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: May 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 May 22, 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.

The information in slides 24 and 26 of such presentation is hereby incorporated by reference into all effective registration statements filed by the Company under the Securities Act of 1933, as amended.

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: May 22, 2023

Exhibit 99.1

Akari Therapeutics

May 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 p

Potential for use
diseases; comm
due to multiple r
administration (s
topical, intravitre

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-C

Pre-clinical progr
PAS-nomacopan
atrophy (GA) witl
interval of 3 mon
without increase
neovascularizatio

Leadership Team

Rachelle Jacques
President & CEO

ENZYVANT
ALEXION
Shire
Baxalta
Baxter
Board member
CORBUS
uniQure

John Neylan, MD
Chief Medical Officer

angion
KERYX
SANOFI
genzyme
Wyeth
EMORY TRANSPLANT CENTER
UCDAVIS

Melissa Bradford-Klug
Chief Operating Officer

RARESTONE
MAYFIELD
KERYX
amag
Mallinckrodt Pharmaceuticals
Baxter
Lilly
Board member
MassBio

Miles Nunn, DPhil
Chief Scientific Officer

Discovered nomacopan
Evolutec
Volution
UKRI Natural Environment Research Council

Torsten Ho
Chief Finar

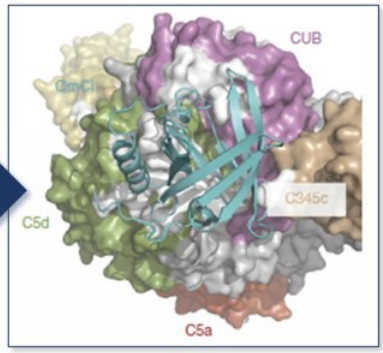
ogennix
GPC

Nomacopan Is a Dual Action Recombinant Protein Discovered In Ticks

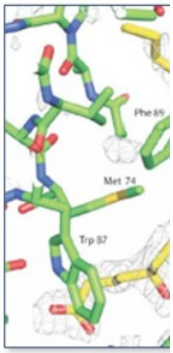
Novel, bispecific nomacopan is a recombinant protein derived from nature via discovery in ticks

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**



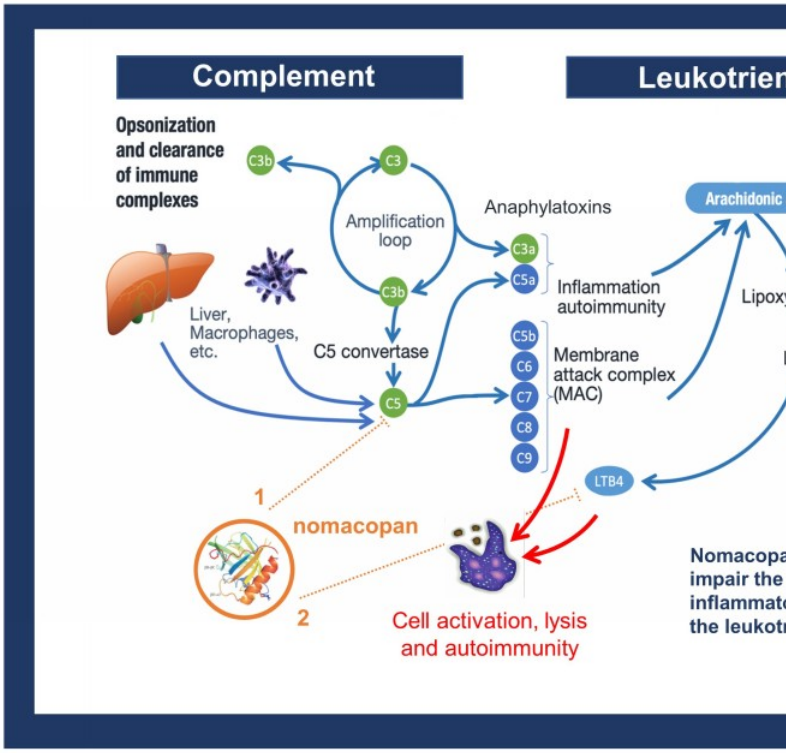
- Inhibits complement C5 activation simi market complement inhibitor ablating e terminal complement activation
- Sequesters LTB4, disrupting activation recruitment of immune modulating cell for damaging inflammation

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



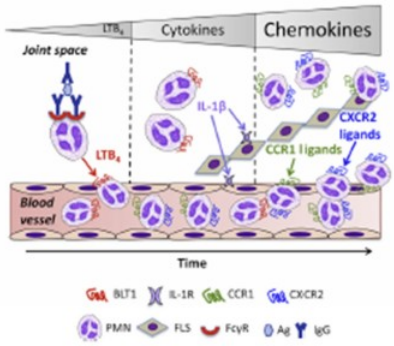
1. Nunn MA, Sharma A, Paesen GC et al. Complement inhibitor of C5 activation from the soft tick Ornithodoros moubata. *J Immunol.* 2005; 174:2084-2091

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

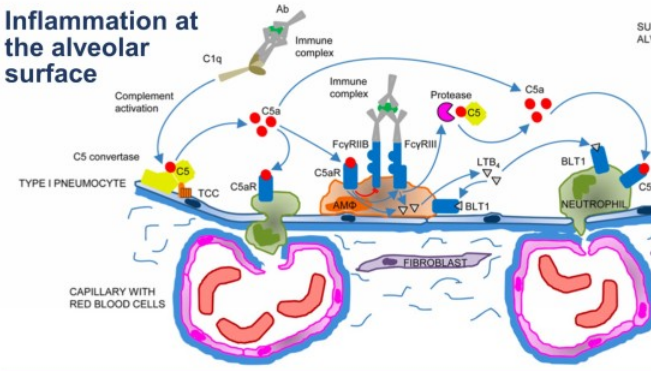
LTB4 initiates cytokine and chemokine cascade in the joint



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

- C5 and LTB4 contribute equally to this model of IC-ALI
- C5a receptor signaling regulates Fc receptors promoting
- Activated alveolar macrophages produce proteases, cyto
- C5a and LTB4 receptor activation upregulate adhesion m degranulate neutrophils releasing super-oxides, causing f 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

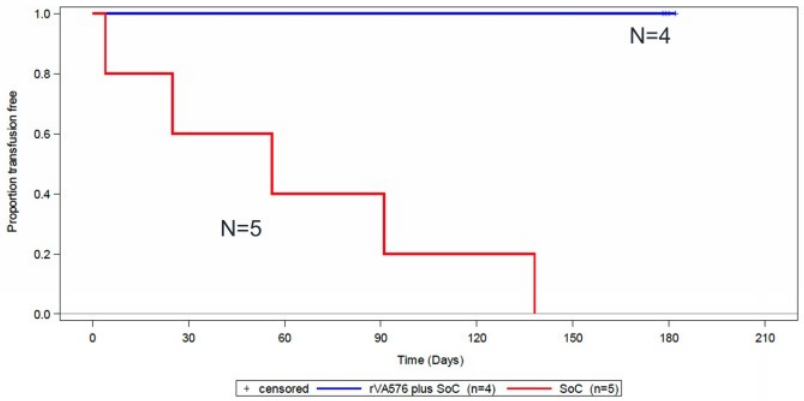
Indication	Candidate/ Formulation	Designations	Pre-Clinical	Phase 1	Phase 2	
Pediatric hematopoietic stem cell transplant–related thrombotic microangiopathy (HSCT-TMA)	Nomacopan/ subcutaneous	FDA Orphan Drug, Fast Track and Rare Pediatric Disease designations				**Adva Part coho begi Part enrol age**
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 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

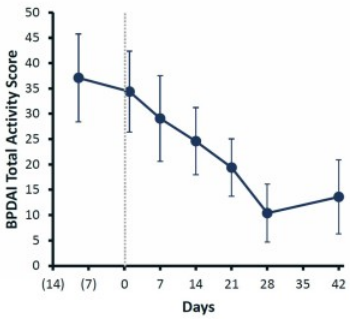
- **In a Phase 3 study in PNH, 100% of untreated patients were transfusion dependent while 0% of nomacopan patients were transfusion dependent**
  - >32 patient years of nomacopan exposure in PNH in 19 patients

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

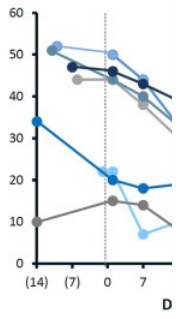


- **In clinical studies of nomacopan in BP, 7 c responded to nomacopan[1]**
  - 3 showed >80% reduction in BPDAI by day 42 (E

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 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 ecu
supported in HSCT-TMA[2] and atypical hemo
syndrome (aHUS) [3,4], another TMA; efficacy
C5 inhibition supported by clinical PNH rese

2. Dosing

Nomacopan clinical trials are establishing a s
dose for each age category; rapid offset of ac
complement re-activation if/when needed

3. GVHD

Graft versus host disease (GVHD) is commor
patients with severe HSCT-TMA[6]; LTB4 is oft
patients with GVHD and inhibition of LTB4 ma
progression[7]

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

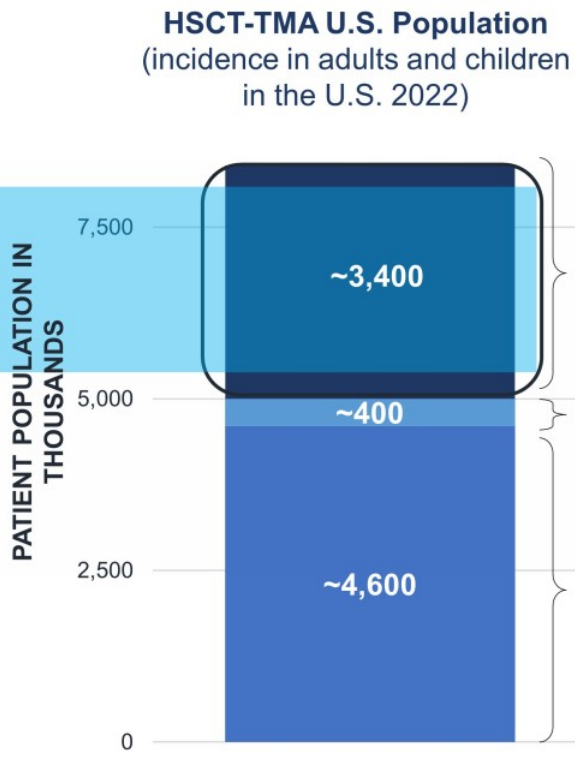
~22,000 HSCTs occur annually in the U.S.

~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**

Enrollment for you
group (0.5 to <2 y
Part A is not comp
study will stay ope
these patients wh
Part B study in the
pediatric patients
Part A data readou
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

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

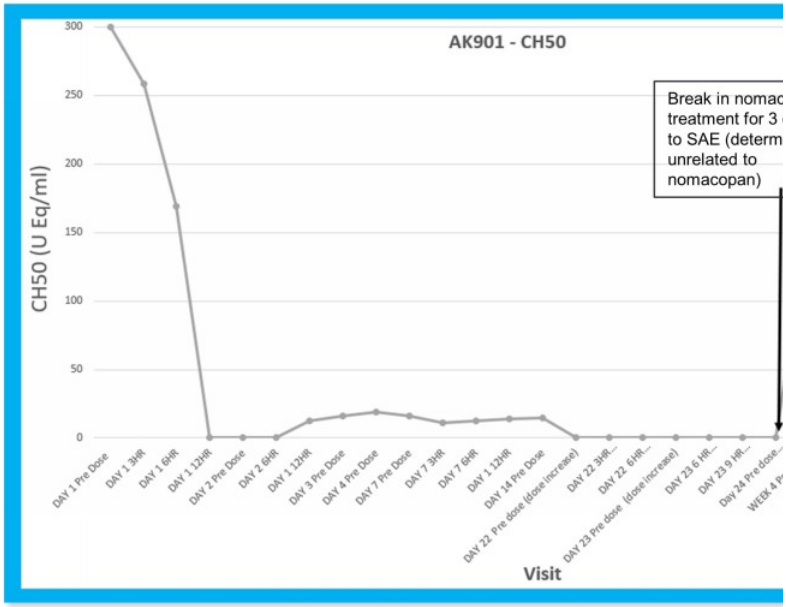
An expanded PK/PD model using data fro
patients treated with nomacopan was revie
the recent Type C interaction with the FDA
with PK/PD data from Part A and found to
predictive supporting simple, fixed dosing
upcoming Phase 3 pivotal Part B clinical tr

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

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

GEOGRAPHIC ATROPHY (GA)

Geographic Atrophy (G

- Geographic atrophy (GA) manifests as a chronic progressive degeneration of the macula, which o during late-stage dry age-related macular degene (dAMD) and can lead to irreversible vision loss
- Approximately 5 million people worldwide are affe 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 for 2020 and 2040: a systematic review and meta-analysis. Lancet Glob Health. 2014;2(2):e
2. Rudnicka AR, et al. Age and gender variations in age-related macular degeneration prevalen 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 [pu correction appears in Arch Ophthalmol. 2011 Sep;129(9):1188]. Arch Ophthalmol. 2004;122

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 at 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 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

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.

1. Efficacy

Efficacy of complement C3 and C5 inhibiti
progression of GA lesions is well understo

2. Frequency of Intravitreal Inj

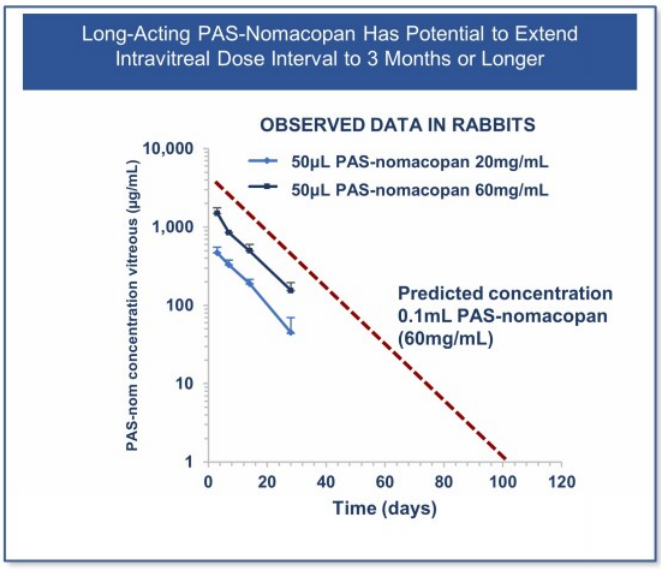
Frequent needle injections into the back c
source of fear, discomfort and disruption f
potential for 4 or fewer injections with
nomacopan each year

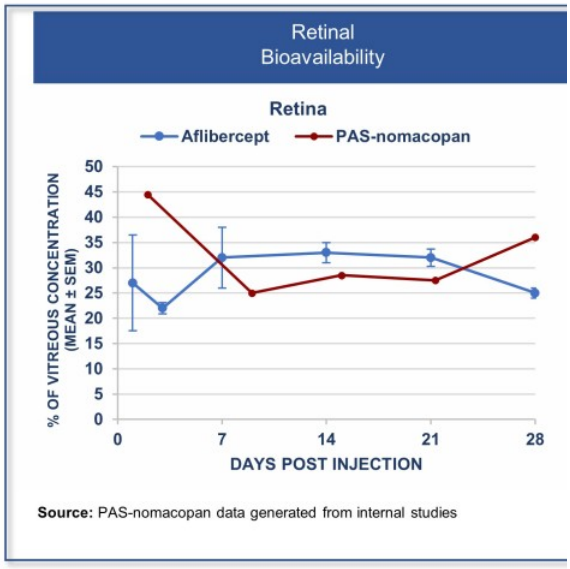
3. Safety

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

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]

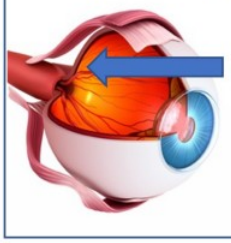




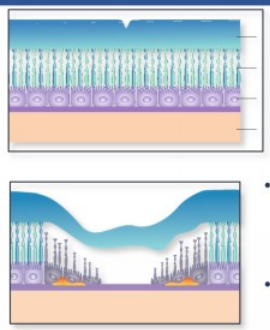
Reference:
1. Weston-Davies, W., et al. Development of long-acting PAS-nomacopan for treatment of GA and other retinal diseases. Poster presentation ARVO, 2022.

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]

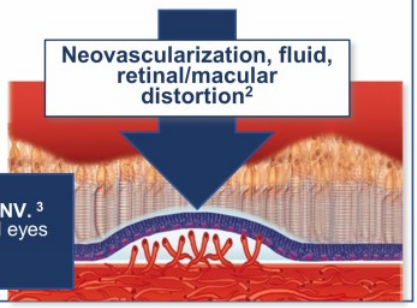
2 LTB4 activa to over exp VEGF-A

- 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

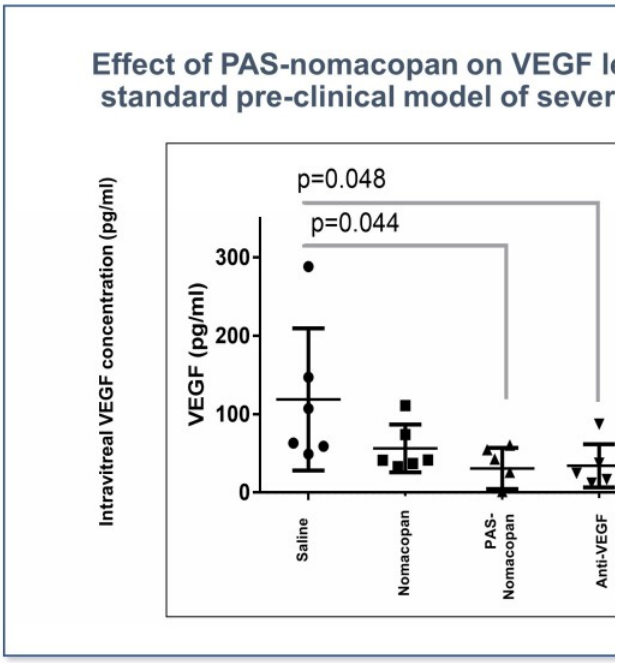
References:

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]

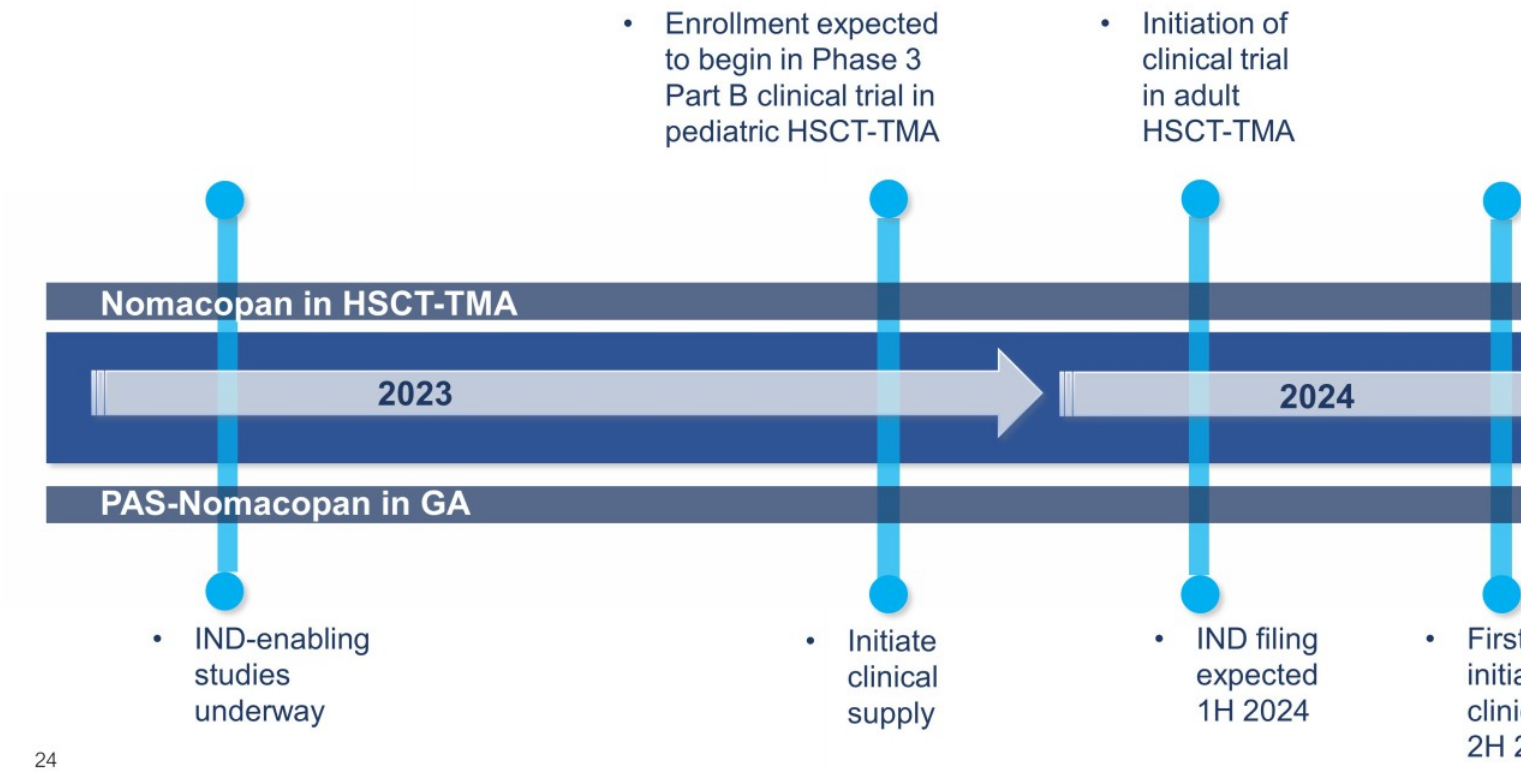


References

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; 191:3
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
Nomacopan in HSCT-TMA
2023
2024
PAS-Nomacopan in GA
• IND-enabling studies underway
• Initiate clinical supply
• IND filing expected 1H 2024
• First initia clini 2H 2

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