Combined Inhibition Of Complement & Leukotriene Pathways To Treat Inflammatory Diseases

July 2021

Forward-Looking Statements

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 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 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 impact of the outbreak of COVID-19; 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 March 31, 2020.

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Akari Overview
Targeting Inflammatory Diseases With Unmet Need

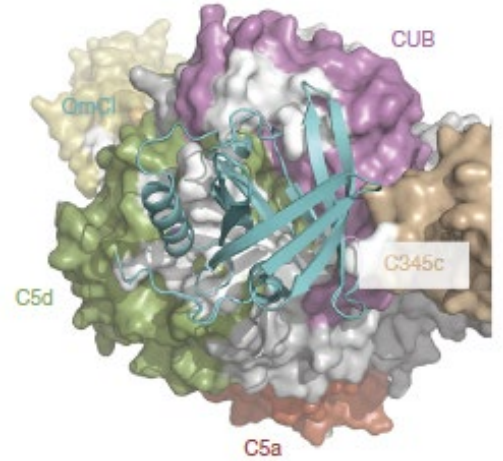
- **First in class, differentiated, dual MOA within the complement mediated disease space:**
  - Nomacopan inhibits complement C5 and leukotriene B4 (LTB4)
  - Ability to target multiple inflammatory indications where inhibiting both proinflammatory mediators may be more effective than treating either pathway alone

- **Positive clinical and safety data – with two Phase 3 clinical studies in 2021:**
  - **Bullous Pemphigoid** – a severe dermatological orphan condition
  - **Pediatric HSCT-TMA** – an ultra orphan indication and gateway into broader TMA space

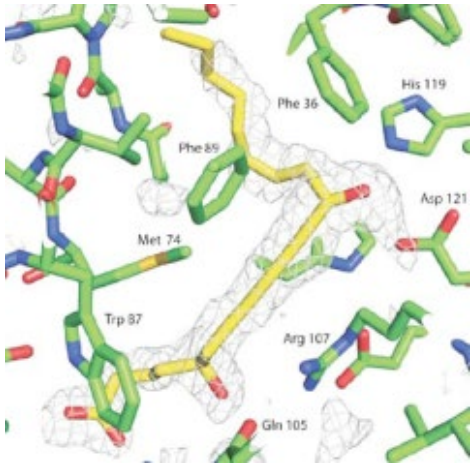
- **Development pipeline:**
  - **Ophthalmic** – large markets with partnering upside in both back of eye and topical indications
  - **Pulmonary** – multiple potential targets with high unmet need

HSCT-TMA: Hematopoietic Stem Cell Transplant-Related Thrombotic Microangiopathy

First-In-Class Bifunctional Anti-Inflammatory Biological



Top view of nomacopan (Cyan) bound to C5 Jore et al., Nat Struct Mol Biol. 2016



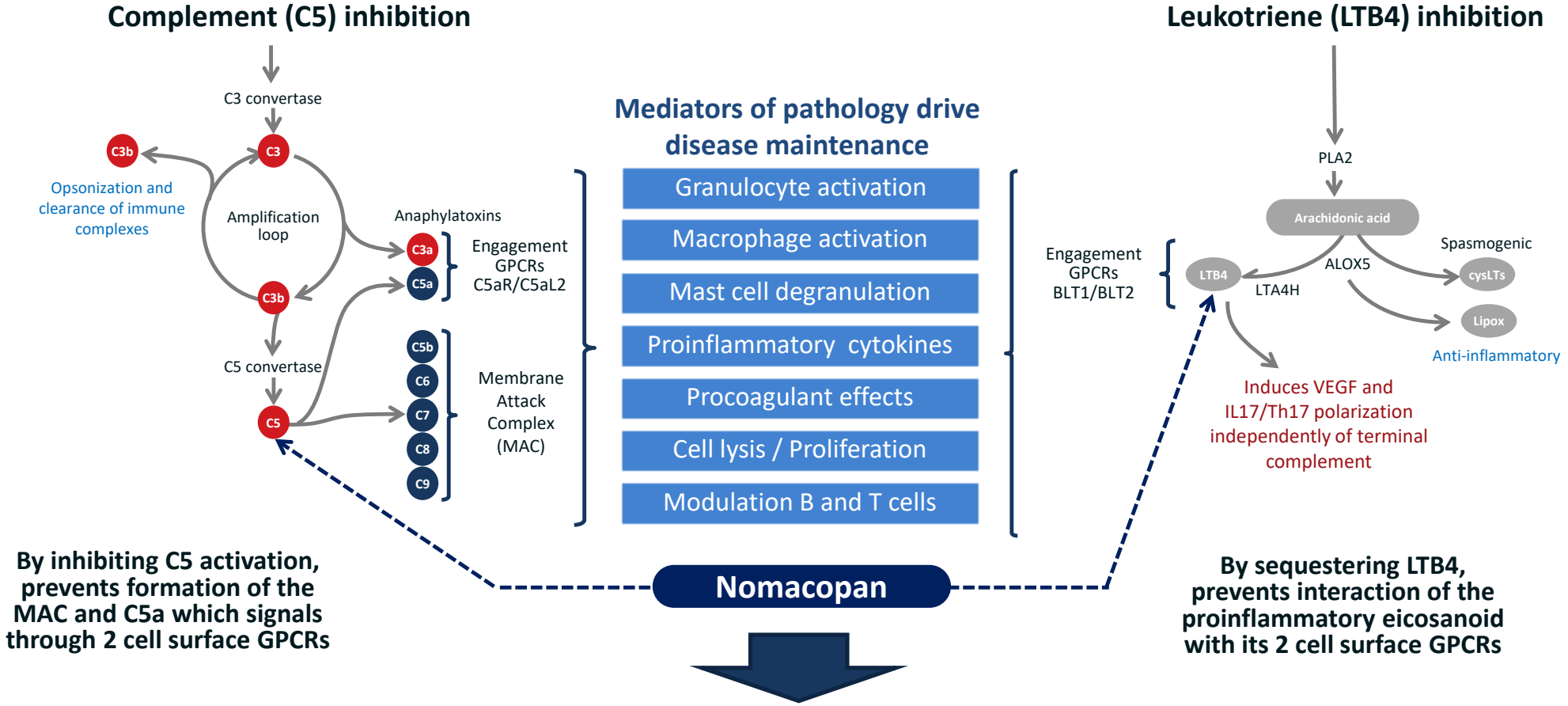
Detail of nomacopan binding to LTB4 Roversi et al., J Immunol. 2013

Two Modes of Action

- Nomacopan, by binding C5 and LTB4, inhibits both complement and leukotriene immune pathways, which are both active in a wide range of inflammatory disorders
- Inhibits complement C5 activation in a similar way to eculizumab, but binds a different highly conserved region of C5 **(K_D <1nM**)
- Unique* mode of action against leukotriene LTB4 by very tightly sequestering LTB4 within body of protein 'ligand capture' (**K_D 0.13nM**) thereby preventing receptor mediated cell activation

**Other LTB4 inhibitors are small molecules. Only currently approved LTB4 inhibitor is zileuton (Zyflo) for severe asthma*

Nomacopan's Competitive Advantage: Synergistic Inhibition of Two Key Effectors Driving Autoinflammation

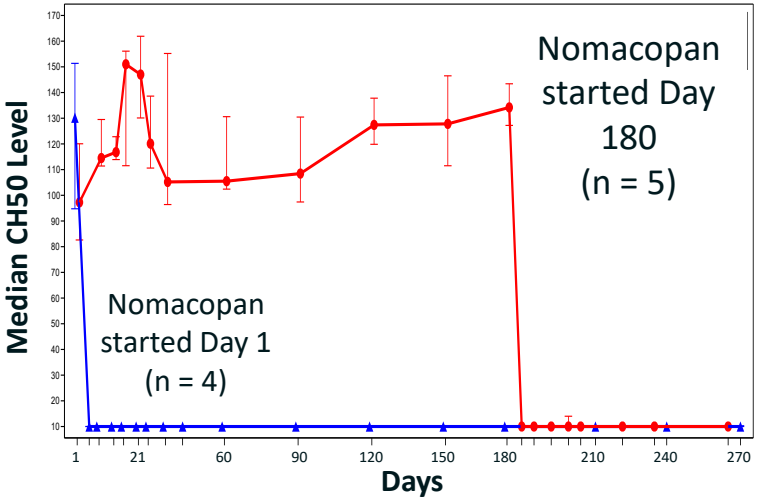


- Two independent but related activation pathways
- Both complement and leukotriene pathways active in multiple inflammatory diseases
- Nomacopan achieves additive, synergistic effect by inhibiting both

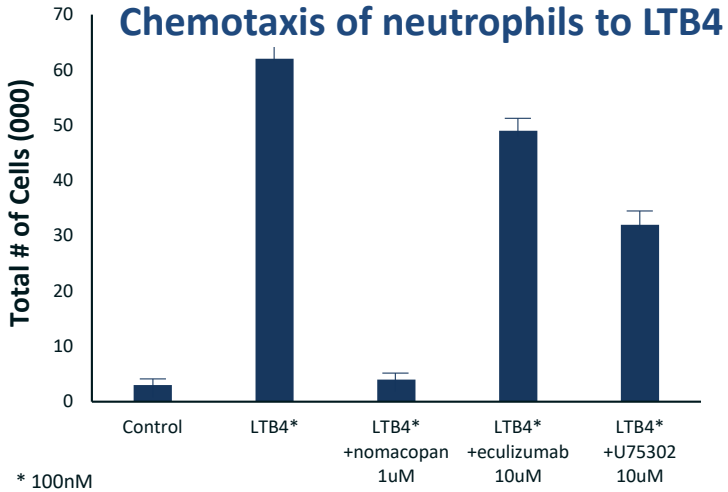
Nomacopan's Effectiveness Inhibiting C5 & LTB4 Demonstrated In PNH And Other Models

AKARI THERAPEUTICS

TCA PNH patients treated with nomacopan



Chemotaxis of neutrophils to LTB4



* 100nM

Terminal complement inhibition demonstrated in PNH patients

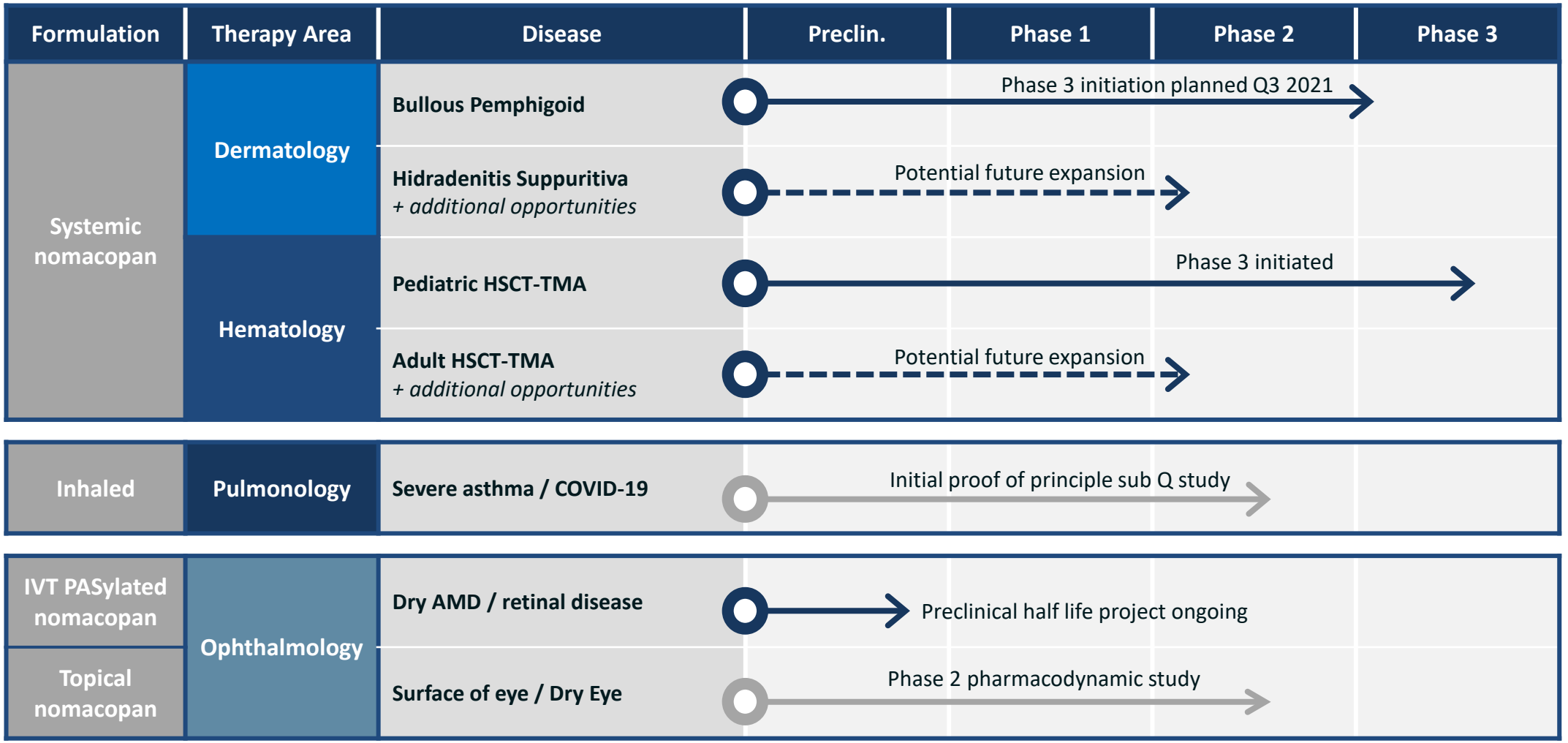
- **Drug effect:** Terminal complement activity (TCA) fully inhibited during long-term self-administration of nomacopan by PNH patients
- **Efficacy**: Nomacopan reduced transfusion dependence by 79% in 14 formerly transfusion dependent PNH patients treated for ≥6 months. Compares to 50-60% transfusion independence with eculizumab*
- **Safety: T**otal of >35 cumulative patient years nomacopan exposure with only one reported possibly drug-related SAE (a urinary tract infection), no meningococcal infections, no major adverse vascular events

Binds LTB4 , thereby preventing receptor-mediated cell activation

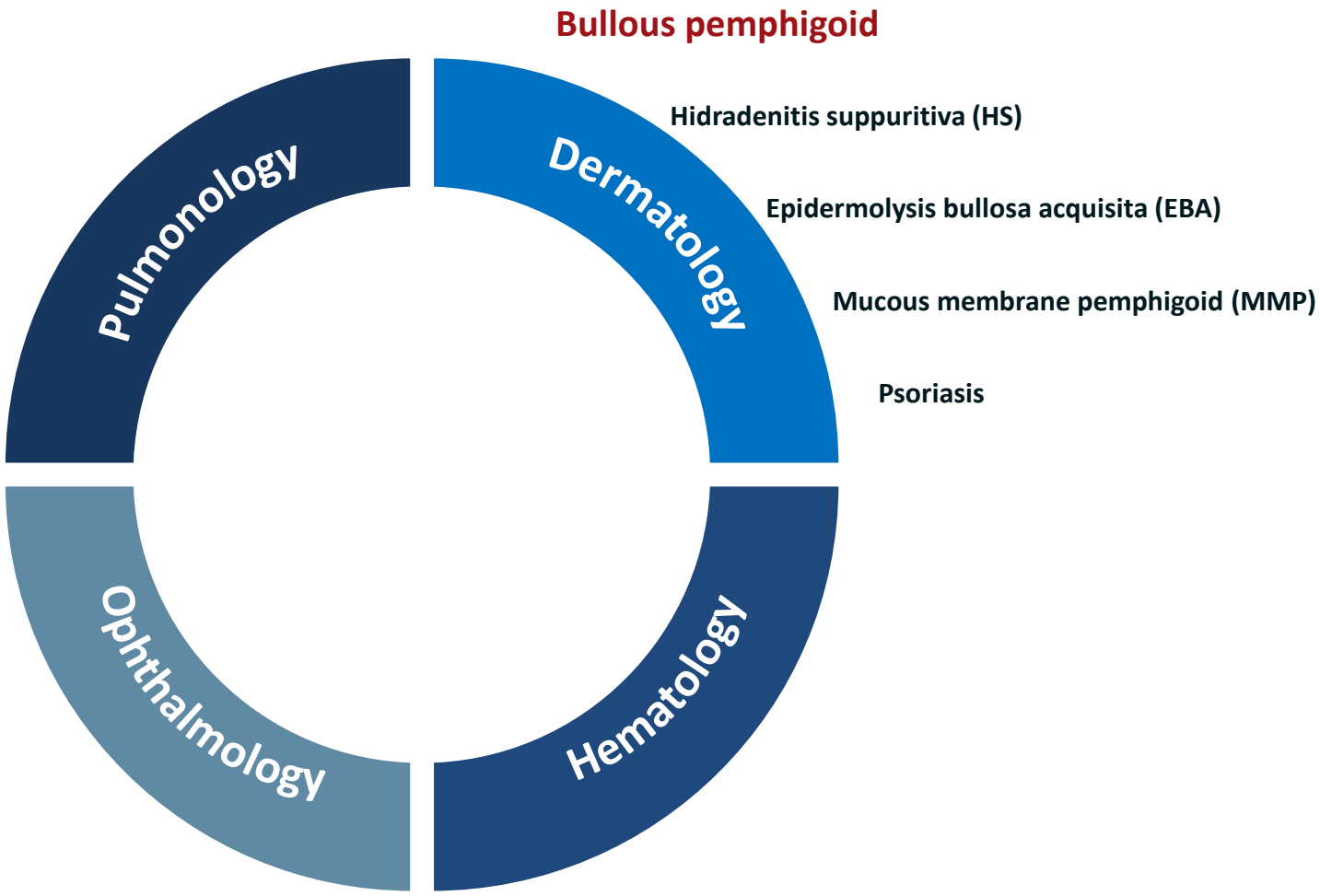
- **Drug effect:** Nomacopan significantly more effective in *ex vivo* model than U75302, another leukotriene inhibitor (U75302 is a BLT-1 receptor antagonist which is the high affinity receptor for LTB4)

- Brodsky et al., 2008, SHEPHERD study, Hillmen et al 2013 - Phase III study of eculizumab and ravulizumab (Wook Lee et al., 2019) transfusion independence respectively increased from 21% to 66% of and 23% to74% of patients over first 6 months of treatment

Clinical Pipeline Progressing Into Phase 3 Studies



Bullous Pemphigoid:
Chronic Disease, No Specific Approved Therapies



Bullous Pemphigoid: Auto-Immune Blistering Disease With No Specific Approved Therapy

Unmet Need

High dose oral corticosteroid (OCS) use associated with approximately 3-fold increase in mortality in this patient group

- Need for rapidly efficacious steroid-sparing therapy

Prevalence

Most common auto-immune blistering skin disease

- 120K patients in US+EU; 75% are moderate/ severe
- Majority of cases in elderly

Cause

Auto-antibodies to epidermal basement membrane proteins leads to separation of dermis / epidermis

- C5 & LTB4 initiate and maintain inflammatory drive in BP

Treatment

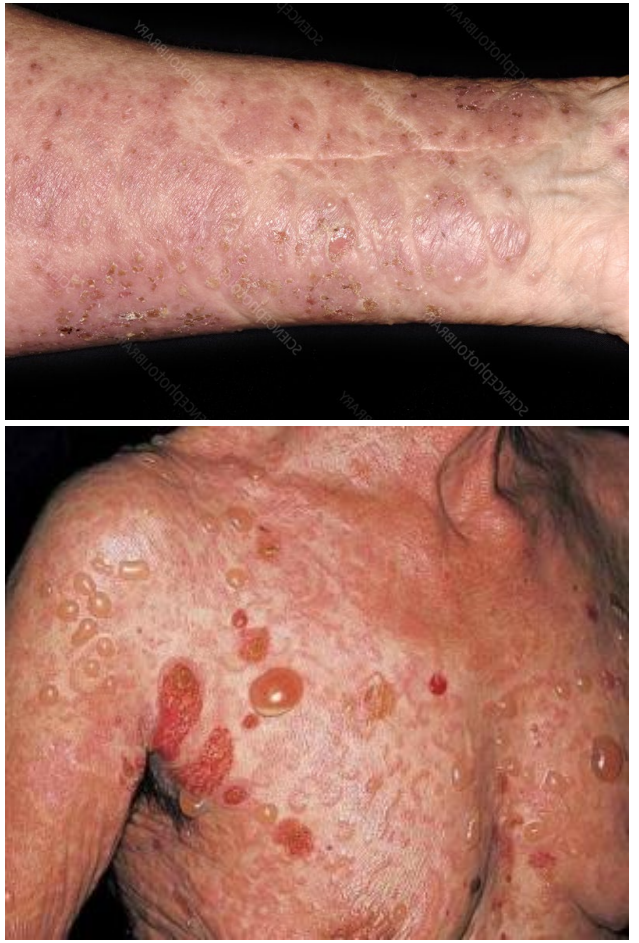
No specific approved therapies

- Mod/severe patients: high dose OCS / superpotent topical steroids

Status

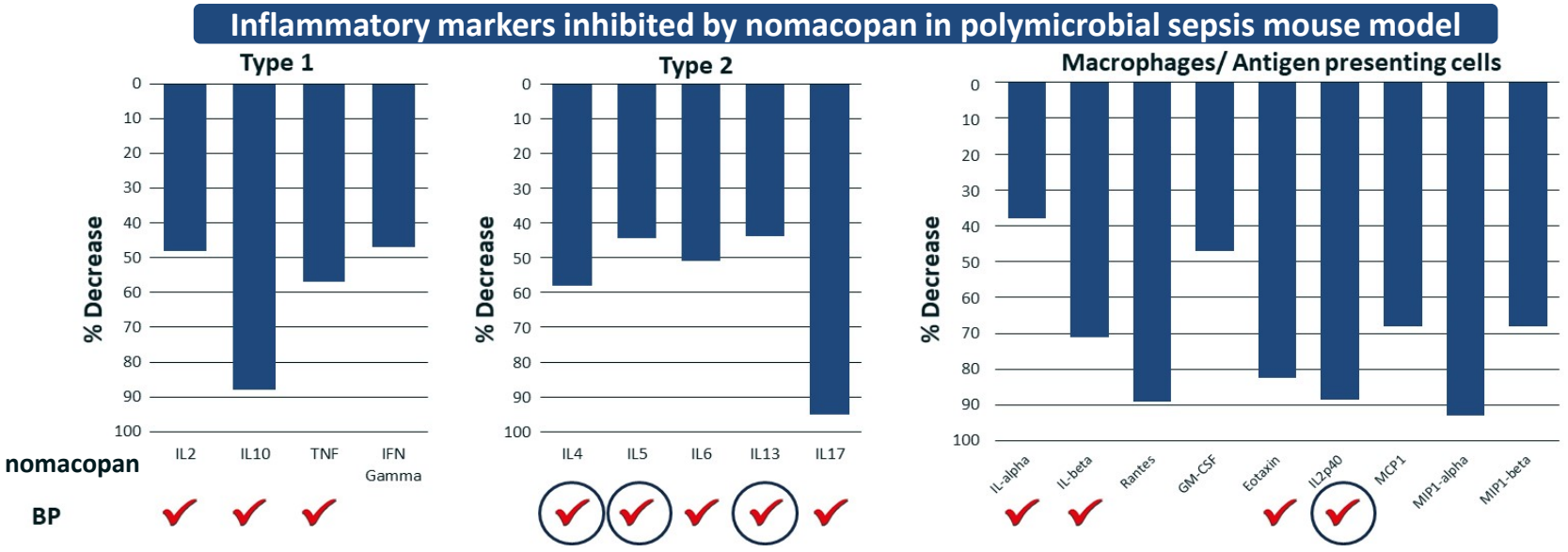
Phase 3 expected to commence mid 2021

- Orphan and Fast Track status.



Central Role For C5 & LTB4 in Bullous Pemphigoid

- **Preclinical:** BP-like model of epidermolysis bullosa acquisita showed c.50% reduction in surface area affected by blisters by inhibiting LTB4 alone but c.80% reduction by inhibiting both LTB4 and C5
- **Clinical biomarkers:** C5 and LTB4 levels elevated in blister fluid from BP patients illustrating local activation of both pathways. Mean level of LTB4 in plasma of phase 2 BP patients more than 2.5-fold higher than in PNH patients



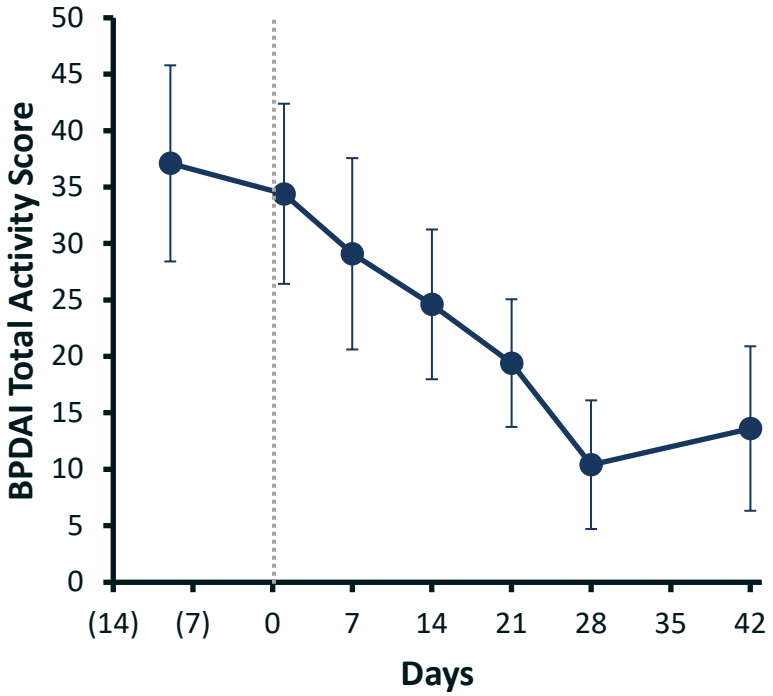
- ***Nomacopan inhibits multiple cytokines implicated in BP as well as the direct effector mechanisms of C5+LTB4 such as neutrophil and eosinophil activation***
- *Other drugs in development for BP primarily operate downstream of nomacopan, inhibiting specific cytokines/chemokines or immunoglobulins*
- *Dupilumab (Dupixent) inhibits IL-4 & IL-13 and benralizumab (Fasenra) inhibits IL-5, and ustekinumab (Stelara) inhibits IL12 and IL23 (via IL12p40).* ***See left***

Nomacopan (coversin) data adapted from Figs. 1, 2 & 3 of *Huber-Lang et al. J Immunol 2014*; *192:5324 – 5331*;
BP activity taken from *Le Jan et al 2019*: IL17, IL22, IL6 and IL23 raised in blister fluid; TGFbeta raised in serum; *Salz et al 2017*: IL31 elevated in serum and blister fluid;

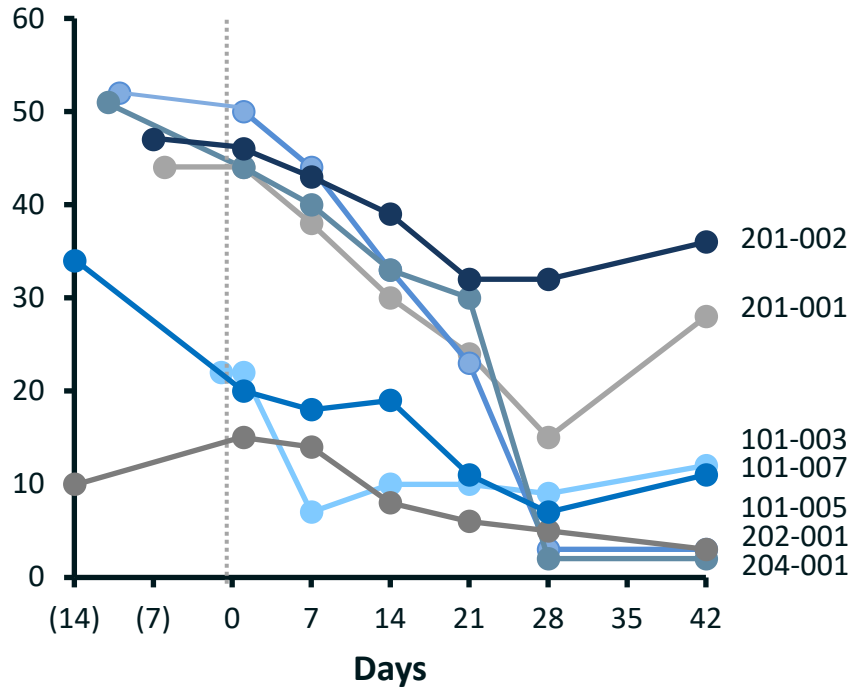
Phase 2 Outcome: Rapid Reduction in BPDAI Activity Scores of 7 Responders On Nomacopan

Single arm (n = 9); 42 days treatment; mild - moderate active BP; endpoints safety (primary) and BPDAI/QoL at Day 42

Mean BPDAI Activity + 90% CI



Individual Patients BPDAI Activity

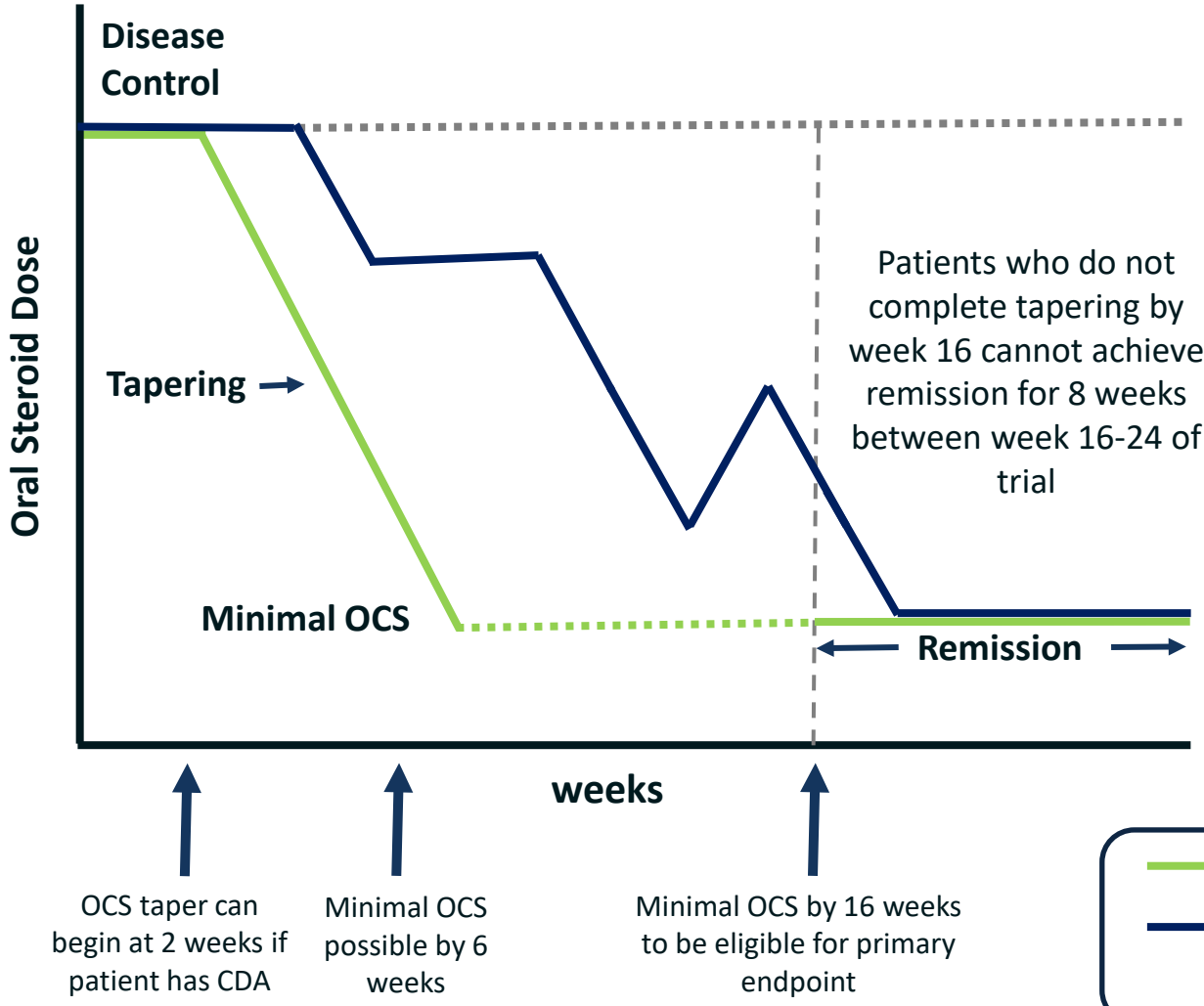


Phase 2 Observations

- **Rapid clinical response**
- **7 of 9 patients were responders**
- Clinical response similar to potent oral steroids
- Indication of remission within 6 weeks of treatment
- No reported grade 3, 4 or 5 treatment-related Adverse Events
- Supports Phase 3 design of rapid steroid tapering

- All prior treatment, including steroids, withdrawn circa one week prior to initiation of treatment with nomacopan
- Addition of <30g/week low dose 0.1% llesional mometasone, a moderate topical steroid, was permitted from study initiation through Day 21. Mometasone may stabilize mild BP - but is not sufficiently potent to control moderate or severe BP

BP Phase 3 Design – Visual Summary



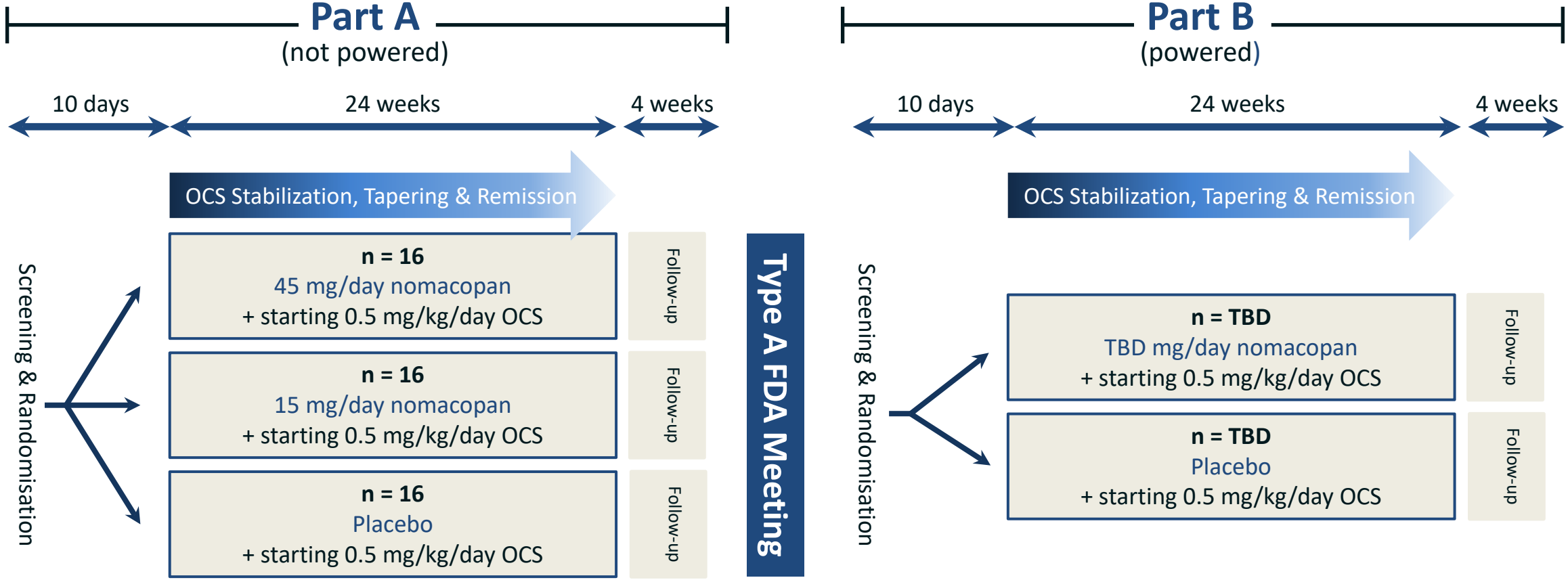
- Recruiting moderate and severe patients
- 24-week treatment vs 6-week for phase 2
- Systemic oral steroid - starting 0.5mg/kg/day
- Steroid tapering and disease remission phases
- Home monitoring to decrease patient burden
- Nomacopan likely to be additive to OCS which impact different immune pathways
- Testing 45mg (fully inhibiting C5 & LTB4) and 15mg (partially inhibiting C5 & fully inhibiting LTB4) vs single 30mg dose used in Phase 2

Patient 1: tapers to minimal OCS by 8 weeks and remains in disease remission

Patient 2: does not taper to minimal OCS by 16 weeks and fails primary endpoint

Note : if achieve wk16 remission but not maintained to wk 24 also fail primary end point

BP Phase 3 Design Provides Interim Readout After Part A



Primary endpoint: Achievement of Complete Disease Remission (Cr_{on}) on minimal OCS therapy (0.1mg/kg/day) for 8 weeks or more by Week 24

[CRon = Absence of new or established lesions and absence of pruritic symptoms while patient is receiving minimal therapy (≤ 0.1 mg/kg/day OCS) for at least 8 weeks]

Broad Applications Across Dermatology

Multiple market opportunities with $bn+ aggregate peak sales where both C5 and LTB4 implicated

Potential follow on dermatology targets

Hidradenitis suppurativa (HS)

Nomacopan uniquely inhibits three critical pathways

C5a

InflaRx: IFX-1 - C5a inhibition - Phase 3

Chemocentryx: Avacopan - C5aR inhibition Phase 2

MAC (C5b9)

C5b9 elevated and activates multiple pathways including TNF which is target of only HS approved drug adalimumab

LTB4

Novartis: Phase 2 (n=120) with LYS006 an LTA4H inhibitor (the enzyme that synthesises LTB4)

LTB4 is elevated in HS patients and in a trial with ustekinumab (anti-IL12/IL23) good responders had low LTA4H

Epidermolysis bullosa acquisita (EBA)

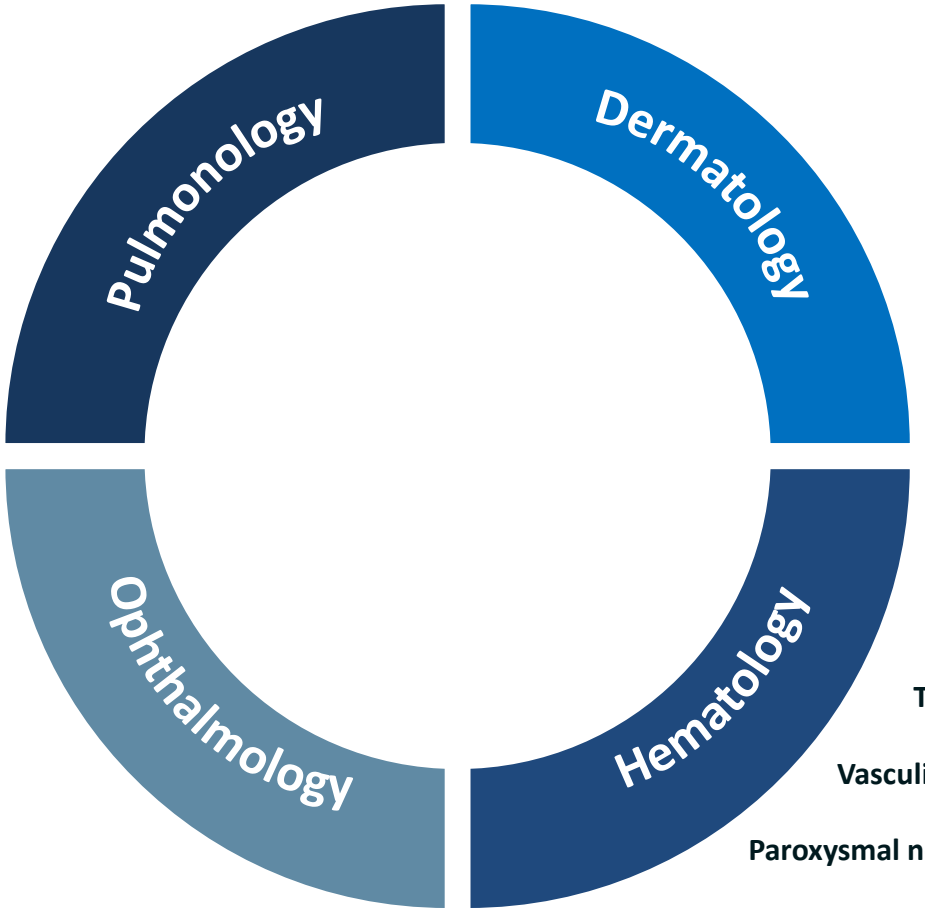
Mucous membrane pemphigoid (MMP)

Blistering skin diseases with similar pathology to BP and where C5 and LTB4 both implicated

HS : Other mediators being directly targeted include IL17, IL23, IL1alpha and CXCR1/2 which are downstream to and likely to be inhibited by nomacopan (in addition to its direct effect on C5 and LTB4

REFERENCES: *Kanni et al., 2018*: Complement activation in hidradenitis suppurativa; *Frew et al., 2019*: Topical, systemic and biologic therapies in hidradenitis suppurativa; *Penno et al., 2020*: Lipidomics profiling of hidradenitis suppurativa skin lesions reveals lipoxygenase pathway dysregulation and accumulation of proinflammatory leukotriene B4; *Blok et al., 2016:* Ustekinumab in hidradenitis suppurativa

HSCT-TMA:
High Clinical Impact, No Approved Therapies



Pediatric & adult HSCT-TMA

Anti-phospholipid syndrome

TMA in SLE

Vasculitis

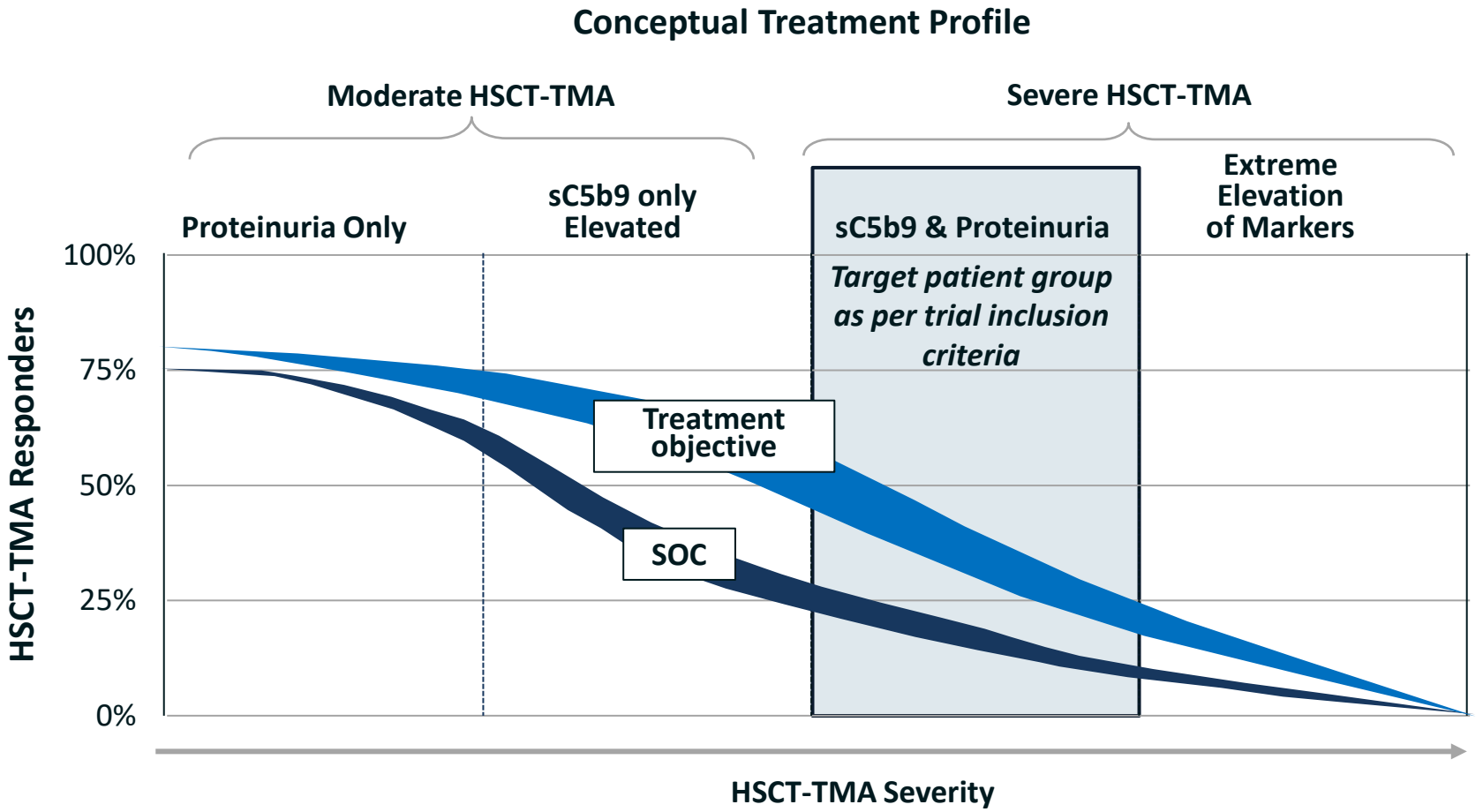
Paroxysmal nocturnal hemoglobinuria

Atypical hemolytic uremic syndrome

HSCT-TMA (Pediatric & Adult): Potential To Reduce Mortality In Aggressive Disease

Unmet Need	**High mortality rate of up to 80% associated with severe pediatric HSCT-TMA** • Need for rapid, complete & sustained C5 & potentially LTB4 inhibition
Prevalence	**~10K pediatric HSCT and ~60K adult HSCT performed in US + EU annually** • TMA diagnosed in up to 30% of HSCT • 50% of the HSCT-TMA cases are severe
Cause	**Vicious cycle of inflammation leads to progressive organ damage and death** • Complement activation leads to endothelial tissue injury, a prothrombotic state • LTB4 shown to impact GVHD progression and activate endothelial surfaces and neutrophil extracellular traps (NETs) exacerbating prothrombotic state and elevating inflammation
Treatment	**No approved therapies (SOC transfusions and immunosuppressants)** • Off-label use of eculizumab especially in US – used at higher dosing level than in PNH
Status	**Phase 3 study ongoing in pediatric population** • Orphan and Fast Track status

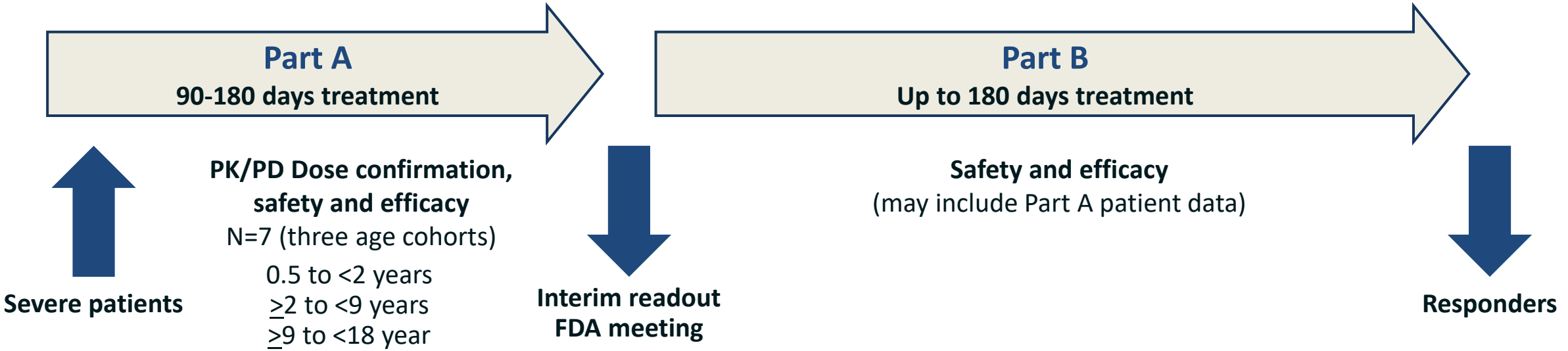
Pediatric HSCT-TMA Phase 3 Program Goals: Transfusions, Renal Function, Survival



- Targeting severe HSCT-TMA with elevated sC5b9 and proteinuria
- Early diagnosis and intervention critical to optimize responder outcomes
- Target population *c.*50% of patients with HSCT-TMA
- Treatment goals
  - Decrease transfusions
  - Renal improvement
  - Increase survival

Source: Conceptual graphic based on review of treatment literature, including Jodele et al Blood 2014 and Jodele et al Transfus Apher Sci 2016

Pediatric HSCT-TMA Open-Label Phase 3 Study:

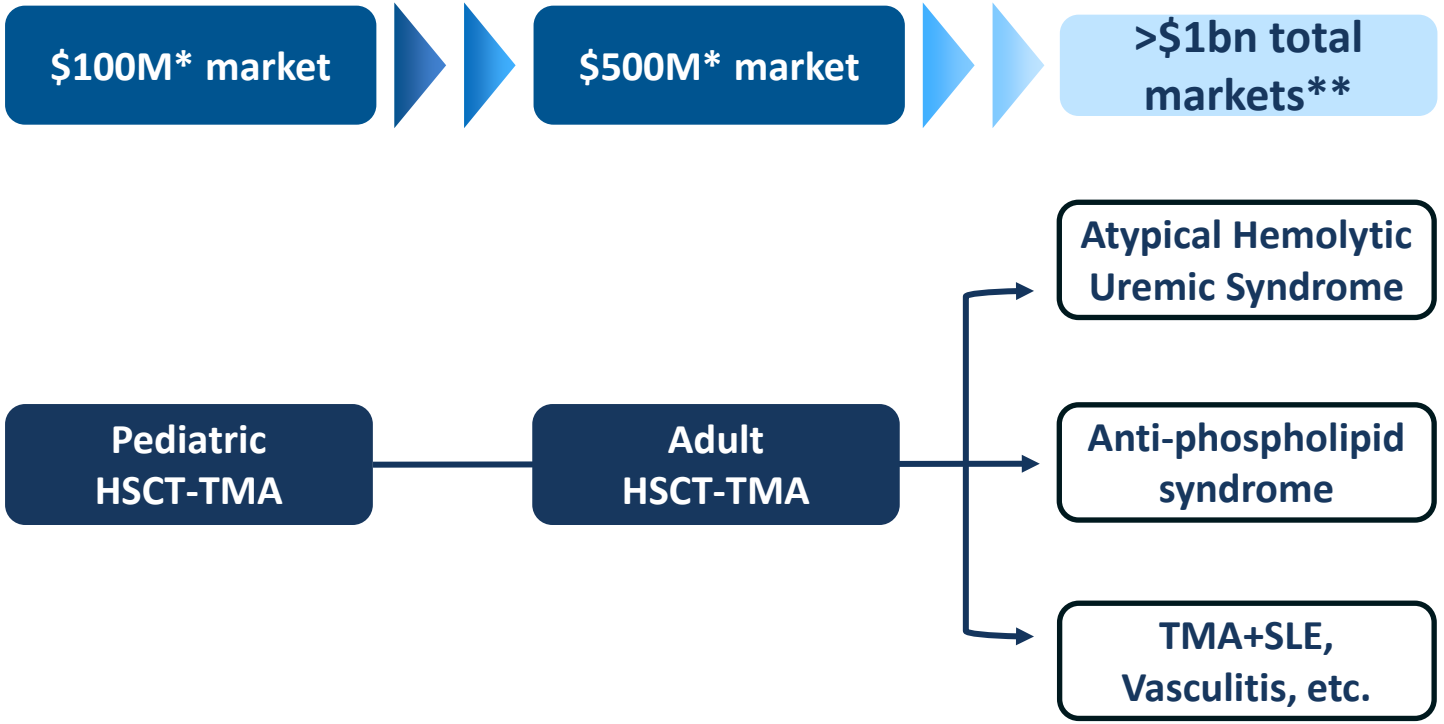


Primary Endpoints

- Response-based end point
- Reduced transfusion dependence and renal improvement

Broad Applications Across Hematology Revenue Potential > $1 Billion

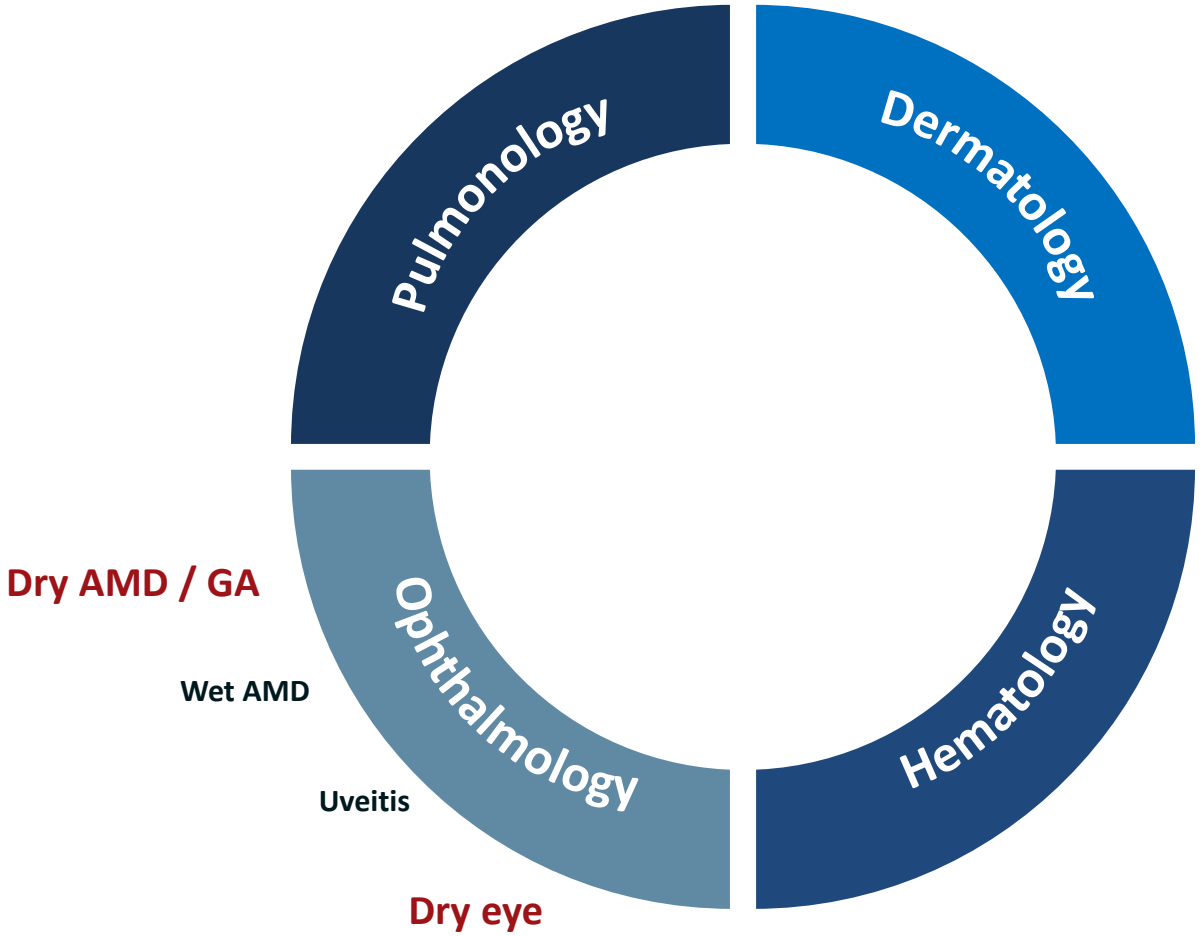
Success in HSCT-TMA leads to multiple follow-up market opportunities where role for nomacopan being investigated



- In aHUS *ex-vivo* model C5b9 elevated on endothelial cells exposed to aHUS patient serum. LTB4 also increases C5b9 deposition. Nomacopan reduces C5b-9 deposition to normal levels.
- In anti-phospholipid disease model nomacopcan reduces induced thrombi in mouse by 90% compared to normal human serum. Both C5 & LTB4 likely to play a role in disease.
- C5 and LTB4 implicated in wide range of endothelial and kidney diseases. Ongoing *ex vivo* study at OSU.

* US and EU5 markets only
** Initial and follow-on indications; company sources
Note : market size projection are company estimates based on existing treatments and prevalence

Ophthalmology Program In Dry AMD/GA & Dry Eye
Large Markets, Partnering Opportunities



Ophthalmology Programs: Partnering Potential

	Surface of the eye - Topical	Back of Eye (intra vitreal)
Historical	**Clinical :** • Several positive pre-clinical studies demonstrated rapid onset and effect equivalent to cyclosporin • LTB4 elevated in tear fluid, C5 and LTB4 in conjunctiva • AKC Phase 1/2 study showed no ocular treatment SAEs and nomacopan was comfortable and well tolerated	**Preclinical :** • C5 & LTB4 receptors both identified in retina • In EAU* model, PAS-nomacopan** significantly reduced inflammation; comparable to dexamethasone • Inhibition of LTB4 significantly reduces VEGF production the primary treatment for Wet AMD
Future	**Dry eye and related diseases** **More severe patients poorly treated** **$3bn + market** **Exploring phase 2 program**	**Dry AMD** **No approved treatment** **$5bn + market** **Ongoing work confirming injection interval**

* EAU: Experimental Autoimmune Uveitis

** PAS-nomacopan: PASylated nomacopan with longer half-life

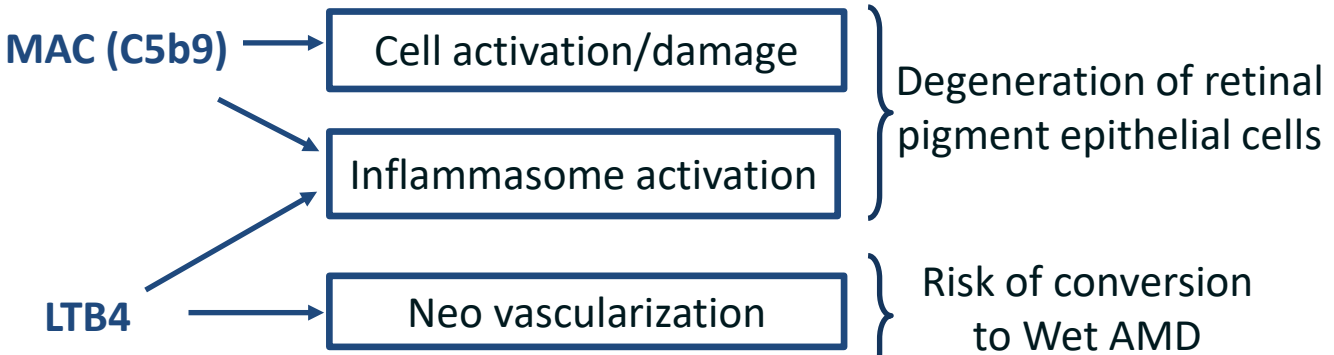
Note : market size projection are company estimates based on existing treatments and prevalence

PASylated-Nomacopan (Long-Acting) Provides Unique Treatment Approach In Back Of Eye Inflammatory Indications

Current Unmet Need

- Geographic Atrophy (GA): no approved therapy treatment
- Dry AMD is estimated as a $5+ billion market opportunity

Nomacopan Unique MOA



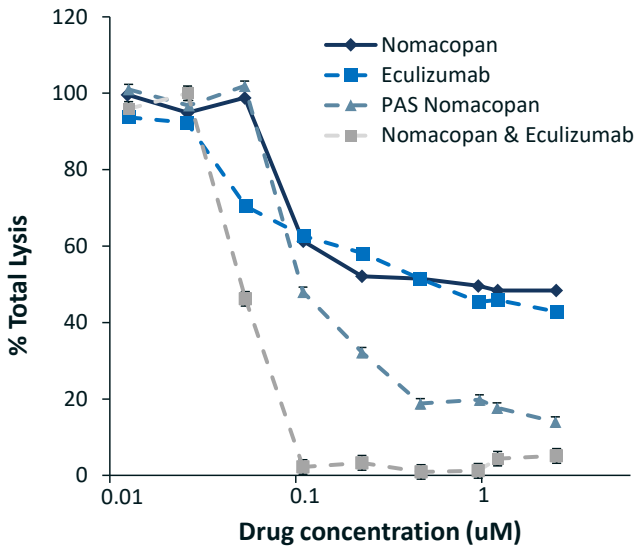
Extended Dosing

- Potential for low injection frequency and higher efficacy
- PAS-nomacopan: actual MW of 68kDa; apparent MW of >600kDa - greatly extending half life without reducing C5 or LTB4 binding

PAS-nomacopan Properties

- Manufacturing established
- Acute IV toxicity complete
- Activity demonstrated in preclinical models

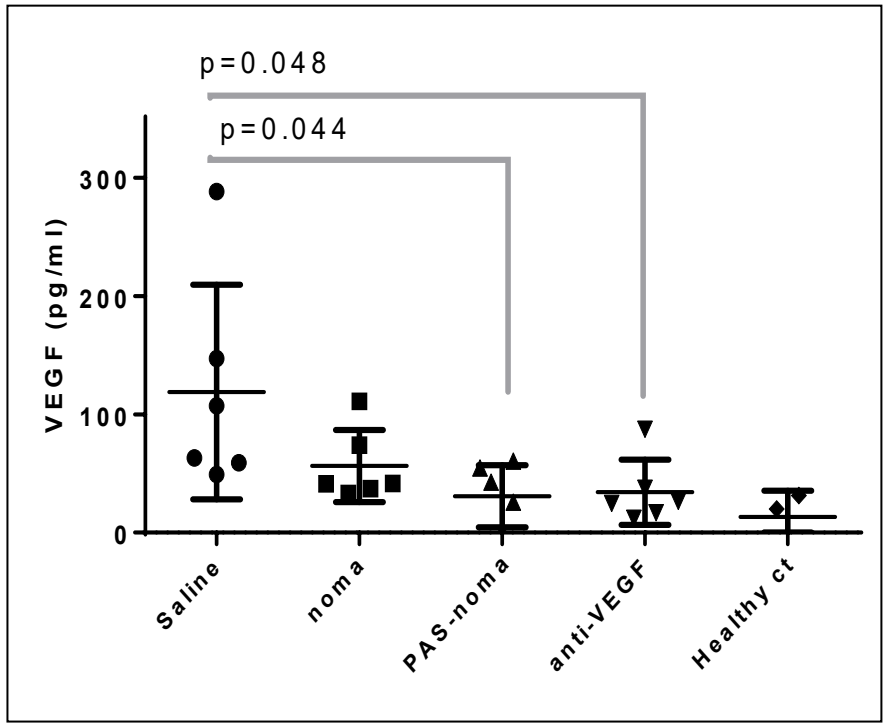
Inhibition of complement alternative pathway rabbit red blood cell lysis



IVT Nomacopan: Role Of C5 & LTB4 Established In Ocular Inflammatory Conditions

Nomacopan and PAS-nomacopan

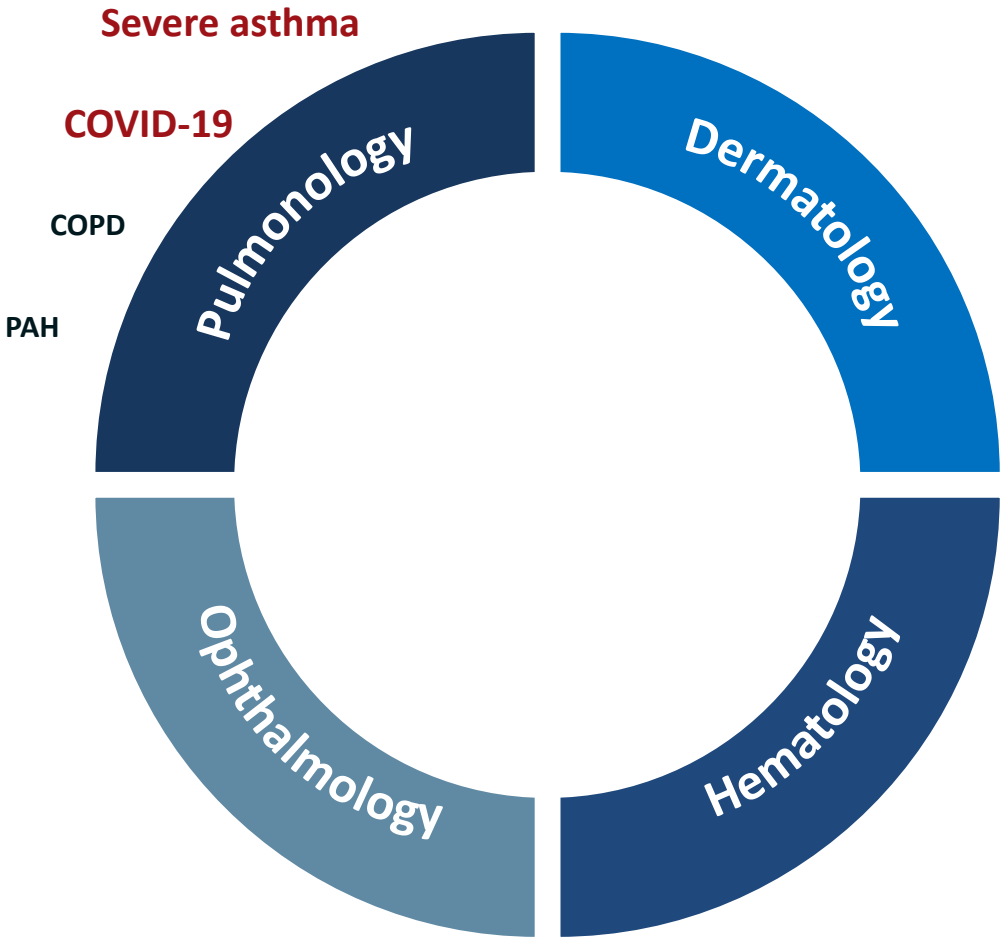
Effect on VEGF levels in mouse eye inflammation



- **In mouse eye inflammation model (1, 2)**
  - Significant VEGF reduction by PAS-nomacopan, equiv. to anti-VEGF
  - Additionally retinal inflammation reduced by 58% & 42% with PAS-nomacopan, versus saline & anti-VEGF respectively
- **In choroidal neovascularisation (CNV) model**
  - PAS- nomacopan (single dose) reduced neo-vascularisation in line with Eylea® (four doses), a VEGF inhibitor treatment for wet AMD
- **Ongoing rabbit study with PAS-nomacopan to determine optimal dose interval for IVT injection**
  - Quantify PAS-nomacopan in vitreous, choroid and retina after single IVT dose
  - Safety and histology
- **Opportunity in Dry AMD / GA**: where nomacopan may simultaneously reduce complement drive of GA and risk of CNV through LTB4 inhibitory activity on VEGF

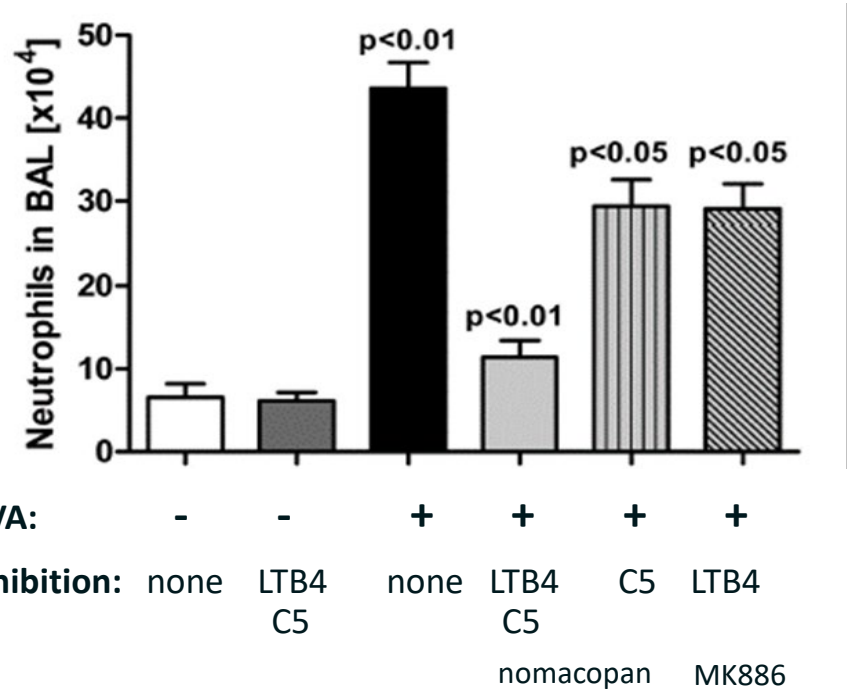
(**1**) Eskandarpour et al., *Am J Pathol*. 2021; 191:320-334. (**2**) Eskandarpour et al., *Cells* 2021;10:396. https://doi.org/10.3390/cells10020396

Potential Across Multiple Pulmonary Indications Large Markets, Partnering Opportunities



Additive Role Of C5 & LTB4 Established Across Multiple Pulmonary Indications

Lung model of immune-complex alveolitis
Dual inhibition superior to C5 or LTB4 inhibition alone



- Leukotriene pathway validated in the lung. Zileuton® approved in **severe asthma**
- Preclinical lung models show additive effects of C5 and LTB4 inhibition by nomacopan
- Nebulized nomacopan provides median particle size <5µM for deep lung delivery

- *Two forms of nomacopan one inhibiting C5+LTB4, the other inhibiting C5 only (pre-saturated with LTB4)*
- *LTB4 inhibition only is MK886 a leukotriene inhibitor that inhibits 5-lipoxygenase activator protein (FLAP)*
- *Source : Roversi et al (2013) JBC 288: 18789 - 18802*

Complex Pathology & Progression Of Severe Lung Inflammatory Diseases Reflects Potential For Inhibiting Multiple Pathways

AKARI THERAPEUTICS

COVID-19 Pneumonia

Severe Asthma

- Potential in a range of lung disorders, characterised by excessive neutrophil activity and exacerbations
- Leukotriene inhibition is an established treatment which can be optimized
- Growing clinical evidence for role of complement in COVID-19 pneumonia and severe asthma
- Opportunity for inhaled administration of nomacopan
- Partnering is likely strategy following proof of principal validation

- Biomarker & longitudinal study completed. LTB4 elevated and potential to identify COVID-19 patients suitable for nomacopan
- FDA approved multi-center investigator-led randomized study adopting a more intense dosing regimen following study in Brazil

- Lack of control in severe asthma - recurrent exacerbations requiring either oral steroids, visit to ER, or hospital admission
- Potential clinical study will assess efficacy in moderate-severe asthma refractory to long-acting beta-agonist (LABA) and inhaled corticosteroids (ICS)

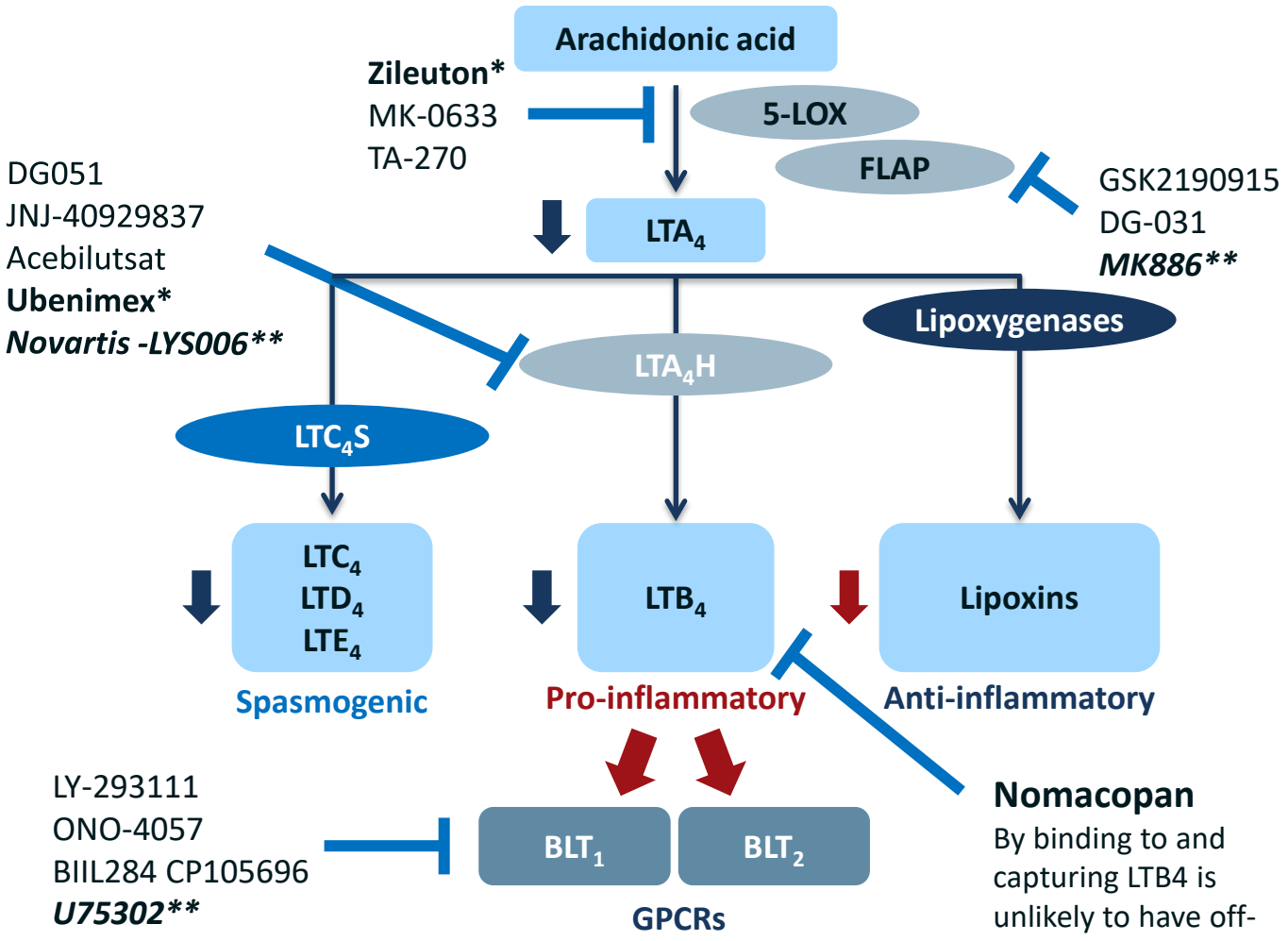
Akari Summary

- Therapeutic targets in C5 and LTB4 validated by recent $1 billion+ acquisitions* of companies targeting complement-mediated diseases
- Multiple inflection points in the near term
  - **BP**: Phase 3 recruitment expected Q3 2021
  - **TMA-HSCT**: Phase 3 pediatric study ongoing
  - **Ophthalmology**: Readout back of the eye residency data expected H2 2021
  - **Pulmonology:** Proof-of-principle activities
  - **R&D:** Multiple exploratory programs
- Lead indications expected to be gateways to high potential follow-on indications

* Acquisitions of RA Pharma by UCB, Achillion by Alexion and Alexion by AstraZeneca

Appendix

LTB4 Ligand Capture: Advantageous & Unique MOA



Note : *approved, **referenced in presentation deck

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

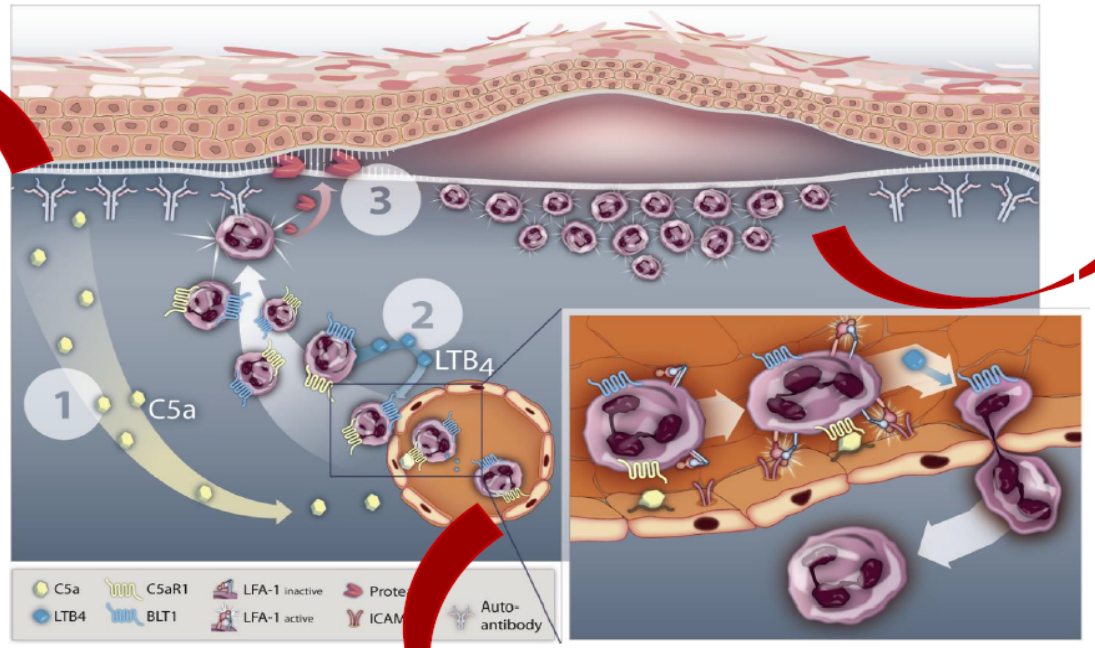
LTA_4H inhibitors

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

Note: LTB4 capture by nomacopan is external to cell so will not interfere with recently described anti-inflammatory effects of LTB4 within cells

Interaction Of C5 & LTB4 in Bullous Pemphigoid Pathology

1. Autoantibodies directed against dystonin (BP230) and type XVII collagen (BP 180) are deposited at the dermal-epidermal junction (DEJ); this leads to the formation of C5a



2. C5a binds with C5aR1 on neutrophils in dermal blood vessels and induces the release of LTB4. LTB4 further amplifies the recruitment of neutrophils and directs their migration to the DEJ.

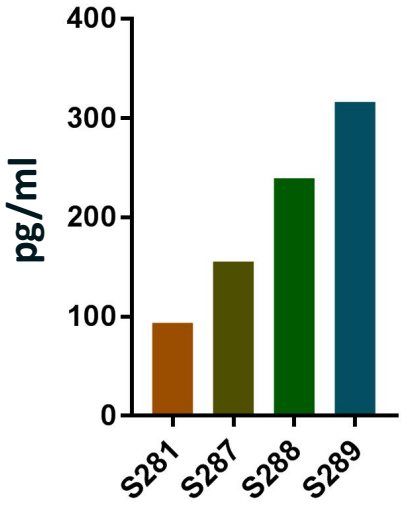
3. Activation of neutrophils at the DEJ by the deposited autoantibodies induces the release of proteases which degrade proteins of the DEJ and compromise dermal-epidermal adhesion.

Sadik et al., Semin Immunol 37:21-29 (2018)

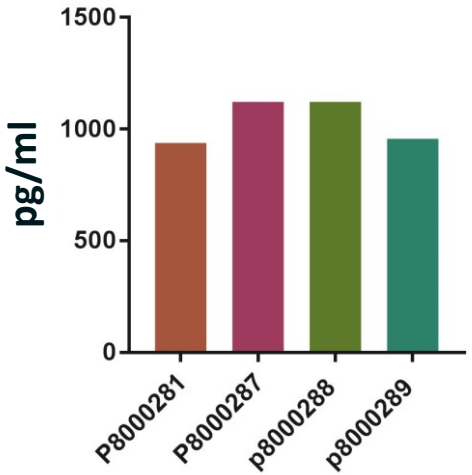
Rational for Nomacopan in Bullous Pemphigoid Preclinical Efficacy & Elevated C5a/LTB4 (In BP Patients)

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

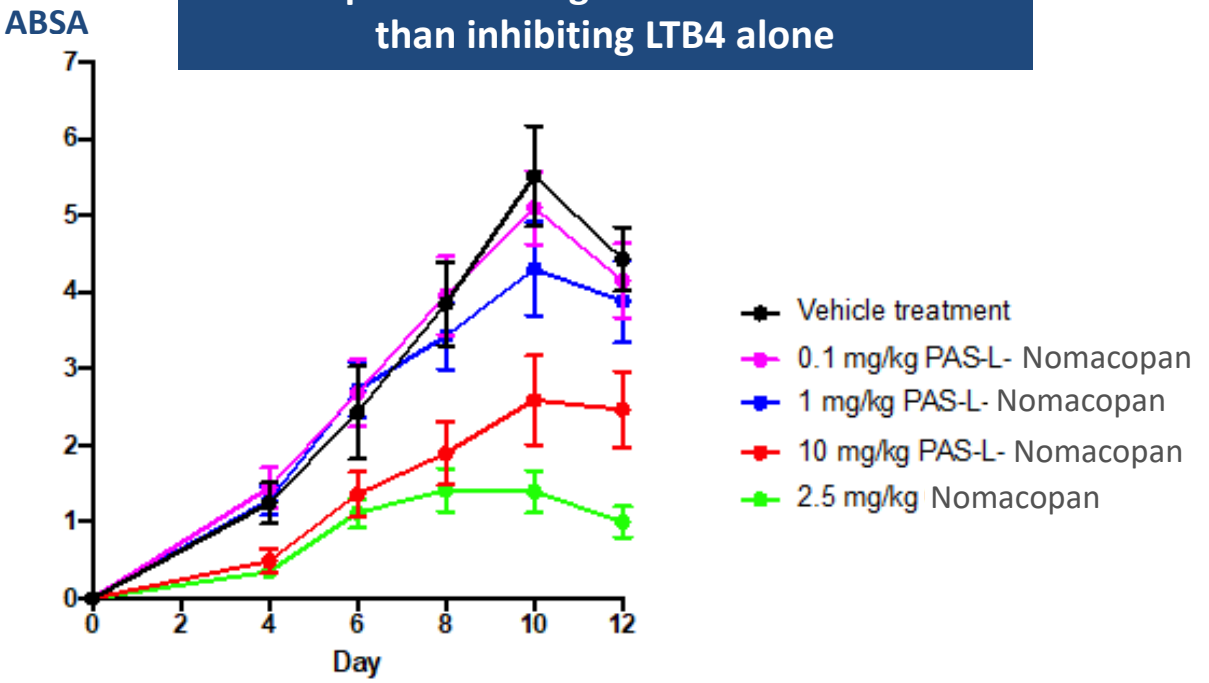
LTB4 levels
Human blister fluid



C5a levels in
blister fluid diluted 1:25



Nomacopan inhibiting C5+LTB4 more effective than inhibiting LTB4 alone



- Mouse model of BP-like epidermolysis bullosa acquisita (EBA)
- ~80% reduction in absolute body surface area affected by blisters (ABSA) on Nomacopan (SQ) compared to vehicle
- Long acting LTB4-only Nomacopan (10 mg/kg) ameliorates blister formation, but less effective than molar equivalent dose of Nomacopan (2.5 mg/kg) inhibiting C5 + LTB4

Preclinical passive mouse model of epidermolysis bullosa acquisita (EBA)

Role Of LTB4 In Neovascularization Of AMD

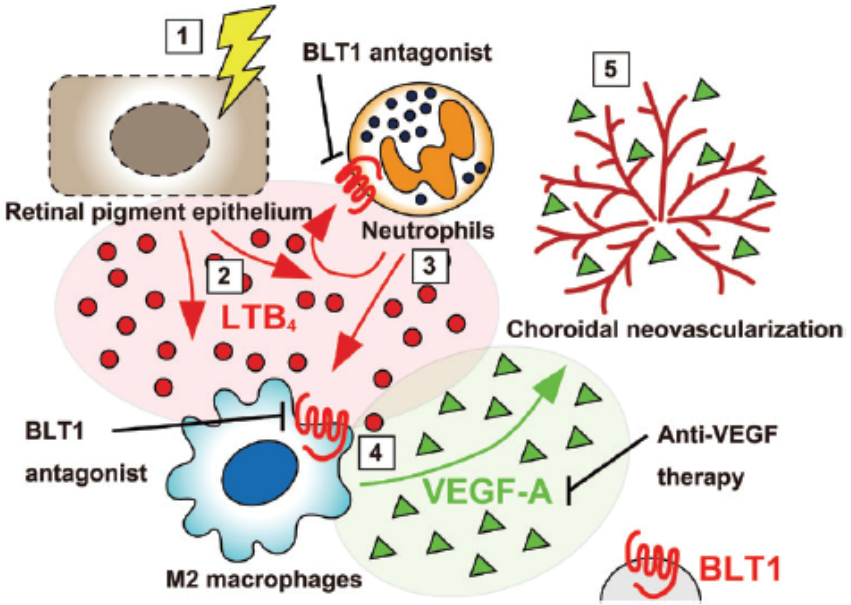


Fig. 2. M2 macrophages (M2 Mϕ) promote neovascular age-related macular degeneration (AMD) via the LTB_4–BLT1 axis (1). Retinal pigment epithelium damaged by lipid oxidation products such as malondialdehyde (2) releases leukotrienes such as LTB_4, resulting in (3) the recruitment of immune cells including neutrophils and inflammatory monocytes/Mϕ to the injured retina and leading to autocrine/paracrine loop of LTB_4 production in these cells (4). M2 Mϕ are also recruited and secrete VEGF-A, followed by (5) acceleration of choroidal neovascularization (CNV).

- LTB4 are released by damaged RPE in AMD
- LTB4 recruits immune cells including macrophages
- Recruited macrophages produce VEGF
- VEGF leads to CNV to form wet AMD

- Anti-VEGF is the standard of care for wetAMD.
  It works mainly by reducing choroidal neovascularization, (CNV) leakage / edema

- Anti-LTB4 can prevent CNV or stop further leakage

Sasaki & Yokomizo: The leukotriene receptors as therapeutic targets of inflammatory diseases. *International Immunology 2019, 31(9):607-615*

Cooperative Research & Development Agreement (CRADA) With US Army In Trauma

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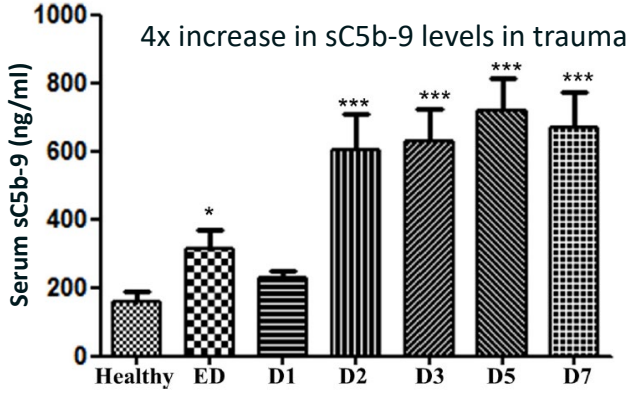
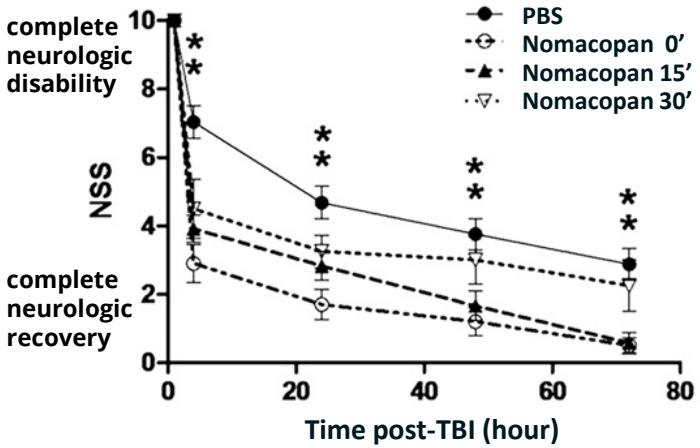
USAISR study of complement level in trauma patients



Nomacopan (OmCI) reduces neural damage in mouse trauma model



- Ongoing collaboration with U.S. Army Institute of Surgical Research (USAISR) to evaluate nomacopan activity in pig model of blast injury and haemorrhagic shock
- Nomacopan reduced secondary neuroaxonal loss and promoted neurologic recovery after traumatic brain injury (in mice)
- Nomacopan does not require special handling and can be carried at ambient temperature; may facilitate use in prehospital settings
- C5 and LTB4 inhibition by nomacopan for treatment of trauma is supported by a large body of literature reflecting the harmful role for both these inflammatory mediators in early pathophysiology of trauma & haemorrhagic shock

Early complementopathy predicts the outcomes of patients with trauma; Li Y, et al. Trauma Surg Acute Care Open 2019;4:e000217

Inhibition of the Membrane Attack Complex of the Complement System Reduces Secondary Neuroaxonal Loss and Promotes Neurologic Recovery after Traumatic Brain Injury in Mice; J Immunol 2014; 192:2339-2348

Active Pipeline Program Expansion

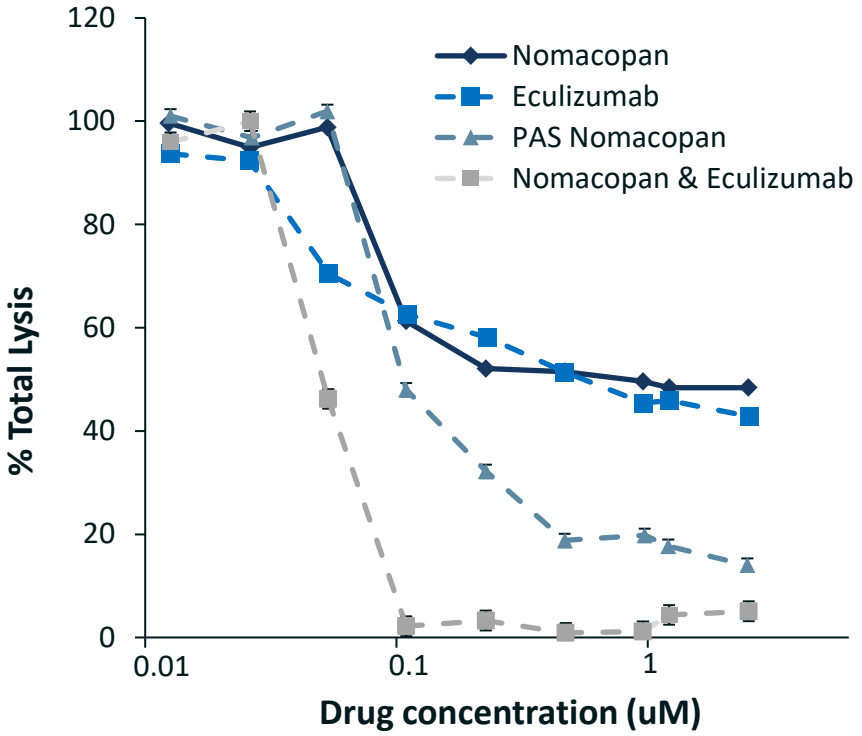
Engineered Nomacopan

- Long-acting PASylated nomacopan with potential for weekly SQ / IVT dosing
- Tissue targeted : nomacopan linked to peptides which bind to specific disease related tissue such as neuromuscular junction
- Ligand specific: C5 or LTB4 inhibition only for diseases that do not require inhibition of both mediators

Other lipocalin molecules

- Votucalis captures histamine uniquely preventing activation of all four histamine receptors which can induce diverse pathophysiological processes, including chronic pain, itch and inflammation. Potential for topical delivery.

Inhibition of complement alternative pathway rabbit red blood cell lysis



Combined Inhibition Of Complement & Leukotriene Pathways To Treat Inflammatory Diseases

July 2021