UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

Form 6-K

Report of Foreign Private Issuer
Pursuant to Rule 13a-16 or 15d-16
under the Securities Exchange Act of 1934

For the month of: July 2023

Commission file number: 001-36288

AKARI THERAPEUTICS, PLC
(Translation of registrant's name into English)

75/76 Wimpole Street
London W1G 9RT
United Kingdom
(Address of principal executive offices)

Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F.

Form 20-F ☒ Form 40-F ☐

On July 25, 2023, Akari Therapeutics, Plc, a public company with limited liability incorporated under the laws of England and Wales (the "Company"), posted an updated investor presentation on its website. A copy of the Company's presentation is furnished as Exhibit 99.1 to this Report on Form 6-K and is incorporated herein by reference.

Exhibit No.	
99.1	Slide Presentation.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the Registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

Akari Therapeutics, Plc
(Registrant)

By: /s/ Rachelle Jacques
Name: Rachelle Jacques
Title: President and Chief Executive Officer

Date: July 25, 2023

Akari Therapeutics

July 2023

Forward-Looking Statements

Certain statements in this presentation constitute “forward-looking” statements within the meaning of Section 27A of the Securities Act of 1933 and Section 21E of the Securities Exchange A
forward-looking statements reflect the current views of Akari Therapeutics, Plc (the “Company”, “we”,. “our” and “us”) and its plans, intentions, expectations, strategies and prospects, which a
information currently available to it and on assumptions it has made. Although we believe that our plans, intentions, expectations, strategies and prospects as reflected in or suggested by tho
statements are reasonable, we can give no assurance that the plans, intentions, expectations or strategies will be attained or achieved. Furthermore, actual results may differ materially from
forward-looking statements and will be affected by a variety of risks and factors that are beyond our control. Such risks and uncertainties for our company include, but are not limited to: need
fund our operations; our ability to continue as a going concern; uncertainties of cash flows and inability to meet working capital needs; an inability or delay in obtaining required regulatory app
(Coversin) and any other product candidates that may result in unexpected cost expenditures; our ability to successfully develop nomacopan as a treatment for COVID-19 related pneumonia
commercialize any product in that indication; our ability to obtain orphan drug designation in additional indications; risks inherent in drug development in general, and risks specific to the deve
treatments for COVID-19-related illnesses; uncertainties in obtaining successful clinical results for nomacopan and any other product candidates and unexpected costs that may result from d
patients in our clinical trials; failure to realize any value of nomacopan and any other product candidates developed or being developed in light of inherent risks and difficulties involved in suc
product candidates to market; inability to develop new product candidates and support existing product candidates; the approval by the FDA and EMA an any other similar foreign regulatory
competing or superior products brought to market; risks resulting from unforeseen side effects; risk that the market opportunity for nomacopan may not be as large as expected; risks associa
the COVID-19 pandemic; inability to obtain, maintain and enforce patents another intellectual property rights or the unexpected costs associated with such enforcement or litigation; inability t
commercial manufacturing arrangements with third party manufacturers or establish commercial scale manufacturing capabilities; the inability to timely source adequate supply of our active p
ingredients from third-party manufacturers on whom the company depends; unexpected cost increases and pricing pressures and risks and other risk factors detailed in our public filings with
Exchange Commission; including our most recently filed Annual Report on Form 20-F filed with the SEC.

The statements made in this presentation speak only as of the date stated herein, and subsequent events and developments may cause our expectations and beliefs to change. Unless othe
applicable securities law, we do not intend, nor do we undertake any obligation to update or revise any forward-looking statements contained in this presentation to reflect subsequent inform
circumstances or otherwise. While we may elect to update these forward-looking statements publicly at some point in the future, we specifically disclaim any obligation to do so, whether as a
information, future events or otherwise, except as required by law.

Unless otherwise indicated, information contained in this presentation concerning our industry, competitive position and the markets in which we operate is based on information from indepe
research organizations, other third-party sources and management estimates. Management estimates are derived from publicly available information released by third-party sources, as well
research, and are based on assumptions made by us upon reviewing such data, and our experience in, and knowledge of, such industry and markets, which we believe to be reasonable, bu
independently verified the accuracy of this information. Any industry forecasts are based on data (including third-party data), models and experience of various professionals and are based o
all of which are subject to change without notice. In addition, projections, assumptions and estimates of the future performance of the industry in which we operate and our future performanc
to uncertainty and risk due to a variety of factors, including those described in “Cautionary Note Regarding Forward-Looking Statements.” These and other factors could cause results to diffe
expressed in the estimates made by the independent parties and by us.

The trademarks included herein are the property of the owners thereof and are used for reference purposes only. Such use should not be construed as an endorsement of such products.

This presentation shall not constitute an offer to sell or the solicitation of an offer to buy, nor shall there be any sale of these securities in any state or other jurisdiction

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 Phas

Phase 3 clinical trial in pedi hematopoietic stem cell tra thrombotic microangiopathy TMA); no approved therapie mortality; FDA Orphan, Fas designations; Rare Pediatri designation with potential fo Review Voucher upon appr opinion from EMA on orpha designation; potential for ac

5. GA Pre-Clinical

Pre-clinical program investig nomacopan in geographic a with target dose interval of 3 longer without increased ris choroidal neovascularizatio

Leadership Team

Rachelle Jacques
President & CEO

ENZYVANT
ALEXION
Shire
Baxalta
Baxter
Board member
CORBUS PHARMACEUTICALS
uniQure

Miles Nunn, DPhil
Chief Scientific Officer

Discovered nomacopan
Evolutec
Volution
UKRI Natural Environment Research Council

John Neylan, M.D.
Chief Medical Officer

angion
SANOFI
KERYX
genzyme
Wyeth
EMORY TRANSPLANT CENTER
UCDAVIS

Melissa Bradford-Klug
Chief Operating Officer

RARESTONE
KERYX
MAYFIELD
amag
Mallinckrodt Pharmaceuticals
Baxter
Lilly
Board
MassBio

Wendy
Chief Financia

rocket pharma
CENTINEL SPINE
Camber Spine
Board
ExpressCells
NACD

Complement Technologies Continue to Garner Significant Investment

8 acquisitions 2017-2022 **14** collaborations 2017-2022

Company*	Company Value	Product(s)	Status/Phase	Type	Indications
Astra Zeneca / Alexion	$39 billion completed acquisition	Soliris®/Ultomiris®	On market	C5	PNH, aHUS, gMG, N
Apellis	$3.85 billion market cap**	Empaveli®/Syfovre®	On market	C3	PNH/GA
Astellas / Iveric	$5.9 billion definitive agreement	Zimura	Awaiting 2023 approval	C5	GA
Amgen / ChemoCentryx	$3.7 billion completed acquisition	Tavneos®	On market	C5	ANCA-Vasculitis
UCB / Ra Pharma	$2.3 billion completed acquisition	zilucoplan	Phase 3	C5	gMG

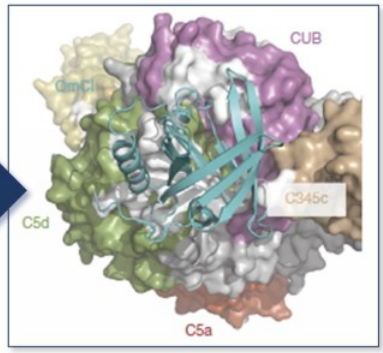
* A selection of companies with complement therapeutics on market or in development ** As of July 24, 2023
Sources: Needham January 2023 Complement report, company public disclosures. Accessed June 7, 2023.

Nomacopan Is a Dual Action Recombinant Protein Discovered In Ticks

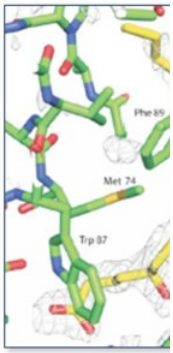
Novel, bispecific nomacopan is a recombinant protein derived from nature via discovery in ticks

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 struc
capture of **LT**



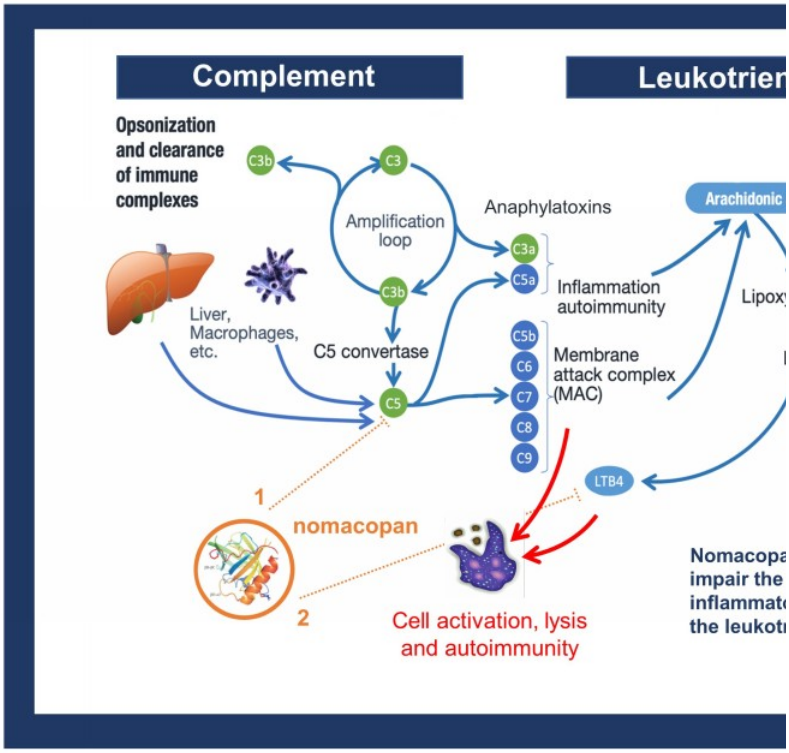
- Inhibits complement C5 activation simil on-market complement inhibitor ablatin terminal complement activation
- Sequesters LTB4, disrupting activation recruitment of immune modulating cells for damaging inflammation

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

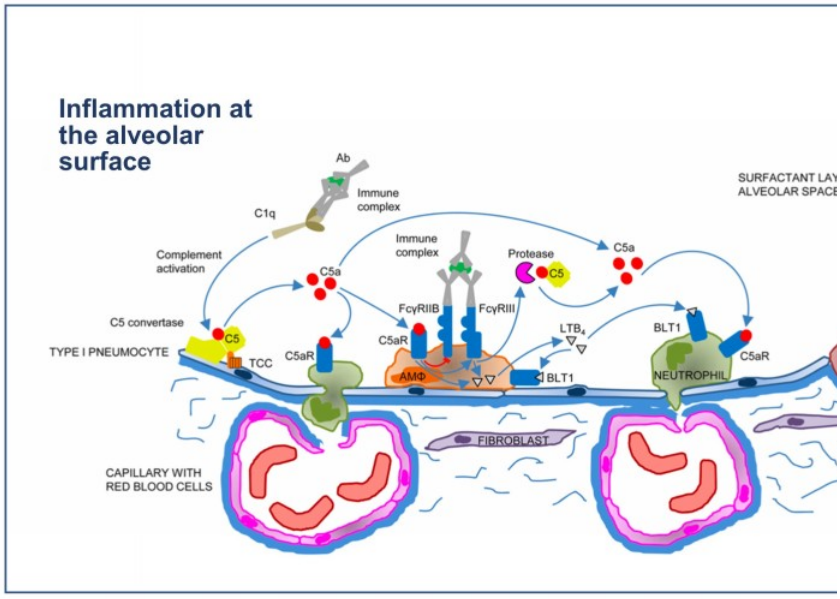


Nunn MA, Sharma A, Paesen GC et al. Complement inhibitor of C5 activation from the soft tick Ornithodoros moubata. *J Immunol.* 2005; 174:2084-2091

C5 and LTB4 Contribute Equally to Inflammation in an In Vivo Model of Immune Complex-Induced Acute Lung Injury

- 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

In vivo study of immune complex-induced acute lung inj

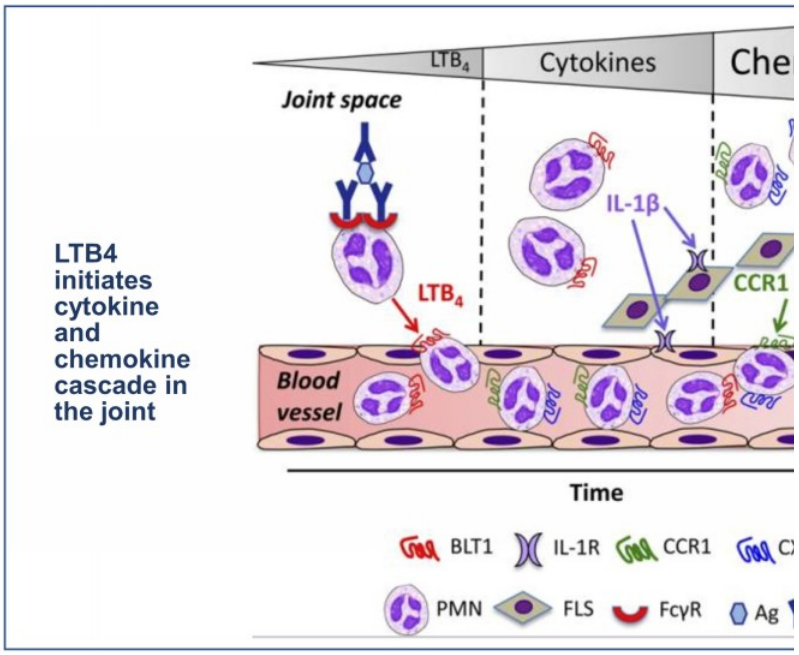


Roversi P, et al. Bifunctional lipocalin ameliorates murine immune complex-induced acute lung injury. *J Biol Chem*. 2013;288(26):18789-18802

In Vivo Data Point to Signalling Interplay Between C5 and LTB4 That Leads to Damaging Inflammation

- 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

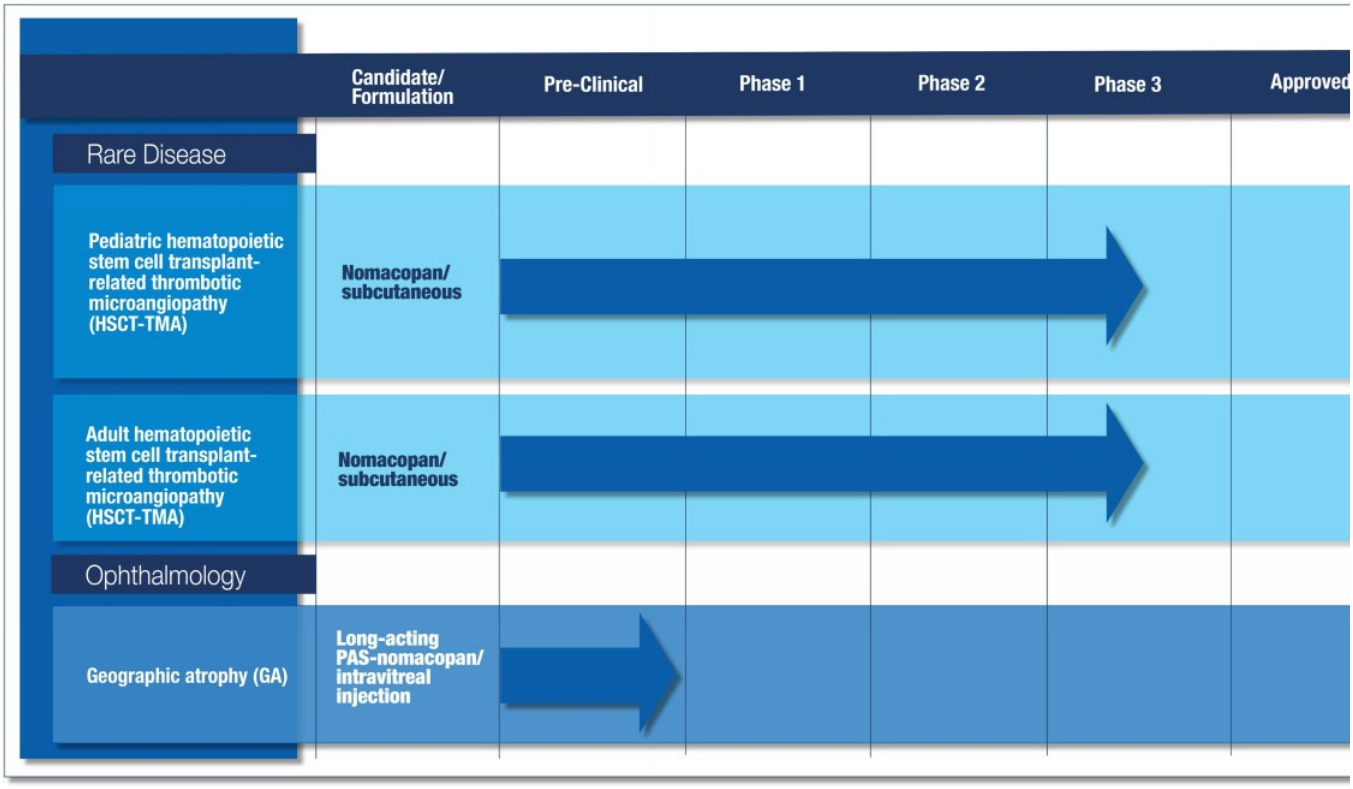
In vivo study of autoantibody-induced inflammatory



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.;

The Akari Pipeline Includes Near-Term Potential, Promising Pre-Clinical Program

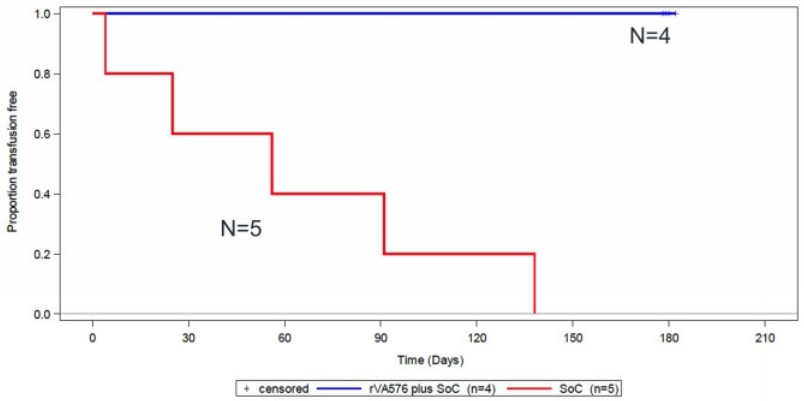


Previous Areas of Clinical Development, Including PNH and BP, Support Current Development Pathways

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

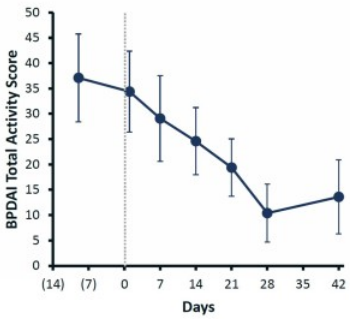
- **In a Phase 3 study in PNH, 100% of untreated patients were transfusion dependent while 0% of nomacopan patients were transfusion dependent**
  - >32 patient years of nomacopan exposure in PNH in 19 patients

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

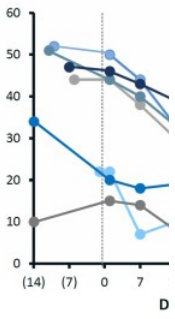


- **In clinical studies of nomacopan in BP, 7 c responded to nomacopan[1]**
  - 3 showed >80% reduction in BPDAI by day 42 (E

Mean BPDAI Activity + 90% CI



Individual Patie



All prior treatment, including steroids, withdrawn ~one week prior with nomacopan. Lesional mometasone was administe

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)

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

- HSCT-TMA is a rare but serious complication of HSCT involving complement activation, inflammation, tissue hypoxia and blood clots, leading to progressive organ damage and death
- Graft versus host disease is commonly present in patients with severe HSCT-TMA[1]
- Mortality is 80% across adults and children (severe)[2]
- No approved treatment options

Nomacopan in HSCT-TM

1. Complement C5 inhibition efficacy

Nomacopan C5 inhibition supported by clinical PNH res

2. Simple, fixed dosing

Nomacopan clinical trials are establishing a simple, fixe children; ease of dosing at home or in hospital for adults

3. Rapid onset & offset of action

Rapid onset/offset of action allows complement re-activa needed

4. LTB4 inhibition may slow GVHD progr

LTB4 is often elevated in patients with GVHD and noma inhibition of LTB4 may slow GVHD progression[4]

1. 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.
2. Rosenthal J. Hematopoietic cell transplantation-associated thrombotic microangiopathy: a review of pathophysiology, diagnosis, and treatment. *J Blood Med*. 2016;7:181-186.
3. 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.
4. 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.

Diagnostic Criteria International Consensus Guidelines Informed Akari Phase 3 Clinical Trials Design

There is consensus that prospective screening and early diagnosis of TMA following transplantation can save lives by heading off the multiorgan dysfunction that is too often irreversible and fatal.

Sonata Jodele, M.D.,
Cincinnati Children's
Hospital Medical Center

Transplantation and Cellular Therapy

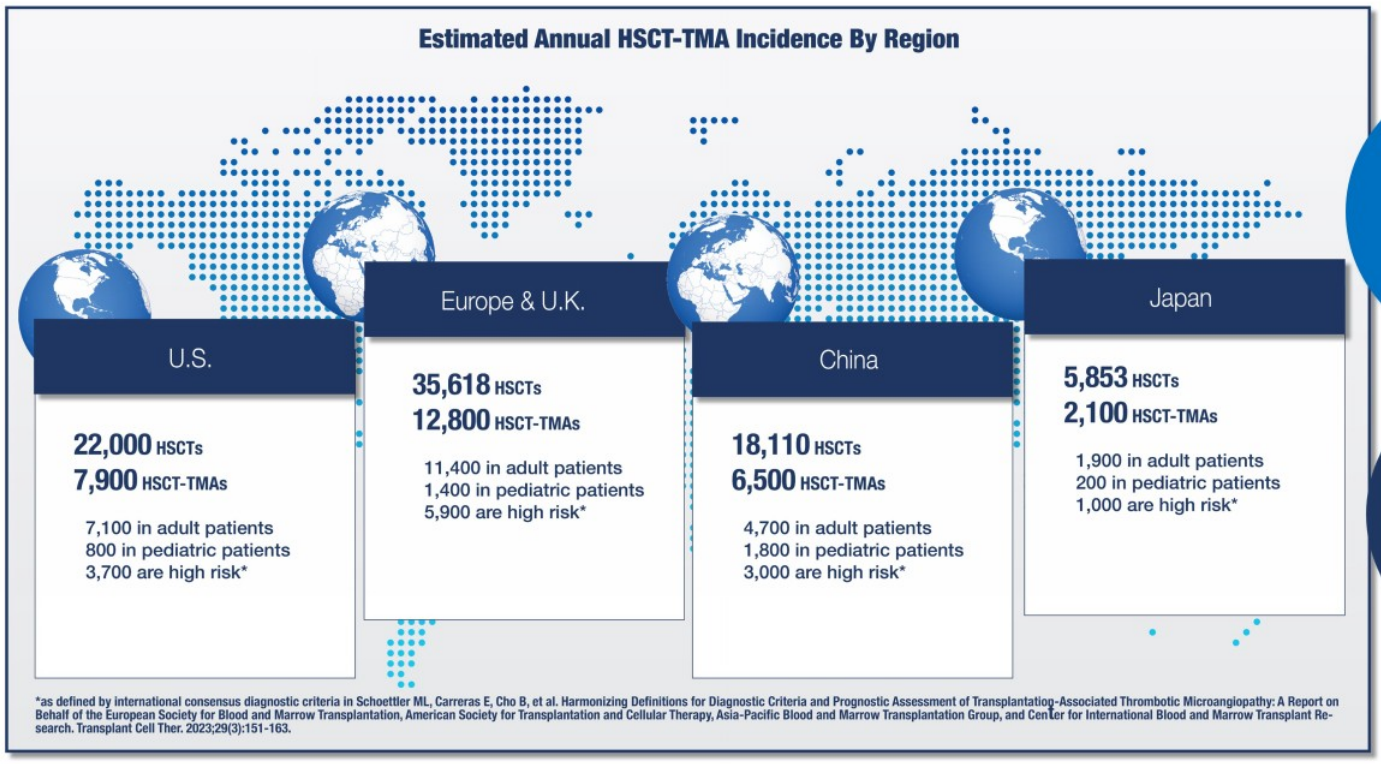
journal homepage: www.tctjournal.org

Guideline

Harmonizing Definitions for Diagnostic Criteria and Prognostic Assessment of Transplantation-Associated Thrombotic Microangiopathy: A Report on Behalf of the European Society for Blood and Marrow Transplantation, American Society for Transplantation and Cellular Therapy, Asia-Pacific Blood and Marrow Transplantation Group, and Center for International Blood and Marrow Transplant Research

- Transplant-associated TMAs are with significant mortality once m dysfunction occurs
- Consensus guidelines develope international panel of experts tha harmonize diagnosis criteria and earlier screening and diagnosis of patients
- The consensus criteria stratify ri standard to high risk, which inclu elevated complement C5 (sC5b- than the upper limit of normal)
- Akari's HSCT-TMA Phase 3 clin design has been significantly inf these consensus criteria for earl diagnosis of high-risk (severe) p

Estimated HSCT-TMA Incidence By Region



Nomacopan in HSCT-TMA Development Programs

Pediatric

- Registrational Phase 3 study of nomacopan in pediatric HSCT-TMA expected to produce safety and efficacy data supportive of a potential regulatory filing and approval on track to **begin enrollment by end of 2023**
- FDA Orphan Drug, Fast Track, and Rare Pediatric Disease designations have been granted
- Positive opinion by the European Medicines Agency (EMA) on orphan drug designation

“It’s important there is harmonization between adult and pediatric studies with the study design, such as inclusion criteria, because comparability in our clinical trials data will not only help future researchers but will also help bring clarity to regulators as they review our applications for approval.”

John Neylan, M.D.
Akari Chief Medical Officer

Adult

- Akari is mov
into a Phase
blind placeb
clinical trial
nomacopan
HSCT-TMA
begin enrol
2024
- FDA Orphar
designation

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

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 prev
clinical studies and healthy volunteers) sup
PK/PD model simulations used to select d
for the nomacopan Phase 3 Part A clinical
HSCT-TMA

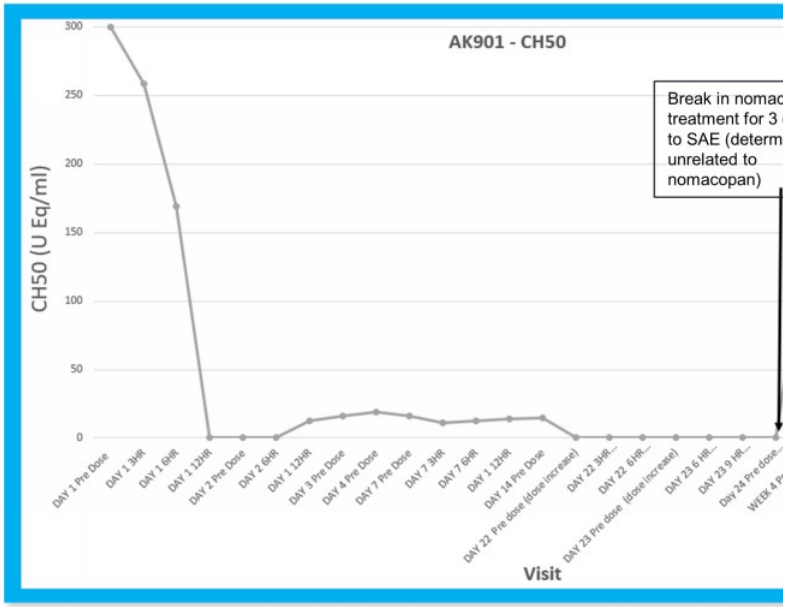
An expanded PK/PD model using data fro
patients treated with nomacopan was revie
the recent Type C interaction with the FDA
with PK/PD data from Part A and found to
predictive supporting simple, fixed dosing
upcoming Phase 3 pivotal Part B clinical tr

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

Clinical Trial Patient Case Study Presented at Two Transplantation and Cellular Therapy Meetings

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

- 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



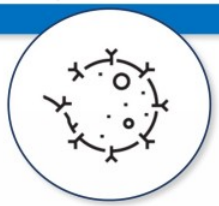
Clinical Response to Nomacopan in the Pediatric HSCT-TMA Setting presented Feb. 16, 2023, at the Transplantation & Cellular Therapy Tandem Meetings. Poster available http://inves events/presentations

Nomacopan May Have Potential As a Treatment for Other TMA Indications in the Future

Immune & autoimmune

- A hallmark of atypical hemolytic uremic syndrome (aHUS) is TMA
  - Incidence of aHUS is ~3,000 U.S. cases a year[1]
  - >600 aHUS-related TMA events each year [6-8]
- Autoimmune conditions such as systemic lupus erythematosus (SLE) are associated with TMA
  - Incidence of autoimmune conditions is ~200,000 per year in the U.S.[3]
  - >1000 TMAs each year are associated with these conditions[4]

Transplant



- Other types of transplant-related TMA, such as solid organ transplant (kidney, liver, lung)
  - ~36,000 solid organ transplants in the U.S. each year[5]
  - >4,300 solid organ transplant-related TMAs each year [6-8]

Cell &

- TMAs are associated CAR T-cel therapies

References

1. aHUS Alliance Ac
2. Bayer, Clin J Am
3. Izmirly, Arthritis R
4. Pivovarova, Disea
5. Health Resources
6. Ávila, Front Med (
7. Shindoh, Am J Tr
8. Verbiest, Blood. 2
9. FDA Cellular Tiss
10. Gavriilaki, Int. J. M
11. Taneja, eJhaem,

GEOGRAPHIC ATROPHY (GA)

Geographic Atrophy (GA)

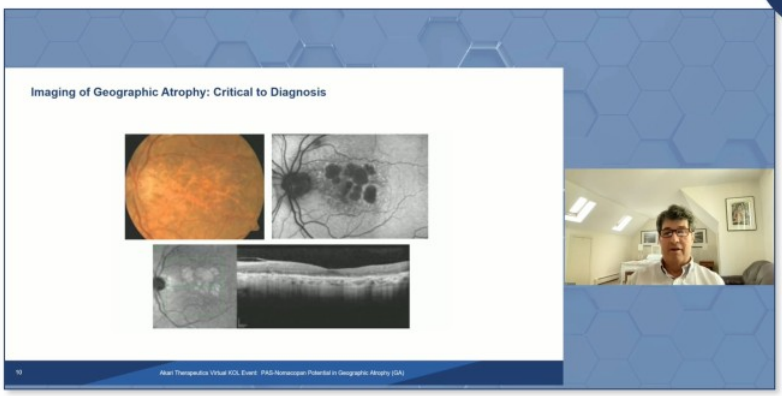
- Geographic atrophy (GA) manifests as a chronic progressive degeneration of the macula, which c during late-stage dry age-related macular degen (dAMD) and can lead to irreversible vision loss
- Approximately 5 million people worldwide are af with nearly 1 million in the U.S.[3]
- The first-and-only treatment for GA was approve FDA in 2023

References

1. Wong WL, et al. Global prevalence of age-related macular degeneration and disease burde for 2020 and 2040: a systematic review and meta-analysis. Lancet Glob Health. 2014;2(2):
2. Rudnicka AR, et al. Age and gender variations in age-related macular degeneration prevale 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 [p correction appears in Arch Ophthalmol. 2011 Sep;129(9):1188]. Arch Ophthalmol. 2004;12

KOL Insights on GA Treatment Landscape and Unmet Needs

The recent key opinion leader event hosted by Akari discussed GA diagnosis, treatment, and significant unmet needs



https://lifescievents.com/event/akari-event/

Despite FDA approval of the first trea for GA, there are still significant unm needs. It's important that we reduce t frequency of therapy, which must be administered through intravitreal inje into the eye. In addition, treating geo atrophy while preventing choroidal neovascularization from developing i another important unmet need.

Elias Reichel, M.D.
Professor of Ophthalmology
Tufts University School of Medicine

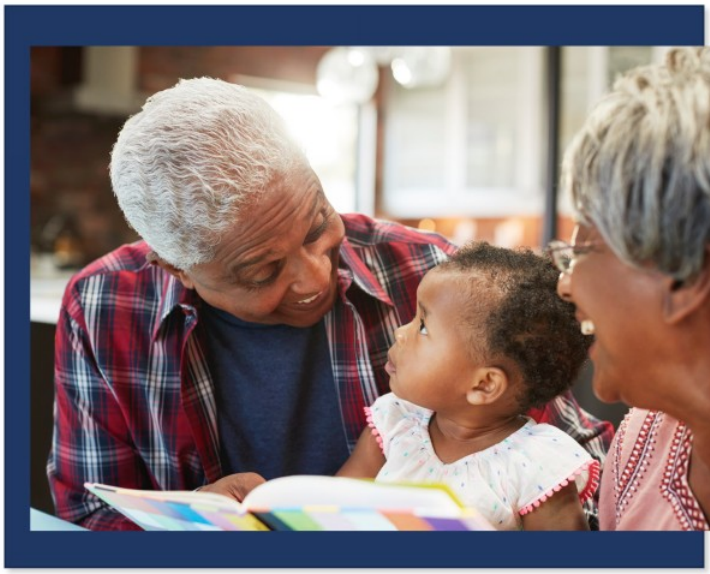
Complement-Only Inhibitors Have Demonstrated Promising Efficacy in GA, Yet Significant Treatment Burdens Exist

Drug	MOA	Stage	Dose interval	Reduction GA growth (mm^2) vs sham[1, 2]	Incidence of CNV[2,3]	Injections/yea with no 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
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
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

- 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 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 a 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 Asses Treatment by Intravitreal Injections (QUALITII). BMJ Open Ophthalmol. 2021;6(1):e000669.

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



PAS-nomacopan in GA

1. Complement C5 inhibition to slow GA

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

2. Fewer needle injections into the eye

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

3. LTB4 inhibition may reduce risk of CN

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

References

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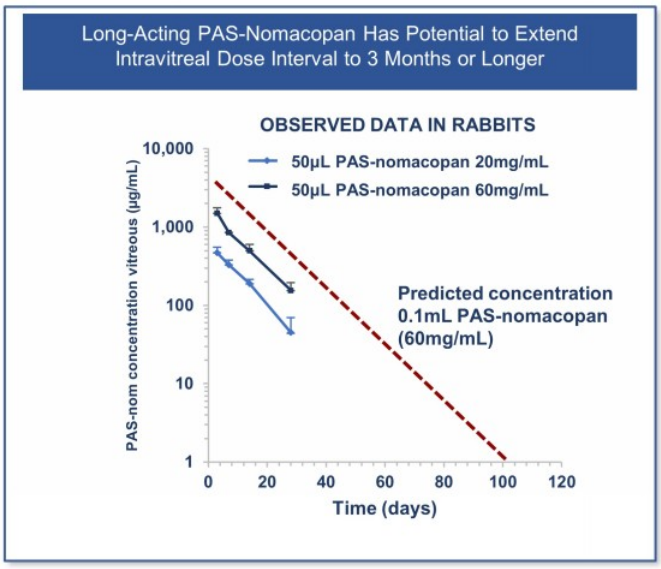
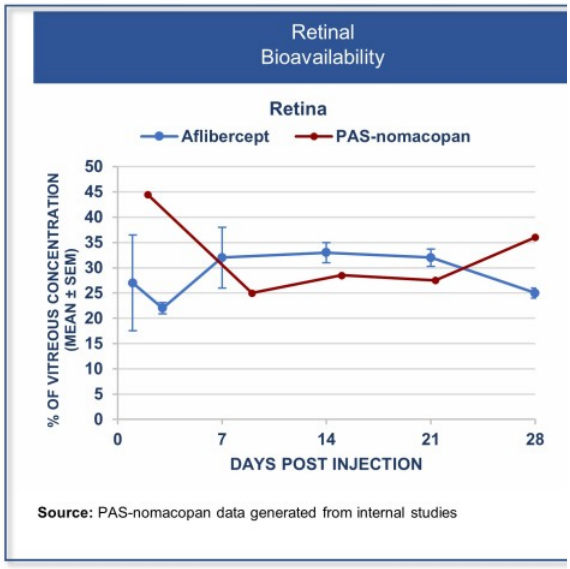
2. Jaffe GJ, et al., C5 inhibitor avacincaptad peg for geographic atrophy due to age-related macular degeneration - a randomised pivotal phase 2/3 trial. *Ophthalmology* 2021; 128: 576-586.
3. McClard CK, et al. Questionnaire to Assess Life Impact of Treatment by Intravitreal Injections (QUALITII): Development of a patient-reported measure to assess treatment burden of repeat intravitreal injections. *BMJ Open Ophthalmol.*
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.
  suggesting the dose interval may be 3 months or longer[1]





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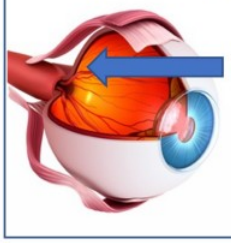
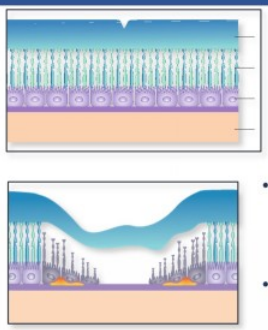
LTB4 Inhibition May Prevent CNV Associated with Approved & Late-Stage Complement-Only Inhibitors

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]

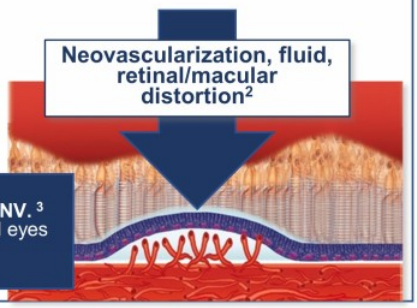
2 LTB4 activa to over exp VEGF-A

- In a pre-clinical moc induced CNV LTB4 **inflammatory imm** into the retina[3]
- M2 macrophages w and activated via LT receptors leading to **of vascular endoth growth factor–A (V**

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



3 Normal expression o healthy

- VEGF-A is one of the key fact responsible for **endothelial c** proliferation and migration
- Endothelial cells **form the inn layer of blood vessels** and p a key role in function, includin exchanges between blood vessels and surrounding tissu

References:

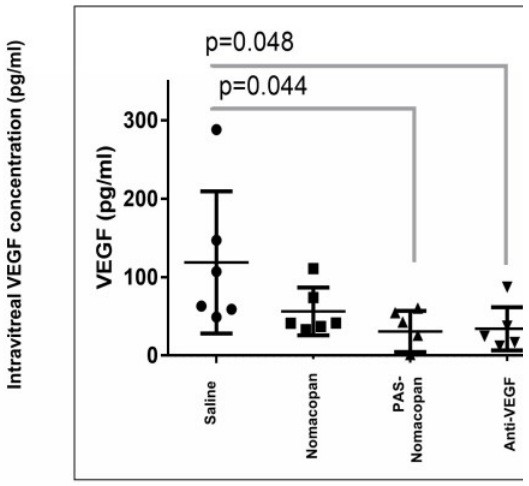
1. Hejtmancik JF, Nickerson JM. Overview of the Visual System. *Prog Mol Biol Transl Sci.* 2015;134:1-4..
2. Grossniklaus HE, Green WR. Choroidal neovascularization. *Am J Ophthalmol.* 2004;137(3):496-503.
3. Sasaki F, Koga T, Ohba M, et al. Leukotriene B4 promotes neovascularization and macrophage recruitment 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
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 l
standard pre-clinical model of sever



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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

PAS-Nomacopan in GA Development Program

Candidate selection

- Selected the PAS-nomacopan candidate that will move forward into clinical trials for treatment of geographic atrophy (GA)
  - Fully active drug potency
  - Planned small (<100µL) injection volume, viscosity enabling intravitreal injection with a fine needle
  - Pre-clinical half-life that supports a potential clinical dose interval of 3 months or longer

Start of clinical trials

- On track for an IND submission in the first half of 2024 and the start of clinical trials in the second half of 2024

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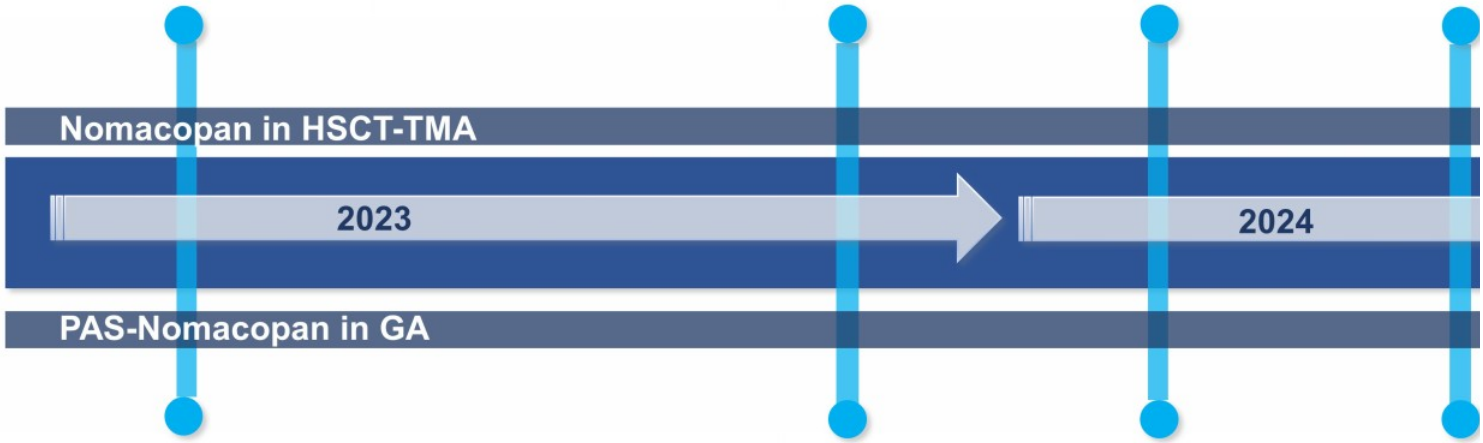
“As we complete the final stages before anticipated submission of an IND in first half of 2024, we are confident we’ve chosen the asset that positions us to succeed in the clinical trials we expect initiate in the second half of 2024.

Rachelle Jacques, Akari President & CEO

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 initia clini 2H 2

FINANCES

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