Akari Therapeutics

May 2023

AKARI
THERAPEUTICS

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Akari Overview

1. Novel C5+LTB4 inhibitor

Nomacopan is a unique asset inhibiting 2 co-dependent, proinflammatory targets: complement C5 and leukotriene B4 (LTB4)

2. Broad potential

Potential for use in several diseases; commercial flexibility due to multiple routes of administration (subcutaneous, topical, intravitreal, IV)

3. Robust clinical dataset

Extensive clinical and safety data from multiple clinical trials

4. HSCT-TMA Phase 3

Phase 3 clinical trial in pediatric hematopoietic stem cell transplant-related thrombotic microangiopathy (HSCT-TMA); no approved therapies and ~80% mortality; FDA Orphan, Fast Track, and Rare Pediatric Disease designations; potential for adult follow-on indication

5. GA Pre-Clinical

Pre-clinical program investigating PAS-nomacopan in geographic atrophy (GA) with target dose interval of 3 months or longer without increased risk of choroidal neovascularization (CNV)

Leadership Team

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ALEXION
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Discovered nomacopan
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Volution
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Torsten Hombeck, PhD
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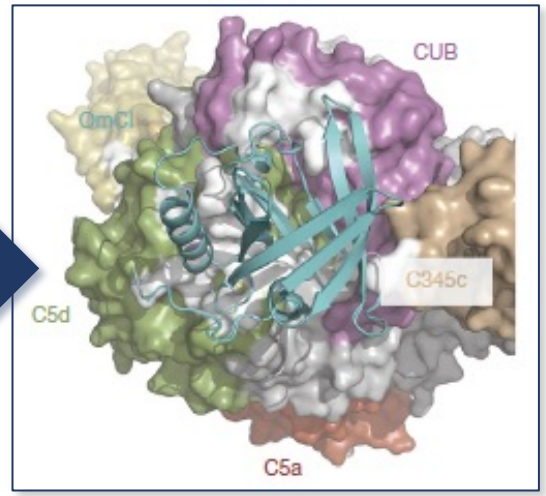
cytonet
GPC biotech

Nomacopan Is a Dual Action Recombinant Protein Discovered In Ticks

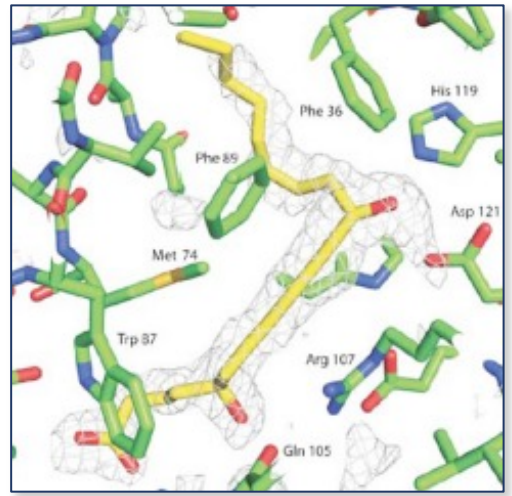
Novel, bispecific nomacopan is a recombinant protein derived from nature via discovery in ticks

Ticks secrete immunomodulatory proteins that help them control host responses (inflammation, pain, itch and blood flow). These are the same responses that may be out of control in certain human autoimmune and inflammatory conditions.

High resolution structure of nomacopan (blue) bound to human complement **C5**[1]



High resolution structure of nomacopan capture of LTB4 (yellow)[2]



- Inhibits complement C5 activation similarly to an on-market complement inhibitor ablating effects of terminal complement activation
- Sequesters LTB4, disrupting activation and recruitment of immune modulating cells responsible for damaging inflammation

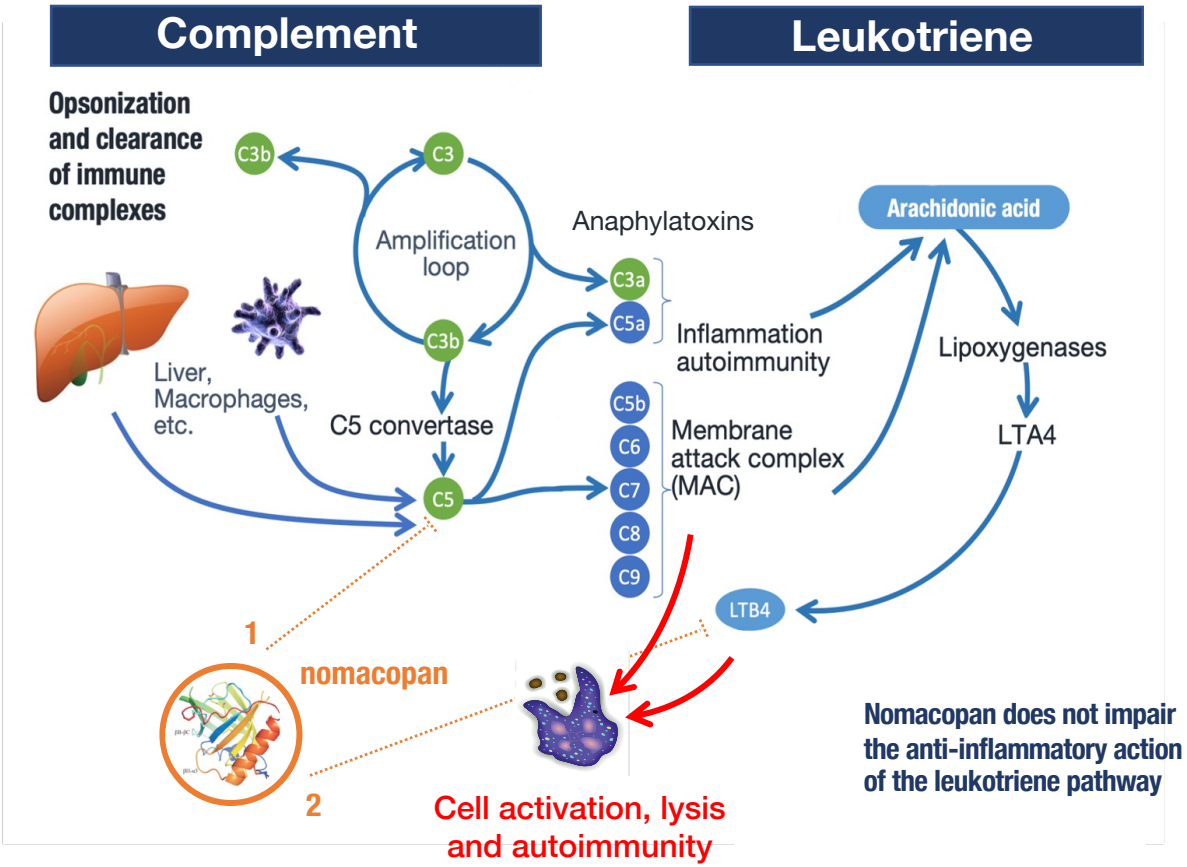
References

1. Jore MM, Johnson S, Sheppard D, et al. Structural basis for therapeutic inhibition of complement C5. *Nat Struct Mol Biol.* 2016;23(5):378-386.
2. Roversi P, Ryffel B, Togbe D, et al. Bifunctional lipocalin ameliorates

Nomacopan Inhibits Two Pathways That Can Cause Damaging Inflammation, While Preserving Important Immune Functions

C5a, LTB4 and MAC act jointly on neutrophils, macrophages and other cell types that can cause inflammation and damage

- Bispecific mechanism prevents two separate, but related, tissue-damaging effects
- Opsonization (antibody binding) and role of complement in clearance of immune complexes that are needed for healthy immune response remain intact
- LTB4 is a key mediator of inflammation that:
  - Is independently activated from complement
  - Can amplify the effects of complement activation
  - Has independent potent inflammatory actions



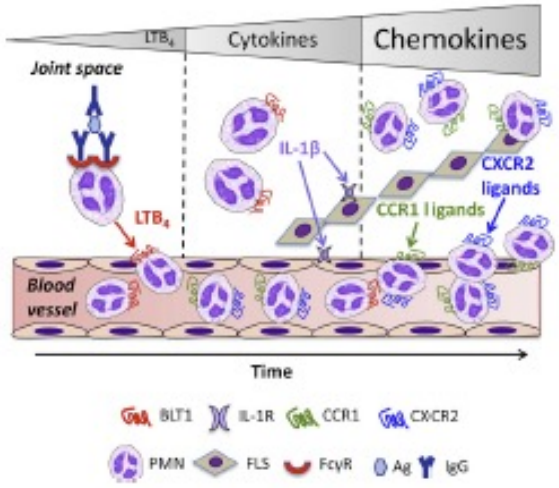
1. Nunn MA, Sharma A, Paesen GC et al. Complement inhibitor of C5 activation from the soft tick Ornithodoros moubata. *J Immunol.* 2005; 174:2084-2091

LTB4 & C5 Are Separate Pathways But In Vivo Data Point to Signalling Interplay That Leads to Damaging Inflammation

In vivo study of autoantibody-induced inflammatory arthritis[1,2]

- Neutrophils infiltrate joints by way of multiple chemoattractant receptors, including LTB4 (BLT1) and chemokine receptors
- In the joint, neutrophils perpetuate their own recruitment by releasing LTB4 and IL-1β
- Complement C5aR activation of neutrophils is required for LTB4 release and early neutrophil recruitment into the joint

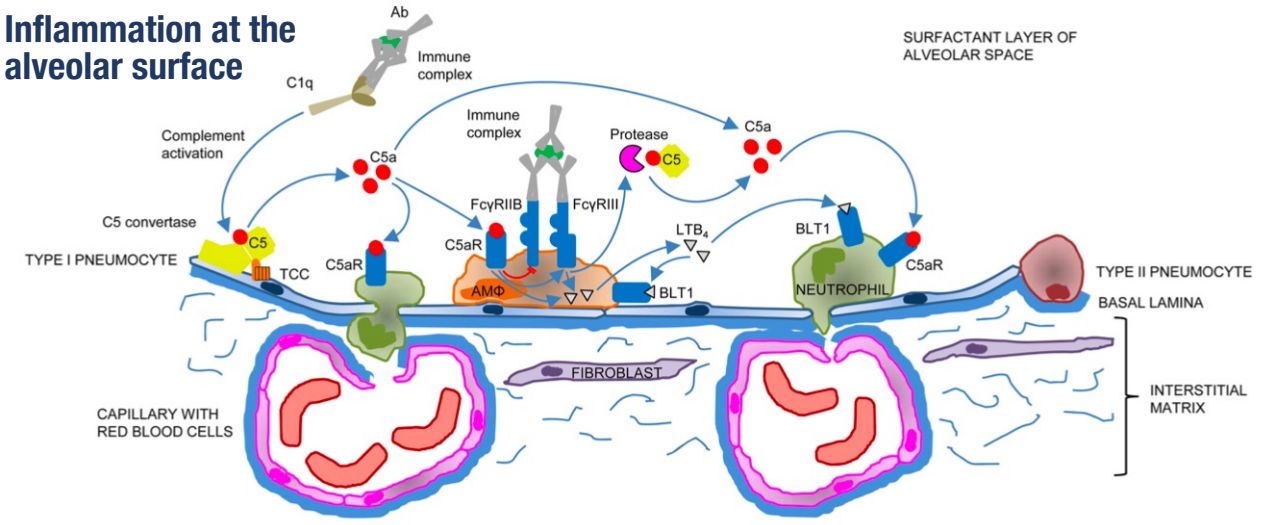
LTB4 initiates cytokine and chemokine cascade in the joint



In vivo study of immune complex-induced acute lung injury (IC-ALI)[3]

- C5 and LTB4 contribute equally to this model of IC-ALI
- C5a receptor signaling regulates Fc receptors promoting inflammation
- Activated alveolar macrophages produce proteases, cytokines & LTB4
- C5a and LTB4 receptor activation upregulate adhesion molecules, recruit & degranulate neutrophils releasing super-oxides, causing further inflammation and microvascular damage

Inflammation at the alveolar surface



References

1. Sadik CD, et al. Neutrophils orchestrate their own recruitment in murine arthritis through C5aR and FcγR signaling. *Proc Natl Acad Sci U S A*. 2012;109(46):E3177-E3185.
2. Sadik CD et al. Lipid-cytokine-chemokine cascades orchestrate leukocyte recruitment in inflammation. *J Leuk Biol.* 2012; 91(2:207-215.;
3. Roversi P, et al. Bifunctional lipocalin ameliorates murine immune complex-induced acute lung injury. *J Biol Chem*. 2013;288(26):18789-18802

Near-Term Potential, Promising Pre-Clinical Program

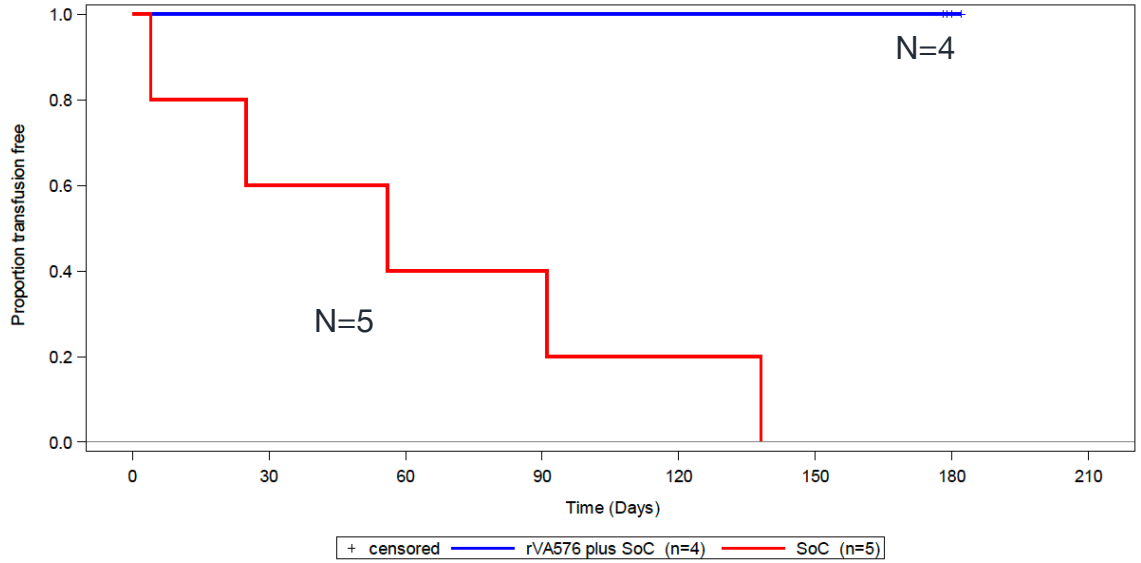
Indication	Candidate/ Formulation	Designations	Pre-Clinical	Phase 1	Phase 2	Phase 3 Parts A & B
Pediatric hematopoietic stem cell transplant–related thrombotic microangiopathy (HSCT-TMA)	Nomacopan/ subcutaneous	FDA Orphan Drug, Fast Track and Rare Pediatric Disease designations				**Advancing to pivotal Part B in >2 years age cohorts, on track to begin enrollment in Q4. Part A remains open for enrollment in <2 years age cohort**
Adult hematopoietic stem cell transplant–related thrombotic microangiopathy (HSCT-TMA)	Nomacopan/ subcutaneous	FDA Orphan Drug Designation			**Study enrollment expected to open in 2024**	
Geographic atrophy (GA)	Long-acting PAS-nomacopan/ intravitreal injection		**IND filing expected in 1H 2024**			

Previous Areas of Clinical Development, Including PNH and BP, Support Current Development Pathways

- In addition to current areas of focus, Akari has conducted clinical research in several other areas, including Phase 2/3 clinical trials of subcutaneous nomacopan for treatment of bullous pemphigoid (BP) and paroxysmal nocturnal hemoglobinuria (PNH)
- This research set a solid foundation for the current Phase 3 clinical trial in pediatric HSCT-TMA

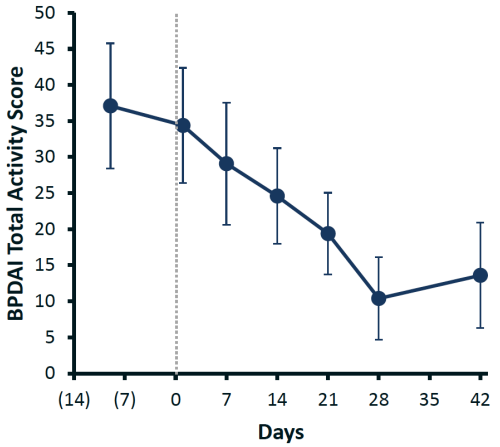
- **In a Phase 3 study in PNH, 100% of untreated patients were transfusion dependent while 0% of nomacopan patients were transfusion dependent**
  - >32 patient years of nomacopan exposure in PNH in 19 patients

Proportion of PNH patients who were transfusion independent following entry to trial

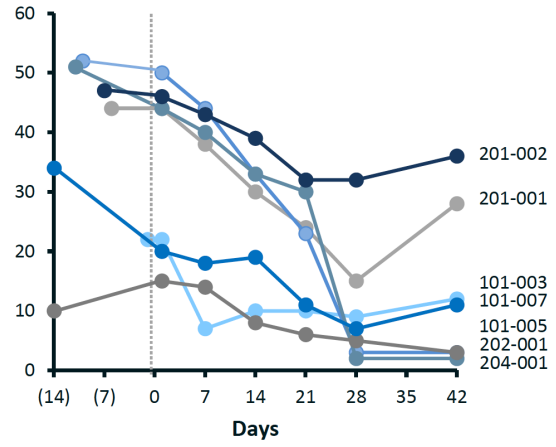


- **In clinical studies of nomacopan in BP, 7 of 9 patients responded to nomacopan[1]**
  - 3 showed >80% reduction in BPDAI by day 42 (BP disease activity)

Mean BPDAI Activity + 90% CI



Individual Patients BPDAI Activity



All prior treatment, including steroids, withdrawn ~one week prior to initiation of treatment with nomacopan. Lesional mometasone was administered to Day 21.

1. Sadik CD, et al. Evaluation of nomacopan for treatment of bullous pemphigoid a phase 2a non0randomized controlled trial. JAMA Dermatol. 2022; 158: 641-649

THROMBOTIC MICROANGIOPATHIES (TMAs)

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Nomacopan May Be the First Treatment for Pediatric HSCT-TMA, a Condition with Mortality Up to 80%

- TMA following a stem cell transplant procedure is a rare but serious complication of HSCT that appears to involve complement activation, inflammation, tissue hypoxia and blood clots, leading to progressive organ damage and death
- Mortality in patients who develop severe transplant-related TMA is 80% (across both adults and children)[1]
- Currently, there are no approved treatment options in the U.S. or Europe

1. Rosenthal J. Hematopoietic cell transplantation-associated thrombotic microangiopathy: a review of pathophysiology, diagnosis, and treatment. *J Blood Med.* 2016;7:181-186.
2. Jodele S, et al. Eculizumab therapy in children with severe hematopoietic stem cell transplantation-associated thrombotic microangiopathy. *Biol Blood Marrow Transplant.* 2014;20(4):518-525.
3. Licht C, et al. Efficacy and safety of eculizumab in atypical hemolytic uremic syndrome from 2-year extensions of phase 2 studies. *Kidney Int.* 2015;87(5):1061-1073.
4. Greenbaum LA, et al. Eculizumab is a safe and effective treatment in pediatric patients with atypical hemolytic uremic syndrome. Kidney Int. 2016;89:701-711. *Kidney Int.* 2016;90(3):709
5. Schols S, Nunn MA, Mackie I et al. Succesful treatment of a PNH patient non-responsive to eculizumab with novel complement C5 inhibitor covers (nomacopan). *Br J Hematol*. 2020; 188: 332-340.
6. Jodele S, et al. Complement blockade for TA-TMA: lessons learned from a large pediatric cohort treated with eculizumab. *Blood*. 2020;135(13):1049-1057.
7. Takatsuka H, et al. Predicting the severity of intestinal graft-versus-host disease from leukotriene B4 levels after bone marrow transplantation. *Bone Marrow Transplant.* 2000;26(12):1313-1316.

1. Efficacy

Efficacy of complement C5 inhibition by eculizumab is supported in HSCT-TMA[2] and atypical hemolytic uremic syndrome (aHUS) [3,4], another TMA; efficacy of nomacopan C5 inhibition supported by clinical PNH research[5]

2. Dosing

Nomacopan clinical trials are establishing a simple, fixed dose for each age category; rapid offset of action allows complement re-activation if/when needed

3. GVHD

Graft versus host disease (GVHD) is commonly present in patients with severe HSCT-TMA[6]; LTB4 is often elevated in patients with GVHD and inhibition of LTB4 may slow GVHD progression[7]

Pediatric & Adult HSCT-TMA U.S. Market Opportunity

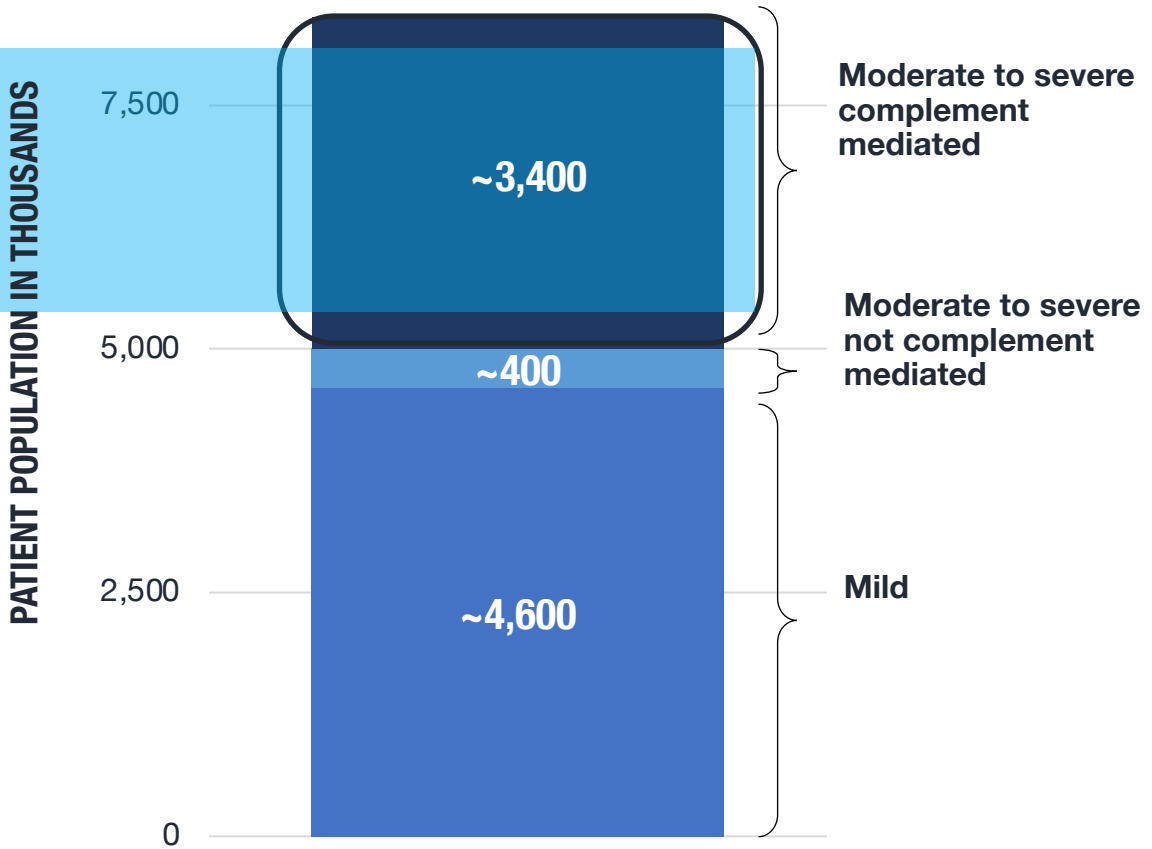
~22,000 HSCTs occur annually in the U.S.

~8,400 (38%) lead to TMAs

~3,400 TMAs are moderate to severe complement-mediated

- 200-300 pediatrics
- 3,100-3,200 adult

Adult population >10X pediatric



References

- Health Resources and Services Administration (HRSA), 2020
- Jodele S, et al. Diagnostic and risk criteria for HSCT-associated thrombotic microangiopathy: a study in children and young adults. Blood. 2014;124(4):645-653.
- Jodele S, et al. Complement blockade for TA-TMA: lessons learned from a large pediatric cohort treated with eculizumab. Blood. 2020;135(13):1049-1057.

Nomacopan in HSCT-TMA Recent Progress

1 **Acceleration into pivotal, registrational Phase 3 Part B**

Based on FDA guidance, moving forward to design and planning for pivotal Part B of Phase 3 clinical trial in pediatric HSCT-TMA patients 2 years to <18 years of age

2 **Addition of new pipeline program in adult HSCT-TMA**

Initiating new program for indication in adults with HSCT-TMA, which will include a study supportive of both adult and pediatric regulatory pathways; adult HSCT-TMA population is >10 times pediatric

3 **Phase 3 Part A to remain open for youngest pediatric patients**

Enrollment for youngest group (0.5 to <2 years) in Part A is not complete and study will stay open for these patients while pivotal Part B study in the older pediatric patients advances; Part A data readout upon completion

Program Acceleration in Pediatric HSCT-TMA

MIDD Participation

Akari was selected to participate in the FDA Model-Informed Drug Development program that is helping accelerate development of treatments, including those for rare pediatric diseases

Predictive PK/PD Model

Akari's robust, predictive PK/PD model simulated 10,000 virtual patients informing FDA MIDD interactions that helped confirm PK/PD model suitability and doses selected for Phase 3 Part A nomacopan study in severe pediatric HSCT-TMA

Rich Data Set

Akari clinical data from 38 subjects (in previous clinical studies and healthy volunteers) support PK/PD model simulations used to select doses for the nomacopan Phase 3 Part A clinical trial in HSCT-TMA

An expanded PK/PD model using data from 55 patients treated with nomacopan was reviewed in the recent Type C interaction with the FDA along with PK/PD data from Part A and found to be predictive supporting simple, fixed dosing in the upcoming Phase 3 pivotal Part B clinical trial

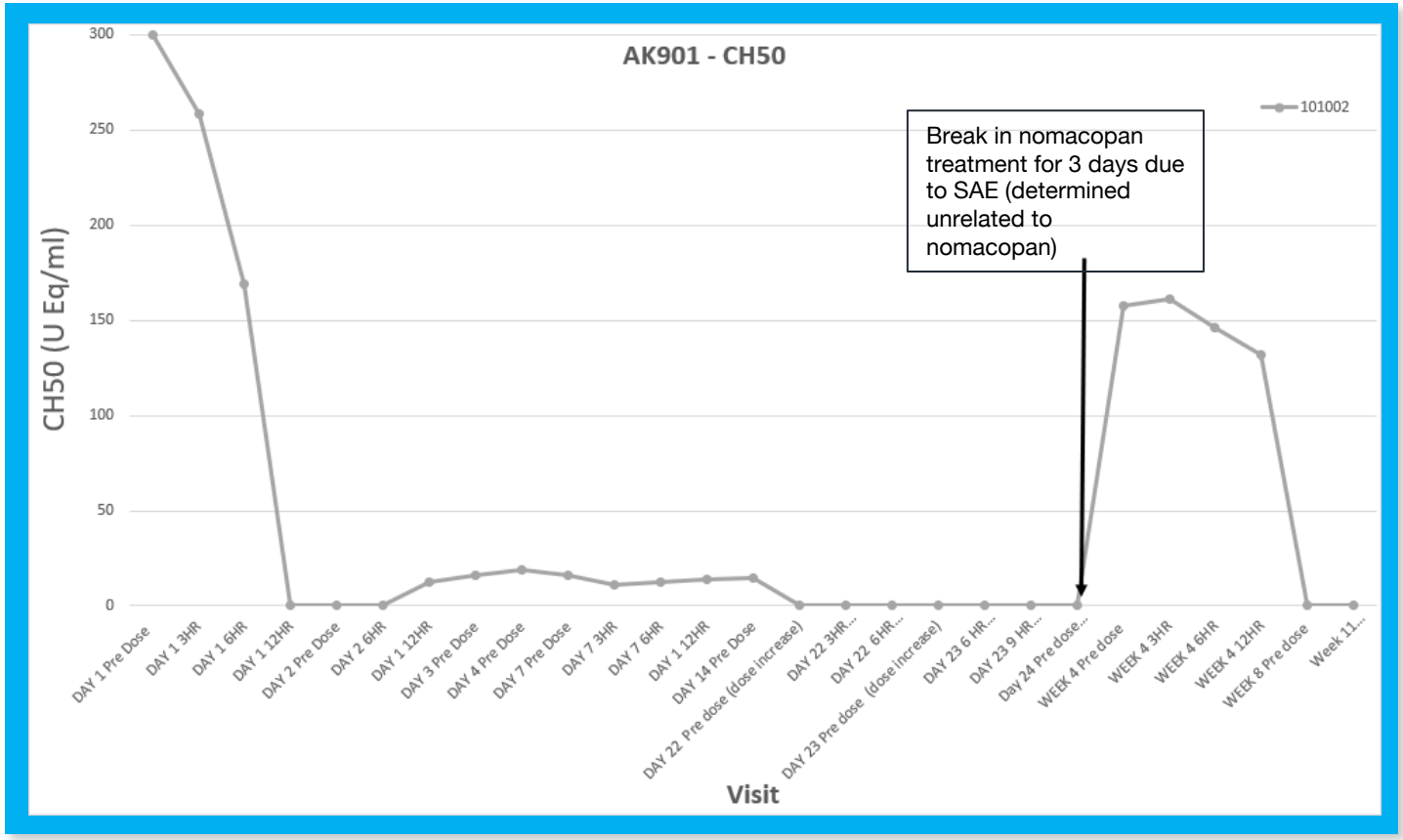
References

1. FDA website. https://www.fda.gov/drugs/development-resources/model-informed-drug-development-pilot-program.

Clinical Trial Patient Case Study Presented As Late-Breaker at Transplantation and Cellular Therapy Tandem Meetings

A patient with severe pediatric HSCT-TMA, which typically involves multi-organ failure and other acute consequences, was discharged home from the hospital following treatment with nomacopan

- 6-year-old male received a cord blood HSCT for relapsed refractory acute myelogenous leukemia (AML)
- Post-transplant acute gut graft-versus-host disease (GVHD)
- TMA at day +66 post-transplant
- Treatment with a single-age, weight-based ablating dose of nomacopan day +74 followed by maintenance dosing for 21 days
- After a 3-day break in treatment for encephalopathy unrelated to nomacopan, treatment continued for a further 46 days until the end of the study with correction of the patient's urine protein creatinine ratio for ≥28 days
- Gut pathology and thrombocytopenia resolved
- No adverse events related to nomacopan



Clinical Response to Nomacopan in the Pediatric HSCT-TMA Setting presented Feb. 16, 2023, at the Transplantation & Cellular Therapy Tandem Meetings. Poster available http://investor.akaritx.com/news-and-events/presentations

GEOGRAPHIC ATROPHY (GA)

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Geographic Atrophy (GA)

- Geographic atrophy (GA) manifests as a chronic progressive degeneration of the macula, which occurs during late-stage dry age-related macular degeneration (dAMD) and can lead to irreversible vision loss
- Approximately 5 million people worldwide are affected,[1,2] with nearly 1 million in the U.S.[3]
- One treatment was approved by the FDA in 2023

References

1. Wong WL, et al. Global prevalence of age-related macular degeneration and disease burden projection for 2020 and 2040: a systematic review and meta-analysis. Lancet Glob Health. 2014;2(2):e106-e116.
2. Rudnicka AR, et al. Age and gender variations in age-related macular degeneration prevalence in populations of European ancestry: a meta-analysis. Ophthalmology. 2012;119(3):571-580.
3. Friedman DS, et al. Prevalence of age-related macular degeneration in the United States [published correction appears in Arch Ophthalmol. 2011 Sep;129(9):1188]. Arch Ophthalmol. 2004;122(4):564-572.

AKARI THERAPEUTICS

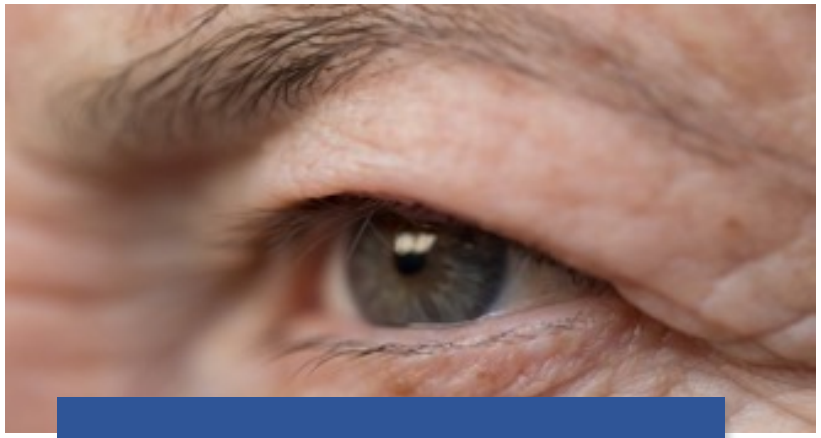
Complement-Only Inhibitors Have Demonstrated Promising Efficacy in GA, Yet Significant Treatment Burdens Exist

Drug	MOA	Stage	Dose interval	Reduction GA growth (mm^2) vs sham[1, 2]	Incidence of CNV[2,3]	Injections/year with no CNV	Injections/year with CNV
pegcetacoplan (Syfovere™)	anti-C3 PEGylated peptide, IVT	FDA approved Feb 2023	25 to 60 days	EM: 17% EOM: 14% (pooled 12-month data DERBY & OAKS)	EM: 12% EOM: 7% SHAM: 3.1% (at 24 months)	6 -14 injections	12 - 22 injections (6 – 8 anti-VEGF)
avacincaptad pegol (Zimura®)	anti-C3 PEGylated aptamer, IVT	PDUFA Aug 2023	Monthly	EM: 17.3% (12-month data GATHER2)	EM: 7.2% SHAM: 3.6% (at 12 months)	12 injections	18 - 20 injections
PAS-nomacopan / Akari	anti-C5 PASylated small protein, IVT	Pre-clinical	3+ months	TBD	Address via LTB4 inhibition/ target equivalent to sham	4 injections or fewer	N/A

- In clinical trials discontinuation for an approved complement-only inhibitor for GA treatment reported up to 20%[4]
- For anti-VEGF CNV treatments, up to 1/3 of patients may discontinue/ not adhere[5]

References: 1. Presentation DERBY and OAKS trial results Oct 11 2021, American Society of Retina Specialists 2021, San Antonio, Texas; 2. Iveric GATHER-2 press release 6 Sept 2022 - table in Supplement showing GATHER-1 and GATHER-2 results at 12-months; 3. Apellis DERBY and OAKS 24 month data press release August 24 2022; 4. Medscape article on 24-month data presentation at AAO 2022 With Approval Pending, Pegcetacoplan Shows Mixed Results for Treating Geographic Atrophy https://www.medscape.com/viewarticle/981813#vp_2 5. McClard CK, et al. Questionnaire to Assess Life Impact of Treatment by Intravitreal Injections (QUALITII). BMJ Open Ophthalmol. 2021;6(1):e000669.

PAS-Nomacopan May Provide 3 Key Benefits: Complement Inhibition, Fewer Doses and LTB4 Inhibition to Address CNV Risk



New PAS-nomacopan construct composition of matter patent filed Dec. 2022; if granted provides patent protection to 2042

1. Efficacy

Efficacy of complement C3 and C5 inhibition slowing progression of GA lesions is well understood[1,2]

2. Frequency of Intravitreal Injections

Frequent needle injections into the back of the eye, a source of fear, discomfort and disruption for patients[3]; **potential for 4 or fewer injections with PAS-nomacopan each year**

3. Safety

LTB4 inhibition may prevent VEGF-A overexpression, a key driver of sight-threatening CNV,[4] a safety risk (treated with VEGF inhibitors) associated with approved and late-stage complement-only inhibitors

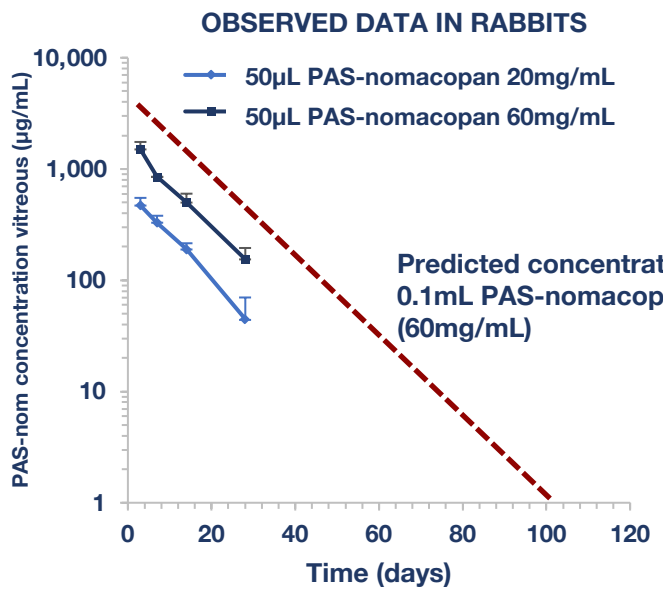
References

1. Liao DS, et al., Complement C3 inhibitor pegcetacoplan for geographic atrophy secondary to age-related macular degeneration - a randomised phase 2 trial. *Opthalmology* 2019; 127: 586-195.
2. Jaffe GJ, et al., C5 inhibitor avacincaptad peg for geographic atrophy due to age-related macular degeneration - a randomised pivotal phase 2/3 trial. *Ophthalmology* 2021; 128: 576-586.
3. McClard CK, et al. Questionnaire to Assess Life Impact of Treatment by Intravitreal Injections (QUALITII): Development of a patient-reported measure to assess treatment burden of repeat intravitreal injections. *BMJ Open Ophthalmol*. 2021;6(1):e000669.
4. Sasaki F, et al., Leukotriene B4 promotes neovascularisation and macrophage recruitment in murine wet-type AMD models. *JCI Insight* 2018; 3: e96902.

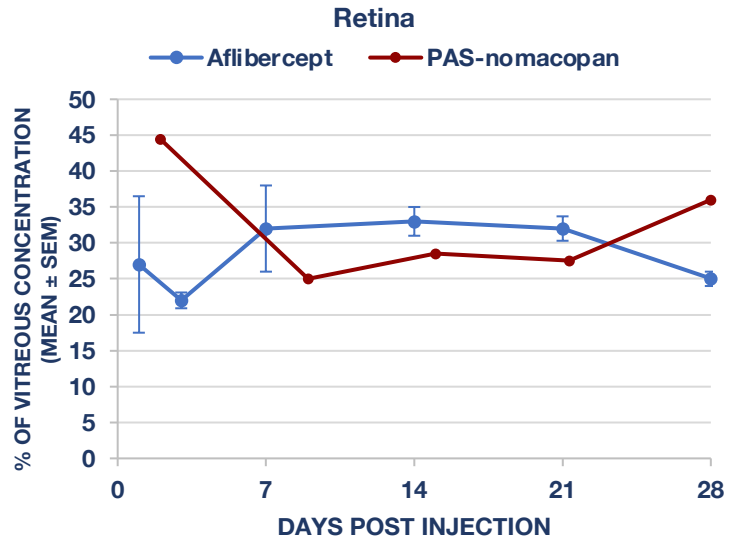
Long-Acting PAS-Nomacopan Has Potential for 4 or Fewer Injections Into the Eye Per Year

- PK/PD data show PAS-nomacopan has extended half-life in the eye after intravitreal injection (7.4 to 8.4 days), suggesting the dose interval may be 3 months or longer[1]

Long-Acting PAS-Nomacopan Has Potential to Extend Intravitreal Dose Interval to 3 Months or Longer



Retinal Bioavailability



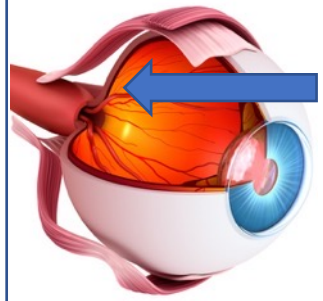
Source: PAS-nomacopan data generated from internal studies

Reference:

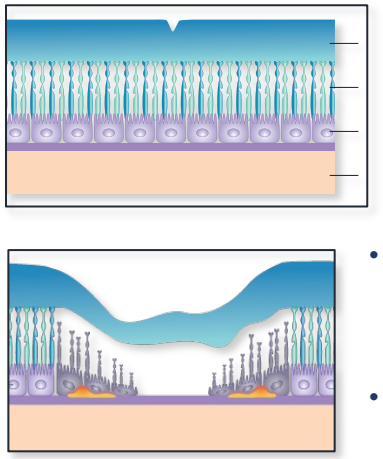
1. Weston-Davies, W., et al. Development of long-acting PAS-nomacopan for treatment of GA and other retinal diseases. Poster presentation ARVO, 2022.

LTB4 Inhibition May Prevent Choroidal Neovascularization

1 CNV starts with inflammation in the choroid and retinal pigment epithelium (RPE)



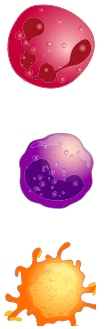
- The **choroid** is part of the vascular layer of the eye[1]
- The **RPE,** adjacent to the choroid, is constantly exposed to high levels of metabolic and oxidative stress[1]



- The RPEs ability to cope with stress decreases with age and the subsequent inflammation damages the RPE and photoreceptors[2]
- Damaged RPE releases leukotrienes, including **LTB4**[2,3]

2 LTB4 activation can lead to over expression of VEGF-A

- In a pre-clinical model of laser-induced CNV LTB4 recruited **inflammatory immune cells** into the retina[3]
- M2 macrophages were attracted and activated via LTB4 receptors leading to **production of vascular endothelial growth factor–A (VEGF-A)** [3]

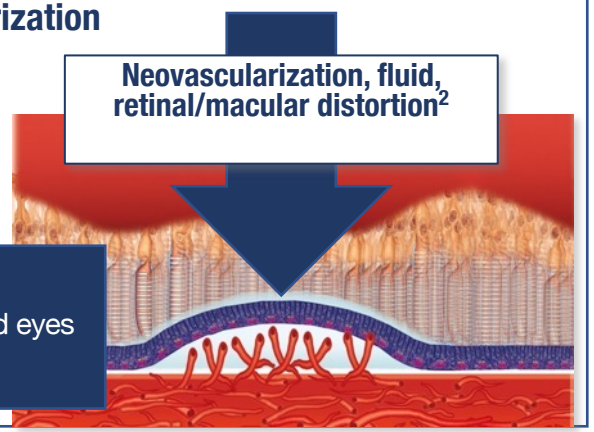


3 Normal expression of VEGF-A is healthy

- VEGF-A is one of the key factors responsible for **endothelial cell** proliferation and migration
- Endothelial cells **form the inner layer of blood vessels** and play a key role in function, including exchanges between blood vessels and surrounding tissues

4 Overexpression of VEGF-A drives choroidal neovascularization

- **CNV is an overdevelopment of blood vessels** in the retina [2]
- New blood vessels are **leaky**, fluid from blood/red blood cells enter the retina [2]
- Fluid can **distort/damage the retina**, including photoreceptors [2]



LTB4 can upregulate the production of VEGF-A, a key driver of CNV. [3]
CNV is responsible for 90% of severe vision loss in AMD patients and eyes with CNV experience greater vision loss than GA only

References:

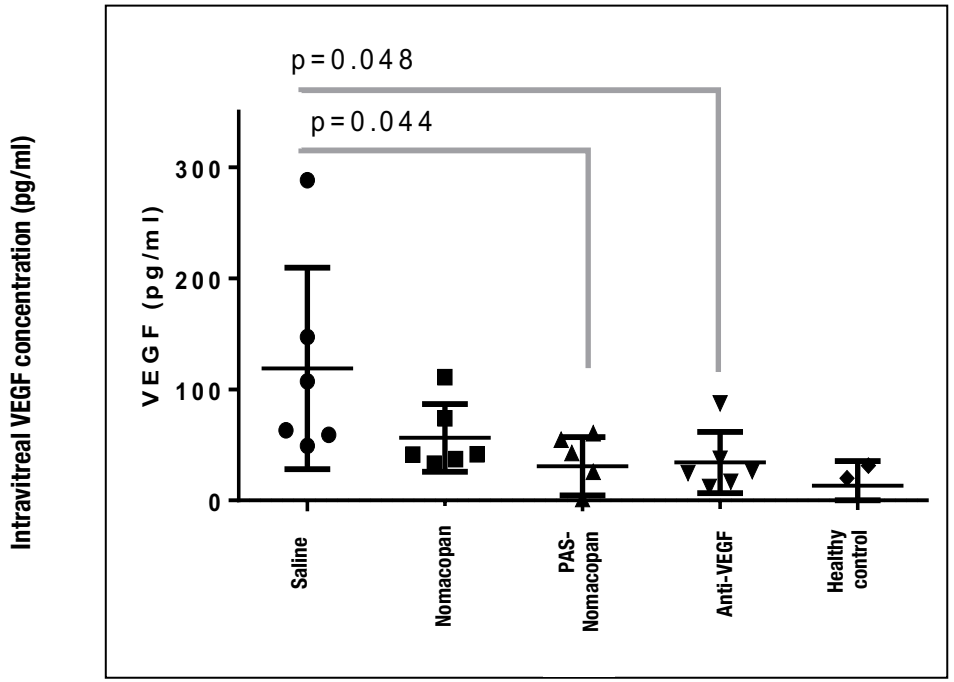
1. Hejtmancik JF, Nickerson JM. Overview of the Visual System. *Prog Mol Biol Transl Sci.* 2015;134:1-4..
2. Grossniklaus HE, Green WR. Choroidal neovascularization. *Am J Ophthalmol.* 2004;137(3):496-503.
3. Sasaki F, Koga T, Ohba M, et al. Leukotriene B4 promotes neovascularization and macrophage recruitment in murine wet-type AMD models. *JCI Insight* 2018;3(18):e96902. Published 2018 Sep 20.
4. Guyer D.R., et al. Subfoveal choroidal neovascular membranes in age-related macular degeneration. Visual prognosis in eyes with relatively good initial visual acuity. *Arch Ophthalmol*. 1986;104:702–705
5. Wong T.Y., et al. The natural history and prognosis of neovascular age-related macular degeneration: A systematic review of the literature and meta-analysis. *Ophthalmology* 2008;115:116–126.

PAS-Nomacopan Decreased VEGF Levels As Effectively As An Anti-VEGF Antibody In a Pre-Clinical Model

In a pre-clinical model of severe uveitis, long-acting PAS-nomacopan (single IVI) decreased VEGF levels (VEGF-A is a key driver of CNV) as effectively as anti-VEGF antibody treatment [1,2]

LTB4 promotes laser induced CNV in a pre-clinical model of wet age related macular degeneration[3]

Effect of PAS-nomacopan on VEGF levels in a standard pre-clinical model of severe uveitis



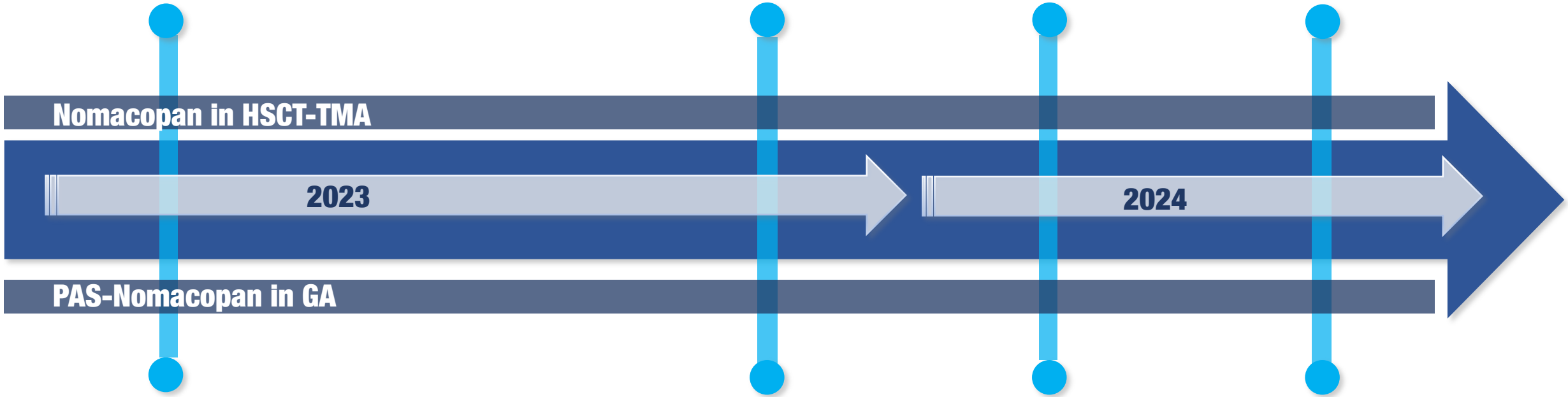
References

1. Eskandarpour M, et al., Leukotriene B4 and its receptor in experimental autoimmune uveitis and in human retinal tissues – clinical severity and LTB4 dependence of retinal Th17 cells. *Am J Pathol.* 2021; 191:320-334
2. Eskandapour M, et al., Immune mediated retinal vasculitis in posterior uveitis and experimental models: the leukotriene (LT)B4-VEGF axis. *Cells* 2021; 10:396
3. Sasaki F, et al., Leukotriene B4 promotes neovascularization and macrophage recruitment inn murine wet-type AMD models. *JCI Insight* 2018; 3:e96902

NEXT STEPS

Next Steps

- Enrollment expected to begin in Phase 3 Part B clinical trial in pediatric HSCT-TMA
- Initiation of clinical trial in adult HSCT-TMA



- IND-enabling studies underway
- Initiate clinical supply
- IND filing expected 1H 2024
- First-in-human/ initiation of clinical trials 2H 2024

FINANCIAL UPDATE

Financial Update

- Ticker: AKTX (NASDAQ)
- 101.1M ADS outstanding
- Cash of $13.2M as of December 31, 2022
- Additional gross proceeds of $4M raised in a registered direct offering in March 2023
- Estimated cash runway into Q4 2023

THANK YOU