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

September 2019

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 ☐

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulations S-T Rule 101(b)(1):_____

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulations S-T Rule 101(b)(7):_____

CONTENTS

On September 10, 2019, Akari Therapeutics, Plc updated its corporate presentation that it intends to use in conferences and meetings with investors. A copy of the presentation is attached hereto as Exhibit 99.1 and is incorporated herein by reference.

Exhibit No.

99.1 Corporate presentation dated September 2019.

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/ Clive Richardson
Name: Clive Richardson
Chief Executive Officer and
Chief Operating Officer

Date: September 10, 2019

Investor Presentation

September 2019

Disclaimers

Certain statements in this presentation constitute contains "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 and 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, which may result in unexpected cost expenditures; our ability to obtain orphan drug designation in additional indications; risks inherent in drug development in general; uncertainties in obtaining successful clinical results for nomacopan and any other product candidates and unexpected costs that may result therefrom; difficulties enrolling patients in our clinical trials; failure to realize any value of nomacopan and any other product candidates developed and 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 and 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 for nomacopan may not be as large as expected; risks associated with the departure of our former Chief Executive Officers and other executive officers; risks associated with the SEC investigation; inability to obtain, maintain and enforce patents and other 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 on April 23, 2019.

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 laws, 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.

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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 in which such offer, solicitation or sale would be unlawful prior to registration or qualification under the securities laws of any such state or other jurisdiction.

Treating Orphan Diseases Caused by Complement & Leukotriene Dysregulation

Unique MOA

- **Nomacopan (Coversin):** Bifunctional inhibitor of C5 & LTB4
  Focused on orphan inflammatory & autoimmune diseases where treatment options limited and C5 and LTB4 implicated

Diversified Targets

- **Four orphan conditions currently in Phase II and Phase III studies**
  – Subcutaneous (TMA, BP, & PNH) and topical delivery (AKC)

Clinical Data

- **Positive initial clinical readouts across clinical studies**
- Excellent safety profile; no drug-related SAEs over 20 cumulative patient-years of cumulative patient treatment data

Market Potential

- **$500 million peak sales targets, including follow-on indications**
- Platform molecule with a large and active pipeline

Pathway to Approval

- Potential upcoming value inflection points via multiple mid-stage clinical study readouts in 2019-2020 from TMA, BP, and AKC

TMA: Thrombotic Microangiopathy; BP: Bullous Pemphigoid; AKC: Atopic Keratoconjunctivitis; PNH: Paroxysmal Nocturnal Hemoglobinuria

Dual C5 & LTB4 MOA Provides Unique Option for Multiple Poorly Treated Inflammatory Diseases

Background

- **C5 & LTB4 act synergistically**:
  C5 activates granulocytes, LTB4 promotes granulocyte migration
- **Each pathway proven as treatment option:**
  Eculizumab (Soliris®) – inhibits C5, and Zileuton® inhibits LTB4

Evidence for Treatment

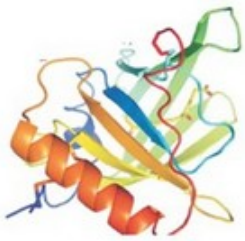
- **Additive effect** demonstrated in preclinical RA, lung and BP models with Nomacopan: significantly more effective than LTB4 or C5 alone
- **Elevated levels of C5a and LTB4** seen in human and animal tissue - blister fluid (BP), eye (uveitis), GVHD (HSCT-TMA)
- **Clinical response seen** across the three target conditions (BP, AKC, HSCT-TMA) where C5 and LTB4 implicated

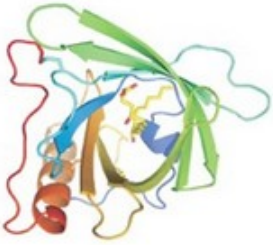
Opportunity

- **Nomacopan: good safety profile in clinical studies to date**
- **Potential to address unmet need across a wide range of conditions**

Nomacopan: Well-Designed by Nature Tightly Binding Both C5 & LTB4

Independent, Highly Specific C5 & LTB4 Inhibitory Activity



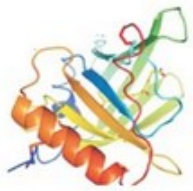


- Nomacopan: biological with high specificity for C5 & LTB4
- Avoids off-target effects by binding ligands not receptors
- No immunogenicity to date in human studies
- Multiple modes of administration (sub Q, topical, nebulized)
- Delivers high degree of patient comfort
  - Sub Q: Highly soluble & low viscosity
  - Topical drops: pH neutral / comfortable

Nomacopan: Extensive and Growing Positive Efficacy Data Across Four Indications

2015-2018		2019	Projected Next Step
9 patients sub Q **~20 cumulative patient-years*** of treatment data **PNH Phase II** **Achieved primary Phase II end point** Six Phase II patients transfusion dependant; two-thirds now transfusion independent	**TMA**	2 patients sub Q – chronic Named patient program	Initiate **pivotal study** Q4 2019
	BP	3 patients sub Q – chronic ongoing Phase II	Completion of 9 patient **Phase II** Q4 2019
	AKC	3 patients topical – chronic ongoing Phase II	Completion of 16 patient **Phase I/II** Q4 2019
	PNH	Phase III study naïve patients	Objective to introduce potential pen injector

** as of September 2019*



Subcutaneous Nomacopan:

Clinically Testing C5 & LTB4 Binding in

HSCT – Thrombotic Microangiopathy

HSCT-TMA
High Mortality Rate – and No Approved Treatments

- Arteriolar thrombi associated with intimal swelling, fibrinoid necrosis, thickening of arterioles and capillaries, and subendothelial accumulation of proteins & cell debris
- Cause of TMA after bone marrow transplant not well understood
  - Can occur in up to 30% patients
  - Typical diagnostic markers include elevated sC5b9 and proteinuria
- Diagnosis & treatment of pediatric TMA-HSCT refined by Dr. Jodele at Cincinnati Children's Hospital
- No approved therapy; current treatment by immunosuppressants
- In severe pediatric cases, mortality ≥80%
- Orphan condition with a prevalence of circa 5,000 treatable patients

Upcoming Pediatric HSCT-TMA Pivotal Trial Potential Expansion to Related Indications

- Complement's role highlighted by elevated terminal complement components (sC5b9) in many patients.
- LTB4 believed to have a role in TMA-HSCT in terms of graft vs. host disease (GVHD) progression
- Nomacopan used on a named patient basis for pediatric HSCT-TMA
- Positive FDA meeting, trial designed around multiple responder endpoints
- Nomacopan has received pediatric Fast Track and Orphan status for adult and pediatric HSCT-TMA
- **Pivotal trial anticipated to commence Q4 2019; primary endpoint TMA response**

Potential expedited approval with a single pivotal study
Gateway condition into other TMAs
Potential first approval for nomacopan

Rosenthal (2016) *Journal of Blood Medicine 7:* 181-186

Activity Demonstrated in HSCT–TMA Across Multiple Responder End Points (First 2 Named Patients)

TMA Marker	Patient	Baseline	Day 7	Day 14	Day 28	Day 60
Hemolytic anemia	1	Yes →	→	→	→	Resolved
	2	Mild				
Red blood cell fragments	1	Yes →	→	→	→	Resolved
	2	Yes →	→	→	Resolved	
Thrombocytopenia	1	Yes →	→	→	→	Resolved
	2	Yes →	→	Resolved		
Increased LDH	1	Yes →	→	→	Resolved	
	2	Yes →	→	Resolved		
Proteinuria and/or increased creatinine	1	Yes →	→	→	Resolved	
	2	Yes →	→	→	→	N/A
Hypertension	1	Yes →	→	→	→	Resolved
	2	Yes →	→	Resolved		
Neurology	1	Yes →	→	Resolved		
	2	No				
GI bleed	1	No				
	2	Yes →	→	→	Resolved	

Patient 1 : treated at GOSH made a complete recovery and nomacopan was discontinued after seven weeks
Patient 2 : despite resolution of the TMA markers, patient died at day 63 of lung damage considered unrelated to treatment with nomacopan. Note - data for patient 2 is up to day 28

Nomacopan in HSCT-TMA
Differentiation Against Competitors

Clear Positioning

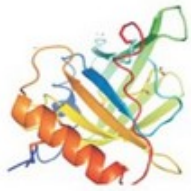
- Patient group to be treated identified via histologic TMA diagnosis in tissue biopsy or laboratory & clinical markers

Differentiated MOA

- Daily dosing facilitates more complete complement suppression
- LTB4 inhibition may further reduce epithelial activation

Next Steps

- **Pivotal pediatric study scheduled to commence Q4 2019**
- Responder-based study based on achieving end points related to disease resolution
- Working with FDA on optimizing dosing through the Model-Informed Drug Development (MIDD) Pilot Program

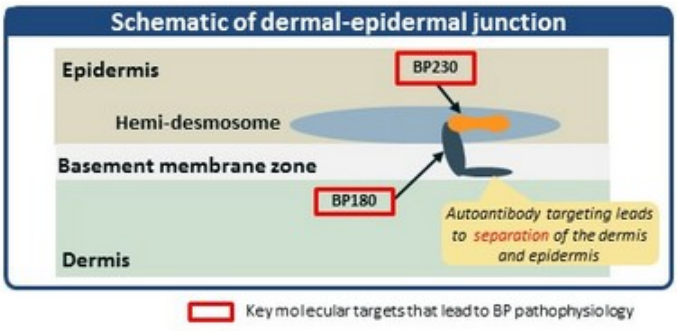


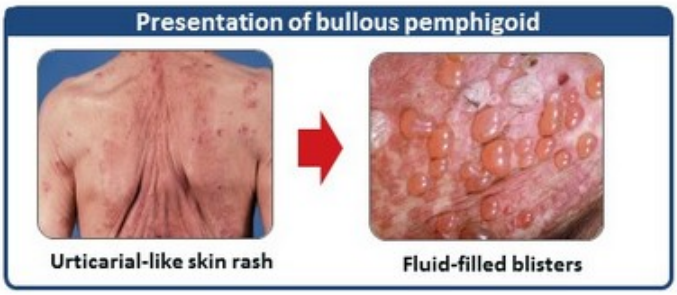
Subcutaneous Nomacopan:

Clinically Testing C5 & LTB4 Binding in

Bullous Pemphigoid

Bullous Pemphigoid
Significant Unmet Need





- Severe autoimmune blistering skin disorder primarily of the elderly
- Caused by immune complex mediated inflammation via autoantibodies
- No approved therapy. Current treatment with chronic corticosteroids poses substantial health concerns
- KOLs: essentially no change in standard of care in decades
- Understanding of BP has grown, but specific therapies still lacking
- Orphan condition, with a prevalence of circa 120K patients in US and EU

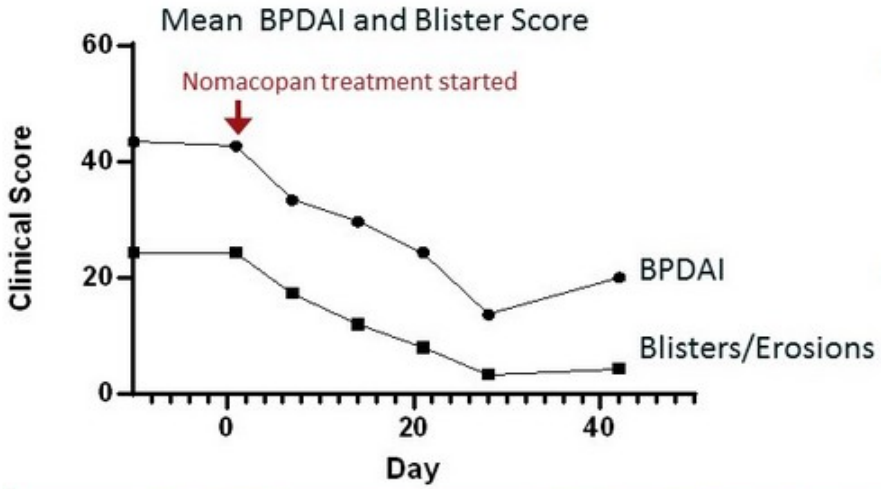
Bullous Pemphigoid
Evidence of Combined C5 & LTB4 Involvement

- Orphan condition in which there is evidence that both terminal complement activation (C5) and LTB4 have a role in driving disease
  - Preclinical studies with nomacopan showed a dose response and combined C5 & LTB4 more effective than LTB4 alone (1)
  - Elevated C5/LTB4 in *ex-vivo* study of BP patients
- Clinical objective is steroid sparing and rapid reduction symptoms
- Ongoing Phase II study : 42 days – mild to moderate patients

Significant market with expansion potential into related conditions

1. Sezin (2019) *JCI insight 4: e*128239

Bullous Pemphigoid Initial Study Data*
First 3 Patients: Global and Blisters BPDAI



- By Day 7, 21 and day 42 of treatment, the BPDAI global score fell by a mean of 31%, 45% and 52%
- By Day 7, 21 and day 42 of treatment, blisters/erosions dropped by a mean of 45%, 75% and 87%

Daily SQ nomacopan well tolerated, no drug-related adverse events

Planned amendment to extend into severe patients

* Prior to treatment, two of three patients were on topical corticosteroids (mometasone), while the third patient was naïve to steroid treatment & received no steroid while on nomacopan. There was no planned tapering of topical steroid dose, but patients did use less than the 30g permitted to day 21. After day 21, any use of mometasone was regarded as rescue therapy, and both patients were only treated with nomacopan.

In the 7-11-day period prior to initiation on nomacopan, the two patients showed either no or minor improvement in BPDAI score (between 0 and 5%), and no improvement in blisters while being treated with topical steroids alone.

Nomacopan in Bullous Pemphigoid Differentiation Against Competitors

Clear Positioning

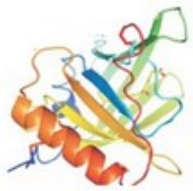
- Positioned as 2nd line treatment for patients with moderate to severe symptoms, previously treated with corticosteroids
- Better safety and side effect profile than steroids and non-specific immunosuppressants
- Used to reduce or eliminate dependency on steroids

Differentiated MOA

- Nomacopan acts upstream of competitors' targets – in particular C5 & LTB4 are part of inflammatory process leading to generation of cytokines via antibody

Next Steps

- Potential post-Phase II regulatory interaction following study completion to help define **pivotal study design**



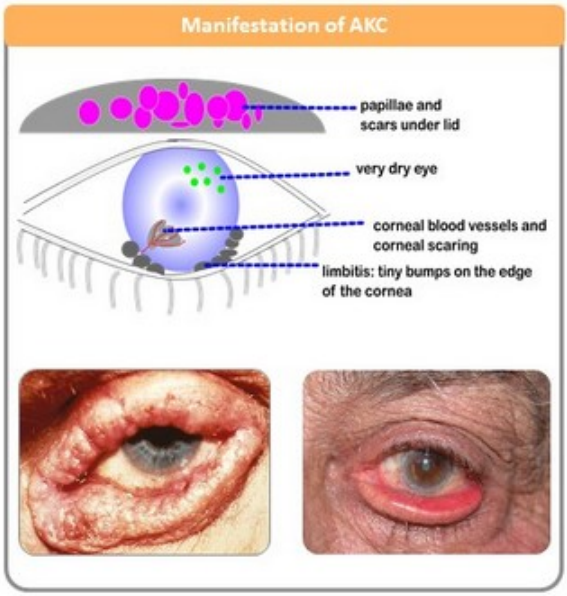
Topical Nomacopan:

Clinically Testing C5 & LTB4 Binding in

Ophthalmology / AKC

Atopic Keratoconjunctivitis (AKC): Chronic Ocular Disease Which Can Lead to Vision Loss

- Atopic Keratoconjunctivitis: severe eye surface inflammation causing infiltration of immune cells (neutrophils & T cells)



- Chronic disease can result in severe dry eye, corneal neovascularisation, & formation of sterile corneal ulcers
- If left untreated, can lead to vision loss; other complications include cataracts, keratoconus, infectious keratitis and tear dysfunction
- Topical drugs, such as steroids or cyclosporine treatments complicated by issues with comfort and side effects of chronic usage
- Orphan condition with a prevalence of 160K patients in the US and EU

Atopic Keratoconjunctivitis (AKC)

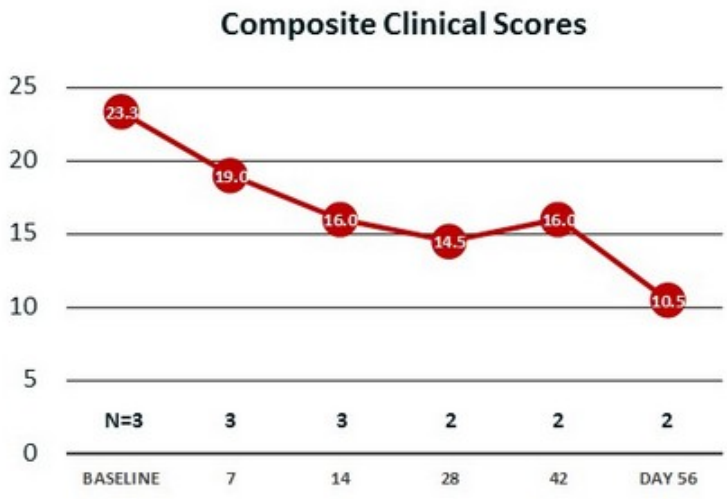
- Infiltration of neutrophils & T cells into conjunctival epithelium
- LTB4 levels – 20X in allergic conjunctivitis patients
- Complement system in tears is functionally active, and concentration of all components is greatly increased in closed-eye tears
- C5, likely generated by macrophages, may be aggressively activated by corneal disease (1)
- Preclinical model demonstrated equivalence to cyclosporine
- **Ongoing Phase I/II study.**

Potential to be first surface of eye biological treatment
Different formulation allows potential pricing & licensing flexibility
Expansion potential into other poorly-treated surface of the eye diseases

1. Bora (2008) *Semin Immunopathol 30:* 85-95

Initial AKC Clinical Data – First 2 Patients to Day 56
Rapid Improvement in Composite Clinical Scores

- Patients had deteriorating moderate – severe AKC despite ≥3 months treatment with cyclosporin t.i.d. ± steroids
- Part A of study – first in man – successfully completed – drug well tolerated
- Overall improvement across both signs and symptoms
  - Patient 1 had a disease flare at Day 42 but then recovered



- No serious drug-related AEs: drops comfortable and well tolerated

- **Next stage of trial (part B) ongoing**
  - **16 patients randomized, double masked**
  - **To enhance recruitment, trial expanded to additional clinical centers**

Nomacopan in AKC
Clear Differentiation Vs. Competitors

Clear Positioning

- Positioned as 1st line treatment for patients with moderate to severe symptoms, given **clear unmet need**

Differentiated MOA

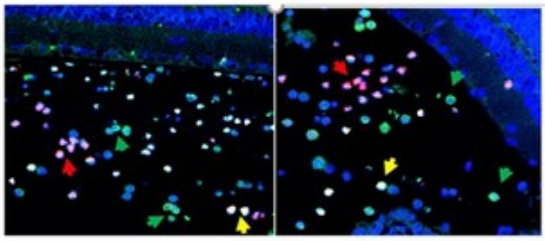
- Comfortable on application (pH neutral)
- Rapid improvement in signs & symptoms in human studies
- Upstream effects on T cells and granulocytes

Next Steps

- Potential regulatory interaction following Phase I/II study completion to help define **pivotal study design**
- Opportunity to expand into broader dry eye

C5 & LTB4 Activity in Back of the Eye Indications
Potential New Target Indication: Uveitis

LTB4 receptor
C5a receptor

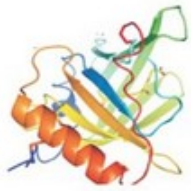


Autoimmune Uveitis (EAU) Model

- Intravitreal nomacopan reported significant improvement in uveitis model versus control
- Significant down-regulation of Th1/Th17 cells and IL-17A production

- Effect approx. equal to intravitreal dexamethasone
- C5 & LTB4 receptors both identified in retina*
- Uveitis treatment currently primarily steroids

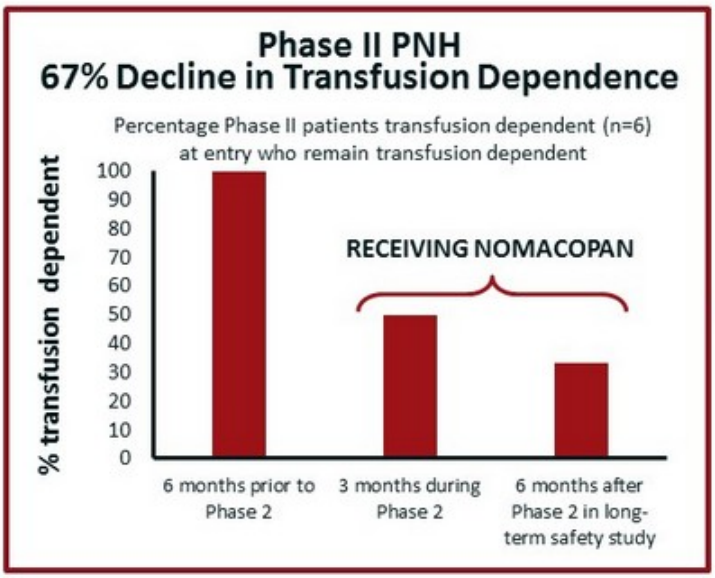
* Eskandarpour M et al. Poster 797 (B0275) ARVO 2019



Subcutaneous Nomacopan:

Paroxysmal Nocturnal Hemoglobinuria

PNH Phase II: Transfusion Reduction Validated Efficacy - Option Value For Continuing to MAA

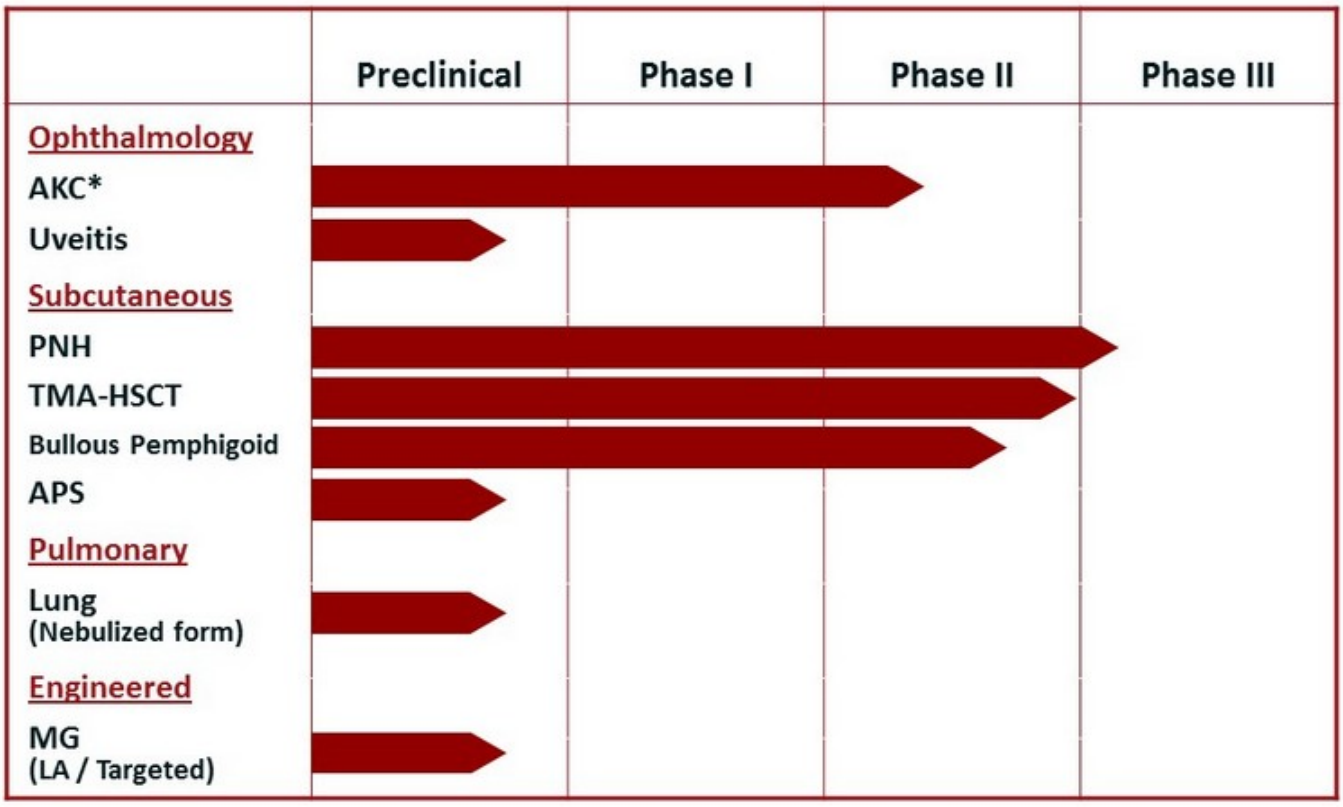


- Six patients transfused in six months prior to entering Phase II
- Three became transfusion independent during Phase II
- Four transfusion independent during follow-up long-term safety study

- Successful Phase II study achieved primary end point LDH ≤1.8X ULN
- Phase III study in naïve patients – primary end point: transfusion independence
- Potential pen injector will hold weekly dose, stable at room temperature with 0.4ml daily injection

Wrap up

Four Primary/Lead Indications in Phase II/III Plus Pipeline of Earlier-Stage Targets



* Phase I/II study

APS: Antiphospholipid Syndrome; MG: Myasthenia Gravis

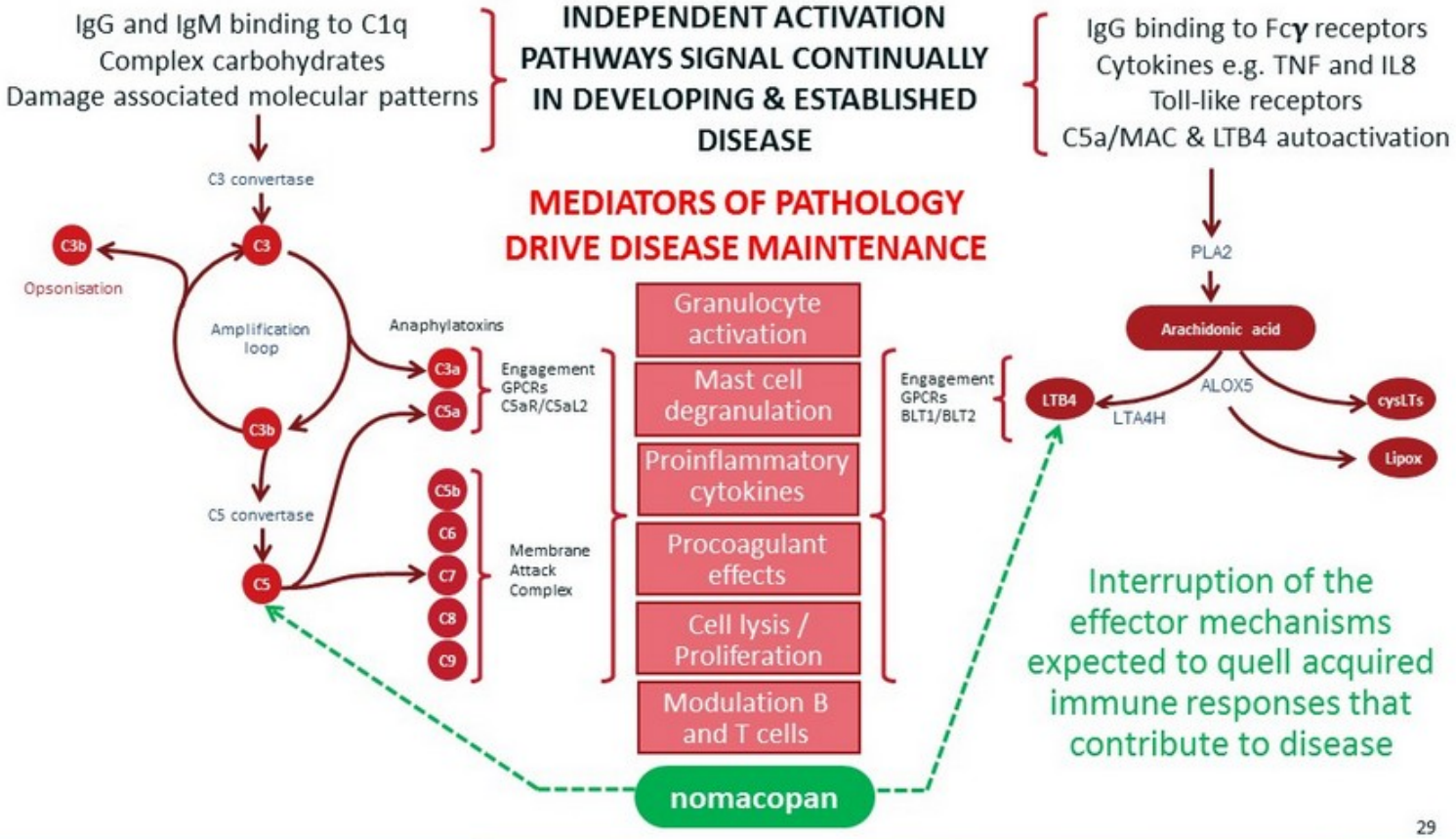
Financial and Clinical Wrap up
Meaningful Upcoming Clinical Milestones

- Growing efficacy data set across four severe orphan conditions
- 20 cumulative patient-years of treatment shows positive safety data and well tolerated
- Multiple expected short- to mid-term clinical readouts:
  - **BP**: Complete mild to moderate patients Q4 2019
  - **TMA**: Q4 2019 - pivotal trial approval/open
  - **AKC:** Part B update Q4 2019
  - **PNH:** Staged readouts
- $20 million financing facility* with Aspire Capital
- ~1.6 billion ordinary shares outstanding / equivalent to 16 million ADSs

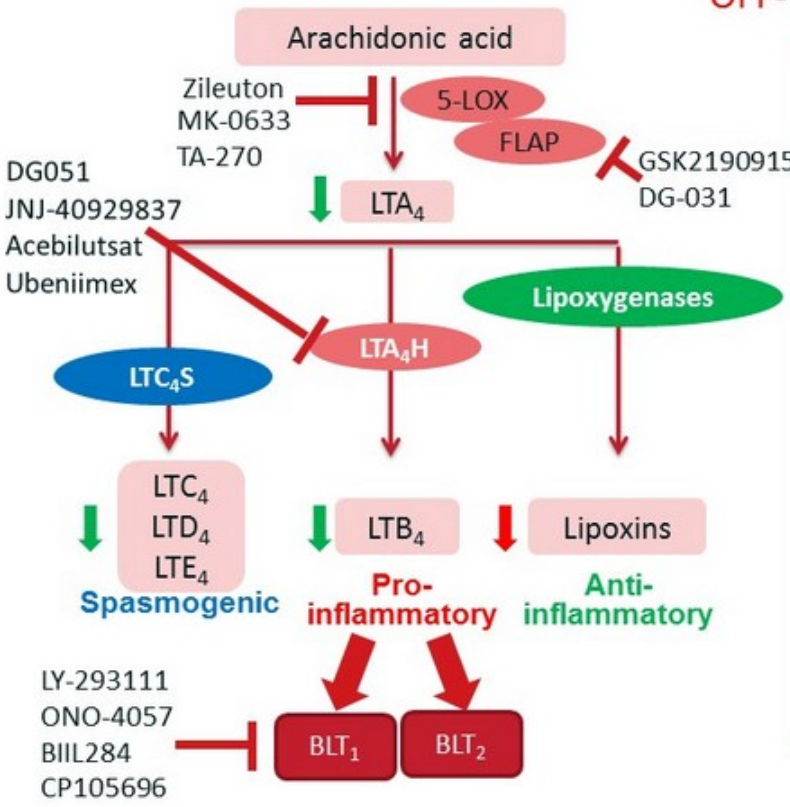
* Circa $5.5 million drawn down to date

APPENDIX

Nomacopan Competitive Advantage: Synergistic C5 and LTB4 inhibition



LTB4 Ligand Capture Advantageous and Unique MOA



OFF-TARGET EFFECTS OF OTHER INHIBITORS

5-LOX / FLAP inhibitors

- Reduces anti-inflammatory lipoxins

BLT1/BLT2 antagonists

- Realization that anti-inflammatory mediators also signal through BLT1/BLT2

LTA_4H inhibitors

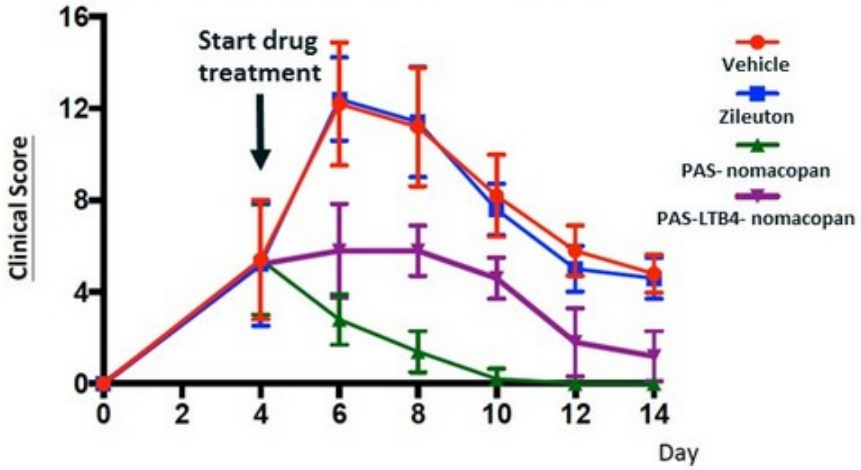
- Secondary anti-inflammatory role for LTA_4H in degrading pro-inflammatory/ remodelling mediator PGP

Expanding Pipeline Focused on Indications Where C5 and LTB4 Both Involved

Dual action C5+LTB4 (PAS-nomacopan) more effective than inhibition of LTB4 only (PAS-LTB4-nomacopan)

Dual action nomacopan more effective than C5-only nomacopan (or Zileuton®) alone

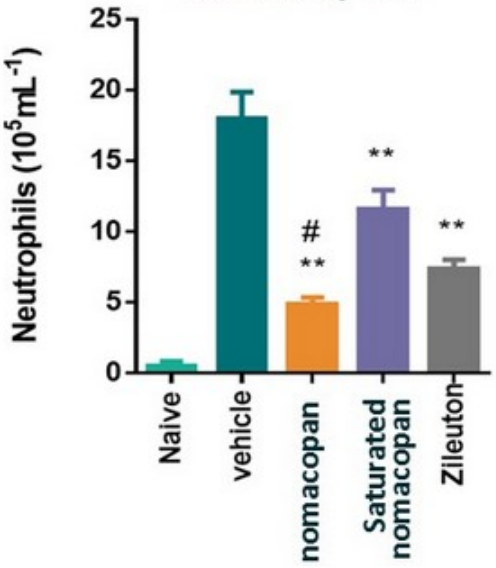
Rheumatoid Arthritis Therapeutic model



N = 5 mice in each treatment group

Work performed in group of Prof Andrew Luster, Mass Gen Hospital, Boston, USA

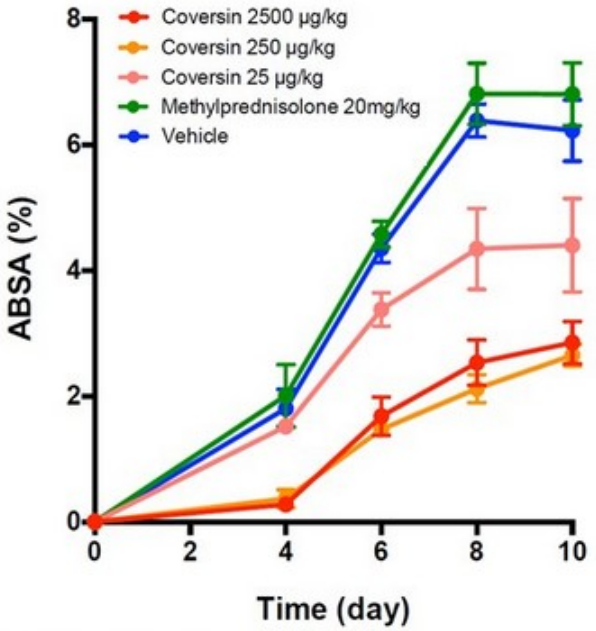
Cell recruitment to Lung Induced by LPS



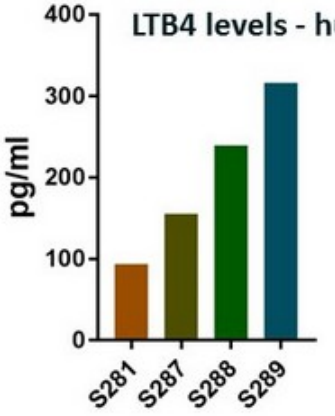
Mouse model by Pneumolabs

Epidermolysis Bullosa Acquisita Preclinical Efficacy & Elevated C5a/LTB4 in Bullous Pemphigoid patients

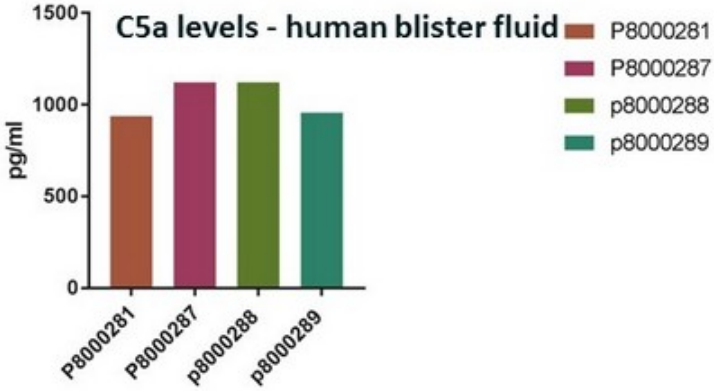
Preclinical passive mouse model of epidermolysis bullosa acquisita (EBA) from Dr. Sadik in Lubeck, Germany a leading Bullous Pemphigoid center



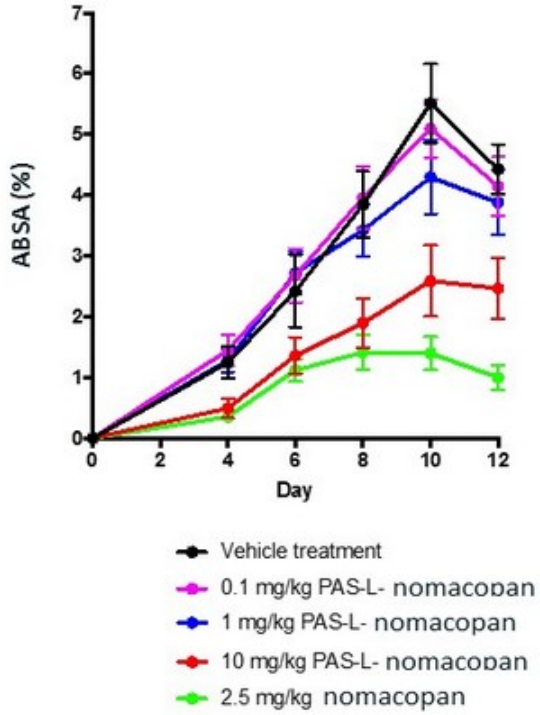
- Clear dose response
- ~60% reduction in affected area on nomacopan (SC) compared to vehicle or steroid
- P=0.0023 between vehicle and 250 µg/kg



Elevated levels of LTB4 and C5a indicate activation of complement and synthesis of LTB4 in blisters



Dual Acting Nomacopan Significantly More Effective Than Long-Acting Nomacopan Only Inhibiting LTB4



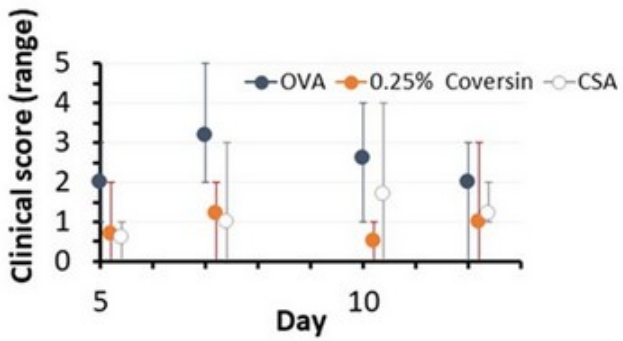
- Long acting LTB4 only nomacopan (10mg/kg) ameliorates blister formation but is less effective than the molar equivalent dose of nomacopan (2.5mg/kg)
- 1mg/kg long acting LTB4 only nomacopan ineffective whereas molar equivalent dose of 0.25mg nomacopan effective (see previous slide)

CONCLUSION:
Both C5 and LTB4 inhibition needed for full efficacy of nomacopan in EBA model

Significant Benefit Demonstrated in Late-Phase Eye Surface Inflammation

Collaboration with Institute of Ophthalmology, London, UK

EIC pre-clinical model of severe eye surface inflammation (Dr Virginia Calder)



- Treatment model of established severe eye surface inflammation
- Significant improvement in clinical scores
- Histology and cytology data support beneficial role of nomacopan
- Reduction in Th9 cells
- Reduction in IL-9

- *C57/Bl6 mice sensitized to OVA for 14 days*
- *Eye surface challenged with OVA for 12 days post sensitization (days 0 – 12)*
- *Nomacopan and cyclosporin A (CSA) applied once daily on days 4 – 12 after inflammatory response well established*
- *Maximum effect seen after 10 days of nomacopan treatment (late phase of inflammation)*

Bullous Pemphigoid Ongoing Phase II Study Patients with Mild-to-Moderate Disease

- **Trial approved Netherlands (2 sites) and Germany (6 sites)**
- **Amendment planned to treat ≤9 additional moderate-severe patients for ≤ 90 days**

Current Study design
- Phase II Single arm (n = 9); 42 days treatment
- Test role of C5 & LTB4 dual inhibition in improving BP outcomes
- Active bullous pemphigoid; newly diagnosed or recurrent

Treatment
- Nomacopan
- Day 1: 60 mg and 30 mg 12 hours later, Day 2-42: 30 mg od

Primary endpoint	Secondary endpoints
Safety	Efficacy evaluated by BPDAI (BP disease activity index) and QoL at day 42

AKC Phase I/II Proof-of-Principle Trial Design

Study design

- Phase I/II randomized, double masked, placebo-controlled, safety and efficacy study
- Part A : 3 patients open label - safety
- Part B : 16 randomized patients
- All patients on maximum cyclosporine

Treatment

- Nomacopan topical eye drops twice daily for 2 months
- Placebo eye drops

Primary endpoint	Secondary endpoints
• Safety, comfort	• Composite score of clinical signs (6) and symptoms (5)

Investor Presentation

September 2019