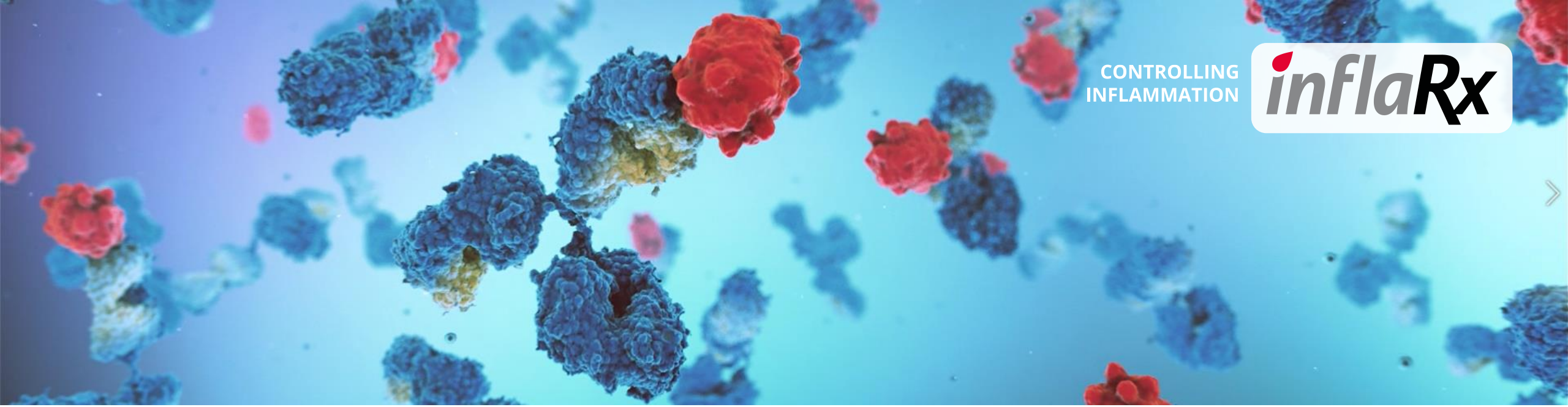
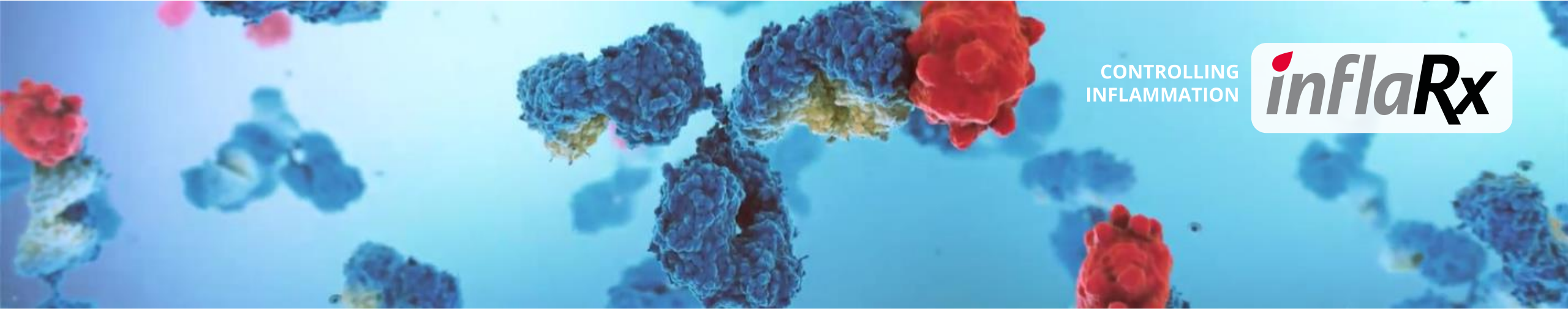


CORPORATE PRESENTATION

MARCH 2024



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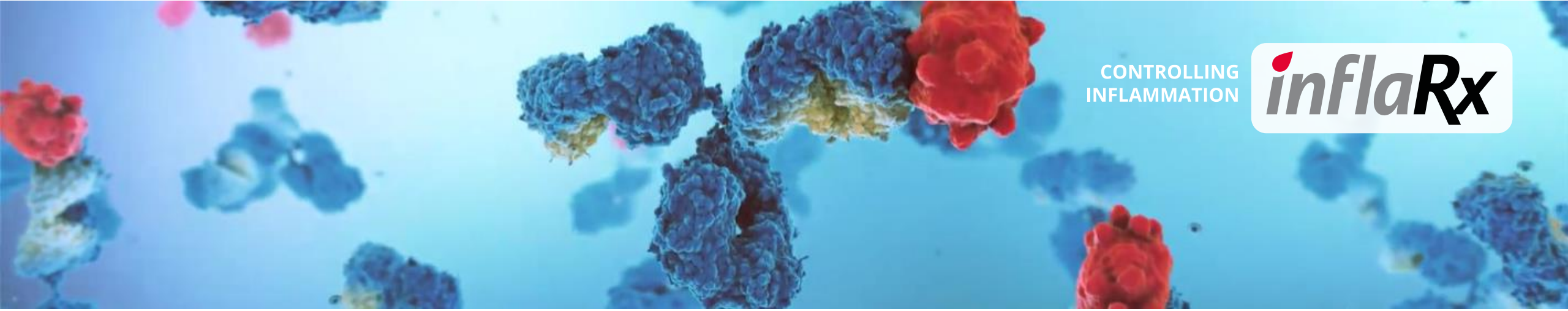
Forward-Looking Statements

This press release contains forward-looking statements. All statements other than statements of historical fact are forward-looking statements, which are often indicated by terms such as "may," "will," "should," "expect," "plan," "anticipate," "could," "intend," "target," "project," "estimate," "believe," "predict," "potential" or "continue," among others. Forward-looking statements appear in a number of places throughout this release and may include statements regarding our intentions, beliefs, projections, outlook, analyses and current expectations concerning, among other things, the receptiveness of GOHIBIC (vilobelimab) as a treatment for COVID-19 by COVID-19 patients and U.S. hospitals and related treatment recommendations by medical/healthcare institutes and other third-party organizations, our ability to successfully commercialize and the receptiveness of GOHIBIC (vilobelimab) as a treatment for COVID-19 by COVID-19 patients and U.S. hospitals or our other product candidates; our expectations regarding the size of the patient populations for, market opportunity for, coverage and reimbursement for, estimated returns and return accruals for, and clinical utility of GOHIBIC (vilobelimab) in its approved or authorized indication or for vilobelimab and any other product candidates, under an EUA and in the future if approved for commercial use in the U.S. or elsewhere; our ability to successfully implement The InflaRx Commitment Program, the success of our future clinical trials for vilobelimab's treatment of COVID-19 and other debilitating or life-threatening inflammatory indications, including PG, and any other product candidates, including INF904, and whether such clinical results will reflect results seen in previously conducted pre-clinical studies and clinical trials; the timing, progress and results of pre-clinical studies and clinical trials of our product candidates and statements regarding the timing of initiation and completion of studies or trials and related preparatory work, the period during which the results of the trials will become available, the costs of such trials and our research and development programs generally; our interactions with regulators regarding the results of clinical trials and potential regulatory approval pathways, including related to our MAA submission for vilobelimab and our biologics license application submission for GOHIBIC (vilobelimab), and our ability to obtain and maintain full regulatory approval of vilobelimab or GOHIBIC (vilobelimab) for any indication; whether the FDA, the EMA or any comparable foreign regulatory authority will accept or agree with the number, design, size, conduct or implementation of our clinical trials, including any proposed primary or secondary endpoints for such trials; our expectations regarding the scope of any approved indication for vilobelimab; our ability to leverage our proprietary anti-C5a and C5aR technologies to discover and develop therapies to treat complement-mediated autoimmune and inflammatory diseases; our ability to protect, maintain and enforce our intellectual property protection for vilobelimab and any other product candidates, and the scope of such protection; our manufacturing capabilities and strategy, including the scalability and cost of our manufacturing methods and processes and the optimization of our manufacturing methods and processes, and our ability to continue to rely on our existing third-party manufacturers and our ability to engage additional third-party manufacturers for our planned future clinical trials and for commercial supply of vilobelimab and for the finished product GOHIBIC (vilobelimab); our estimates of our expenses, ongoing losses, future revenue, capital requirements and our needs for or ability to obtain additional financing; our ability to defend against liability claims resulting from the testing of our product candidates in the clinic or, if approved, any commercial sales; if any of our product candidates obtain regulatory approval, our ability to comply with and satisfy ongoing obligations and continued regulatory overview; our ability to comply with enacted and future legislation in seeking marketing approval and commercialization; our future growth and ability to compete, which depends on our retaining key personnel and recruiting additional qualified personnel; and our competitive position and the development of and projections relating to our competitors in the development of C5a and C5aR inhibitors or our industry; and the risks, uncertainties and other factors described under the heading "Risk Factors" in our periodic filings with the SEC. These statements speak only as of the date of this press release and involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. Given these risks, uncertainties and other factors, you should not place undue reliance on these forward-looking statements, and we assume no obligation to update these forward-looking statements, even if new information becomes available in the future, except as required by law.



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Information and Sources

Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and InflaRx's own internal estimates and research. While InflaRx believes these third-party sources to be reliable as of the date of this presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, all of the market data included in this presentation involves a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Further, while we believe our own internal research is reliable, such research has not been verified by any independent source.

Avacopan Data

We have not conducted a head-to-head comparison of Avacopan to INF904 in a clinical trial but have compared the published data for Avacopan to data from our Phase 1 clinical trial of INF904. For the purpose of conducting pre-clinical studies (hamster neutropenia study), we synthesized Avacopan and did a side-by-side comparison. While we believe this comparison to Avacopan to be useful and appropriate, the value of this and other comparisons to Avacopan in this presentation may be limited because they are not derived from a head-to-head trial and they are from trials that were conducted under different protocols at different sites and at different times. Without head-to-head data, we are unable to make comparative claims between INF904 and Avacopan.

About InflaRx

InflaRx GmbH (Germany) and InflaRx Pharmaceuticals Inc. (USA) are wholly owned subsidiaries of InflaRx N.V. (together, "InflaRx").

InflaRx (Nasdaq: IFRX) is a biotechnology company pioneering anti-inflammatory therapeutics by applying its proprietary anti-C5a and anti-C5aR technologies to discover, develop and commercialize first-in-class, potent and specific inhibitors of the complement activation factor C5a and its receptor C5aR. C5a is a powerful inflammatory mediator involved in the progression of a wide variety of inflammatory diseases. InflaRx's lead product candidate, vilobelimab, is a novel, intravenously delivered, first-in-class, anti-C5a monoclonal antibody that selectively binds to free C5a and has demonstrated disease-modifying clinical activity and tolerability in multiple clinical studies in different indications. InflaRx was founded in 2007, and the group has offices and subsidiaries in Jena and Munich, Germany, as well as Ann Arbor, MI, USA. For further information, please visit www.inflarx.com.

Harnessing C5a/C5aR for Controlling Inflammation in the I&I Space

InflaRx Highlights

Uniquely targeting complement C5a/C5aR, a validated mechanism and critical part of the inflammation cascade with:

- **First-in-class and highly potent anti-C5a monoclonal antibody (vilobelimab + second generation IFX-2)**
- **Best-in-class potential oral C5aR inhibitor INF904**:
  - **Addressing limitations** of marketed comparator (clearly differentiated plasma PK profile and inhibitory potential in phase I study)
  - **Pipeline-in-a-drug** with potential to address several large markets in immuno-dermatology and broader I&I

A targeted development focus on immuno-dermatology where InflaRx can drive pipeline value in larger markets and has strong core IP and medical use IP coverage

- **Vilobelimab in late-stage development for PG** an unmet need with no approved drug in the US or Europe
- **INF904** to initially demonstrate pipeline-in-a-drug potential in large markets of CSU and HS; expected to start Phase II development in 2024

Large upside potential in additional indications in I&I for proprietary drugs with options for collaborations

Strong balance sheet with enough cash to fund operations into at least 2026 and advance programs toward next milestones

Team with proven **track record of delivering clinical and regulatory successes**

CSU [chronic spontaneous urticaria. **HS** [hidradenitis suppurativa]. **PG** [pyoderma gangrenosum]. **I&I** [inflammation and immunology].

Significant Opportunity in Immuno-Dermatology

Why Immuno-Dermatology

- Potential to target **several attractive, billion-dollar+ commercial markets**
- InflaRx has identified **unmet medical needs** that INF904 could strongly address
- **Strong rationale** for the role of C5a/C5aR based on mechanism of action, pre-clinical and clinical data
- **Established endpoints** with the ability of INF904 to potentially achieve a clinical edge and prove to be a differentiated competitor
- INF904 is an oral drug with **no known safety concerns and potential broad therapeutic index**
- As a C5aR antagonist, INF904 acts on a **differentiated pathway with a MoA** not currently addressed by any other treatment approaches in the immuno-dermatology field
- Established **network of experts and in-house trial expertise**
- **Strong IP coverage** for C5aR inhibition in certain immuno-dermatological diseases

Focus INF904 on Immuno-Dermatology: I&I Pipeline-in-a-Drug Potential

Immuno-dermatology

- **Chronic spontaneous urticaria (CSU)**
- **Hidradenitis suppurativa (HS)**
- others

Key initial development focus and area to demonstrate potential of INF904

Neurology

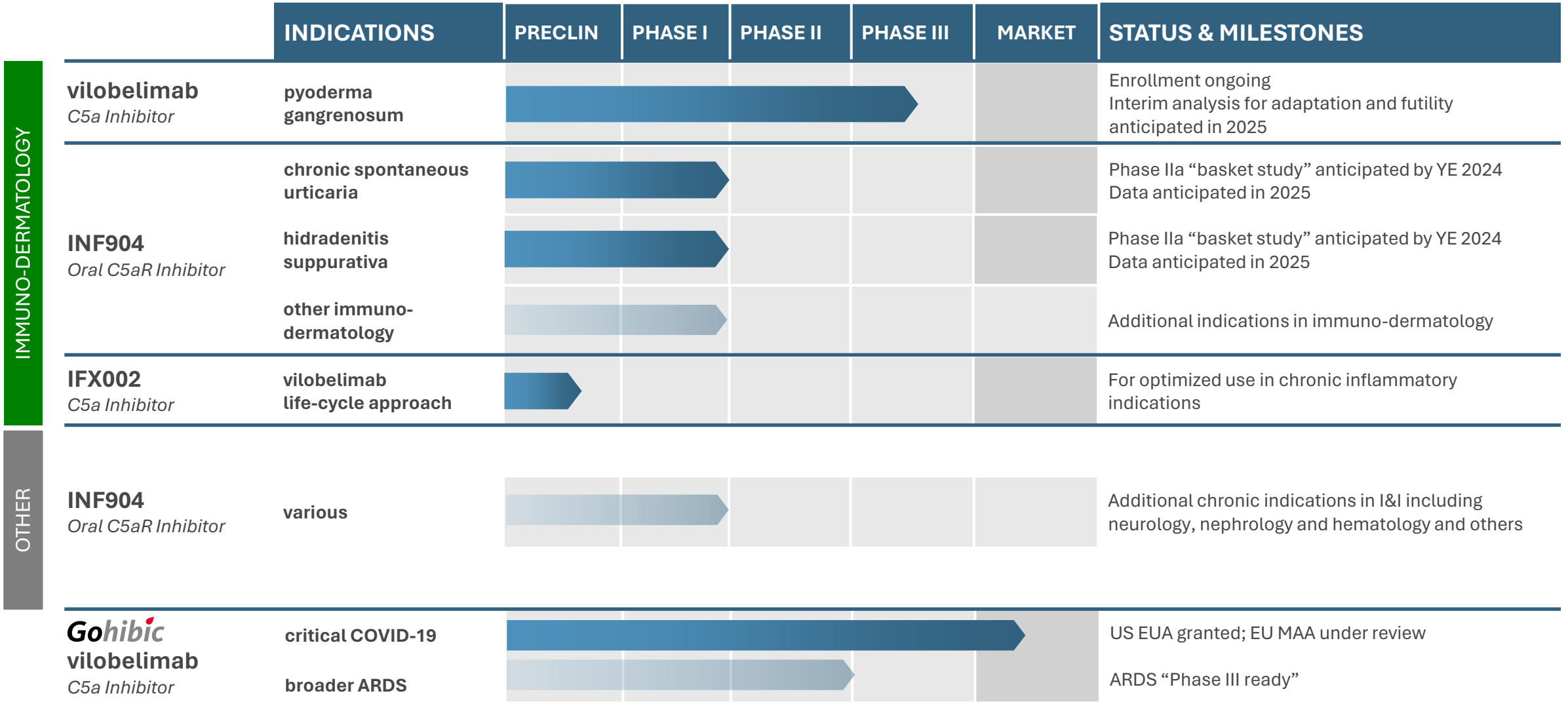
- Chronic inflammatory demyelinating polyneuropathy (CIDP)
- Dermatomyositis

Nephrology & Hematology

- Anti-C3 glomerulopathy (C3G)
- Atypical haemolytic uraemia syndrome (aHUS)
- Immunoglobulin A nephropathy (IgAN)
- ANCA-associated vasculitis (AAV)

Potential for future development or development in collaboration with a partner

Late-Stage Pipeline Targets Multiple Sizable Markets



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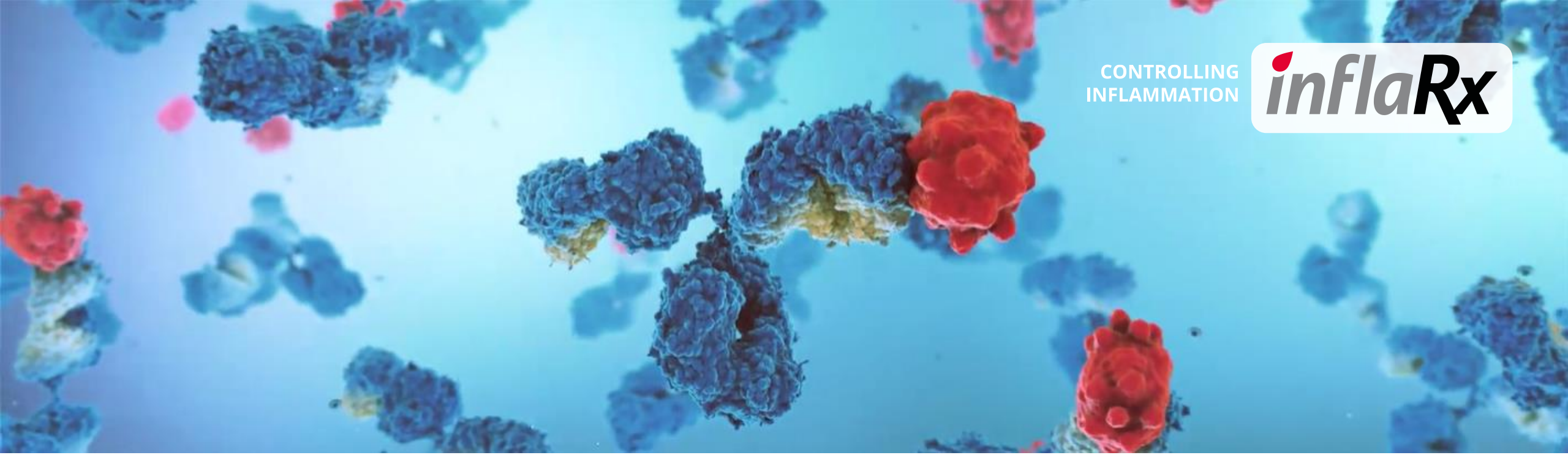


C5a/C5aR: A Strategic Position in the Inflammatory Cascade

Vilobelimab [C5a monoclonal antibody]

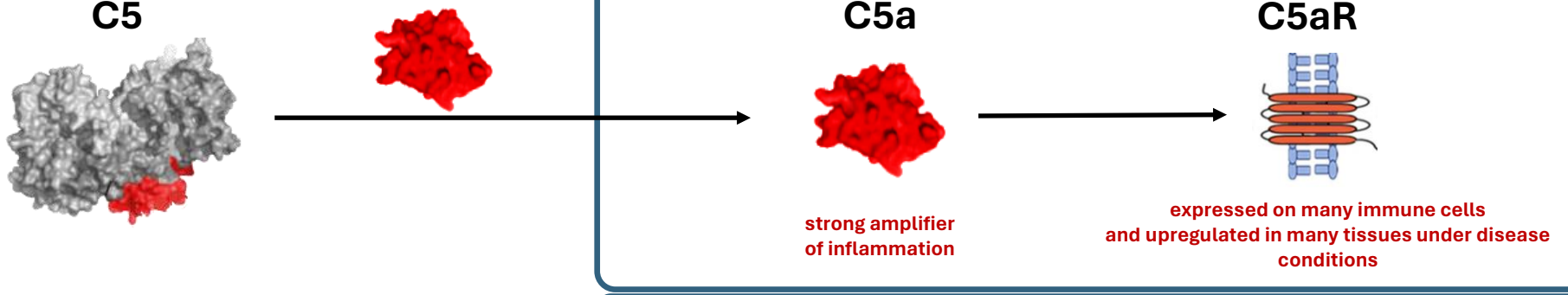
INF904 [oral C5aR inhibitor]

C5a/C5aR are Validated Targets Promoting Inflammation

Targeting strong pro-inflammatory mechanisms

vilobelimab
intravenous mAB

INF904
oral small molecule



Targeting C5 (e.g. marketed C5 blockers) **does not prevent** enzymatic **C5a formation**, but only complement pathway mediated cleavage (classic, lectin, alternative)
⇨ **not suitable** for tightly controlling C5a/C5aR1-driven inflammation

Anaphylatoxin C5a is upstream of the cytokine network

- Boosting effect on various pro-inflammatory cytokines
- IL-17, IL-6, IL-8, IL-1 and others

Strong activator of neutrophils and macrophages

- Chemotaxis of neutrophils
- O_2 radical generation + granular enzyme release
- NETosis (neutrophil extracellular traps)

Essential role in many inflammatory conditions

- Acute and chronic inflammation and other conditions
- Over 6,000 publications on role in numerous diseases

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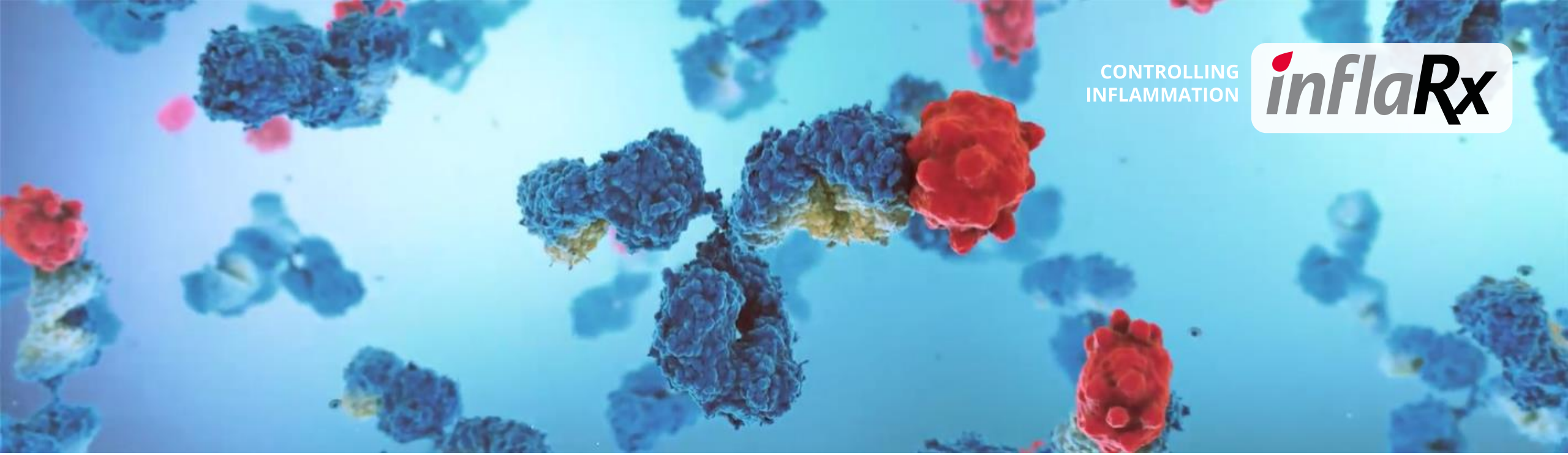
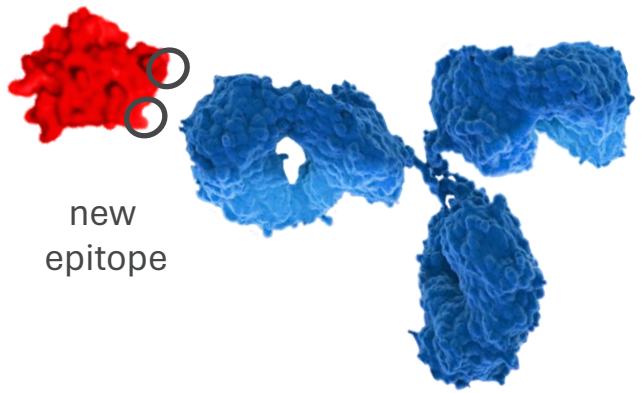


Vilobelimab for Ulcerative Pyoderma Gangrenosum (PG)

Vilobelimab: A First-in-Class Anti-C5a Monoclonal Antibody

Vilobelimab Key Features

- ✓ Highly selective anti-C5a mAB
- ✓ Blocks C5a biological effects up to 100% in human blood
- ✓ Leaves MAC formation intact
- ✓ Fast binding / high affinity to the newly discovered epitope
- ✓ Commercially validated / available under Emergency Use Authorization in certain severely ill COVID-19 patients



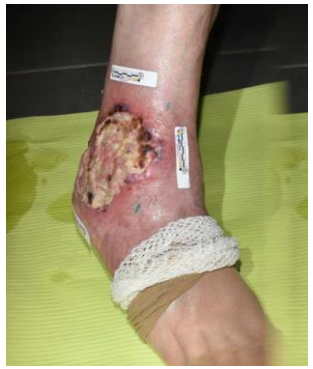
Development Areas in Acute and Sub-Acute Inflammation

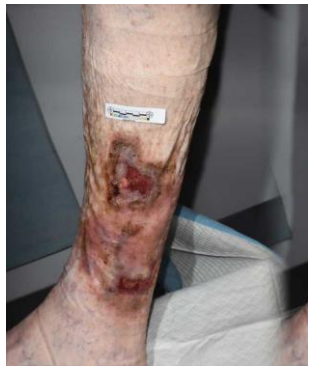
As a fast acting highly specific monoclonal antibody infused, vilobelimab delivers:

- Strong and immediate C5a inhibition in blood
- Fast onset of inhibition of neutrophil activation in human blood
- Potential disease modifying activity for diseases in which C5a signaling may play a key role

PG: An Autoimmune Condition With High Unmet Need

PG Overview and Unmet Need





Clinical features

- PG is a rare but potentially life-threatening skin disorder that can lead to chronic, difficult-to-treat wounds
- Patients frequently suffer from other autoimmune disorders, e.g. ulcerative colitis, rheumatoid arthritis and hematological diseases
- Patients suffer from severe pain, long healing times and frequent relapses

Incidence and market potential

- Rare – estimated that up to 50,000 patients in the US and Europe are affected
- Significant market potential – premium pricing expected based on performed market study

Current treatment and medical need

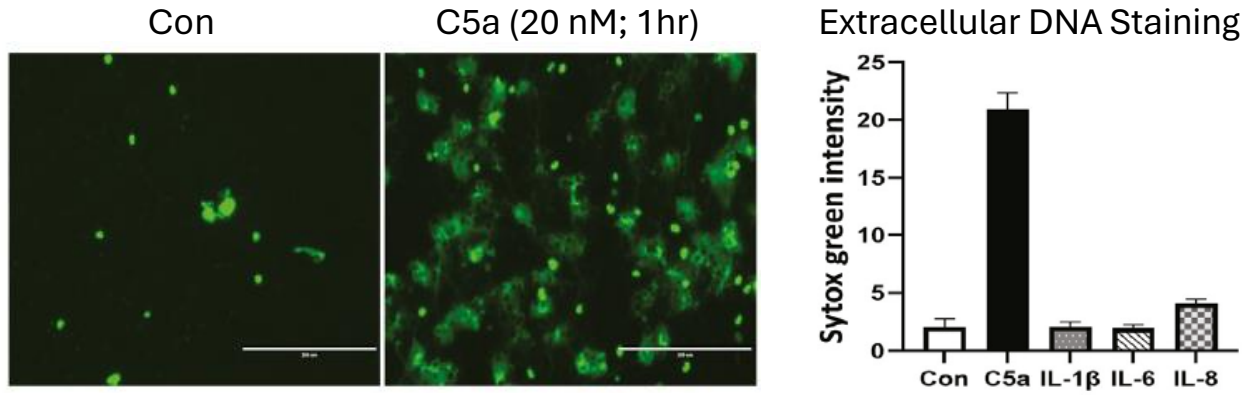
- No drugs currently approved in the US or EU
- For less severe cases, topical or intralesional treatments can be used, including topical steroids
- Use of systemic immunosuppression in rapidly progressing cases
- Mixed reports about efficacy; long treatment durations and relapses are frequently seen

› Strong rationale for treatment with vilobelimab:
PG associated with neutrophilic skin infiltration in affected areas and lesions, potentially triggered by C5a

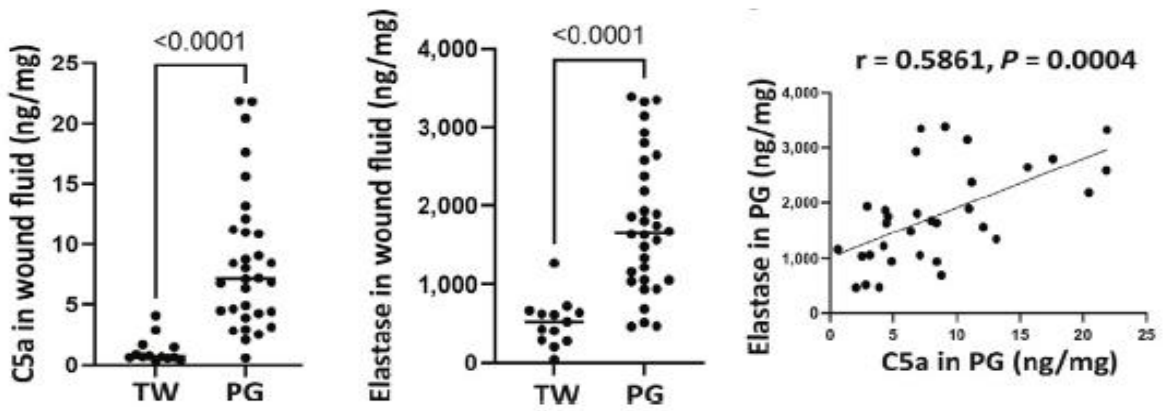
PG Pathogenesis: Potential Role of the C5a/C5aR Axis

- The etiology of PG is believed to be linked to the dysregulation of the immune system, specifically, **altered neutrophil function**
- Evidence suggest that **complement activation and C5a play an important role** in the disease development:
  - High C5a levels were detected in the wound fluids from PG patients
  - C5a levels correlated well with elastase levels in wound fluids, a NETosis marker
  - C5a/C5aR axis activation may be a key driver for NETosis in PG

C5a Induces NETosis in Control Neutrophils



C5a Levels in PG Wound Fluid Correlate With NETosis



Wang et al 2024. J invest Derm. 144; TW = Trauma Wound

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PG Phase IIa Showed No Safety or Tolerability Concerns and Dose-Dependent Drug Activity

Clinical Response

- **High-dose group showed highest rate of target ulcer closure and clinical remission (86%)**
- Out of 17 evaluable patients at end of treatment visit or day of last drug administration
  - **Clinical remission (PGA ≤ 1) reported in 9 patients (53%)**
  - Clinical response (PGA ≤ 3) reported in 1 additional patient (6%)
  - Slight improvement (PGA = 4) reported in 7 patients (41%)

Safety

- No infusion-related reactions observed
- For 2 patients, related SAEs were reported
  - Erysipelas leading to hospitalization (judged as non-related by sponsor)
  - Rash due to delayed hypersensitivity reaction
- Observed AE profile in line with patients' underlying diseases
- No dose-related AEs detected

Phase III Initiated Based on Feedback From FDA

Orphan Drug and Fast Track Status US FDA

Orphan Drug Status EMA

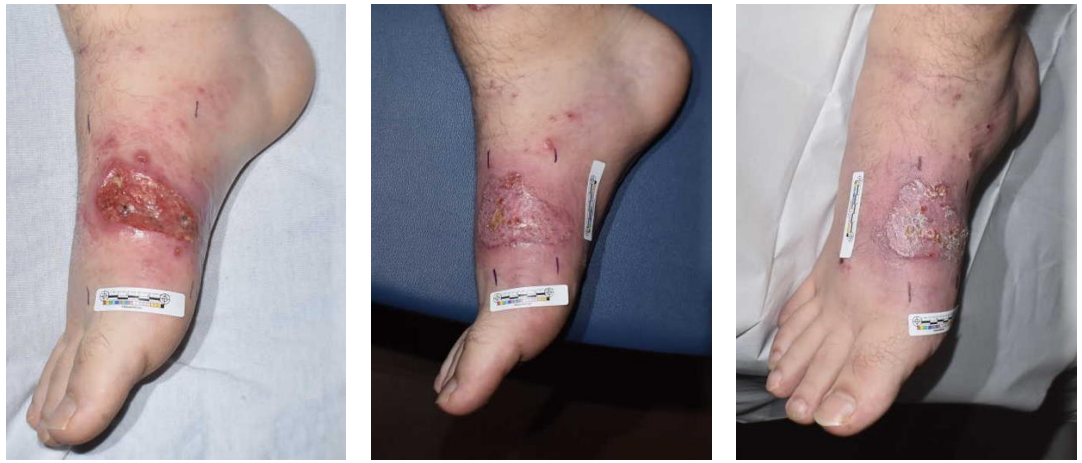
PG Study Phase IIa – Treatment Examples

Patient Case Studies

Target Ulcer Developed While on Adalimumab

- **MH:** PG since August 2020, Psoriasis since 2017
- **Previous PG medication:** None
- **Cohort 3:** 2400 mg Q2W up to Day 85 -> exclusion after 9 doses due to delayed availability of pos. baseline TB testing result (no TB activation)
- **Concomitant medication:** Adalimumab for psoriasis 40mg q2w since 2017

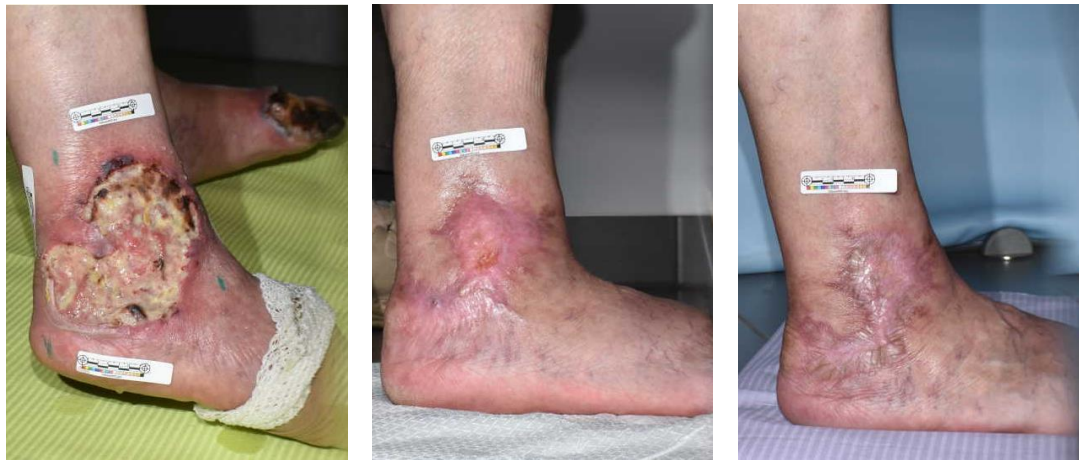
Baseline	Day 85	Day 89
	PGA = 1	PGA = 1
Area: 1136 mm^2	Area: 0.00 mm^2	Area: not yet available



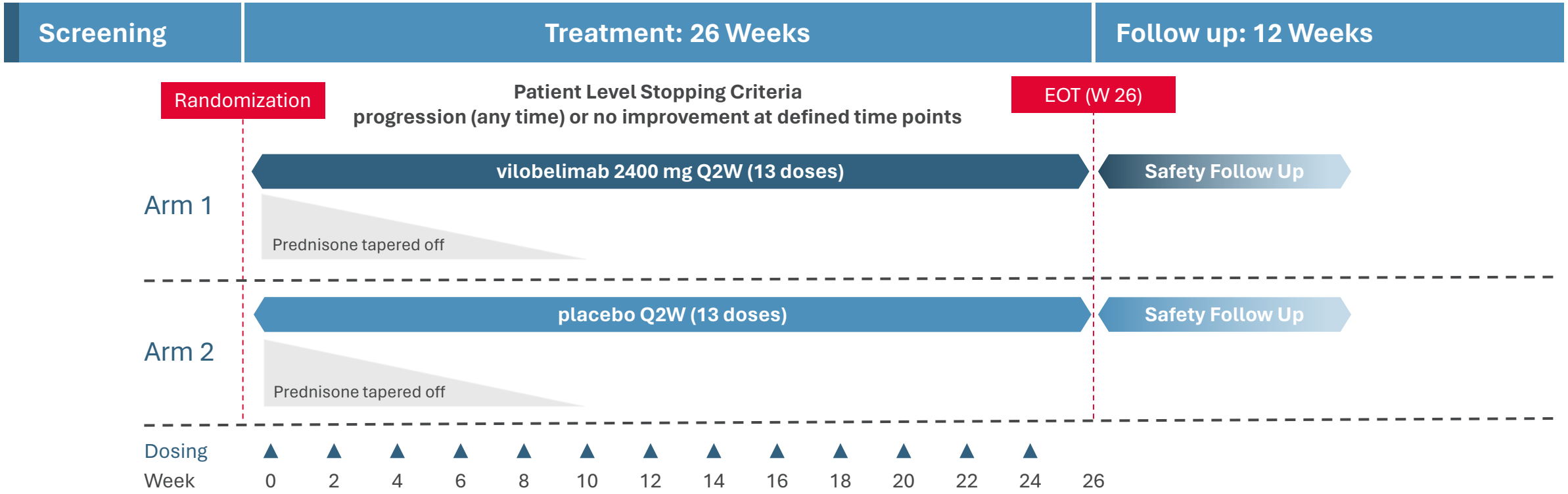
Target Ulcer Reappeared

- **MH:** PG since 2019, Hypertension since 1998
- **Previous PG medication:** Methylprednisolone only in Jun 2019, Dapsone Jun 2019 - Aug 2020, Cyclosporine Oct 2019 - Aug 2020 -> ulcer healed and reappeared after discontinuation of immunosuppressants
- **Cohort 2:** 1600 mg Q2W, individual up-titration to 2400 mg at D57, treatment completed
- **Concomitant medication:** Prednisone 10 mg for PG since October ‘20

Baseline	Day 99	Day 189
	PGA = 1	PGA = 1
Area: 3695 mm^2	Area: 0.00 mm^2	Area: 0.00 mm^2



PG Phase III Study Design: Interim Analysis Expected in 2025



Adaptive Design

Arm 1 n approx. = 15

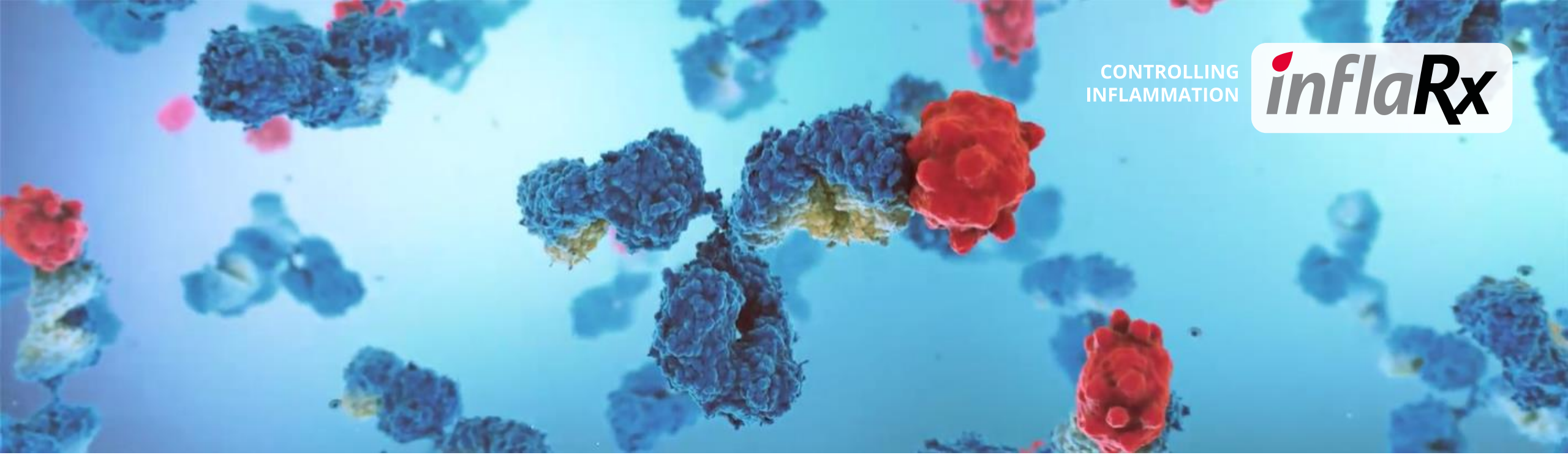
Arm 2 n approx. = 15

Interim Analysis*
set rules for size adjustment or futility stop by IDMC

Total number of patients to be adjusted between 50 – 100**

Primary endpoint: complete target ulcer closure

* Blinded except for independent data safety monitoring committee / **Adjustment of randomization ratio to 2:1 (Arm 1 to Arm 2) after blinded interim analysis



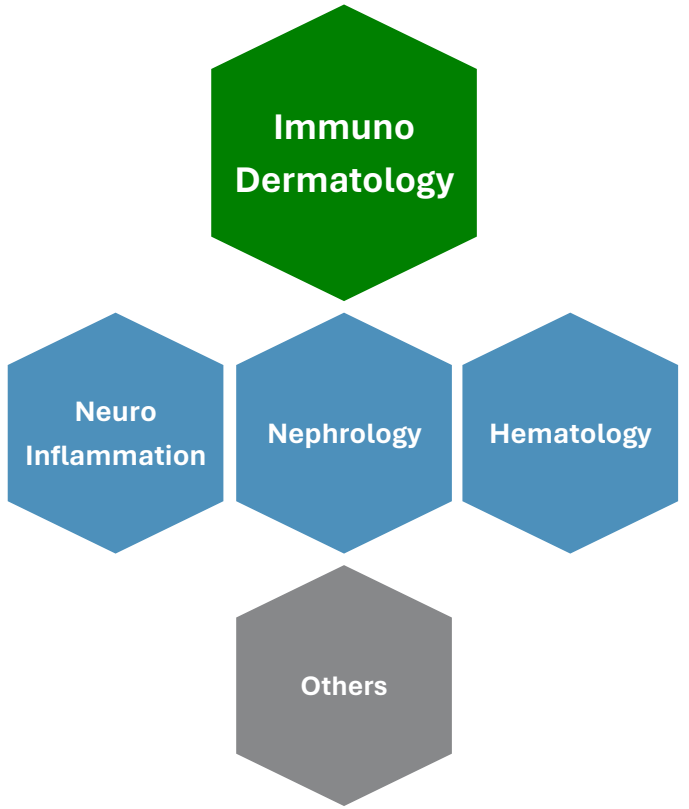
INF904: An Oral Highly Selective C5aR Inhibitor With Best-in-Class Potential

INF904: Oral C5aR Antagonist With Best-in-Class Potential

INF904 Key Features

- ✓ **Favorable drug profile** supported by preclinical studies and data reported from InflaRx's Phase I SAD and MAD trials
- ✓ Phase I PK/PD profile that **could open significant market opportunities** for the C5aR oral inhibitor class
  - well-tolerated and no safety signals over entire tested dose range (no reported SAEs, AE lower than in placebo group)
  - evidence of broad therapeutic index, BID and QD dosing
- ✓ Has **~3-fold higher C_{max}** and **~10-fold higher AUC_{last}** versus published avacopan data, for comparable doses (3, 10, 30 mg)
- ✓ **Significantly increased blocking activity** of C5a-induced neutrophil activation than avacopan's published data
  - Higher plasma exposures and >90% blocking of C5a activity
  - Achieves therapeutic exposures fast which may be needed to successfully treat chronic immuno-inflammatory diseases
- ✓ Potential for **broad range of dosing**
- ✓ Higher drug strength with **reduced capsule intake potential**
- ✓ **Much weaker inhibitor of CYP3A4/5** than avacopan
- ✓ **Strong IP position**, with US patent issued in October 2021

Focusing on Immuno-Derm, Other Options Possible



As a small molecule orally available C5aR inhibitor, INF904 has the potential to deliver…

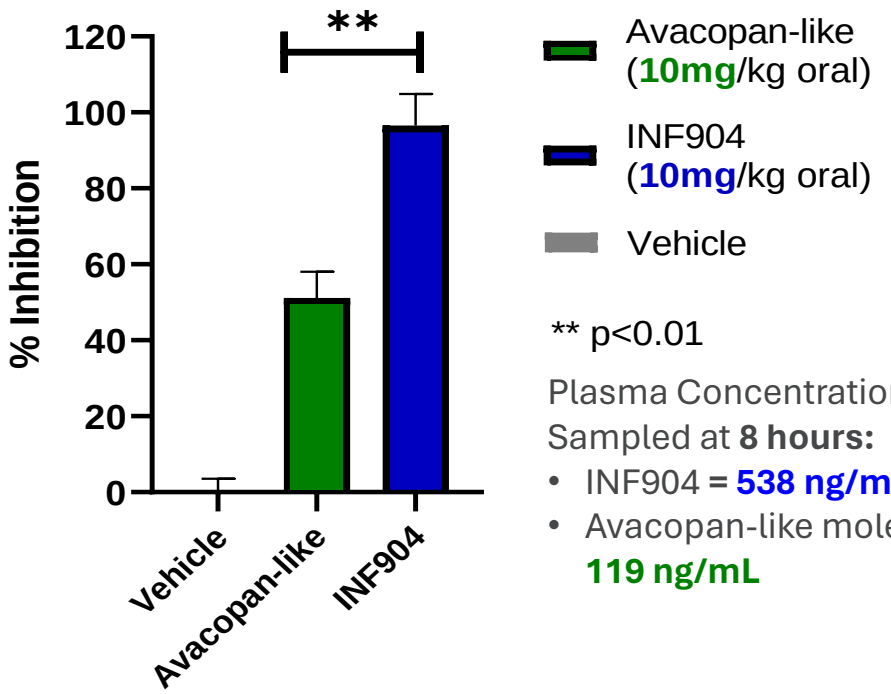
- Favorable tissue penetration and plasma concentrations
- Sustained long-term control over C5a/C5aR activation in a variety of chronic inflammatory diseases
- Convenient oral administration

…to multiple large commercial markets

INF904: Oral C5aR Antagonist With Best-in-Class Potential

INF904 Has Double the Inhibitory Effect in Vivo in a Pre-clinical Model Compared to Avacopan

Inhibition of in vivo neutrophil activation by INF904 compared to avacopan-like molecule*



INF904 doubled the in vivo inhibitory effect at comparable dose when tested head-to-head with avacopan.

The **strongly improved PK features of INF904** (plasma exposure) may drive the ability to increase efficacy in vivo.

Experiment: Challenge of rodents with C5a leads to neutrophil activation and consequent adherence (sticking) of neutrophils to the endothelial cell wall of vessels = mimicking a neutropenia (vehicle). This effect can be completely inhibited when C5aR activation is blocked.

Note: INF904 dosing within this experiment exerts an approximately 4.5-fold higher plasma level 8 h after dosing when compared to the identical dosing with avacopan*

Source: InflaRx data on file.
*Avacopan synthesized based on the published structure and publicly available data.

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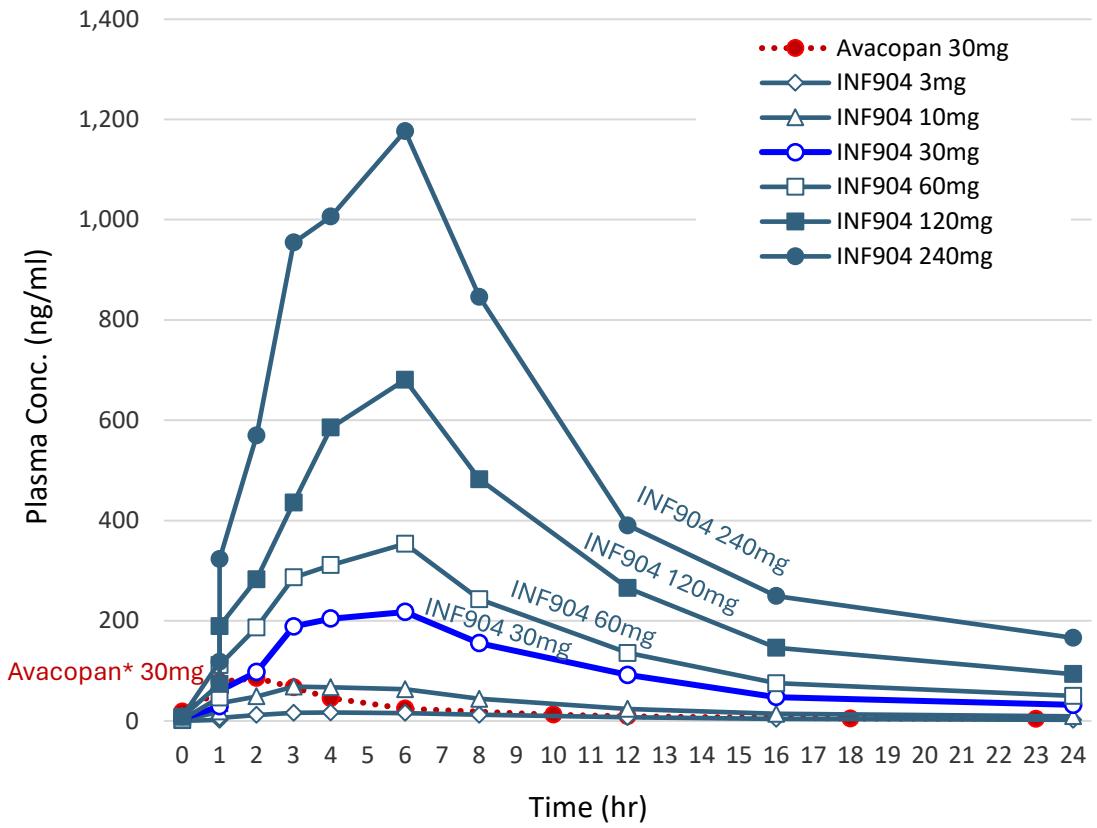
INF904: Oral C5aR Antagonist With Best-in-Class Potential

PK Results From Single Ascending Dose (SAD) Phase 1



Parameter	Unit	*Dose*	INF904	Avacopan*
AUC_{inf}	h.ng/ml	*3 mg*	285	25
		10 mg	1264	130
		30 mg	5956	628
AUC_{last}	h.ng/ml	*3 mg*	254	23
		10 mg	1117	122
		30 mg	5197	557
C_{max}	ng/ml	*3 mg*	21.5	9
		10 mg	74.8	25
		30 mg	289	79
t_{max}	hr	*3 mg*	3.5	1.2
		10 mg	4	1.7
		30 mg	5.01	1.7

› In comparison to published data for avacopan INF904 is approximately 3-fold higher in C_{max} and 10-fold higher in systemic exposure (AUC_{last}) for comparable doses (3, 10, 30 mg)

Source: Bekker et al. 2016, PLoS One; 11(10): e0164646

***Please note:** Avacopan data taken from Bekker et al. 2016, PLoS One; 11(10): e0164646 are superimposed in graph for orientation. Avacopan was not included as a comparator in INF904 Phase I study.

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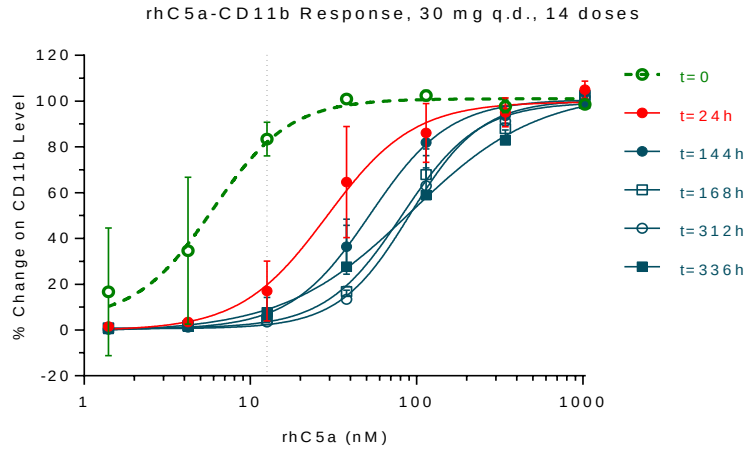
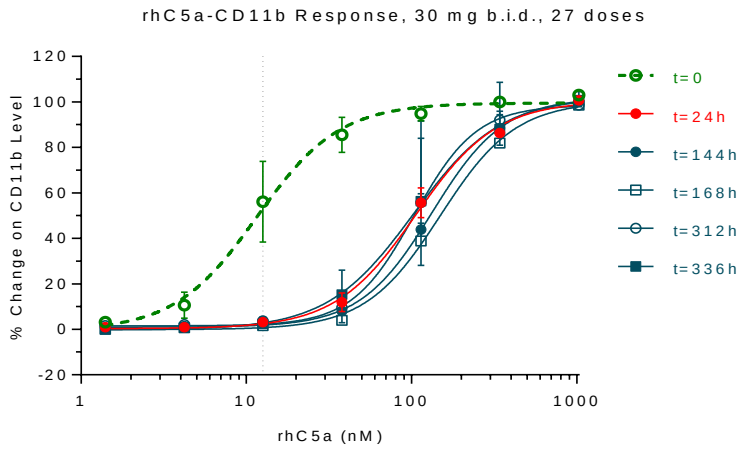
INF904: Oral C5aR Antagonist With Best-in-Class Potential

C5a-Mediated CD11b Upregulation on Neutrophils Ex Vivo up to 14-day Dosing

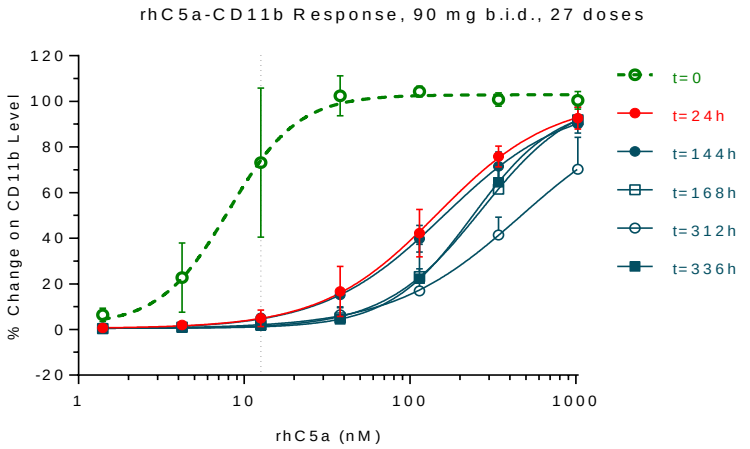
INF904: 30 MG QD



INF904: 30 MG BID



INF904: 90 MG BID



	Upon stimulation with 12.6 nM rhC5a (levels observed in disease state)														
	24 h			144 h (Day 6)			168 h (Day 7)			312 h (Day 13)			336 h (Day 14)		
	30QD	30BID	90BID	30QD	30BID	90BID	30QD	30BID	90BID	30QD	30BID	90BID	30QD	30BID	90BID
Blockade (%)	80	94	90	93	95	94	95	97	97	96	92	97	90	95	97
EC_{50} (nM)	35.6	106.2	145.6	52.4	134.7	160	74.2	149.0	268.2	92.4	126.3	465.7	94.6	110.9	238

PD MAD results confirm strong >90% C5a inhibition at C5a levels found in human diseases – this is clearly differentiated from reported avacopan results which have shown approximately 50% inhibition at a lower challenge of 10nM C5a (7 day dosing – trough)**

*EC_{50} (nM) is the half maximal effective C5a concentration ** Bekker et al. 2016, PLoSOne; 11(10): e0164646

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Investing Into INF904 Development

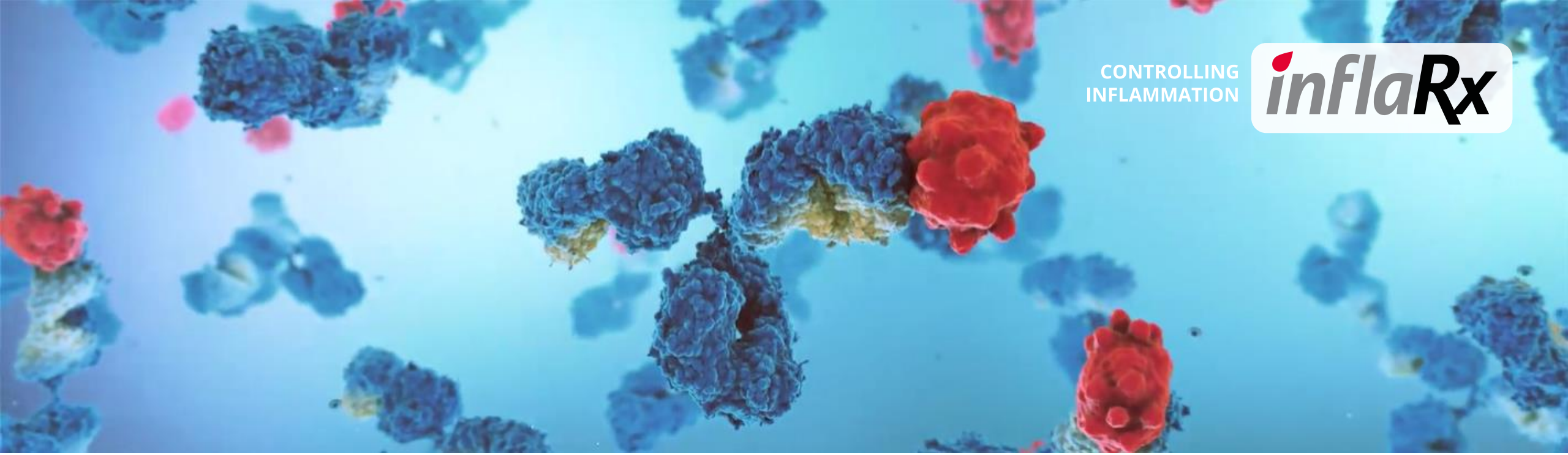
Phase IIa Expected to Begin by EOY 2024

Initial Phase IIa – demonstrating pipeline-in-a-drug potential of INF904

- Open-label PK / PD “basket study” to explore initial efficacy signals
- 4-week treatment period in 2 immuno-derm indications CSU and HS with established endpoints
- Safety and PK / PD assessment planned for at least 3 different doses

Expected catalysts

- Phase IIa expected to begin by EOY 2024
- Phase IIa data anticipated in 2025
- Larger and longer-term Phase IIb study expected to begin in 2025



INF904 for Chronic Spontaneous Urticaria (CSU)

A Strong Rationale for Developing INF904 in CSU

C5aR Signaling is Involved in Histamine Release in an IgE Independent Manner

- Increasing scientific evidence suggests that **C5aR signaling is involved in histamine release** from mast cells and basophils in CSU in **an IgE independent manner**. This mechanism may play an important role for both described endotypes in CSU:
  - Type I (IgE mediated) and
  - Type IIb (IgG autoantibody mediated)

- Despite availability of current treatment options such as anti-histamines and anti-IgE therapy, **approximately 30-60%* of these patients are estimated to remain non-responsive or symptomatic**.

- INF904 could be a **convenient oral therapeutic option** for those underserved with current therapies.

- CSU market potential is **estimated to exceed $3 Bn by 2032****

* Metz et al, Clin Rev Allergy Immunol. 2020; 59(1): 38–45. ** GlobalData and Leerink analyst report

Chronic Spontaneous Urticaria (CSU)

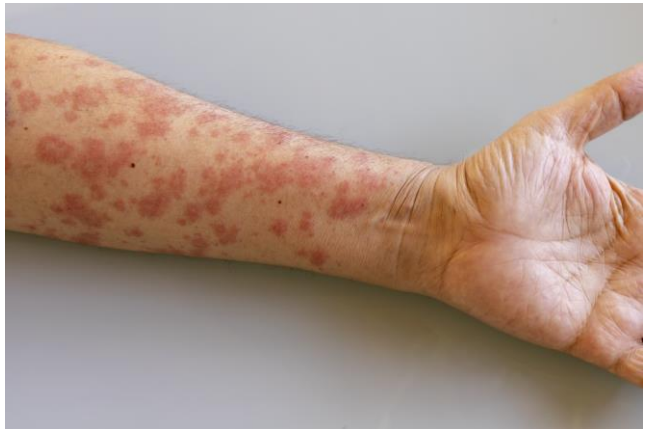
CSU Overview and Unmet Need

Clinical features

- An immune-mediated chronic inflammatory skin disorder, with dysregulated inflammatory cascades that leave patients predisposed to symptom development: debilitating and intensely itchy hives / wheals for > 6 weeks and often associated with angioedema
- Burden of disease is high and impacts sleep, mental health, QoL and productivity due to absences from school and work
- Co-morbidities include atopic disorders, depression, autoimmune and thyroid disorders

Epidemiology

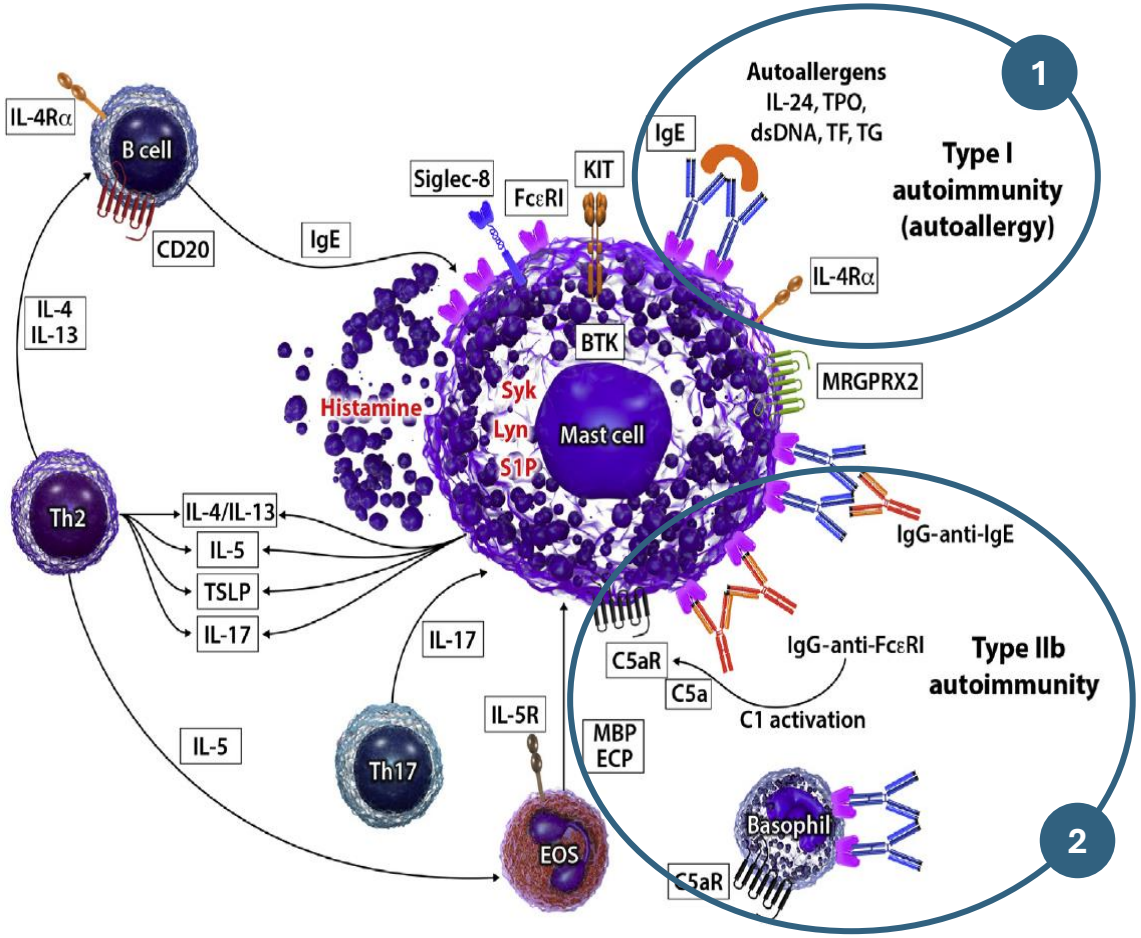
- Estimated prevalence is around 1% of the general population
- 20% of this population experiences symptoms for more than 5 years
- 20 to 40 year-olds are most affected, with women impacted 2x more than men



Current treatment and medical need

- Therapies such as 2nd-generation antihistamines are not effective in a significant number of patients
- Options such as anti Ig-E therapy and immunosuppressants also do not adequately serve the CSU population

CSU Endotypes - Type 1 Auto-Allergens and Type IIb Autoimmunity



Two major endotypes of CSU described as activation of mast cells include:

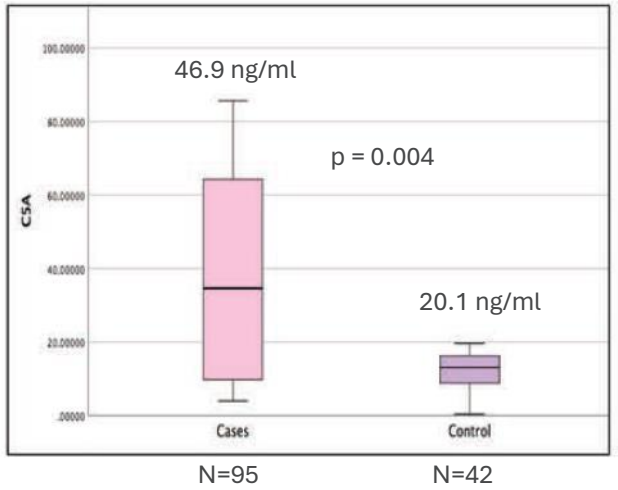
1. Type I autoallergens (IgE mediated)
2. Type IIb autoimmunity (IgG mediated; ~30% of CSU)
   - C5a is activated by the binding of IgG-anti-FcεRI or IgG-anti-IgE to FcεRI on mast cells and basophils

C5aR signaling is suggested to be involved in both, type I and type IIb endotypes

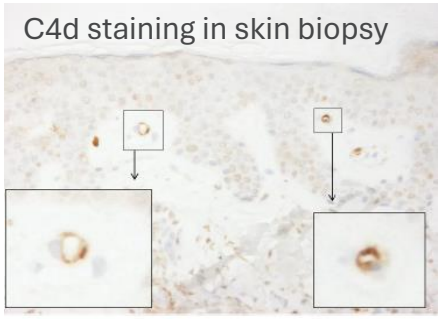
Maurer et al. Clinical reviews in allergy and clinical immunology. 2022.

C5a in CSU and its Role in IgE-Independent Histamine Release

CSU patients have elevated C5a levels

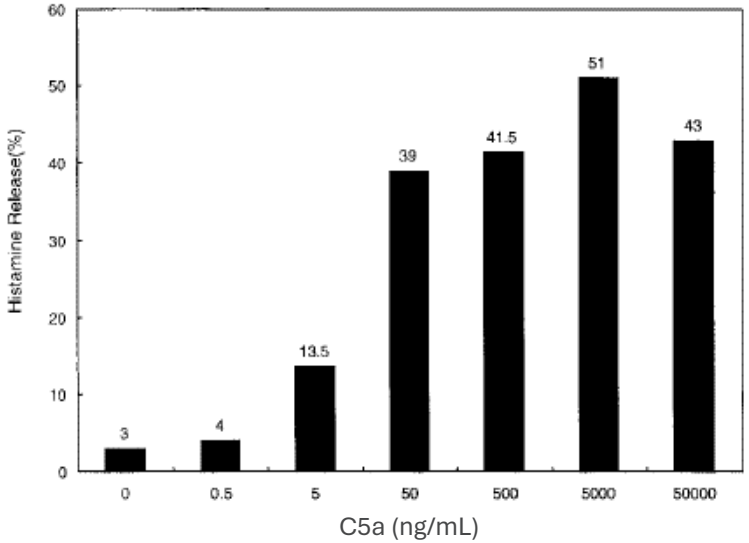


CSU patients show evidence of complement activation in the skin



Bhatia et al. 2024 Asia Pacific Allergy 14 ;
Aghdam et al. 2021 Clin Transl Allergy. 11

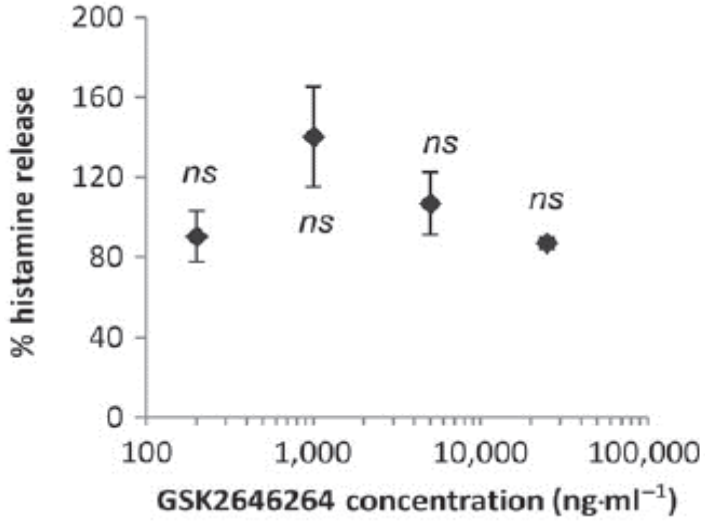
C5a induces histamine release from basophils in a dose-dependent manner



Histamine release (percentage) from donor basophils stimulated with increasing levels of C5a

Kikuchi, 2002 J Allergy Clin Immunol:109

C5a mediated histamine release is independent of the IgE pathway



Human Skin ex vivo Model: microdialysis tubing into the ex vivo human skin with 1nM C5a

C5a stimulation of histamine releases is not affected by IgE pathway / SYK inhibitor GSK2646264

Molina et al; 2019 Br J Pharmacol: 176

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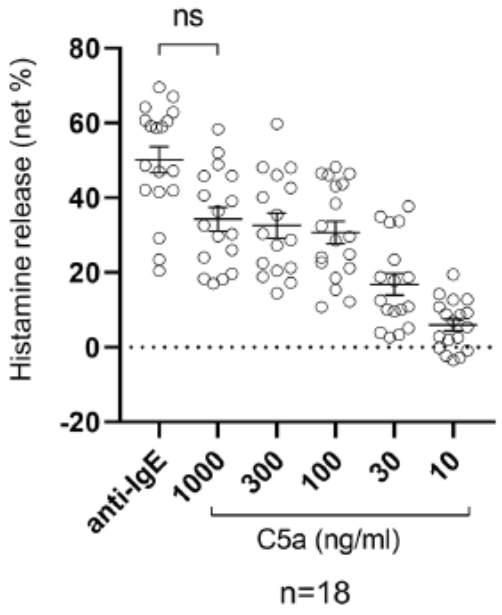
C5a/C5aR Contributes to Histamine Release in an IgE Independent Manner

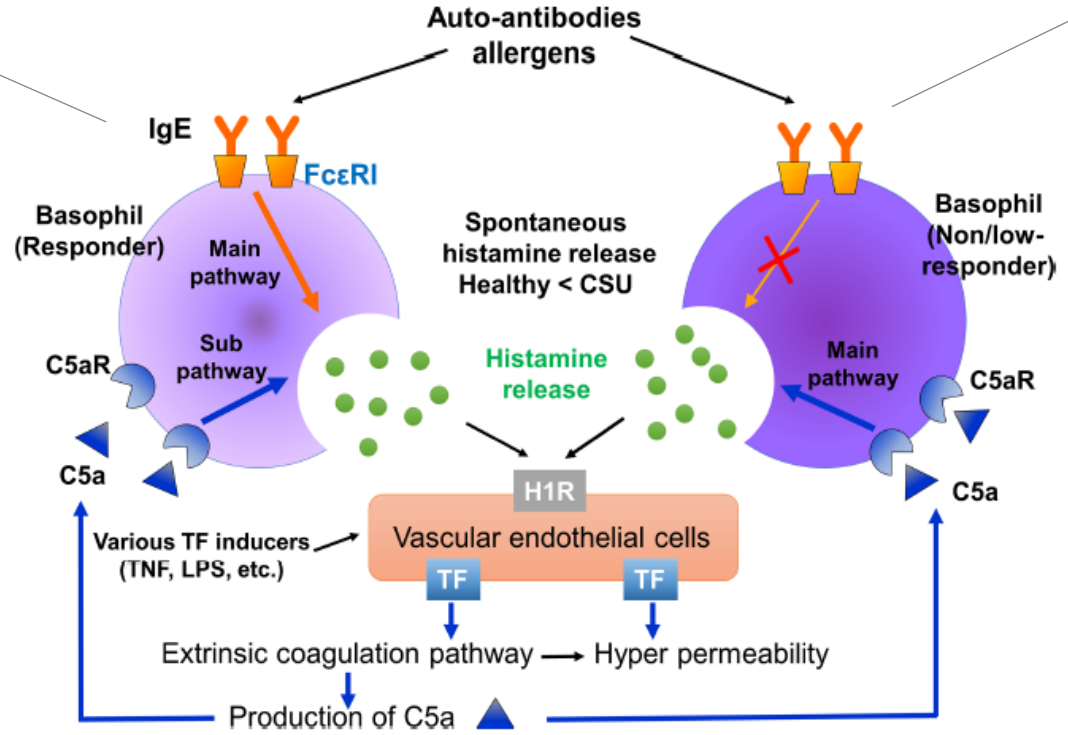
C5a-Induced Histamine Release is Important for Both, IgE Dependent Pathway and IgE Independent Pathway

Anti-IgE Stimulus pos. (>20% HR)



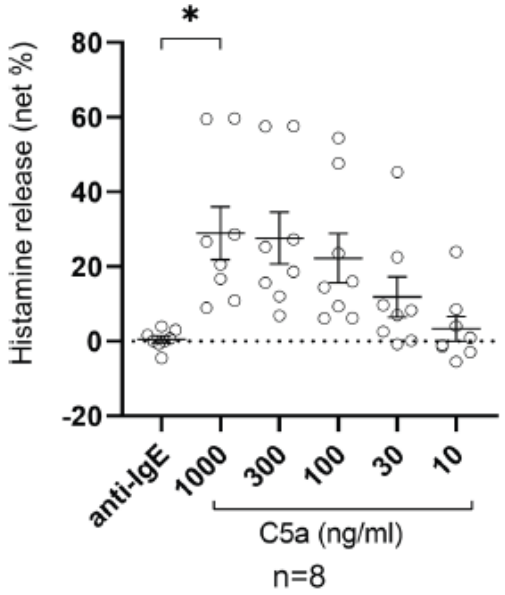
Basophils isolated from CSU patients

C5a stimulation leads to dose-dependent histamine release



C5a generation in CSU may be amplified by the activation of the coagulation pathways (tissue factor [TF] release) in addition to the auto-antibody trigger

Anti-IgE Stimulus neg.



Basophils isolated from CSU patients

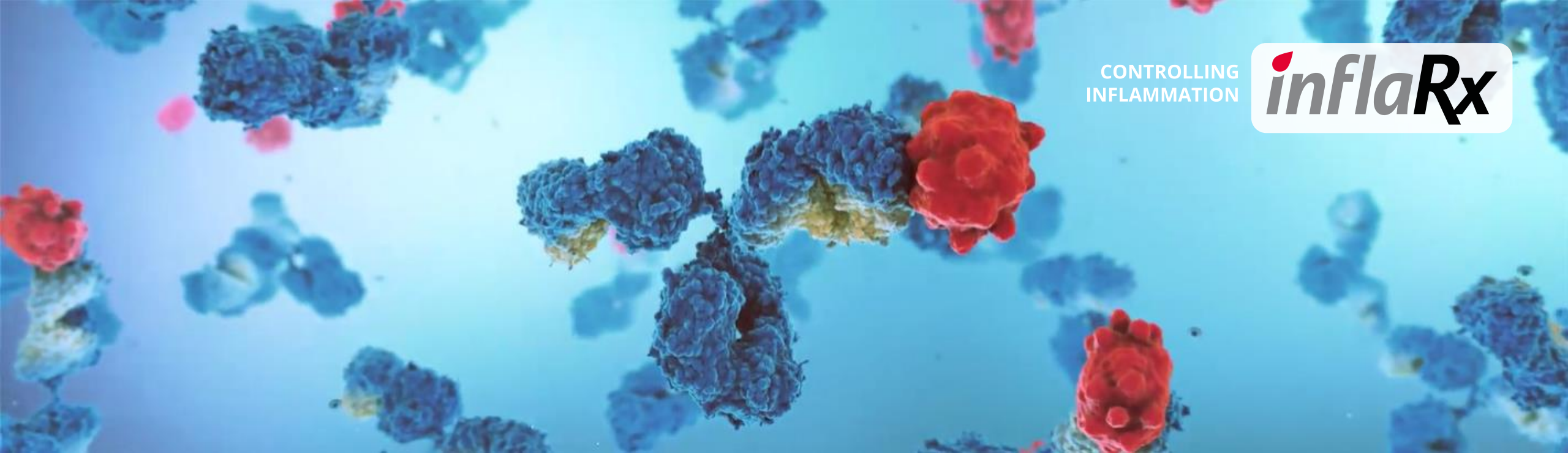
C5a stimulation leads to dose-dependent histamine release

Matsubara et al. Front Immunol 2022, 13.

INF904 Development in CSU

Conclusion:

- C5aR signaling is involved in histamine release from mast cells / basophils in CSU
- This C5a-mediated histamine release is independent of the IgE pathway and has been suggested to play a role in both subtypes of CSU
- C5aR inhibition represents a novel mechanism of action (MoA) to address an unmet medical need in CSU
- **INF904 as an oral potent C5aR inhibitor is ideally positioned for development in CSU**



INF904 for Hidradenitis Suppurativa (HS)

A Strong Rationale for Developing INF904 in HS

New Mechanisms are Needed

- **New mechanisms are needed** to address the disease more completely
  - **E.g. moderate to severe patients with active draining disease** currently have limited approved treatment options which have proven to be effective for them
  - and response to treatment with approved **anti-TNF-alpha or anti-IL17 agents is known to wane over time** in a significant number of cases
- HS patients have a preference for oral medications over injections (and surgical incisions)*
- INF904 is an **oral C5aR inhibitor** with:
  - A mechanism of action which inhibits the known C5a induced effects on neutrophil activation and tissue accumulation of immune cells including induction of NETosis – mechanisms which have been suggested to be involved in HS progression and specifically in HS lesion formation
  - Clinical evidence existing that blocking the C5a/C5aR pathway reduces lesion counts in HS
  - A favorable PK/PD profile with a broad dose range for systemic exposure in patients
- **HS market potential is attractive**, with a market size estimated with >$3.9 Bn by 2032**

* Willems, D., Hinzpeter, EL., Van der Zee, H.H. et al. Patient 16, 153–164 (2023)
** GlobalData and Leerink / Guggenheim analyst reports

Hidradenitis Suppurativa (HS)

HS Overview and Unmet Need

Clinical features

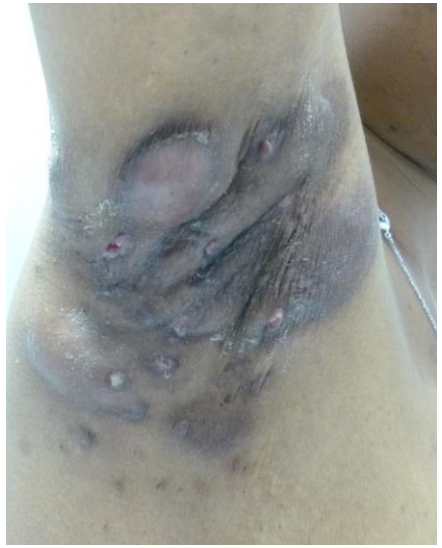
- A chronic, recurring, debilitating neutrophil-driven inflammatory disease, that can persist for years
- Characterized by abscesses, nodules and draining tunnels (dTs) with purulent or bloodstained discharge, that can flare and cause scarring
- Predilection for intertriginous sites such as axillae, groin, buttocks and inframammary areas
- Associated with severe bacterial infections, tremendous QoL impairment and functional disability

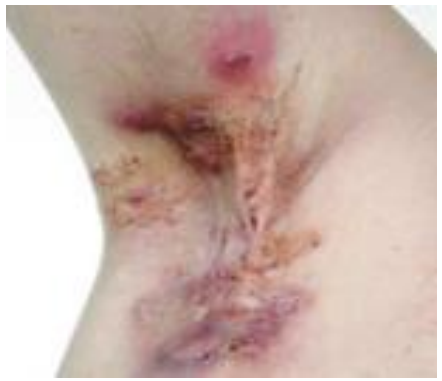
Epidemiology

- Prevalence in the US and EU is estimated to be 0.7% - 1.2%
- Though estimates vary widely, we estimate there are clearly more than 200,000 moderate to severe HS patients in the US alone

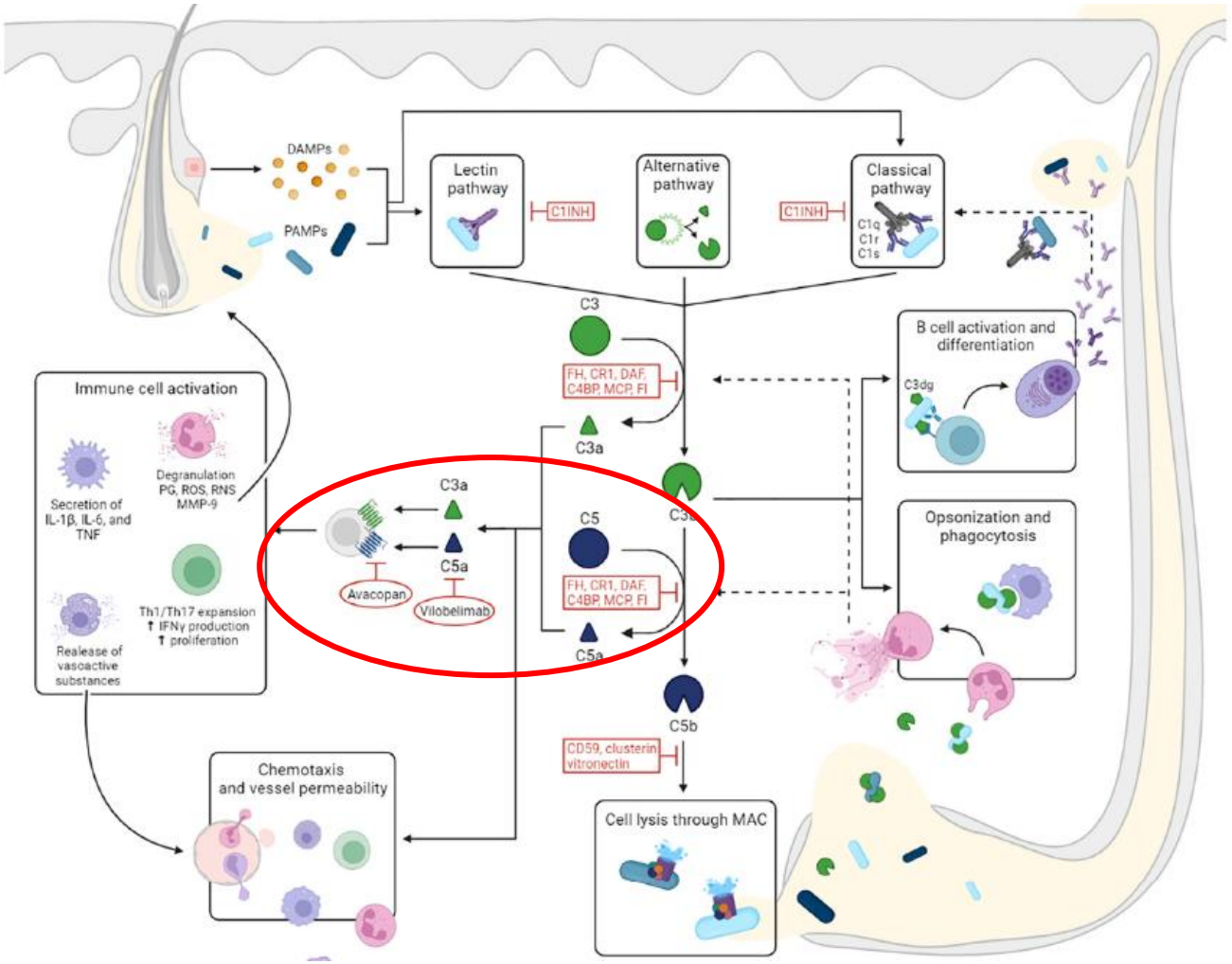
Current treatment and medical need

- Current treatments including pain management, antibiotics, corticosteroids and biologics
- Current approved therapies have shown a waning of effect in a significant number of patients over time
- In addition, high-unmet medical need exists in affected patients with active draining disease





An Important Role for C5a/C5aR is Recognized in HS Pathogenesis



van Straalen KR Front. Immunol. 13:953674.doi: 10.3389/fimmu.2022.953674

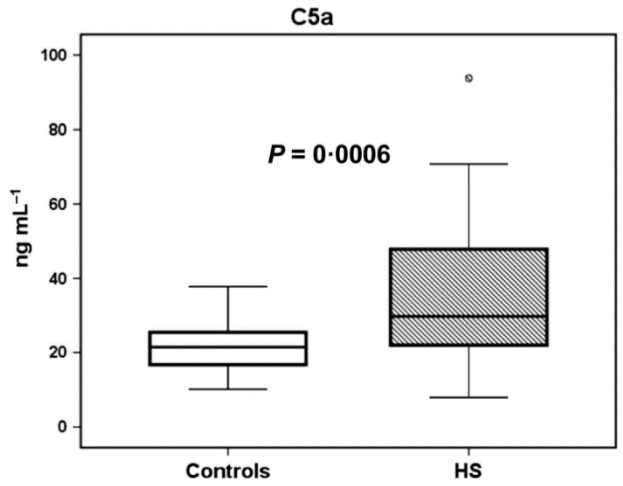
Mechanism in HS development:

Follicular occlusion of the folliculo-pilosebaceous unit, followed by follicular rupture, leading to immune responses which involve complement activation including C5a/C5aR engagement, resulting in the development of clinical HS lesion

Strong Rationale for Developing an Anti-C5a/C5aR in HS

HS Patients Have Elevated C5a, a Major Neutrophil Activator That Can Be Blocked by an Anti-C5a/C5aR

HS patients have **significant complement activation** with elevated C5a levels



Concentrations of C5a in the plasma of 14 healthy controls and of 54 patients with HS. P-values symbolize significant differences between patients and controls.

Kanni et al, 2018

C5a/C5aR activation is a key neutrophil activator in HS patient plasma

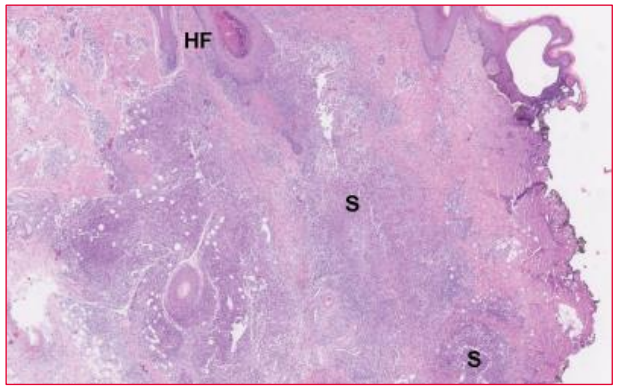
HS patient plasma strongly provokes neutrophil activation in healthy donor blood: this effect could be completely blocked by the addition of:

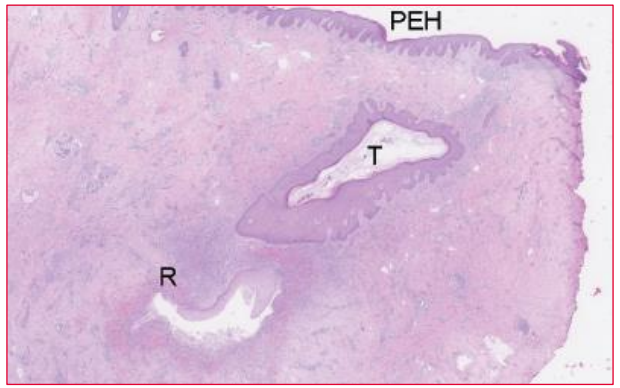
- Vilobelimab (anti-C5a antibody) and
- INF904 (anti-C5aR inhibitor)

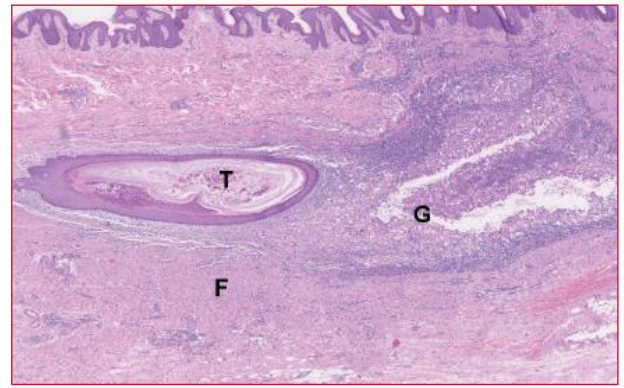
Guo et al. 2019 Aug. US Patent No. 10,376,595
Source: InflaRx in house data on file

Enhanced C5aR Staining in Biopsies From HS Patients at All Disease Stages

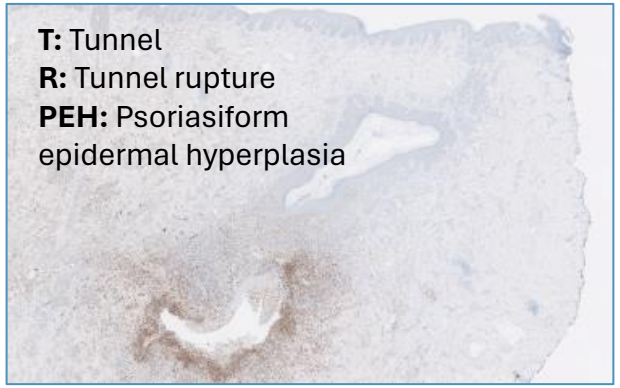
H&E Staining

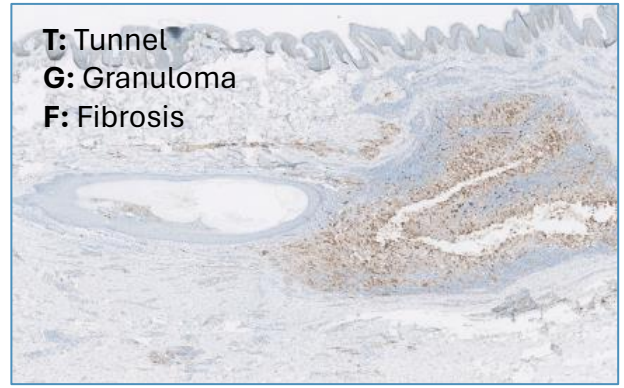






C5aR1 Staining





Hurley stage I patient with extensive deep dermal and subcutaneous **suppurative** abscessing inflammation, surrounding hair follicles with hyperkeratosis. **C5aR1 staining positive – neutrophils**

Hurley stage II patient with tunnel formation and **tunnel rupture area** with epithelium surrounded by sheets of neutrophils. **C5aR1 staining positive – neutrophils**

Hurley stage III patient with tunnel formation and surrounding **granulomatous** inflammation with foreign body giant cells. **C5aR1 staining positive – neutrophils, histiocytes and giant cells**

Van Straalen et al. 2022. Front Immunol 21.

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Clinical Evidence for the Role of C5a/C5aR Signaling in HS

Vilobelimab (anti-C5a mAb)

- SHINE Phase IIb study in moderate-severe HS patients resulted in various signals of efficacy for high dose treatment group (1200mg EOW) including an overall inflammatory lesion reduction *
- In SHINE, dT reduction was higher in patients with tightly controlled C5a levels *
- Key learning from SHINE: **a higher dose of vilobelimab was needed to adequately control C5a/C5aR signaling and increase efficacy in lesion reduction**

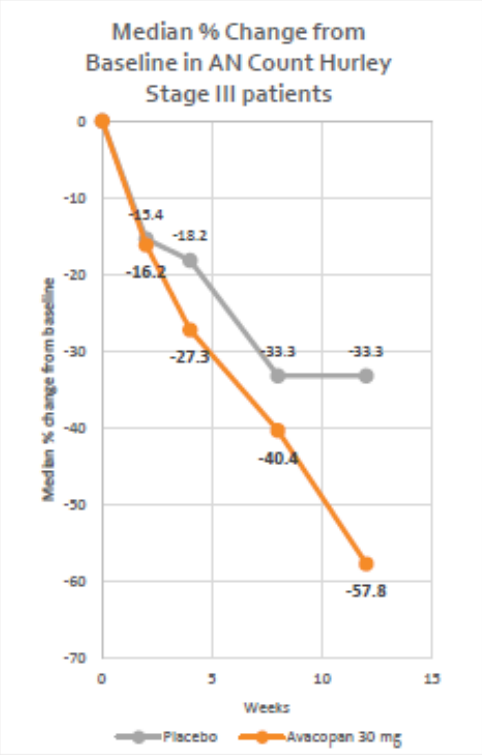
Avacopan (oral C5aR inhibitor)

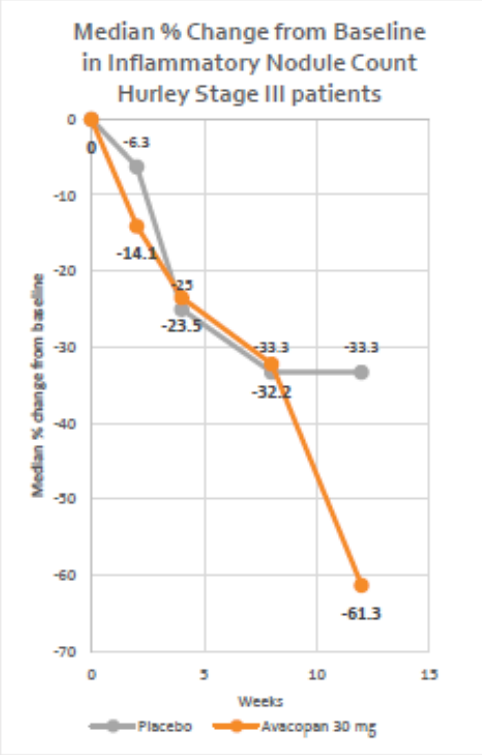
- At standard dose of 30 mg BID, **a p-value positive efficacy signal was detected in severe HS patients (Hurley III) on HiSCR** with clear separation from placebo group emerging at week 12 **
- Of note: in ANCA vasculitis patients steady-state levels of avacopan at 30 mg BID were only reported to be achieved at approx. 3 mths ***
- **30 mg BID dosing regimen may have been too low for adequate HS treatment and late accumulation of avacopan may have prevented earlier onset of efficacy**

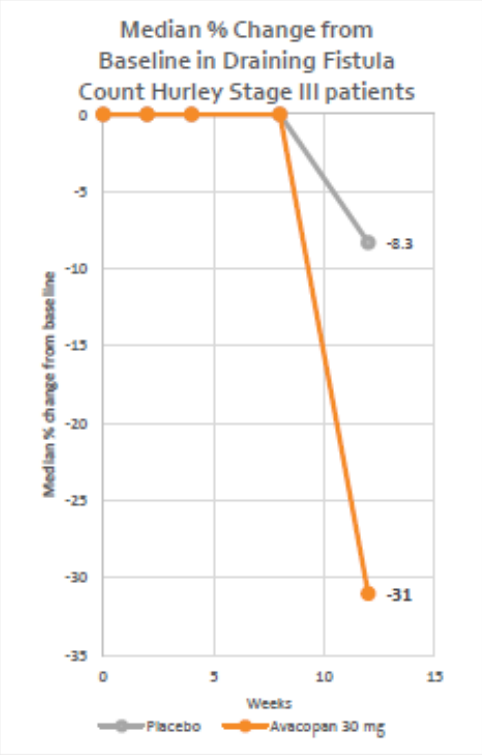
* InflaRx data on file
** Data from Chemocentryx presentation on AURORA trial results, October 28, 2020: note: overall results were not stat. significant for HiSCR in all moderate to severe HS patients (primary endpoint)
*** Data from avacopan NDA filing for ANCA-associated vasculitis

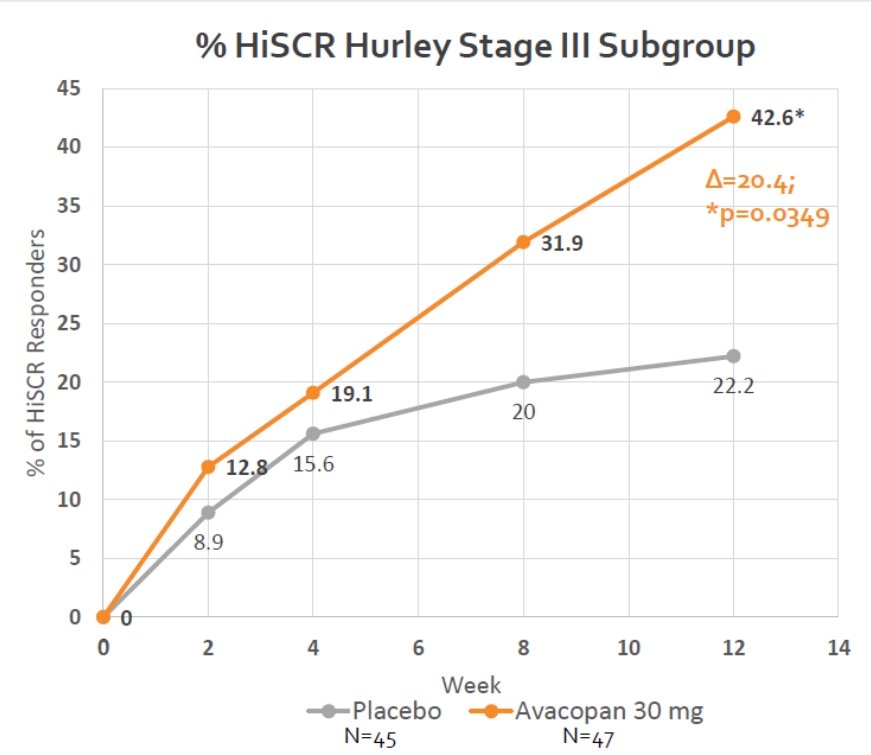
Avacopan Data in Phase II Clinical Trial Shows Efficacy Emerging Only at W12

Results From Avacopan in HS Patients (Hurley III)*









- Avacopan's efficacy (separation from placebo group) in HS only starts to emerge at W12 – please note: steady state reported from ANCA patients was only reached at approx. 3 months due to prolonged drug accumulation (x4)
- Avacopan's 30mg BID dosing regimen may be too low to show adequate clinical efficacy in HS

*Data from Chemocentryx presentation on AURORA trial results, October 28, 2020: note: overall results were not stat. significant for HiSCR in all moderate to severe HS patients (primary endpoint)

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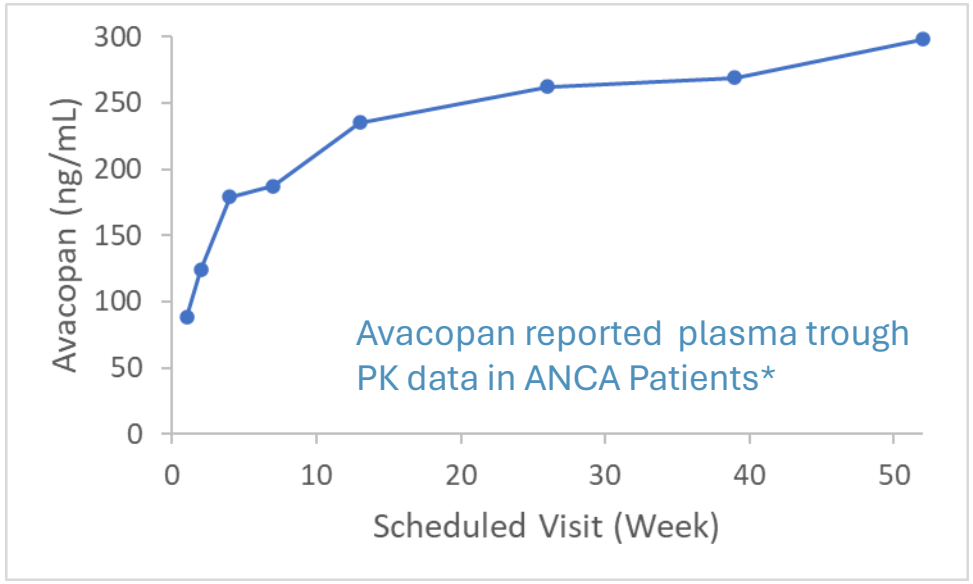
Avacopan Data in ANCA Patients Shows Steady State Reached by 13 Weeks



*Data from avacopan NDA filing for ANCA-associated vasculitis: represented graphically.

- Steady state plasma levels of avacopan 30mg BID are reached by 13 weeks and the accumulation is approximately 4-fold
- Mean steady state plasma exposure estimates of avacopan are: **3466 h*ng/mL for the ($AUC_{0\text{-}12hr}$)** in ANCA patients receiving 30 mg BID

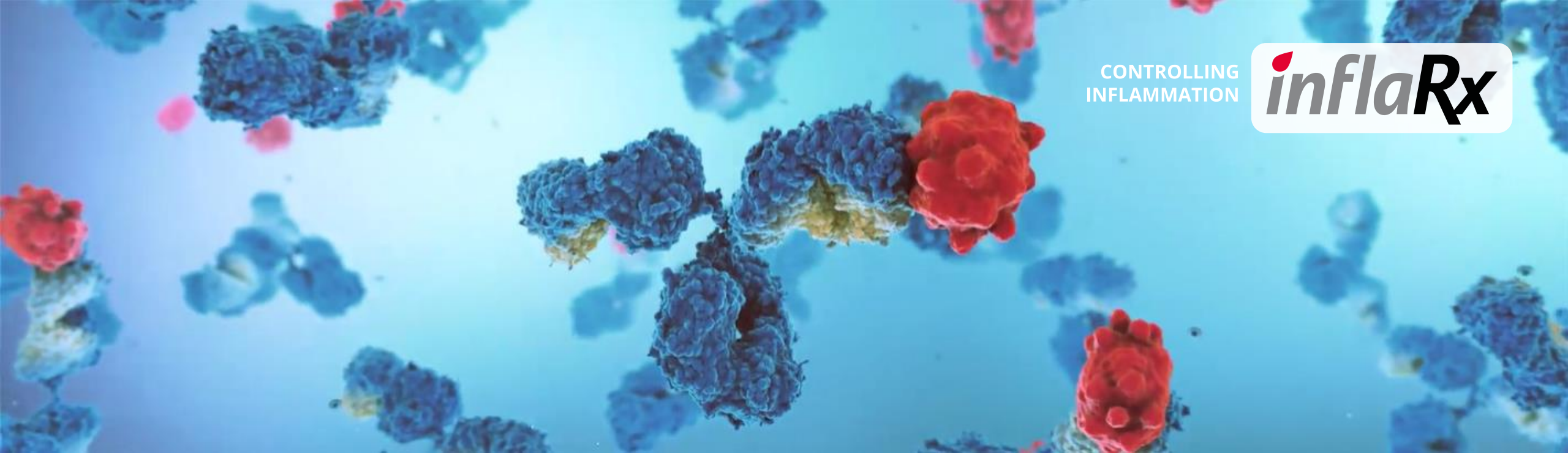
Plasma accumulation may be a prerequisite for reaching blocking activity of C5aR1 on neutrophils, to sufficiently prevent activation and migration into tissue in order to show clinical efficacy

Vilobelimab* and Avacopan** Provide Evidence for Efficacy in HS Patients

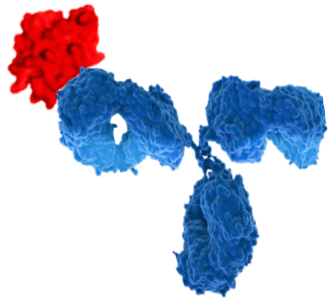
Conclusion:

- There is a strong scientific rationale for the role of C5aR in HS
- C5a/C5aR signaling inhibition has resulted in signals of efficacy in HS patients
- Tight control over C5aR signaling is required to achieve optimal efficacy – dosing is important in HS!
- **INF904 is ideally positioned as an oral C5aR inhibitor with optimized PK / PD profile to address an existing high unmet medical need in HS patients**

* Source: InflaRx data on file. ** Source: Data from Chemocentryx presentation on AURORA trial results, October 28, 2020



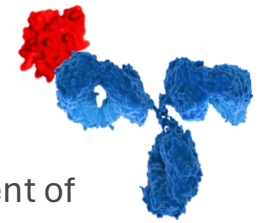
Gohibic (vilobelimab)
Critical COVID-19 & ARDS



Gohibic

Emergency Use Authorization (EUA) Granted for Gohibic

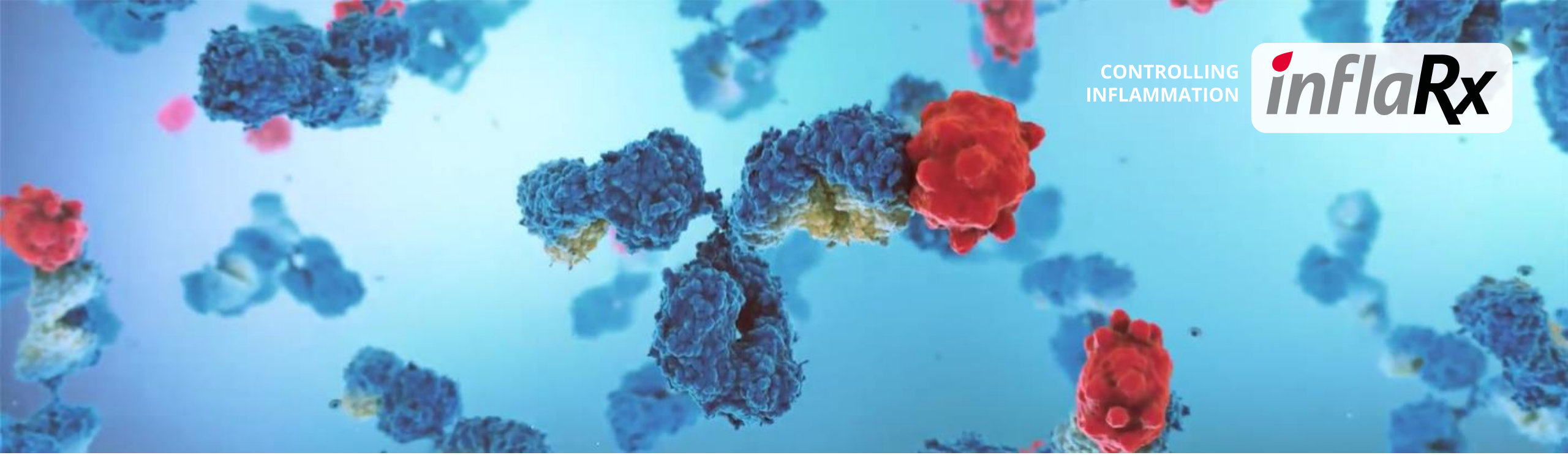
Gohibic (vilobelimab)



- Gohibic (vilobelimab) has not been approved, but has been authorized for emergency use by FDA under an EUA*, for the treatment of COVID-19 in hospitalized adults when initiated within 48 hours of receiving IMV**, or ECMO**
- Authorization granted based on results from a Phase III clinical trial in critically ill, mechanically ventilated COVID-19 patients in which Gohibic treatment reduced mortality by 23.9% vs. placebo.
- Gohibic is the first authorized therapeutic targeting C5a as potential key player in the inflammatory host response
- MAA under review at EMA / CHMP in Europe, discussions with US FDA ongoing related to future BLA submission
- Gohibic has been launched by InflaRx in the US under the EUA:
  - Building an experienced and highly focused commercial team and creating awareness with different healthcare players
  - Building a robust supply chain to allow for uninterrupted supply of Gohibic to US hospitals

For additional and important safety information, please visit www.gohibic.com

* The emergency use of GOHIBIC is only authorized for the duration of the declaration that circumstances exist justifying the authorization of the emergency use of drugs and biological products during the COVID-19 pandemic under Section 564(b)(1) of the Act, 21 U.S.C. § 360bbb-3(b)(1), unless the declaration is terminated or authorization revoked sooner
** IMV = invasive mechanical ventilation, ***ECMO = extracorporal membrane oxygenation



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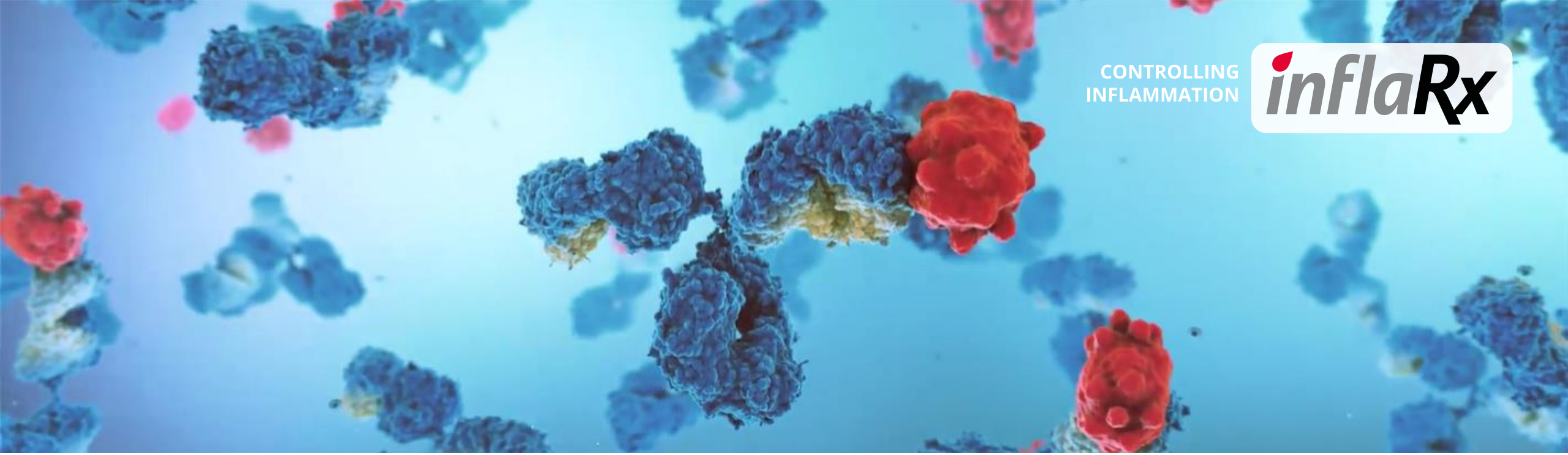


INF904 Appendix

www.inflarx.com

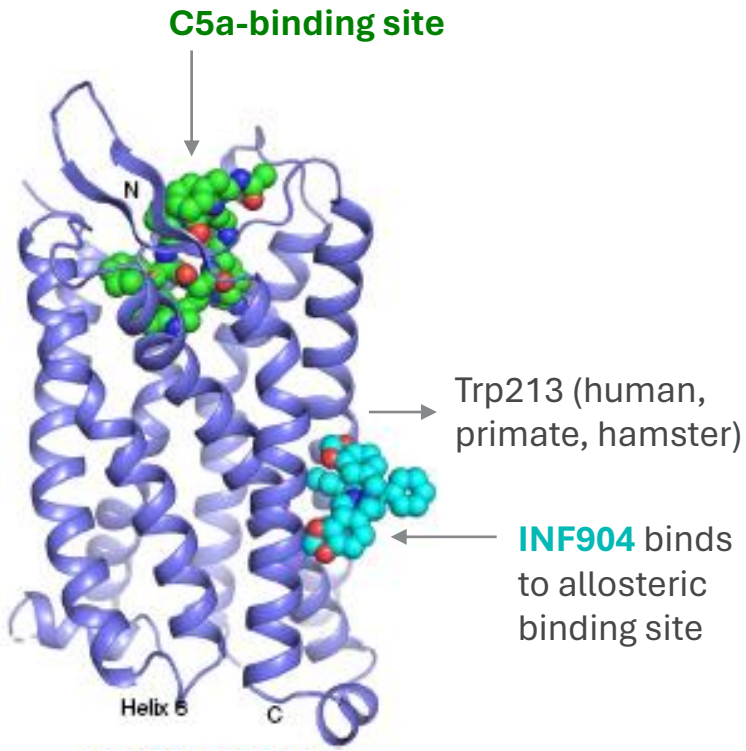
INF904: Pre-Clinical Summary

INF904 Facts

- INF904 binds to the well-defined allosteric site in C5aR transmembrane domain
- INF904 has a novel and proprietary molecular structure
- US patent issued in October 2021; pending in other countries (PCT)

Pre-clinical Findings

- **No toxicity findings**
  even in the highest dose groups (rat and monkey; up to 300 mg/kg)
- **High in vitro potency** with a desired IC50 (<1nM) in calcium mobilization assay
- **Higher plasma exposures** in several in vivo models when compared to avacopan
- **Increased efficacy**
  in hamster neutropenia model when tested at equivalent dose to avacopan
- **Therapeutic effects** in pre-clinical disease models (renal/peritonitis)

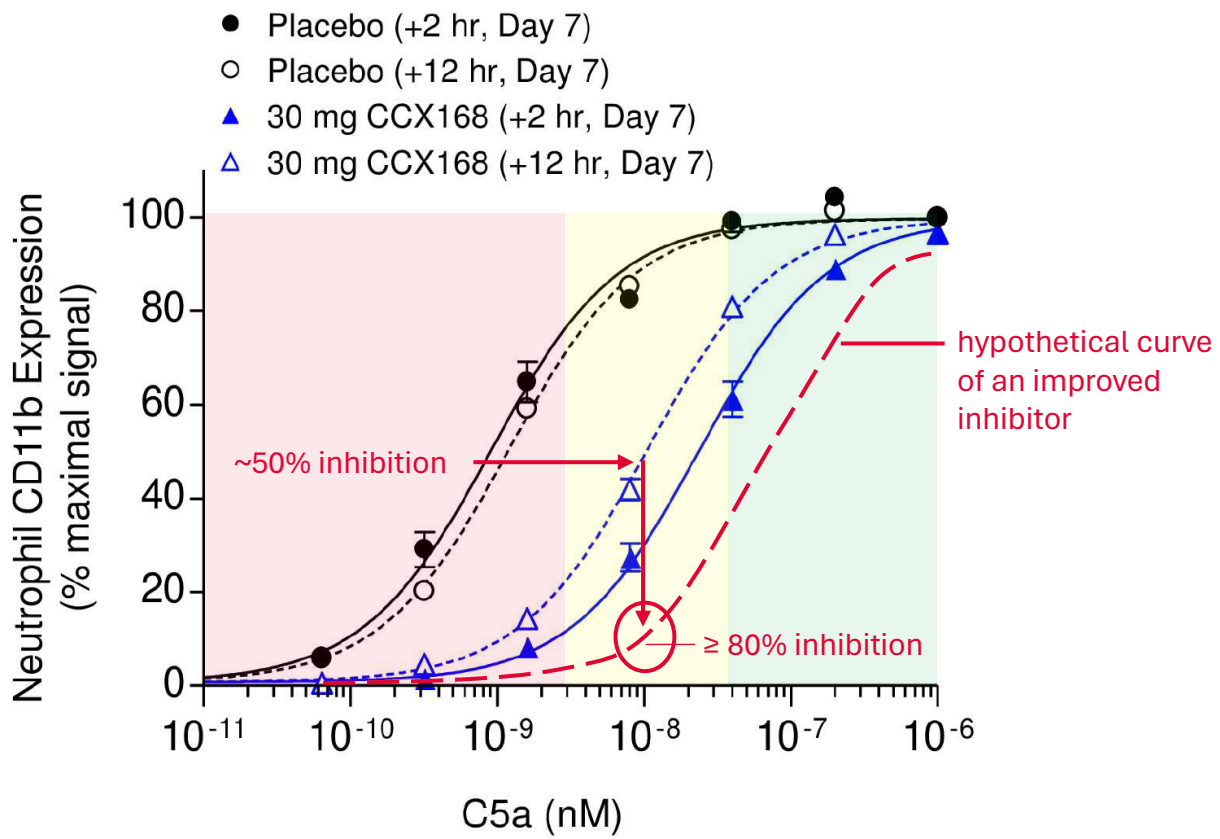


C5aR structure
(Source: Nathan Robertson, 2018 Nature)

Targeted Improvement of C5aR-Inhibition

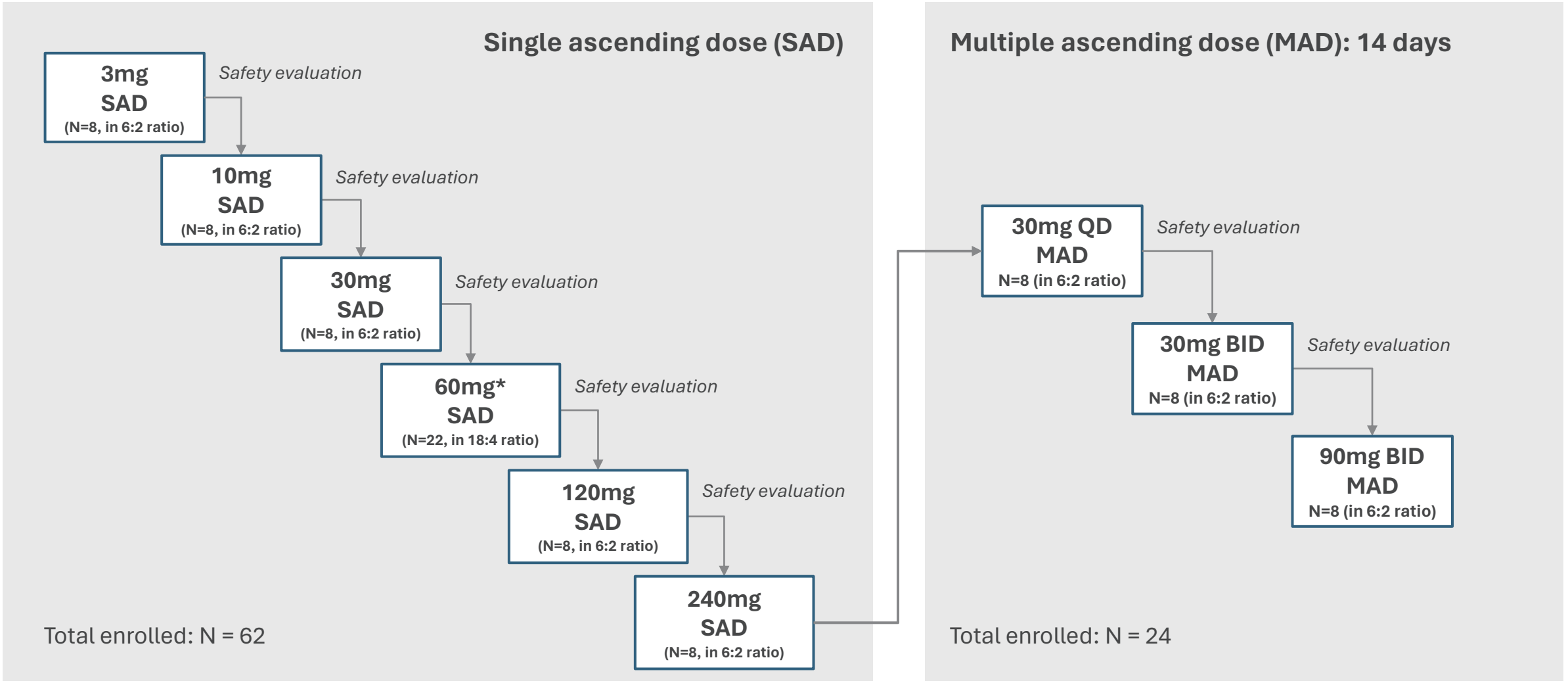
Properties of a Best-in-Class C5aR Antagonist

- **Improved PK properties** with higher plasma trough level (>> 36 ng/mL) to achieve:
- **Improved blocking activity** in vivo in humans (>> 50% blocking at 10 nM C5a)
  = **significantly stronger inhibition of neutrophil activation at C5a levels known to be present in diseases**
- **Improved drug strength** to allow fewer capsules per dosing and potentially less frequent dosing



modified from Bekker et al. (2016, PLoS One; 11(10): e0164646); CCX168 = Avacopan; Whole blood ex vivo assay upon 7 days of 2 x qd dosing with Avacopan measuring up-regulation of CD11b on blood neutrophils upon challenge with addition of different levels of recombinant C5a. CD11b is a marker of neutrophil activation known to rise quickly upon interaction of C5a with the C5a receptor. Measurement were taken at 2 hr or 12h upon last dosing (on day 7) and then ex-vivo challenge with different doses of C5a.

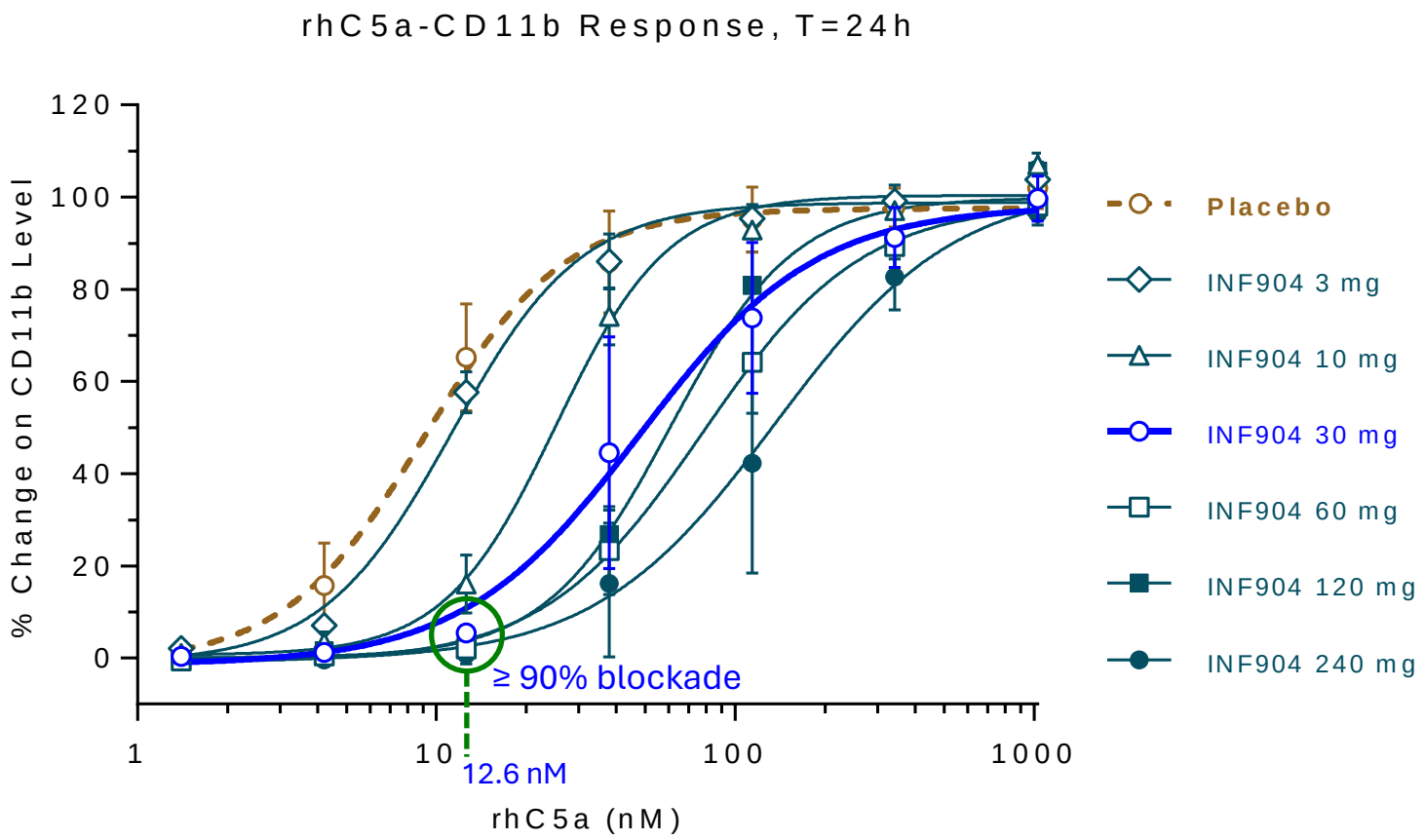
INF904: Phase I Study Design



Footnotes: N= participants per dosing cohort, Ratio for randomization is shown for INF904 : matching Placebo; *3 different capsule strengths tested in a cross-over design

INF904 Phase I Study: PD Results From SAD Part

C5a-Mediated CD11b Upregulation on Neutrophils Ex Vivo at 24h Post Dosing



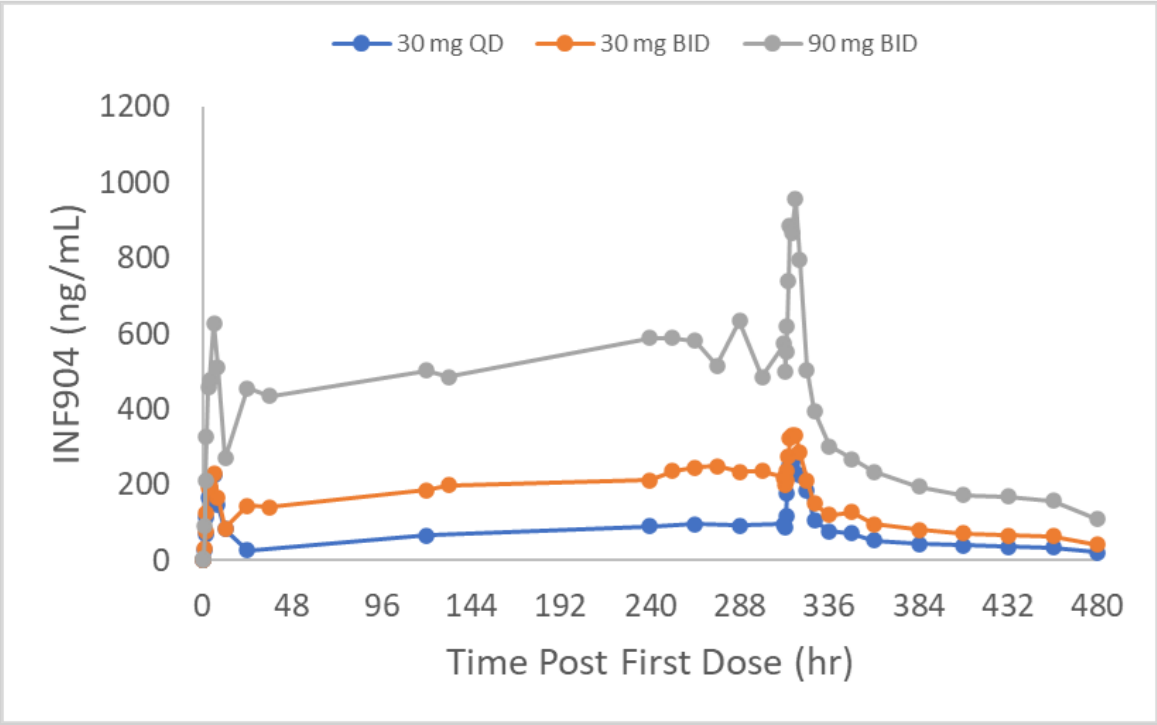
- **INF904**: In response to 12.64 nM of C5a, ≥ 90% blocking activity was observed in the PD analysis in the dose range of 30-240 mg at the time point of **24-hour** post dosing.
- **Avacopan**: In response to ~12.64 nM of C5a, approximately 50% blocking activity was observed in the Phase I published data for 30mg dosing at the time point of **12-hour** post dosing. (Bekker et al. PLoS One 2016; 11(10): e0164646)

C5a (nM)	INF904 (Blocking Activity % vs Placebo)					
	3mg	10mg	30mg	60mg	120mg	240mg
12.6	9.6	74.7	**91.5**	**95.7**	**95.2**	**98.7**

INF904 blocking activity for C5a-induced neutrophil activation in human plasma achieved set goal and is clearly differentiated from the published blocking activity of the only marketed comparator

inflaRx

INF904: PK Results From Multiple Ascending Dose (MAD) Phase I



Dose (Regimen)	Day	C_{max} (ng/mL) ±SD	$AUC_{0\text{-}12hr}$ (ng x hr/mL) ± SD
30 mg QD	1	233 ± 79	1,615 ± 427
	14	284 ± 60	2,609 ± 792
30 mg BID	1	236 ± 97	1,742 ± 648
	14	356 ± 84	3,331 ± 821
90 mg BID	1	653 ± 217	4,815 ± 1,993
	14	1,028 ± 431	8,962 ± 4,247

QD: Once Daily Dosing, BID: Twice Daily Dosing
Results are based on interim data analysis

- INF904 dosing either once daily (QD) or twice daily (BID) exhibits favorable concentration-time profiles (after 14 days dosing)
- INF904 exposure is directly proportional to dose when comparing 30 mg BID versus the 90 mg BID regimens
- In BID regimen, accumulation observed (Day 1 to 14) for C_{max} and $AUC_{0\text{-}12hr}$ average ~ 1.3 and ~1.9-fold
- The first 90 mg dose achieves greater exposure than seen with Day 14 of the 30 mg BID dosing

INF904: Oral C5aR Antagonist With Best-in-Class Potential

MAD

- Well tolerated and with no signals of safety concern in multiple ascending doses involving 30 mg QD, 30 mg BID and 90 mg BID
- Overall percentage of AEs in placebo group was 83.3% compared to 77.8% in active treated subjects
- AE severity: Mild: 52; Moderate: 5; Severe: 0
- No SAEs reported at any dosing level
- 2 AEs, both of mild intensity and both in 1 subject (cohort 3.3/90 mg BID), rated as possibly related to study drug (diarrhea, flatulence)
- No subjects withdrawn from treatment/study

SAD

- Well tolerated and with no signals of safety concern in single ascending doses ranging from 3 mg to 240 mg
- Overall percentage of AEs in placebo group (85.7%) was higher than in active treated subjects (58.3%)
- AE severity: Mild: 78; Moderate: 9; Severe: 0
- No SAEs reported at any dosing level
- 1 moderate AE rated as possibly related to placebo (headache) and 2 mild AEs possibly related to study drug (diarrhea, flatulence)
- 1 subject in cohort 1.4 (60 mg) withdrawn for unrelated AE