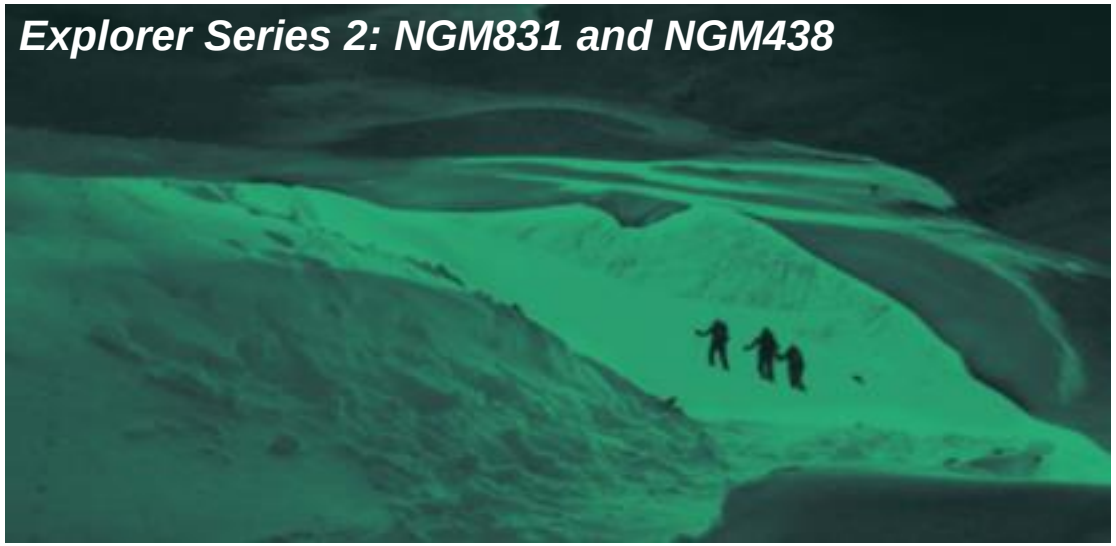
Potential for a large pharma partner with established development and commercialization expertise to drive rapid uptake

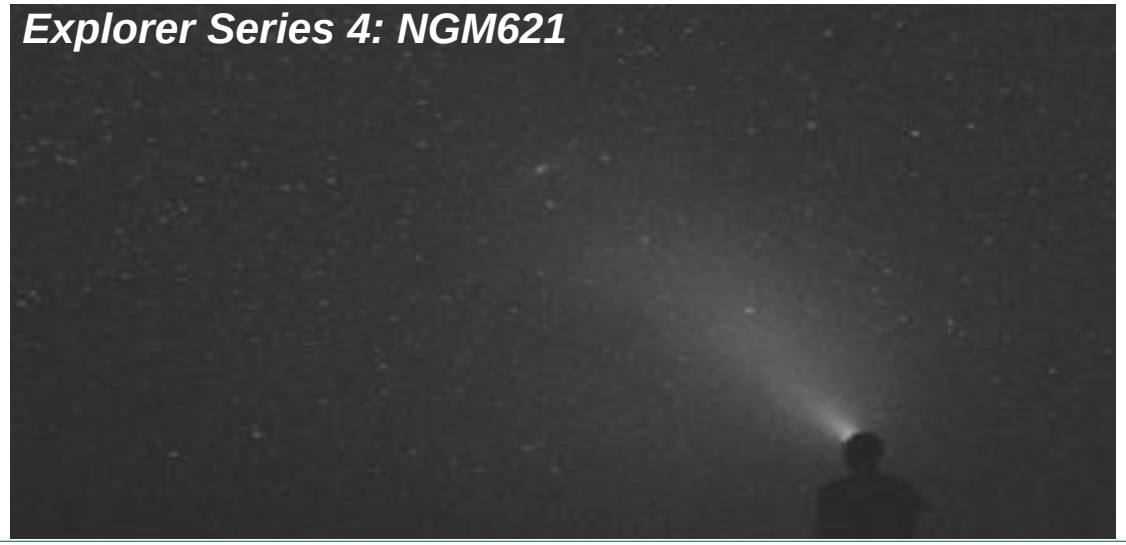
Collaborate closely with motivated, evidence-driven retina specialists

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	**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	Ph2 trial enrolling Ph1a/1b trial ongoing	**Additional Ph1a/1b clinical data readouts in 2H22**	☐
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**	☐

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





Q&A

David Woodhouse, Ph.D.
Chief Executive Officer,
NGM

Erin Henry, Ph.D
Head of Ophthalmology, NGM

Siobhan Nolan Mangini
Chief Financial Officer,
NGM

Charles C. Wykoff, M.D., Ph.D.
Director of Research,
Retina Consultants Houston and the
Greater Houston Retina Research
Foundation