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: February 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 February 15, 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 slide 23 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: February 15, 2023

Akari Therapeutics

February 2023

AKARI
THERAPEUTICS

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 Act of 1934. These forward-looking statements reflect our current views about our plans, intentions, expectations, strategies an prospects, which are based on the information currently available to us and on assumptions we have made. Although we believe that our plans, intentions, expectations, strategies and prospects as reflected in or suggested by those forward-looking 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 those described in the 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: needs for additional capital to 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 approvals for nomacopan (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 and to successfully 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 development of potential 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 difficulties enrolling 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 successfully bringing 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 authorities of other 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 associated with the impact of the outbreak of COVID-19; inability to obtain, maintain and enforce patents another intellectual property rights or the unexpected costs associated with such enforcement or litigation; inability to obtain and maintain commercial manufacturing arrangements with third party manufacturers or establish commercial scale manufacturing capabilities; the inability to timely source adequate supply of our active pharmaceutical 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 the U.S. Securities and 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 otherwise required by 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 information, events, results or 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 result of new information, future events or otherwise, except as required by law.

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

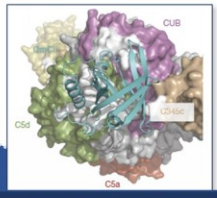
1. Novel bispecific recombinant protein targeting both complement C5 and leukotriene B4 (LTB4)
2. Broad therapeutic potential due to bispecific mechanism of action and multiple routes of administration (subcutaneous, topical, intravitreal, intravenous)
3. Advanced late-stage program with extensive clinical and safety data
4. Lead indication in pediatric hematopoietic stem cell transplant-related thrombotic microangiopathy (HSCT-TMA) accelerated into pivotal Part B of Phase 3 clinical trial; granted Orphan and FDA Rare Pediatric Disease Designation (Priority Review Voucher eligible at approval)
5. Significant unmet need in both adult and pediatric HSCT-TMA with ~80% mortality rate and no approved therapies; added new pipeline program in adult HSCT-TMA, which will support both adult and pediatric regulatory pathways
6. Pre-clinical program investigating long-acting PAS-nomacopan in geographic atrophy (GA) with potential to reduce sight-threatening choroidal neovascularization (CNV) risk via LTB4 inhibition and target dose interval beyond 3 months; IND submission first half of 2024

Nomacopan Is a Novel Bispecific Recombinant Protein

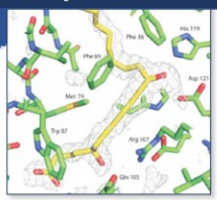
Ticks modulate host responses to their own benefit

- When ticks feed, they must control the host immune response, including inflammation, pain, itch and blood flow to avoid detection and successfully feed
- They do this by secreting many immunomodulatory proteins in their saliva that have been perfected by evolution for potency, specificity and tolerability

High resolution structure of nomacopan (cyan) bound to the CUB, C5d, and C345C domains of **C5** [1]



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



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

Two Modes of Action

- Inhibits complement C5 activation similarly to eculizumab, but binds a different conserved region of C5
- Unique mode of action against leukotriene B4 (LTB4) tightly sequesters LTB4 within the protein 'ligand capture' preventing receptor-mediated cell activation
- Nomacopan LTB4 inhibition is prolonged since nomacopan C5 complex has a half-life of about 60h and is present in excess to LTB4, so nomacopan circulates through the body and absorbs LTB4, disrupting cell recruitment and activation

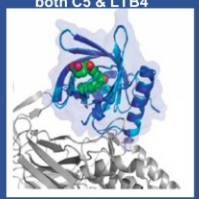
References

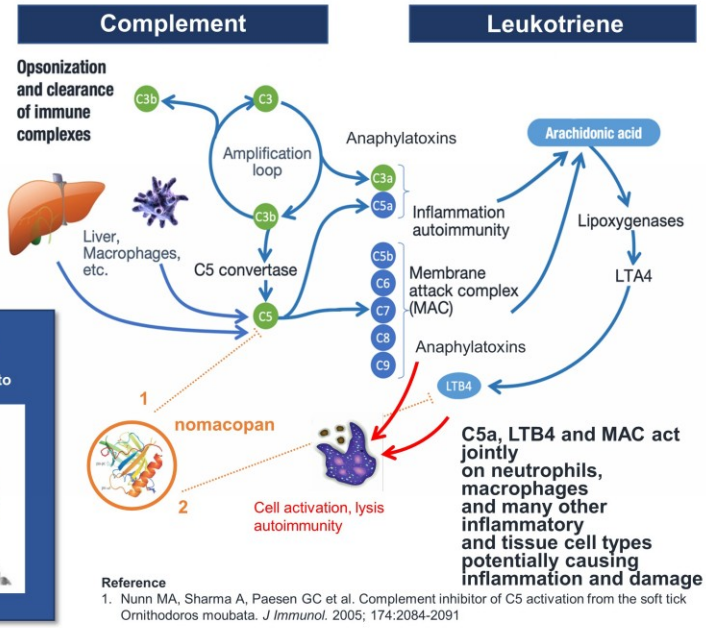
1. Jore MM, Johnson S, Sheppard D, et al. Structural basis for therapeutic inhibition of complement C5. *Nat Struct Mol Biol.* 2016;23(5):378-386.
2. Roversi P, Ryffel B, Togbe D, et al. Bifunctional lipocalin ameliorates murine immune complex-induced acute lung injury. *J Biol Chem.* 2013;288(26):18789-18802.

Nomacopan Inhibits Cell Activation, Lysis and Inflammation Preserving Opsonization and Clearance of Immune Complexes

- Specifically prevents activation of C5 and binds LTB4, preventing proinflammatory and potentially tissue-damaging effects mediated by C5a, the MAC and LTB4[1]
- Opsonization and role of complement in clearance of immune complexes remain intact
- Inhibiting C5 activation prevents formation of the MAC and C5a that signals through 2 cell surface G protein-coupled receptors (GPCRs)
- Sequestering LTB4 prevents interaction of proinflammatory eicosanoid with cell surface GPCRs (BLT1 high affinity and BLT2 low affinity)

Crystal structure of nomacopan simultaneously bound to both C5 & LTB4





Reference

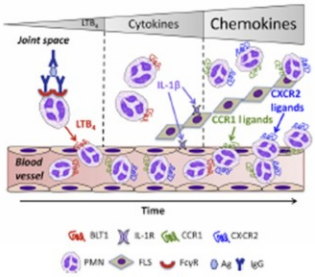
1. Nunn MA, Sharma A, Paesen GC et al. Complement inhibitor of C5 activation from the soft tick Ornithodoros moubata. *J Immunol.* 2005; 174:2084-2091

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

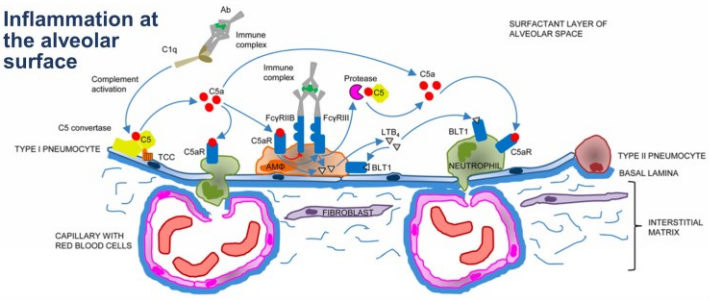
LTB4 initiates cytokine and chemokine cascade in the joint



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

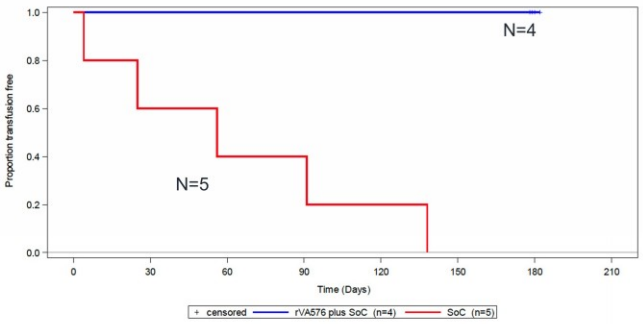
Indication	Candidate/ Formulation	Designations	Pre-Clinical	Phase 1	Phase 2	Phase 3 Parts A & B
Pediatric hematopoietic stem cell transplant–related thrombotic microangiopathy (HSCT-TMA)	Nomacopan/ subcutaneous	FDA Orphan Drug, Fast Track and Rare Pediatric Disease designations				● **Advancing to pivotal 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

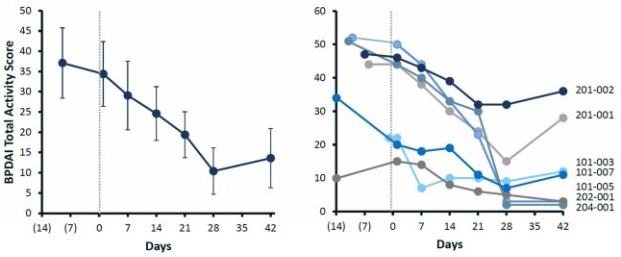
- **In a Phase 3 study in PNH, 100% of untreated patients were transfusion dependent while 0% of nomacopan patients were transfusion dependent**
  - >32 patient years of nomacopan exposure in PNH in 19 patients

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

Leadership Team

Rachelle Jacques President & CEO	John Neylan, MD Chief Medical Officer	Melissa Bradford-Klug Chief Operating Officer	Miles Nunn, DPhil Chief Scientific Officer	Torsten Hombeck, PhD Chief Financial Officer
ENZYVANT	angion	RARESTONE 珑钰集团	Discovered nomacopan	PROMETHERA BIOSCIENCES
ALEXION	KERYX	MAYFIELD	Evolutec	agennix
Shire	SANOFI	KERYX	Volution	cytonet
Baxalta	genzyme	amag	UKRI Natural Environment Research Council	GPC biotech
Baxter	Wyeth	Mallinckrodt Pharmaceuticals		
	EMORY TRANSPLANT CENTER	Baxter		
	UCDAVIS	Lilly		

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 TMAs 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. Published 2016 Sep 2
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 established in HSCT-TMA[2] and atypical hemolytic uremic syndrome (aHUS) [3,4], another TMA (eculizumab); 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]

Nomacopan in HSCT-TMA Progress Update

AKARI THERAPEUTICS

1 Acceleration into pivotal, registrational Phase 3 Part B

Based on FDA Type C guidance Akari is moving forward into design and planning for pivotal Part B of the Phase 3 clinical trial of nomacopan for treatment of pediatric HSCT-TMA in patients between 2 years and <18 years of age

2 Addition of new pipeline program in adult HSCT-TMA

Akari is initiating a new pipeline program for development of nomacopan as a potential treatment for adults with HSCT-TMA, which will include a study supportive of both adult and pediatric regulatory pathways; adult HSCT-TMA population is >10 times the pediatric population

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

Since enrollment for the youngest age group (aged 0.5 to <2 years) in the Part A study is not complete, Akari is keeping the study open for these young pediatric patients while advancing pivotal Part B study in the older pediatric patients; full data readout upon Part A 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 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.

Pediatric HSCT-TMA Phase 3 Advancing Into Pivotal Stage; Adult Phase 2 Supportive of Pediatric Program

AKARI THERAPEUTICS

	Phase 3 Part A	Phase 3 Part B	NDA

Phase 3 Clinical Trial in Pediatric HSCT-TMA	• Dose confirmation/safety
	• Sites in US, UK, Poland
	• Severe HSCT-TMA in 3 age cohorts
	  ○ 0.5 to <2 years
	  ○ ≥2 to <9 years
	  ○ ≥9 to <18 years
	• Predictive PK/PD model compared with PK/PD data from patients enrolled in Part A in >2 years of age
	• <2 year age cohort not yet enrolled; Part A to stay open for these patients
		Data from Part A supports advancement to pivotal Part B with simple, fixed dose for >2 years age cohorts
		• Pivotal registrational study to support NDA for potential regulatory approval
		• Safety and efficacy
		• Two age cohorts
		  ○ ≥2 to <9 years
		  ○ ≥9 to <18 years
		• Study planning, design underway
		• Start of enrollment targeted for end of 2023
		Safety and efficacy data from Part B support a potential regulatory filing
			• Potential pediatric HSCT-TMA regulatory filing with FDA supported by data from pivotal pediatric Phase 3 Part B and supportive Phase 2 clinical trial in adult HSCT-TMA

Phase 2 Clinical Trial in Adult HSCT-TMA	• Supports advancement into adult HSCT-TMA Phase 3 study • Adult Phase 2 and 3 clinical trials support potential adult HSCT-TMA NDA • Adult Phase 2 and pediatric Phase 3 support potential pediatric HSCT-TMA NDA		

Part B Endpoint: TMA response including independence of RBC transfusion and/or urine protein creatinine ratio ≤ 2 mg/mg maintained ≥ 28 days immediately prior to any scheduled clinical visit up to Week 16

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

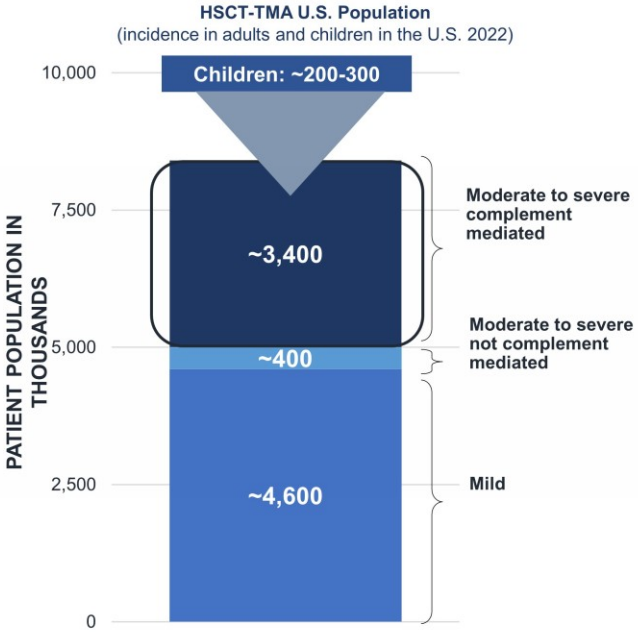
~22,000 HSCTs occur annually in the U.S.

~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.

GEOGRAPHIC ATROPHY (GA)

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

- Long-acting PAS-nomacopan is being developed with a profile that has the potential to deliver efficacy benefits of complement inhibition with a fraction of the number of annual doses (compared with current late-stage complement-only inhibitors for GA)
- Simultaneously, it may help address CNV safety risks reducing the total number of intravitreal injections (IVIs) needed to help preserve vision from GA as well as CNV

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 C5 inhibition slowing progression of GA lesions is well understood[1,2]

2. Frequency of Intravitreal Injections

Less frequent needle injections into the back of the eye, a source of fear, discomfort and disruption for patients[3]; **potential for only 3 to 4 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 current late-stage complement-only inhibitors[1,2]

PAS-Nomacopan in GA Progress Update

1 **PAS-nomacopan development on track for IND submission 1H24**

Based on progress, Akari is targeting an Investigational New Drug (IND) application submission to the U.S. Food and Drug Administration in the first half of 2024

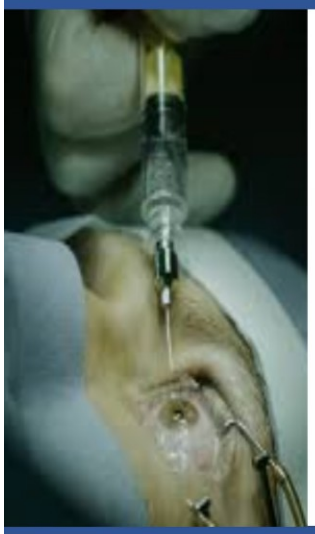
2 **New PAS-nomacopan versions fully active; target dosing interval extended beyond 3 months**

Pre-clinical development on long-acting PAS-nomacopan has successfully achieved multiple new and promising versions that are fully active, show high expression levels and have significantly larger hydrodynamic radii than earlier versions, which potentially increase the PAS-nomacopan target dosing interval beyond 3 months

3 **New versions expressing well and on track toward GMP scale for clinical trials**

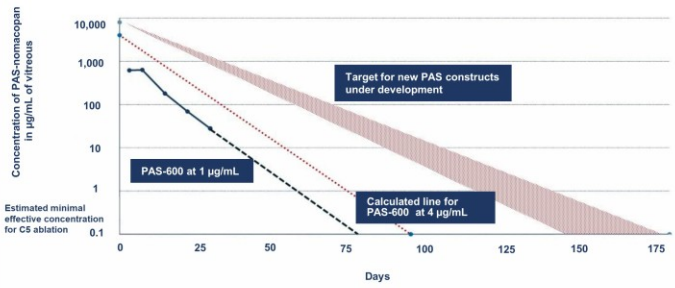
Development has progressed from lab scale to pre-GMP optimization and is on track to select the version of PAS-nomacopan that will advance to GMP manufacturing and non-clinical safety studies in preparation for an IND and the start of clinical trials in GA

PAS-Nomacopan Has Potential to Address the Significant Patient Burden of Frequent Intravitreal Injections for GA/CNV



- Between monthly or every other month (EOM) treatment for GA and CNV, patients could face 18 injections a year[1,2]
- Discontinuation for a late-stage complement-only GA treatment reported up to 20%[1]
- For anti-VEGF CNV treatments, up to 1/3 of patients may discontinue/ not adhere[3]

Long-Acting PAS-Nomacopan Has Potential to Extend Intravitreal Dose Interval to 3 to 6 Months[4]

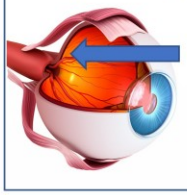


References

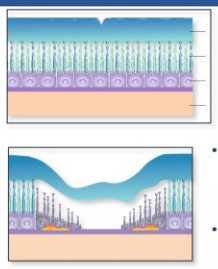
1. Medscape article on 24-month data presentation at AAO 2022 With Approval Pending, Pegcetacoplan Shows Mixed Results for Treating Geographic Atrophy https://www.medscape.com/viewarticle/981813#vp_2
2. Eyelea® Prescribing Information https://www.regeneron.com/downloads/eylea_fpi.pdf
3. McClard CK, et al. Questionnaire to Assess Life Impact of Treatment by Intravitreal Injections (QUALITII). *BMJ Open Ophthalmol*. 2021;6(1):e000669.
4. 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

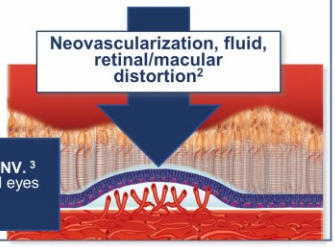
- VEGF-A is one of the key factors responsible for **endothelial cell** proliferation and migration
- Endothelial cells **form the inner layer of blood vessels** and play a key role in function, including exchanges between blood vessels and surrounding tissues

VEGF-A protein

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



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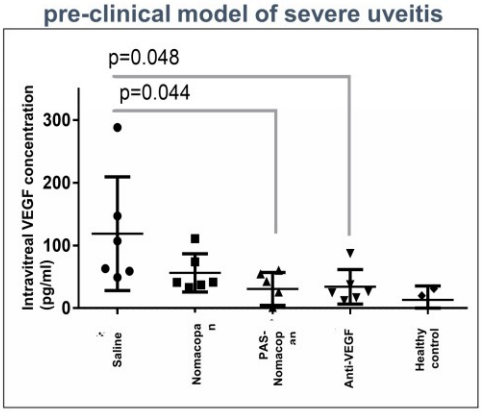
PAS-Nomacopan Decreased VEGF Levels As Effectively As An On-Market Anti-VEGF In a Pre-Clinical CNV Model

- PAS-nomacopan is a bispecific inhibitor of LTB4 and complement (C5)
  - Complement inhibition has shown efficacy in slowing progression of GA[1-3]
  - LTB4 inhibition may also reduce the risk of sight-threatening choroidal neovascularization (CNV), a safety risk associated with clinical trials of current late-stage complement-only inhibitors[1-3]
- In a standard pre-clinical model of laser-induced CNV, long-acting PAS-nomacopan (single IVI) decreased VEGF levels as effectively as anti-VEGF treatment Eylea® (multiple IVIs)[1,2]
- Positive results from the pre-clinical studies[3,4] support the advancement of long-acting PAS-nomacopan toward IND/IMPD for intravitreous injection (IVI) and clinical trials in GA as well as the potential for the investigational treatment to address areas of unmet patient needs, including risk of CNV

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



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FINANCIAL UPDATE

Financial Update

- Ticker: AKTX (NASDAQ)
- 74.4M ADS outstanding
- Cash of $16M as of September 30, 2022
- Estimated cash runway into Q3 2023

THANK YOU