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 of the
Securities Exchange Act
of 1934
For the month of June 2024
Commission File
Number: 001-38283

InflaRx N.V.

Winzerlaer Str. 2
07745 Jena,
Germany
(+49) 3641508180
(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 ☐

INCORPORATION BY REFERENCE

On June 5, 2024, InflaRx N.V. (the "Company") issued a press release titled, "InflaRx Hosts R&D Event Highlighting the Promise of INF904." In connection with such announcement, on June 5, 2024, the Company hosted a virtual R&D event and presented its corporate presentation on immuno-dermatology where the Company provided details and developments on its oral C5aR inhibitor INF904.

A copy of the press release and the corporate presentation are attached hereto as Exhibit 99.1 and Exhibit 99.2, respectively, which shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act.

EXHIBIT INDEX

Exhibit No.	Description
99.1	Press Release, dated June 5, 2024
99.2	Corporate Presentation, dated June 5, 2024

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.

INFLARX N.V.

Date: June 5, 2024

By: /s/ Niels Riedemann
Name: Niels Riedemann
Title: Chief Executive Officer

Exhibit 99.1

InflaRx Hosts R&D Event Highlighting the Promise of INF904

- Thought leaders in complement inhibition, chronic spontaneous urticaria (CSU) and hidradenitis suppurativa (HS) provide compelling new insights into the strong development rationales, potential differentiation and medical role of INF904 in initially targeted indications and inflammation & immunology (I&I) more broadly

- Additional details provided on INF904 Phase 2a trial design in moderate-to-severe CSU and HS, with study initiation expected by the calendar year-end of 2024 and a goal of generating additional safety and pharmacokinetic (PK) data, and showing meaningful clinical benefit

- INF904 Phase 2a data expected in summer 2025, with Phase 2b trial initiation expected in 2025

- Commercial assessment indicates CSU and HS both represent multi-billion-dollar market opportunities, with tremendous patient need for effective new mechanisms of action

- InflaRx's strong financial position is expected to fund company operations into 2026, allowing for advancement of clinical programs towards next milestones

Jena, Germany, June 5, 2024 – InflaRx N.V. (Nasdaq: IFRX), a biopharmaceutical company pioneering anti-inflammatory therapeutics by targeting the complement system, today hosted a virtual R&D event focused on the company's oral small molecule C5aR inhibitor, INF904. Speakers provided additional details on development rationales and plans for INF904, as well as additional insight into its potential role in CSU and HS and its broader therapeutic potential in the immuno-inflammation field.

Presenting key opinion leaders (KOLs) included: Prof. Dr. Marcus Maurer (Professor of Dermatology and Allergology, Institute of Allergology, Charité – Universitätsmedizin Berlin, Germany), Christopher Sayed, MD (Prof. of Dermatology, University of North Carolina, Medical School; and Secretary of the HS Foundation) and Prof. Dr. Jörg Köhl (Director of the Institute for Systemic Inflammation Research, University of Lübeck, Lübeck, Germany).

Supplemental information related to today's event, including presentations conducted by the KOLs and InflaRx management can be found in the accompanying slide deck here.

Prof. Niels C. Riedemann, Chief Executive Officer and Founder of InflaRx, commented: "InflaRx has been eager to provide additional details of its development plans for INF904 and to further showcase the tremendous promise of our approach to C5aR inhibition, initially in CSU and HS, and more broadly in I&I. We see the immense potential of INF904 in its ability to address multiple significant unmet medical needs not addressed by drugs currently in development, as well as the ability for this potentially best-in-class compound to find market acceptance in a number of sizable patient settings. We expect to progress expeditiously in our two initially selected immuno-derm indications, CSU and HS, and look forward to achieving additional milestones with INF904 in 2025."

INF904 CSU and HS clinical development program
As previously disclosed, InflaRx will pursue two initial immuno-dermatology indications with INF904 in a single Phase 2a basket trial that is expected to begin by the end of 2024. The Phase 2a trial will be a multi-center, open-label study dosing 75 patients and evaluating multiple INF904 dosing regimens over 4 weeks of treatment in patients with moderate-to-severe CSU and moderate-to-severe HS.

Outcome measures will be assessed via weekly visits to evaluate safety, PK and preliminary signs of efficacy. After the 4-week treatment period, patients will be followed for an additional 4 weeks. Data from this study are expected in the summer of 2025, with the subsequent initiation of a larger Phase 2b study anticipated in 2025 as well.

In the CSU group, patients in Study Arms 1 and 2 will be dosed with INF904 at 30 mg and 90 mg BID (twice daily), respectively. Patients in Study Arm 3 will be comprised of anti-IgE non-responders and dosed at 90 mg BID. In total, the CSU group will dose 45 patients randomized at a 1:1:1 ratio. In addition to safety and PK parameters, assessed CSU efficacy measures will include change of the Urticaria Activity Score 7 (UAS7), Hives Severity Score (HSS7) and Itch Severity Score (ISS7) from baseline to the end of week 4. Biomarkers and Patient-Reported Outcome (PRO) endpoints related to urticaria control and quality of life will also be assessed.

In the HS group, 30 patients will be randomized at a 1:1:1 ratio to 3 doses of INF904 at 30 mg, 60 mg or 90 mg BID. In addition to safety and PK parameters, assessed HS efficacy measures will include change in total abscess, inflammatory nodule and draining tunnel (dT) count, HS lesions-related scores and Clinician's Global Impression of Change (CGI-C) at 4 weeks. PRO endpoints related to HS disease control and quality of life will also be assessed.

As previously disclosed, the company is currently conducting additional pre-clinical studies with INF904, including chronic toxicology studies, as part of its effort to enable longer-term dosing of INF904 in future clinical trials.

INF904 as a "pipeline-in-a-product"
Given the potential of INF904 to have a broad commercial footprint, InflaRx believes INF904 could address meaningful markets in immuno-dermatology and in immuno-inflammation, including in nephrology, neurology and hematology. While InflaRx intends to focus its resources on its immediate goals addressing CSU and HS, we continue to assess and monitor the value of pursuing additional areas and applications via potential future collaborations with partners.

About INF904
INF904 is an orally administered small molecule inhibitor of C5a-induced signaling via the receptor C5aR. INF904 showed anti-inflammatory therapeutic effects in several pre-clinical disease models. Further, in contrast to the marketed C5aR inhibitor, in vitro experiments demonstrated that INF904 has minimal inhibition of the cytochrome P450 3A4/5 (CYP3A4/5) enzymes, which play an important role in the metabolism of a variety of metabolites and drugs, including glucocorticoids. Reported results from a first-in-human study demonstrated that INF904 is well tolerated in treated subjects and exhibits no safety signals of concern in single doses ranging from 3 mg to 240 mg or multiple doses ranging from 30 mg once per day (QD) to 90 mg twice per day (BID) for 14 days. Pharmacokinetic / pharmacodynamic data support best-in-class potential of INF904 with a ≥90% blockade of C5a-induced neutrophil activation achieved over the 14-day dosing period.

About InflaRx N.V.
InflaRx (Nasdaq: IFRX) is a biopharmaceutical company pioneering anti-inflammatory therapeutics by applying its proprietary anti-C5a and anti-C5aR technologies to discover, develop and commercialize highly 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 is also developing INF904, an orally administered small molecule inhibitor of C5a-induced signaling via the C5a receptor. 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.de.

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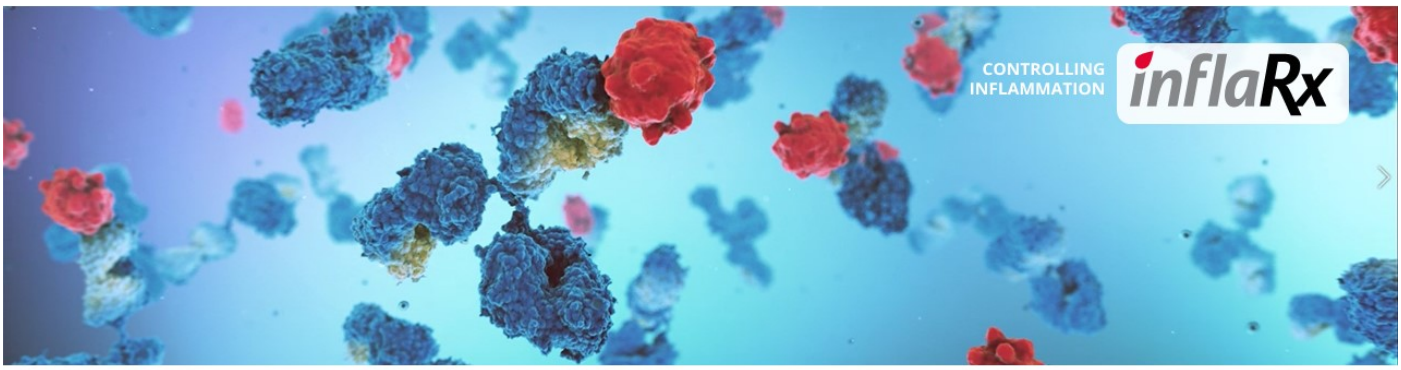
FORWARD-LOOKING STATEMENTS

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 Marketing Authorization Application 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 European Medicines Agency 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.

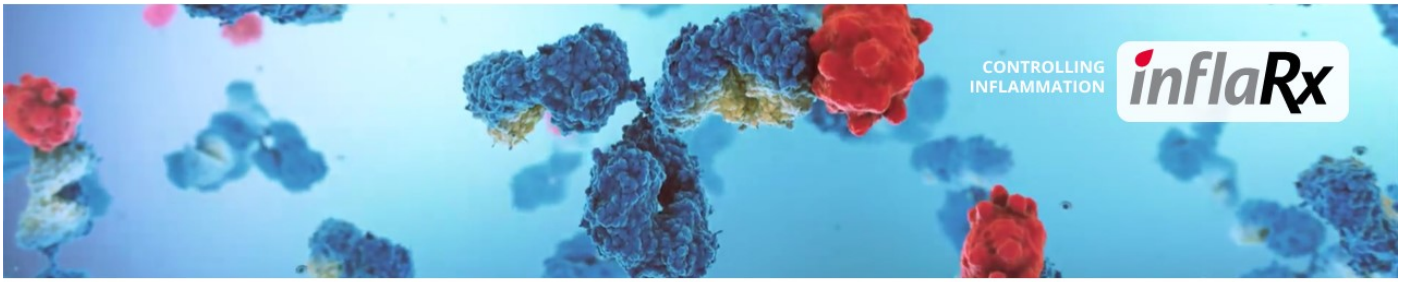
Exhibit 99.2



InflaRx - virtual R&D event

INF904: oral C5aR inhibitor with best-in-class potential in the immuno-dermatology and broader I&I space

June 5, 2024 – 12 p.m. EST (US) / 6pm CET (EU)



IMPORTANT NOTICE AND DISCLAIMER

This presentation has been prepared by InflaRx N.V. ("InflaRx" or the "Company"). This presentation is made for informational purposes only and does not constitute an offer to sell or a solicitation of an offer to buy securities. This presentation may not be relied upon in connection with the purchase or sale of any security and should not be construed as investment advice.

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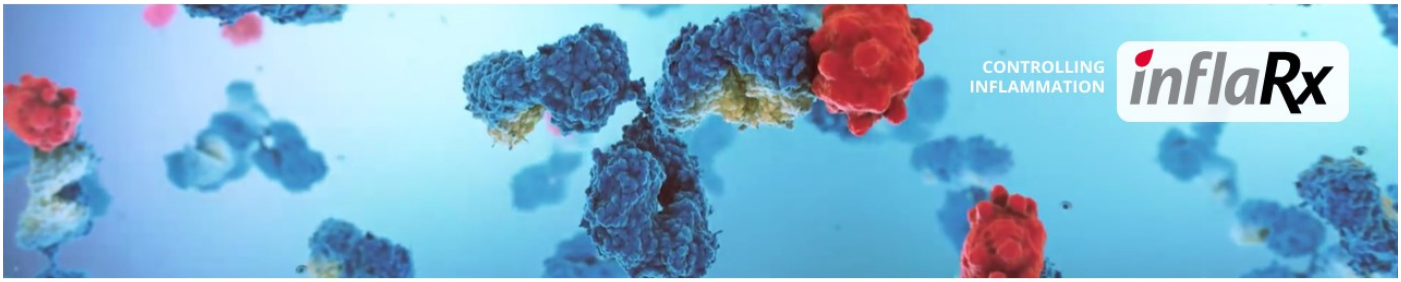

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Information and Sources

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.

About InflaRx N.V.

InflaRx (Nasdaq: IFRX) is a biopharmaceutical 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.

Content

Presentation of today's speakers and company introduction

Current understanding of the C5a/C5aR1 biology
Prof. Dr. med. Jörg Köhl

The Role of C5aR in chronic spontaneous urticaria (CSU)
Prof. Dr. med. Marcus Maurer

The Role of C5aR in hidradenitis suppurativa (HS)
Prof. Chris Sayed, MD

Q&A

Next clinical development steps for INF904

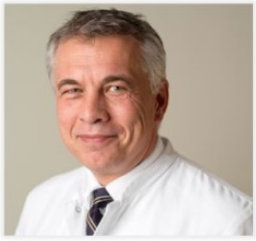
Commercial considerations

Summary and conclusion

Q&A

Today's presenters

Prof. Dr. med. Jörg Köhl
Director, Institute for Systemic Inflammation Research (ISEF)
University of Lübeck, Germany



Prof. Dr. med. Marcus Maurer
Managing Director, Institute for Allergy Research, Charité University Hospital, Berlin, Germany

Dr. Chris Sayed, MD
Prof. of Dermatology
UNC School of Medicine, Dermatology, Chapel Hill, NC
Secretary, HS Foundation, USA

Prof. Niels Riedemann
Founder and CEO

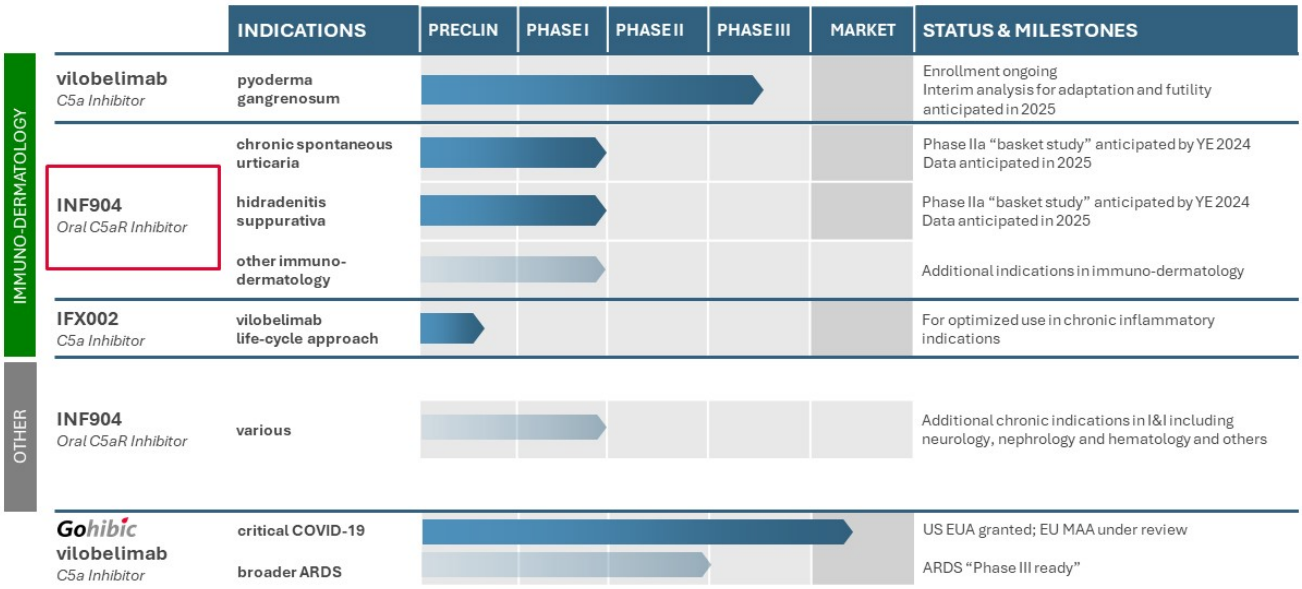
Camilla Chong, MD
CMO

Prof. Renfeng Guo
Founder and CSO

Dr. Thomas Taapken
CFO

Jan Medina
VP Investor Relations

Late-stage pipeline targets multiple sizable markets



IFRX strategic focus: why immuno-dermatology

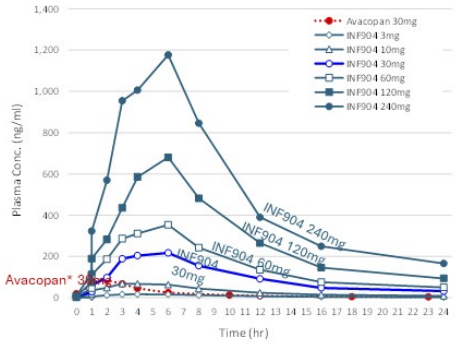
- **Strong rationale for the role of C5a/C5aR based** on mechanism of action, pre-clinical and clinical data
- Potential to target **several attractive, billion-dollar+ commercial markets with an attractive new MoA which is not currently addressed by any other drug under development in this space**
- **For CSU and HS: 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 related to the MoA and potential broad therapeutic index**
- Established **network of experts and clinical development expertise**

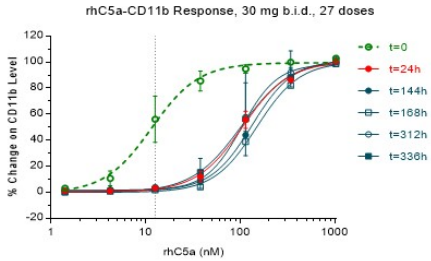
INF904 – highly selective C5aR1 inhibitor with "best in class" potential

- ✓ **Superior PK/PD profile** in Phase I SAD and MAD studies compared to reported data from marketed comparator avacopan:
  - **~3-fold higher C_{max}** and **~10-fold higher AUC_{last}** (at comparable dosing levels)
  - **Significantly increased blocking activity** >90% blocking of C5a activity
  - **Faster achievement of therapeutic exposures** with broad therapeutic index, BID and QD dosing
- ✓ **Favorable drug safety profile** supported by preclinical studies and data reported from InflaRx's Phase I SAD and MAD trials
- ✓ **Other favorable features** compared to avacopan:
  - **Higher drug strength with potential for reduced capsule intake**
  - **Much weaker inhibitor of CYP3A4/5 in pre-clinical studies**

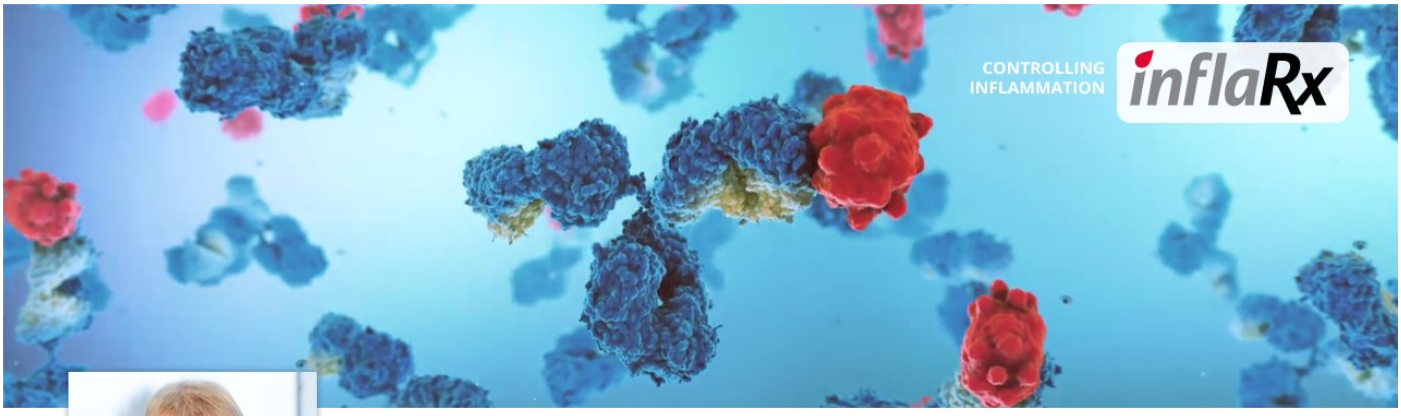
These properties allow for exploring a significantly more potent C5aR1 inhibition in patients and this, ultimately, may lead to higher clinical efficacy for INF904

This **could open significant additional market opportunities**





*InflaRx data on file: PK Results From Single Ascending Dose (SAD) Phase 1 study – note: Avacopan data (Becker et al, 2016, PLoS One) are superimposed in graph for orientation. Avacopan was not included as a comparator in INF904 Phase I study
** InflaRx data on file: PD Results from multiple ascending dose (MAD) Phase 1 study.

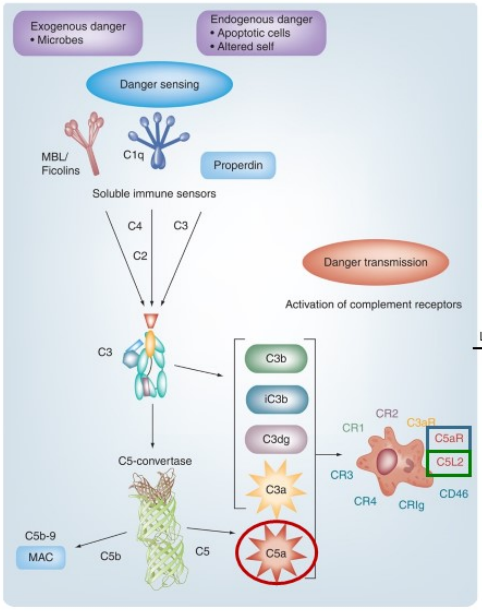


Prof. Dr. Jörg Köhl

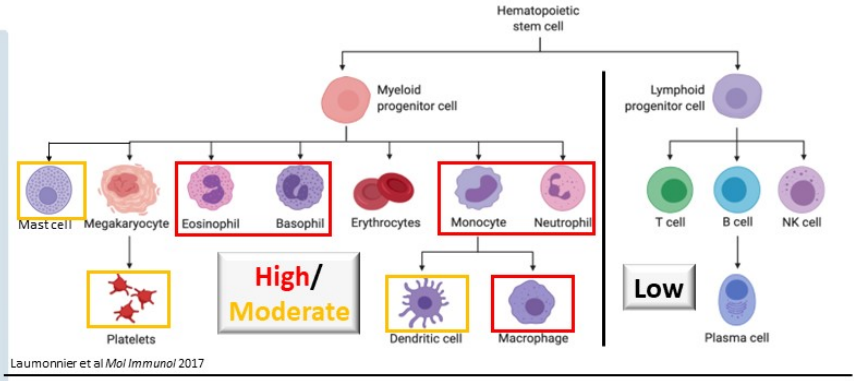
Current understanding of the C5a/C5aR1 biology

Why target C5aR1 in the immuno-dermatologic space?

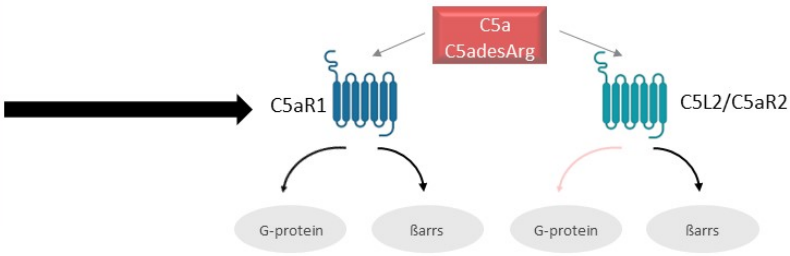
Complement translates danger signals into a broad range of cellular responses



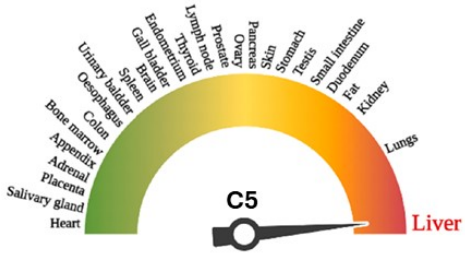
Zhang and Köhl *Expert Rev Clin Immunol* 2010



Laumonnier et al *Mol Immunol* 2017

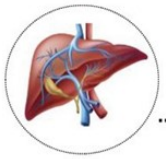


The multiple sources of C5 / C5a and its systemic and cellular functions



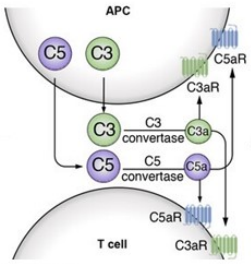
Gosh and Rana *Int. Immunopharmacol* 2023

Liver complement



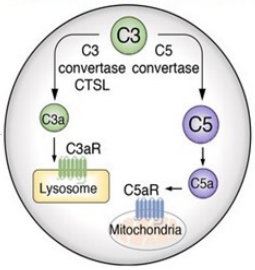
Recognition and removal of pathogens and noxious self in the blood and lymph

Immune cell autocrine complement



Local immune cell activation and function

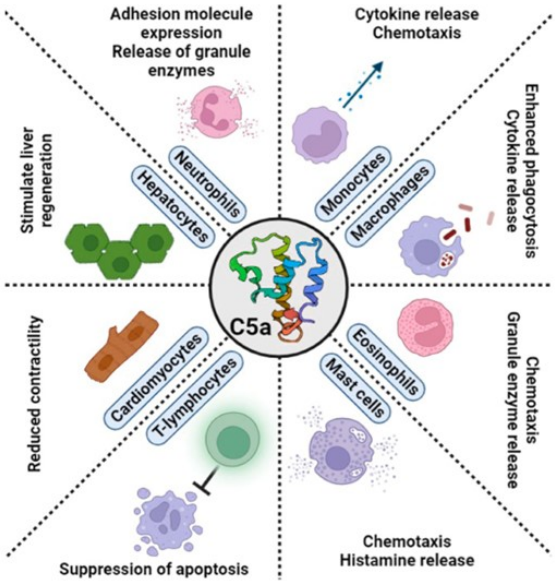
Intracellular complement



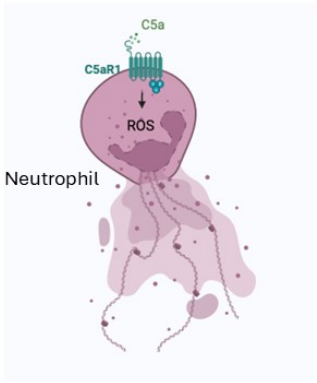
Control of basic cell physiological processes

Singh and Kemper *Eur. J. Immunol.*2023

Multi-functional roles of C5a in distinct myeloid cells via C5aR1 activation



C5aR1 drives NETosis

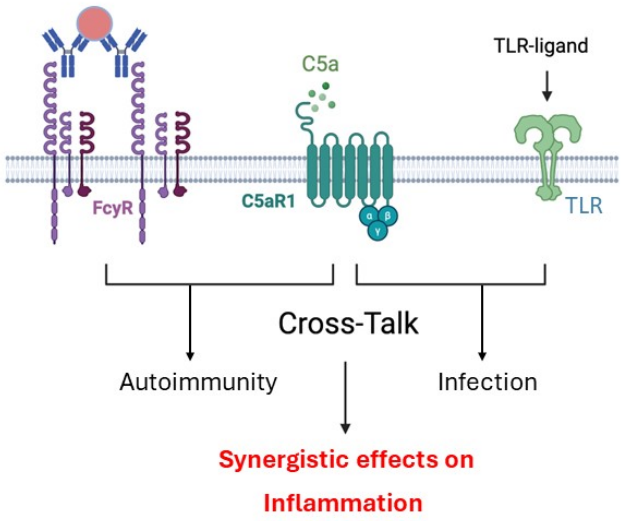


Critical driver of inflammation in Neutrophilic Skin Diseases
(Hidradenitis suppurativa, Pyoderma gangrenosum)

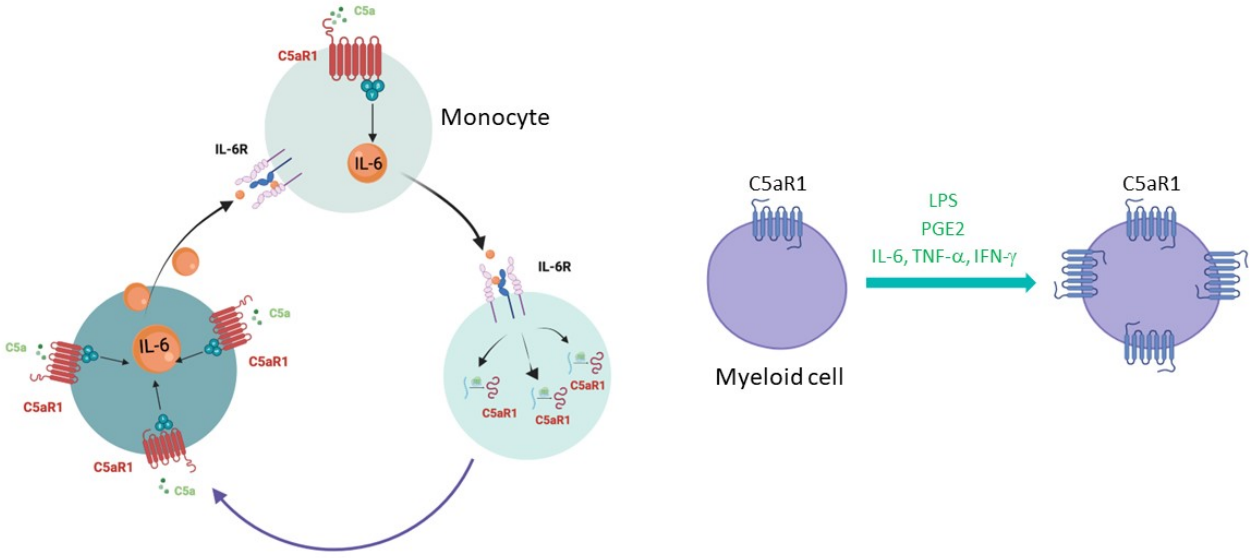
Gosh and Rana *Int. Immunopharmacol* 2023

Silva et al *J Clin Invest* 2023 / Wang et al. *J. Invest. Dermatol.* 2024 / Byrd et al. *Sci. Immunol.* 2019

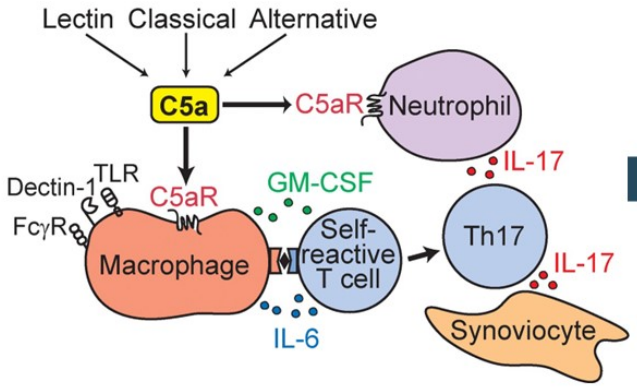
The C5a/C5aR1 axis synergizes with other innate immune receptors to promote a pro-inflammatory environment



C5aR1 signaling induces pro-inflammatory patterns in myeloid immune cells

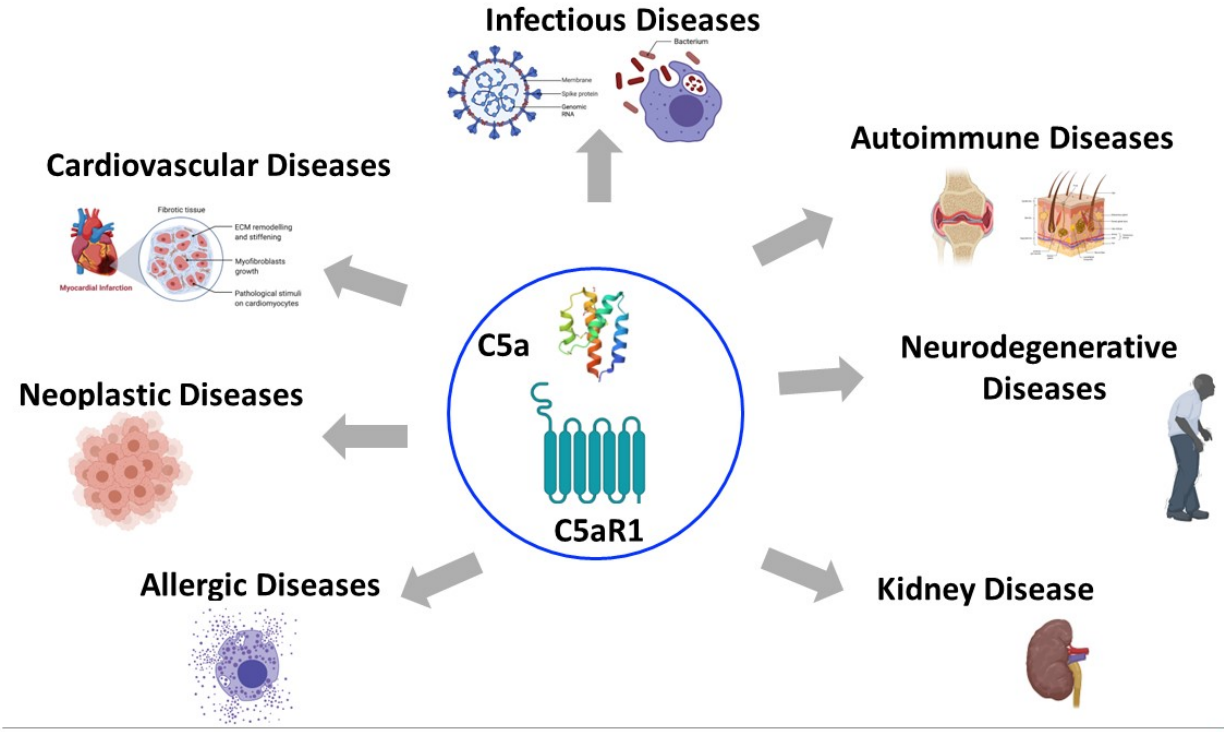


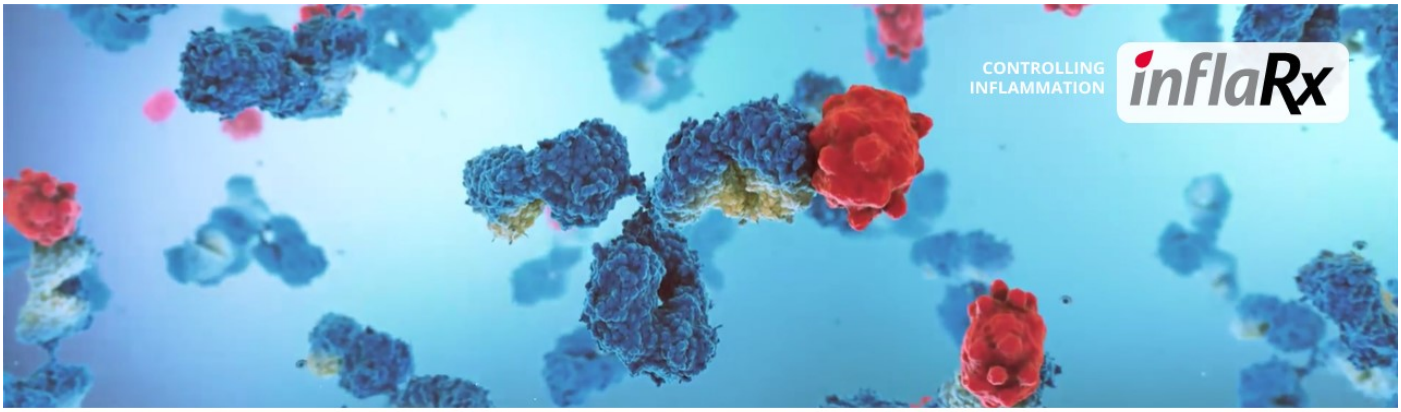
C5a/C5aR1 axis activation in macrophages drives Th17 development



- Th17 responses are frequently found in neutrophilic skin diseases such as HS and PG.
- IL-17 promotes neutrophil migration into affected skin areas.

Hashimoto et al. *J. Exp. Med.* 2010 HS = Hidradenitis suppurativa; PG = Pyoderma gangrenosum

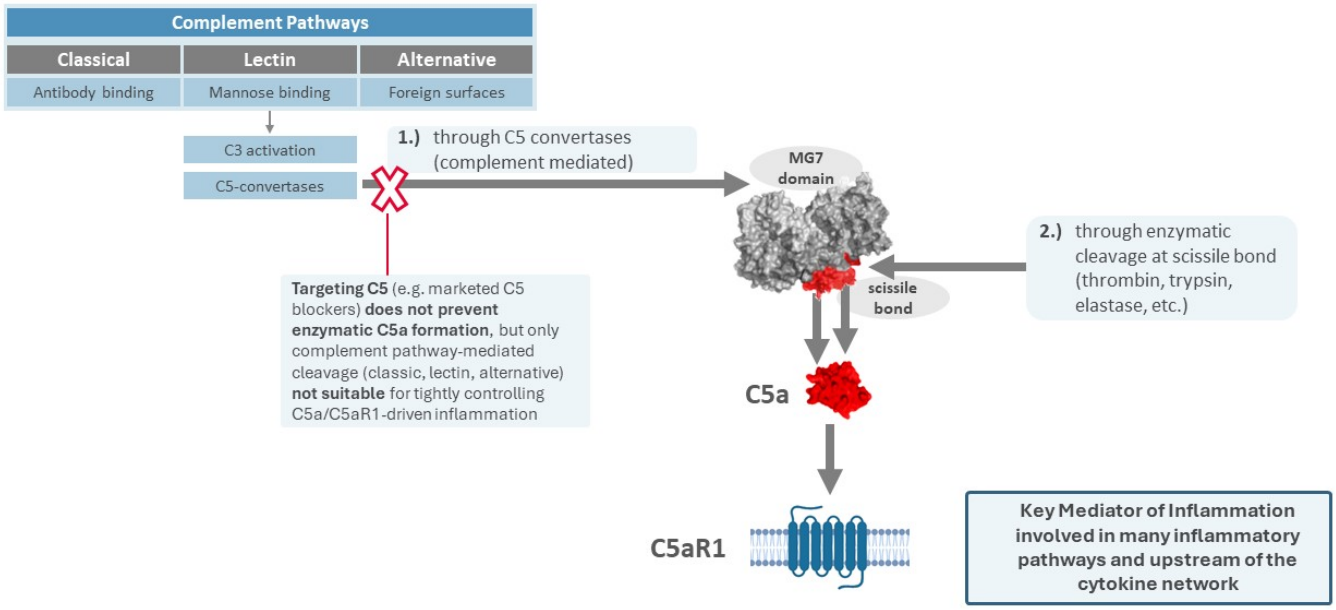

Infectious Diseases
Membrane
Spike protein
Genomic RNA
Bacterium
Cardiovascular Diseases
Fibrotic tissue
ECM remodelling and stiffening
Myofibroblasts growth
Pathological stimuli on cardiomyocytes
Myocardial Infarction
Autoimmune Diseases
C5a
C5aR1
Neurodegenerative Diseases
Neoplastic Diseases
Allergic Diseases
Kidney Disease

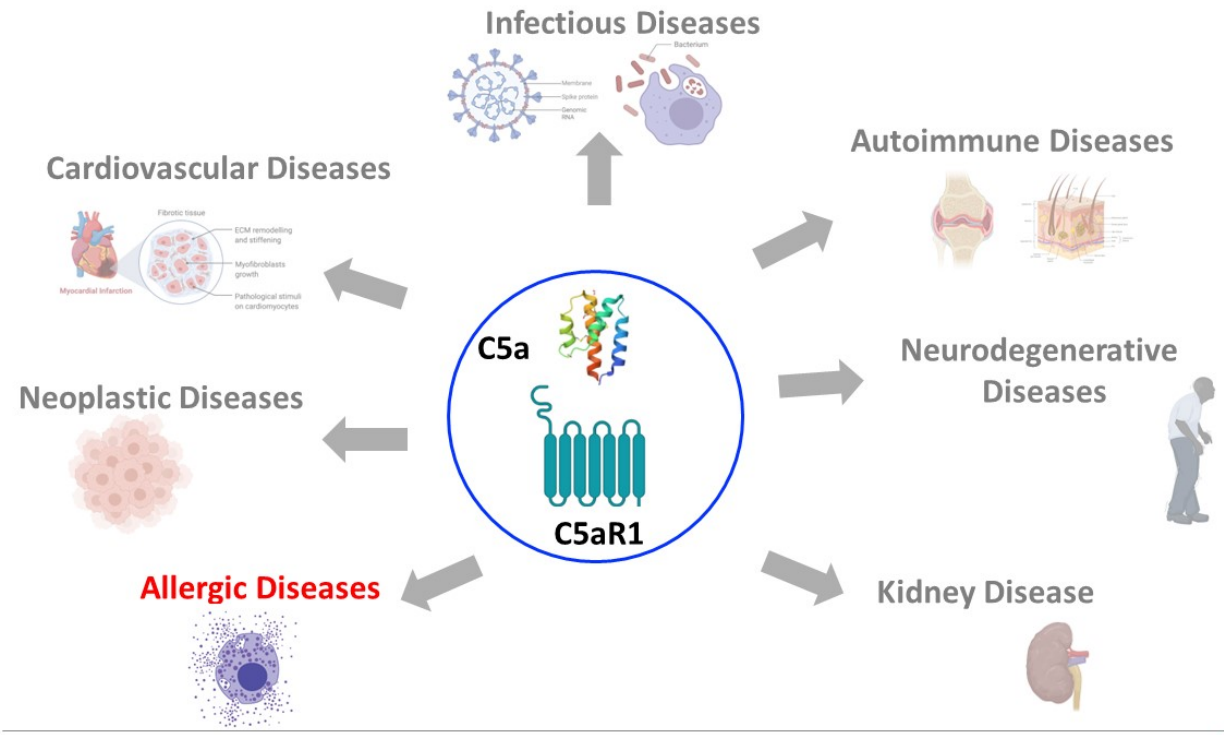


C5aR1 As a Target in the Complement System

Prof. Dr. Jörg Köhl

C5a/C5aR1 signaling inhibition: the importance of a targeted approach

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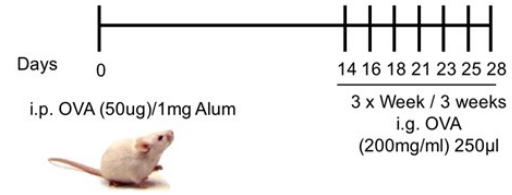
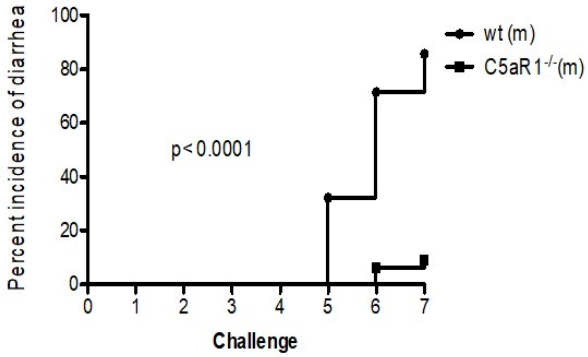
Neurodegenerative Diseases
Neoplastic Diseases
Allergic Diseases
Kidney Disease

C5ar1$^{-/-}$ mice are protected from the development of anaphylaxis

Ovalbumin induced allergic / anaphylactic gut permeability increase leading to diarrhea

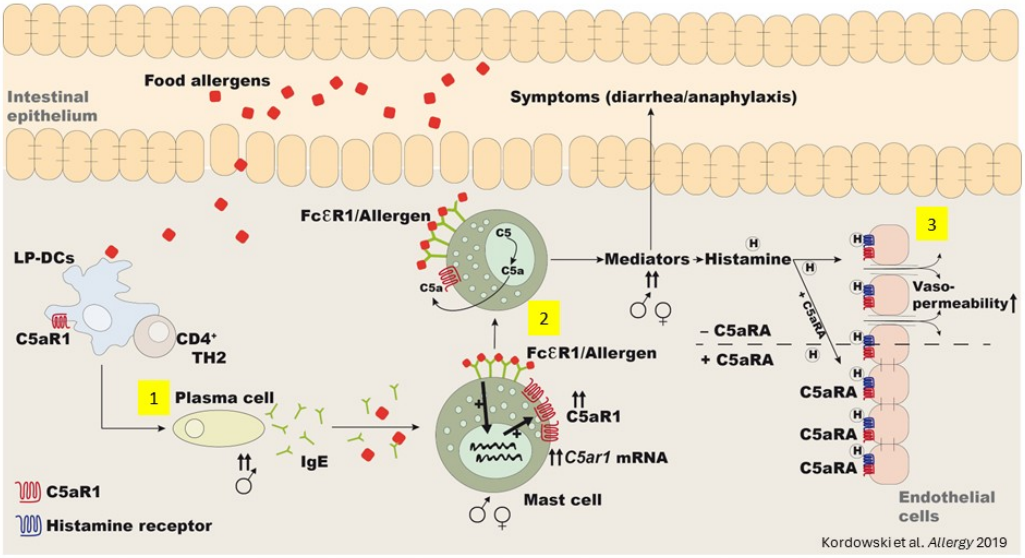


Ahrens et al., *Am J Pathol* 2012



Kordowski et al. *Allergy* 2019

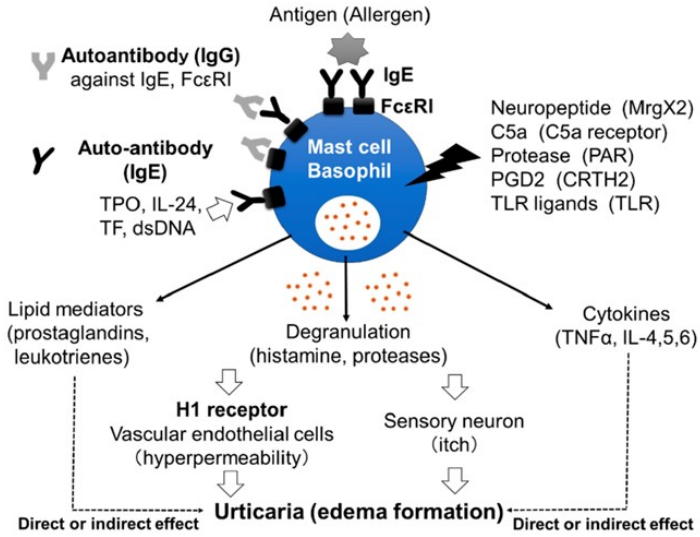
C5aR1-mediated induction of anaphylaxis via mast cell activation



Activation of the C5a/C5aR1 axis drives the development of anaphylaxis at several levels:

1. The regulation of the B cell response in male mice that leads to the production of antigen-specific IgE.
2. The enhancement of FcεR1-dependent degranulation of MCs.
3. The sensitization of the vascular system towards the MC mediator histamine.

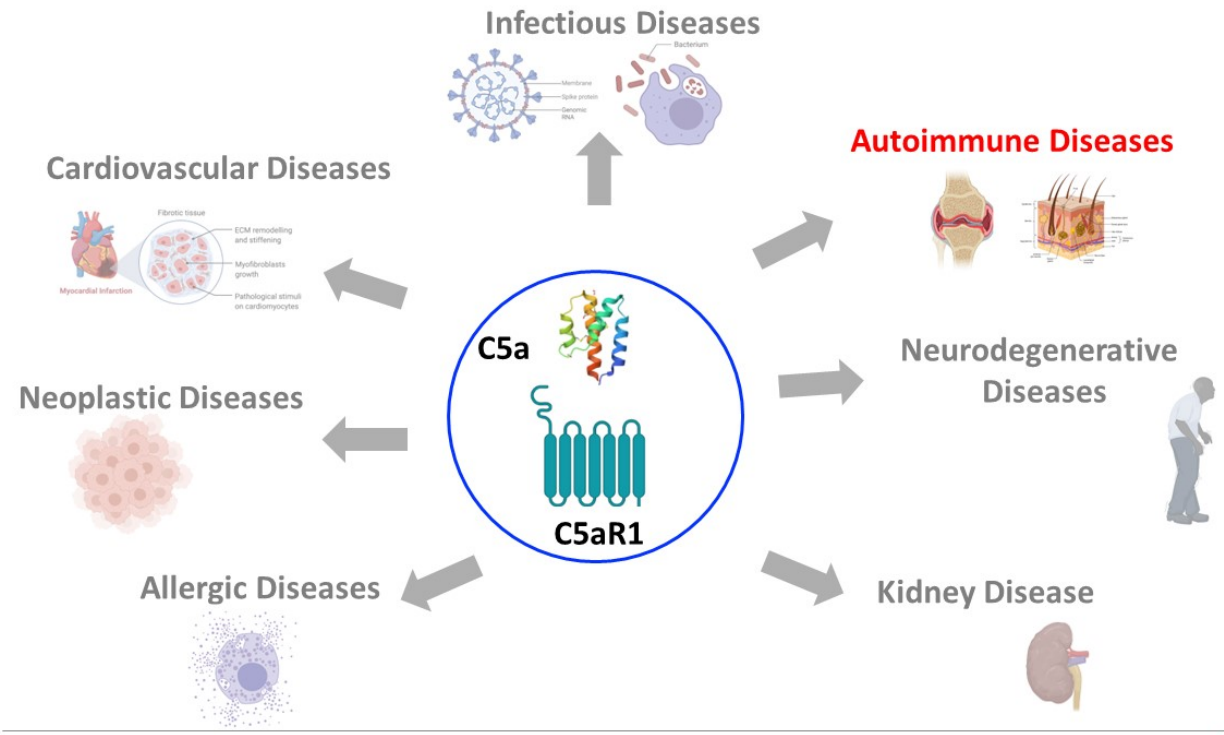
Mast cell activation and its role in chronic spontaneous urticaria (CSU)



addressed by

Prof. Maurer

Yanase et al. *Cells* 2021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Neutrophilic skin diseases are associated with activation of C5aR1

- **Hidradenitis suppurativa***
  - Increased systemic C5a levels
  - Increased local C5aR1 expression
  - Skin microbiota expressing C5-cleaving enzyme

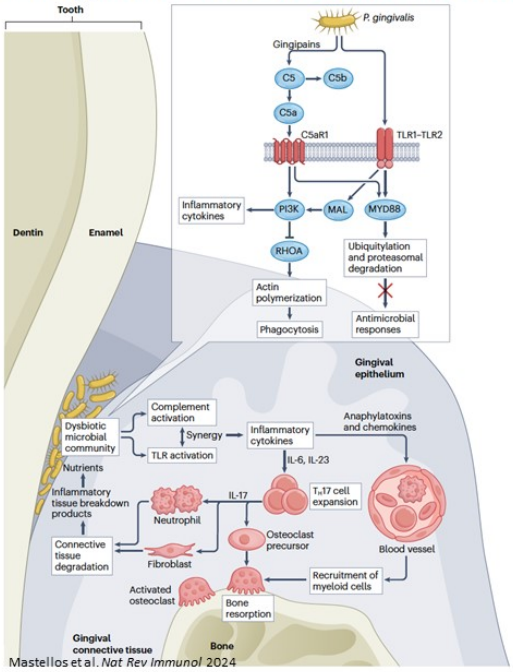
→ Addressed by Prof. Sayed

- **Pyoderma gangraenosum****
  - High levels of local C5a in wound fluid
  - C5a drives strong NET formation and elastase production
  - Increased local C5aR1 expression

- **Pemphigoid diseases*****
  - Complement deposition at the dermal/epidermal junction
  - Increased local C5aR1 expression in skin lesions
  - C5aR1 deletion or targeting protects from the development of skin lesions in preclinical BP models

* Grand et al. *Exp. Dermatol.* 2019 ** Flora et al. *Exp. Dermatol.* 2021 / Wang et al. *J. Invest. Dermatol.* 2024 *** Papara et al. *Front Immunol.* 2022 / Emtenani et al. *Front. Immunol.* 2022

The hypothesis of bacterial-induced C5aR1 / TLR cross-talk driving skin inflammation



Mastellos et al. *Nat Rev Immunol* 2024

- The bacterium *Porphyromonas* in the oral cavity cleaves C5 into C5a and activates TLR2 to subvert complement/TLR-driven anti-microbial responses.
- This leads to a dysbiotic microbial community activating complement and PRRs* to induce inflammatory cytokines (IL-6, IL-23) promoting Th17 cell expansion and neutrophil recruitment.
- The feedforward loop connecting dysbiosis and inflammation is self-sustained and contributes to chronicity.

- Microbiome studies identified Porphyromonas in HS as strongly associated with disease activity.**
- Maladaptive Th17 immunity is a critical driver of disease in HS.
- Of note: the C5a/C5aR1 controls Th17 development in experimental arthritis.

* PRR = pattern recognition receptor **Ring et al. JAMA Dermatol. 2017 / Guet-Revillet et al. Clin Infect Dis 2017

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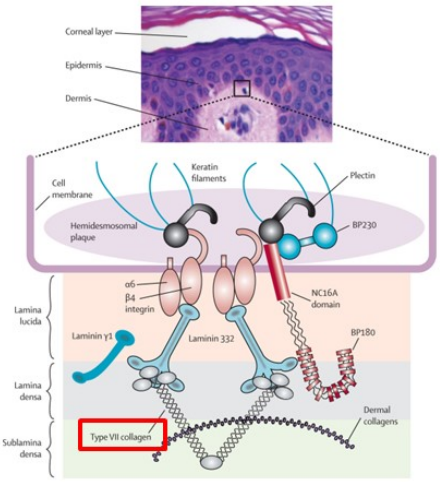
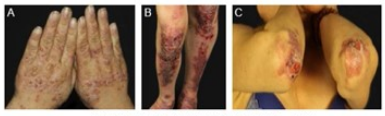
Pathophysiologic role of the C5a/C5aR1 axis in pemphigoid disease



Schmidt and Zillikens, *Lancet*, 2013

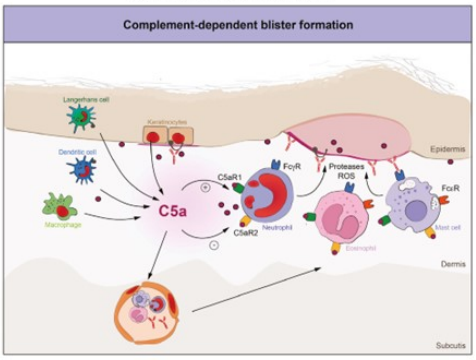
Structural proteins of the dermal/epidermal junction (DEJ) as targets of auto-antibody formation in Pemphigoid Disease

Epidermolysis Bullosa Acquisita (EBA)



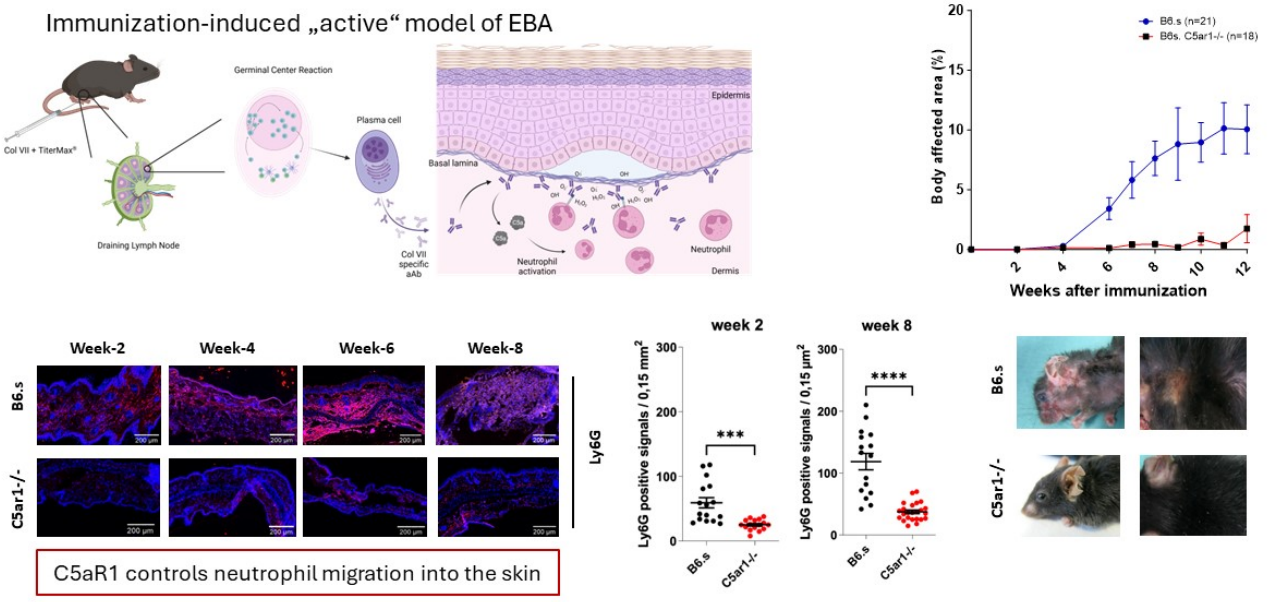
Miyamoto et al., *An Bras Dermatol.*, 2022

Role of C5a/C5aR1 in blister formation and skin inflammation



Papara et al., *Front Immunol.*, 2022

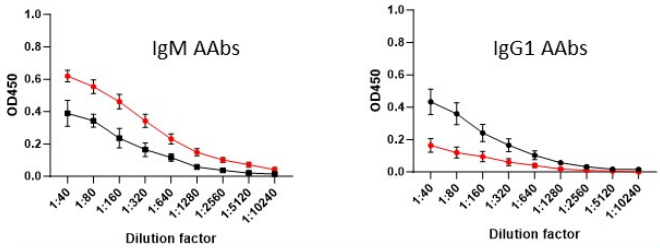
The C5a/C5aR1 axis controls neutrophil migration into the skin



Kenno et al. (unpublished work from the Köhl lab)

C5aR1 controls the early generation of auto-antigen-specific IgG Abs and their inflammatory potential

Auto-antibody (AAb) formation

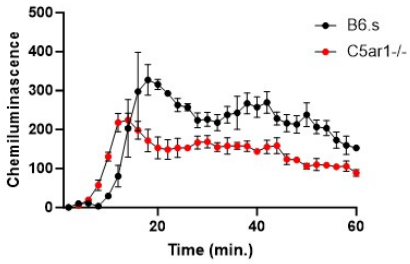


Reduced switch from IgM to IgG auto-antibodies in C5ar1$^{-/-}$ mice

The data are in line with a critical role for C5aR1 in the Germinal Center B cell formation and point towards a more general role for C5aR1 in auto-antibody formation

C5aR1 may also regulate auto-antibody formation in CSU

IgG AAb-driven ROS release from neutrophils



C5aR1 controls IgG Fc (FcγR)-driven ROS* release from neutrophils – i.e. enhances the inflammatory potency of auto-antibodies

Kenno et al. (unpublished work from the Köhl lab) / Verghese et al. JCI Insight 2018 / Cumpelik et al. Nat Immunol 2021

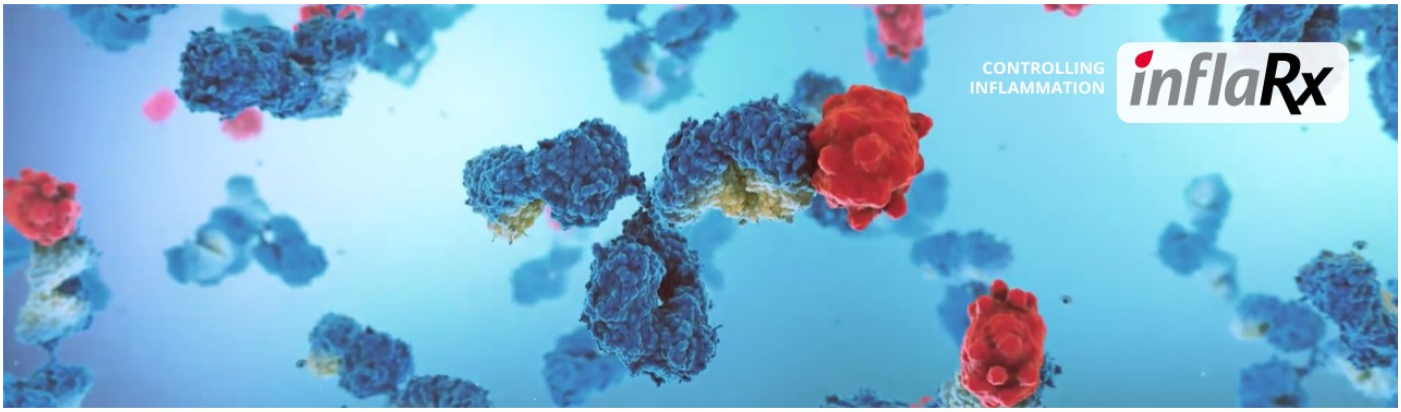
*ROS = Reactive Oxygen Species

C5aR1 drives disease development in experimental EBA at several levels

1. The recruitment of neutrophils into the skin.

2. The early generation of Type VII collagen-specific IgG Aabs.

3. The inflammatory potential (ROS) of Type VII collagen-specific IgG Aabs.



Summary

Why target C5aR1 in the immuno-dermatologic space?

Key Message 1

Evidence for local C5a and C5aR1 expression in disease

Key Message 2

C5aR1 activates and controls key effector cells (neutrophils, eosinophils, basophils, MCs), the B cell response and Th17 development

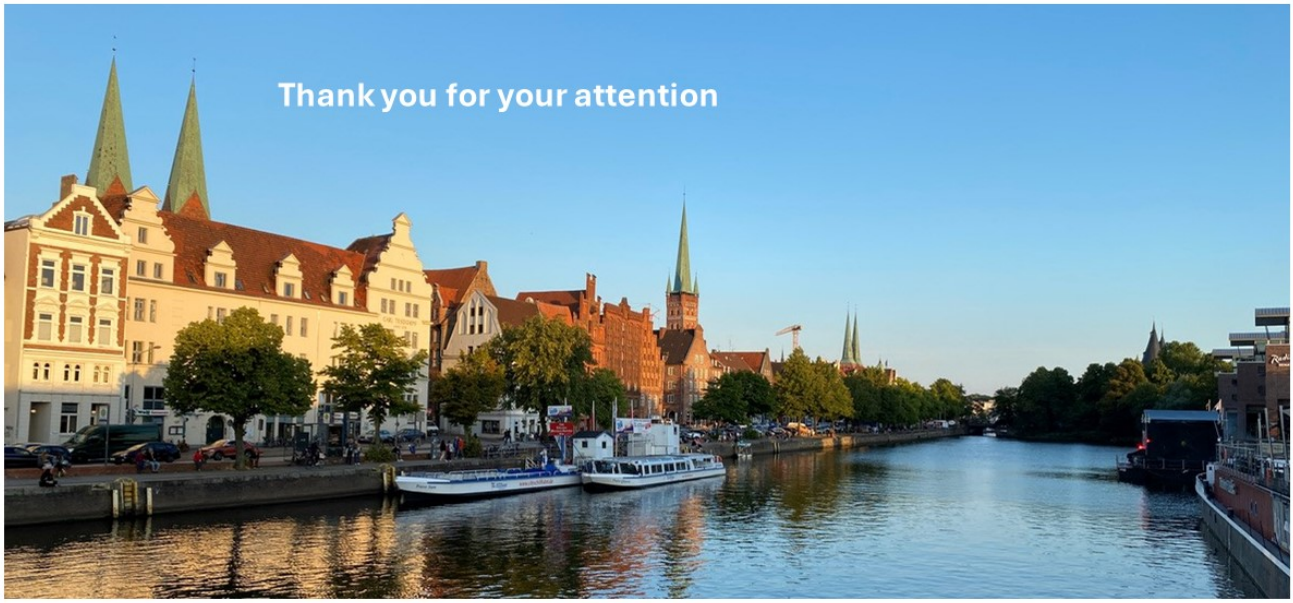
Key Messages

Key Message 3

Targeting C5aR1 in preclinical skin disease models strongly reduces disease development

Key Message 4

Tailored targeting effect: Blocking C5aR1 leaves upstream and downstream complement pathways intact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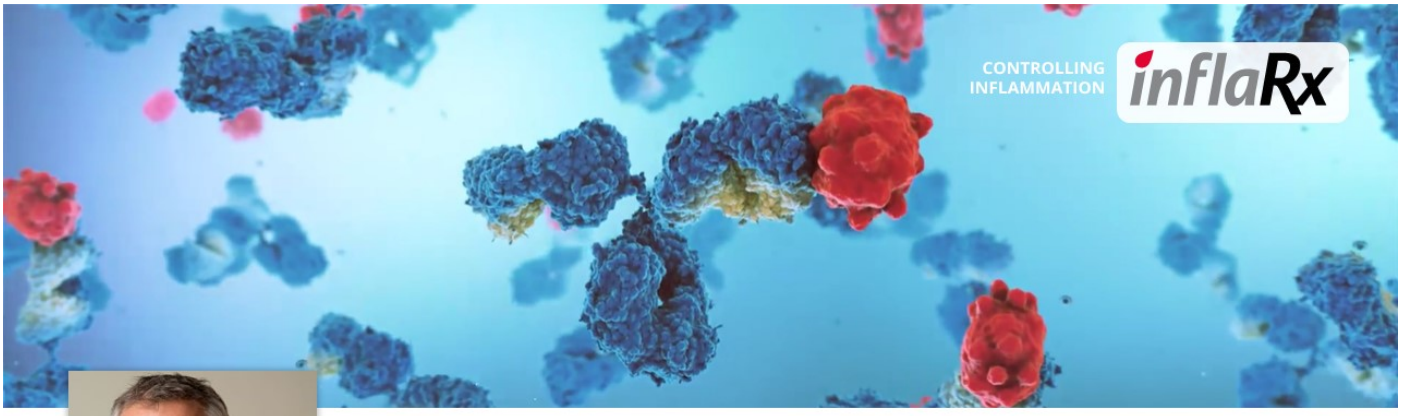
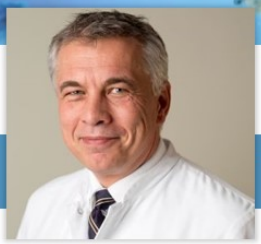




The role of C5aR in chronic spontaneous urticaria (CSU)

Prof. Dr. Marcus Maurer

Objectives for CSU session

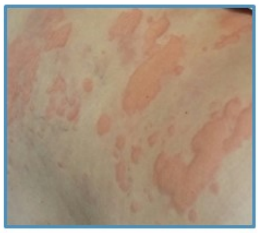
1. Introduction to CSU disease, epidemiology and current unmet medical needs
2. Pathophysiology of the disease : Type I & IIb endotypes
3. The role of C5a/C5aR signaling : IgE dependent and independent pathways
4. The potential role of INF904 in the future treatment landscape

Chronic Spontaneous Urticaria (CSU)

CSU Overview and Unmet Need

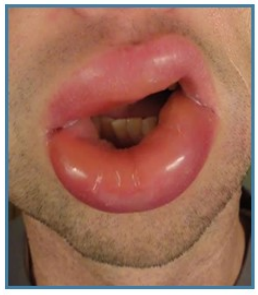
Clinical features

- 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

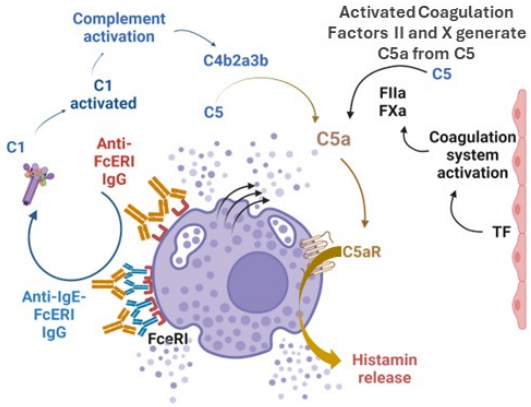
- Therapies such as 2nd-generation antihistamines are not effective in a significant number of patients
- Options such as anti Ig-E therapy and immunosuppressants also do not adequately serve the CSU population

CSU Endotypes : type I auto-allergens and type IIb autoimmunity

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

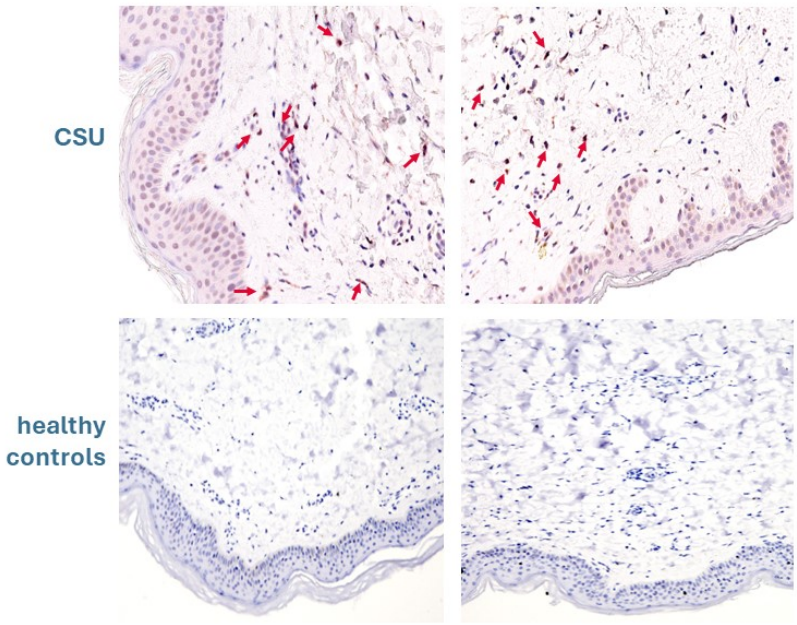
1. **Type I autoallergens (IgE mediated)**
   Close contact of two Fc regions of the IgG anti-FcERI or IgG anti-IgE FcERI complex activate complement factor C1

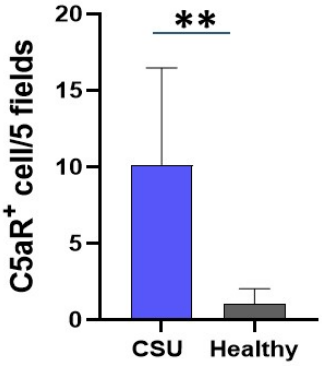
2. **Type IIb autoimmunity (IgG mediated)**
   C5a is activated by the binding of IgG-anti-FcεRI or IgG-anti-IgE to FcεRI on mast cells and basophils
   ~30% of CSU



Modified from Kaplan AP et al Clin Exp Allergy 2009 and Yanase Y, J Allergy Clin Immunol 2021

C5aR is upregulated in the skin of CSU patients

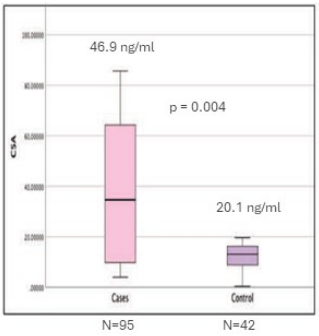




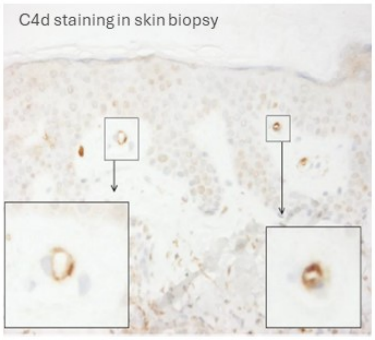
Munoz M. Charite Berlin Data on file

C5a levels are increased in CSU: an important new mechanism for histamine releases

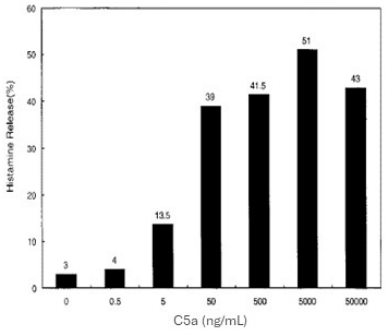
CSU patients have elevated C5a levels



CSU patients show evidence of complement activation in the skin



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



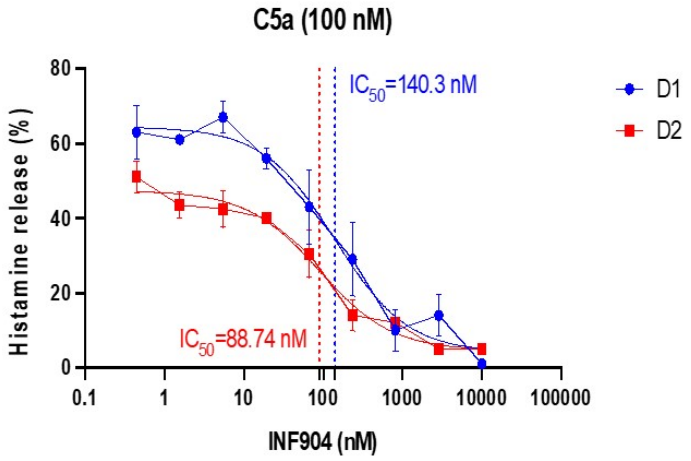
Histamine release (percentage) from donor basophils stimulated with increasing levels of C5a

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

Kikuchi, 2002 *J Allergy Clin Immunol*:109

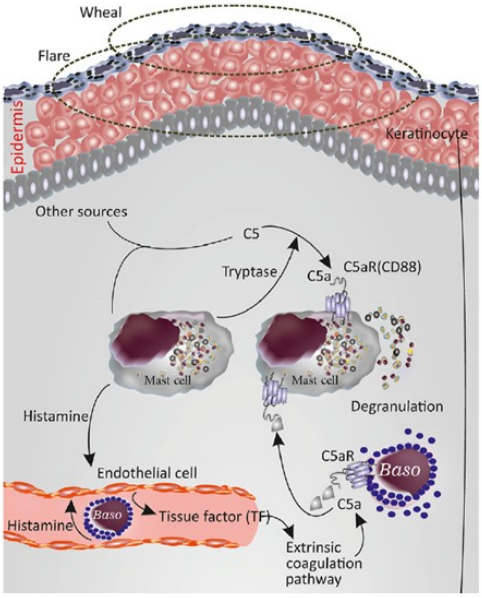
INF904 can effectively block C5a-mediated histamine releases (IgE-independent)

C5a-mediated histamine release from human basophils can be effectively inhibited by INF904



InflaRx Data on file

C5a drives mast cell attraction, activation and degranulation



- The chemoattractant nature of C5a for mast cells explains their accumulation at inflammation sites
- Mast cells amplify this cross talk by producing complement proteins and activating them via their released tryptase
- C5a is also produced by the extrinsic coagulation pathway activated by Tissue Factor

Elieh-Ali-Komi D, Metz M, Kolkhir P, Kocatürk E, Scheffel J, Frischbutter S, Terhorst-Molawi D, Fox L, Maurer M. Chronic urticaria and the pathogenic role of mast cells. *Allergol Int. 2023* Jul;72(3):359-368

Current treatment landscape including Phase 3 clinical compounds

Therapy	MoA	Efficacy: 12 weeks	Limitations
Xolair® (omalizumab) marketed	Anti-IgE mAb	**300 mg Q4w** / **Placebo** UAS7 (CFB) ~-21 / ~-8 UAS7=0 36% / 9%	Black box warning [1] SC injection > 30 % remain symptomatic [5]
Dupixent® (dupilumab) Phase 3	Anti-IL4/13 mAb	**300 mg Q2w** / **Placebo** UAS7 (CFB) -16 / -9	Q2W SC injections Lack of efficacy in Xolair failures [2]
Remibrutinib Phase 3 completed	BTK inhibitor	**25 mg BID** / **Placebo** UAS7 (CFB) -20.2 / -7.9 UAS7 (CFB) -20.1 / -13.8 UAS7 (CFB) -19.6 / -11.7	BTK is expressed on haematopoietic cells including B cells, myeloid cells, platelets[3] potential long-term safety concern [Fenebrutinib – same MoA – on clinical hold by FDA]
Barzolvolimab Phase 3	Anti-cKIT mAb	**150 mg Q4w** / **Placebo** UAS7 (CFB) -23 / -10.5	SC injection c-KIT is expressed on haematopoetic stem cells, melanocytes, CNS and germ cells [4] Hair discolouration, urticaria, neutropenia Unknown impact of long-term mast cell depletion

NB. Apart from anti-histamines, anti-IgE therapy is the only approved therapy for CSU patients.
Off label usage include cyclosporin, hydroxychloroquine, etc

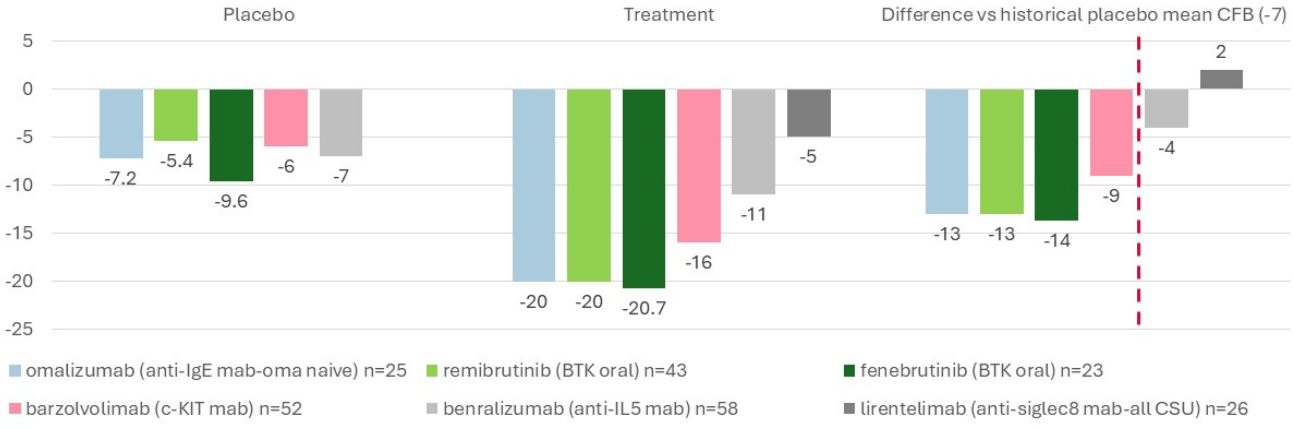
1. Xolair label 2. Sanofi PR 29/07/21 3. Russkamp et al *Experimental Haematology* 2021;95:31-45 4. Garg N et al *J Clin Med* 2022;11(20)6039 5. Metz 2020

There are opportunities for another novel MoA such as anti-C5aR with INF-904

Opportunities
Ease of administration to address patient preference and adherence: ▪ Oral capsules (BD or QD dosing)
New mechanism of action to treat all affected CSU patient populations: ▪ Type I IgE mediated and Type IIb autoimmune non-IgE mediated ▪ Anti-Ig E naïve and refractory patients
Excellent benefit-risk profile ▪ Fast onset of action to alleviate itch and hives within 4 weeks or earlier ▪ Durability of response ▪ No Black Box warning ▪ No hair or skin discoloration during and after treatment ▪ No issues with neutropenia or thrombocytopenia ▪ No recurrence of urticaria

Predictor of efficacy from Phase 2 UAS7 data change from baseline (CFB) at 4 weeks

- Historical UAS7 placebo rates at 4 weeks have ranged from -5.4 to – 9.6 (with an average of -7)
- UAS7 efficacy results at 4 weeks show that those who did not achieve a min. CFB vs placebo of -9 have not succeeded in a longer-term Phase 2B study of 12 weeks or longer



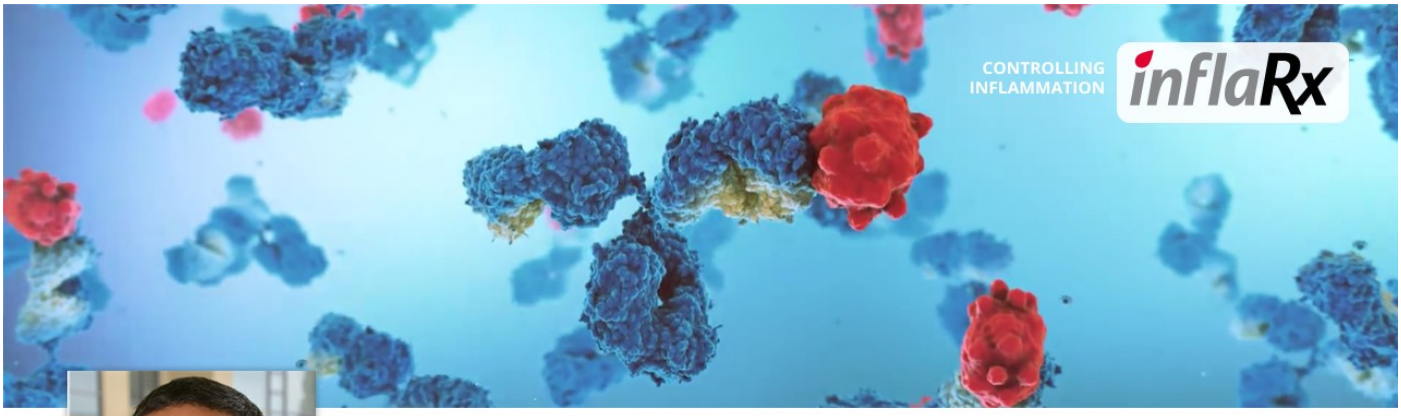
Saini S et al *J Allergy Clin Immunol.* 2011, Maurer M et al. *J Allergy Clin Immunol.* 2022, Metz M et al. *Nat Med. 2021*, Altrichter S et al. *Br J Dermatol. 2024*, Altrichter S et al. *J Allergy Clin Immunol.* 2022, AAAI 2024

A strong rationale for developing INF904 in CSU

Conclusion:

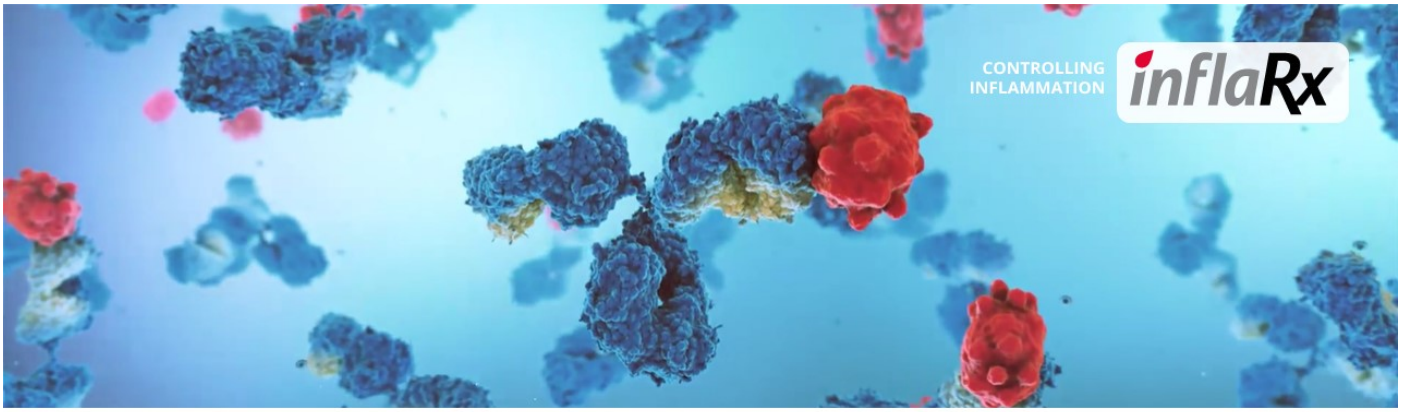
- C5aR signaling is involved in histamine release and mast cell/basophil activation in CSU
- This C5a-mediated histamine release is independent of the IgE pathway and has been suggested to play a role in both subtypes of CSU
- C5aR inhibition represents a novel mechanism of action (MoA) to address an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

Charité Berlin Team



The Role of C5aR in Hidradenitis Suppurativa

Dr. Chris Sayed, MD
Prof. of Dermatology



Disease background

Dr. Chris Sayed, MD
Prof. of Dermatology

Hidradenitis Suppurativa (HS)

HS Overview and Unmet Need

Clinical features

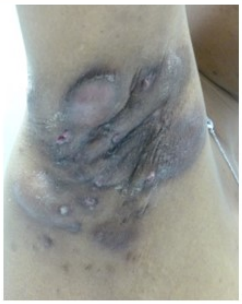
- A chronic inflammatory disease characterized by abscesses, nodules and draining tunnels (dTs) with purulent and bloody drainage in sites such as axillae, groin, buttocks, and breasts
- Flares are unpredictable and cause permanent disfigurement and disability with need for surgery

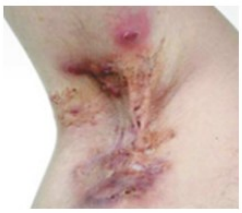
Epidemiology

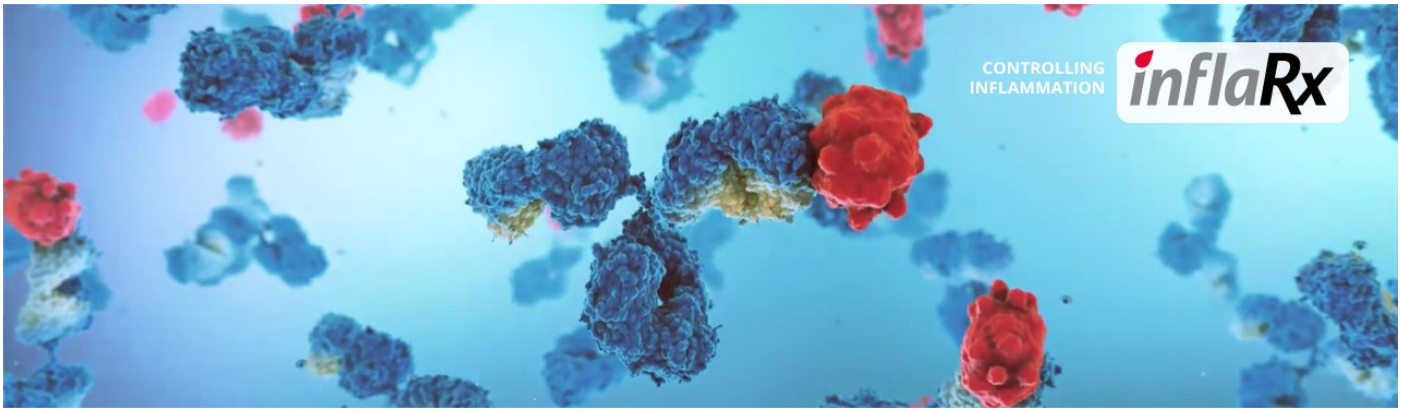
- Prevalence in the US and EU is estimated to be 0.7% - 1.2% with more than 200,000 moderate to severe patients in the US alone

Current treatment and medical need

- Current treatments include pain management, antibiotics, corticosteroids and biologics
- Response rates for most medications average less than 50%, and many patients with standard HiSCR response still have high QOL impact
- Surgery is often necessary for patients with draining tunnels despite current medical management, creating a high unmet need to better manage draining tunnels



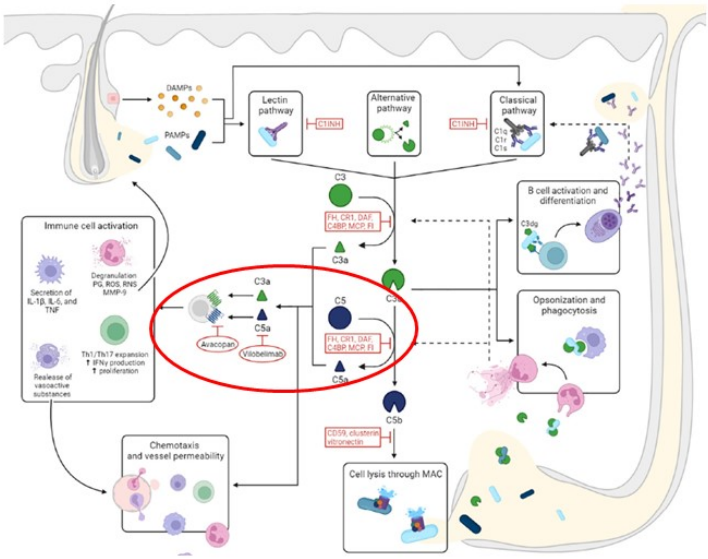




PATHOPHYSIOLOGY – what's new

Chris Sayed, MD
Prof. of Dermatology

C5a is elevated in HS and has a role in HS pathogenesis

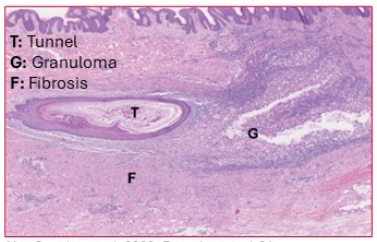


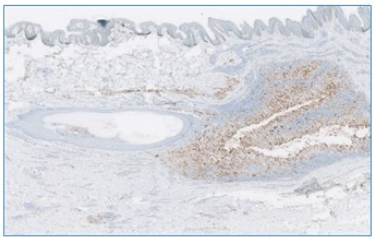
Mechanism in HS development:

- Follicular inflammation and an altered microbiome trigger complement activation including C5a/C5aR engagement
- Inflammation and dysregulated wound healing lead to chronically inflamed and draining tunnels
- This reaction can be blocked by:
  - Vilobelimab (anti-C5a antibody) and
  - INF904 (anti-C5aR inhibitor)

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

Neutrophils play a critical role in HS pathogenesis especially in draining tunnels (DTs)



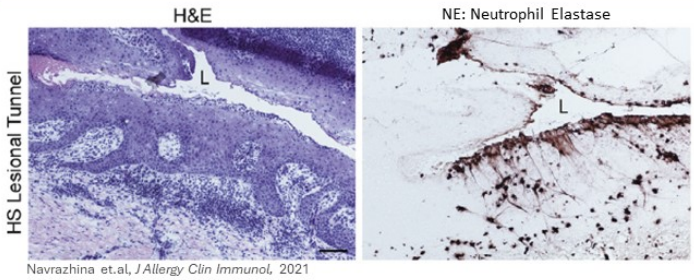


Van Straalen et al. 2022. Front Immunol 21.

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

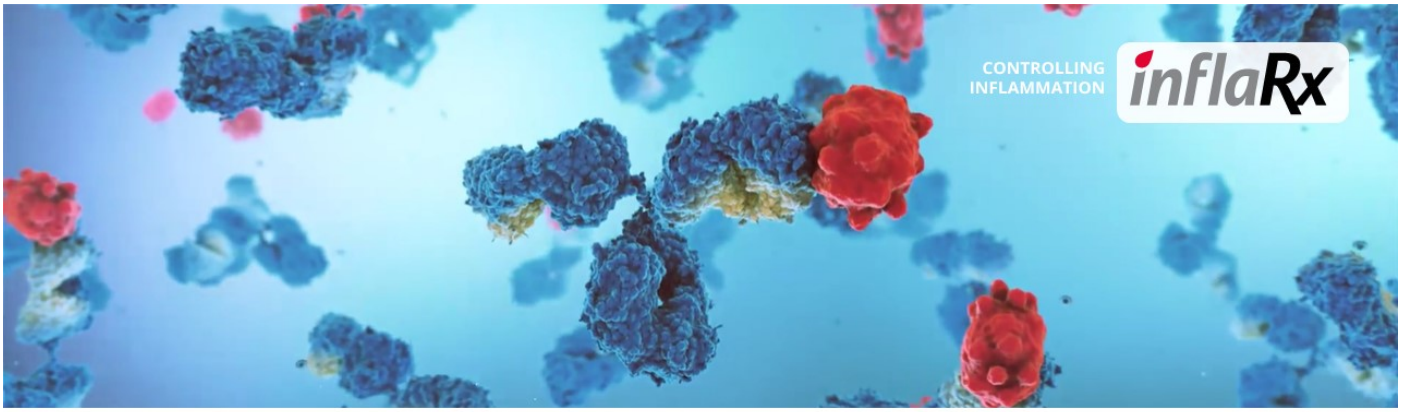
Of note: C5aR positive staining on neutrophils is found in all 3 Hurley stages

› C5a is a key Chemoattractant and a strong activator of neutrophils (which have high C5aR density) leading to Neutrophil Extracellular Traps (NET) which are believed to be a disease driver in HS



Navrazhina et.al, *J Allergy Clin Immunol,* 2021

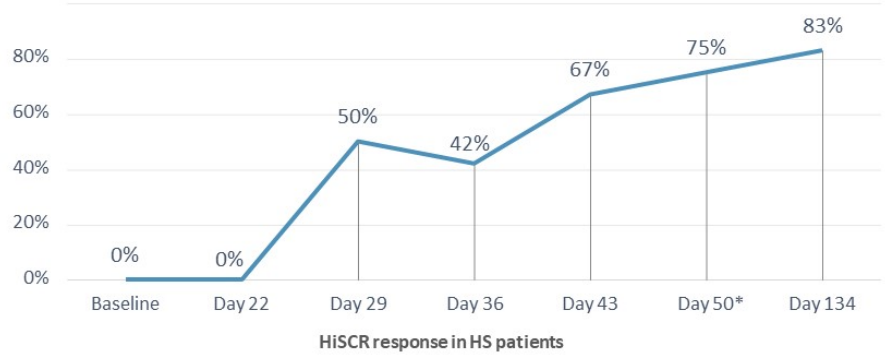
Neutrophils infiltrate inflammatory lesions in HS, including within tunnels and the surrounding tissue



What have we learned from C5a/C5aR signaling inhibition in HS

Dr. Chris Sayed, MD
Prof. of Dermatology

Phase 2a open label study vilobelimab in HS : HiSCR results



HiSCR response in HS patients

Male	8 (66.7%)
Age [y]	48 ± 15 50 (22; 69)
Hurley Stage III	12 (100%)
BMI	27.3 ± 4.9 26.6 (19.6; 34.5)
Weight [kg]	82.2 ± 14.7 78.0 (63.0; 105.0)
Duration of HS [y]	20 ± 9 20 (3; 35)
AN count	6.4 ± 2.5 6 (3; 11)
Failure to TNF-alpha blockade	9/12

DESIGN

Open label / single center / 12 patients / 1 dose group with weekly i.v. 800 mg until week 8 (plus one additional loading dose on day 4)

EFFICACY OUTCOME

75% of patients HISCR responders at week 8 and 83% at end of trial (late-stage patients who previously failed to respond to SOC incl. TNF-alpha blockade)

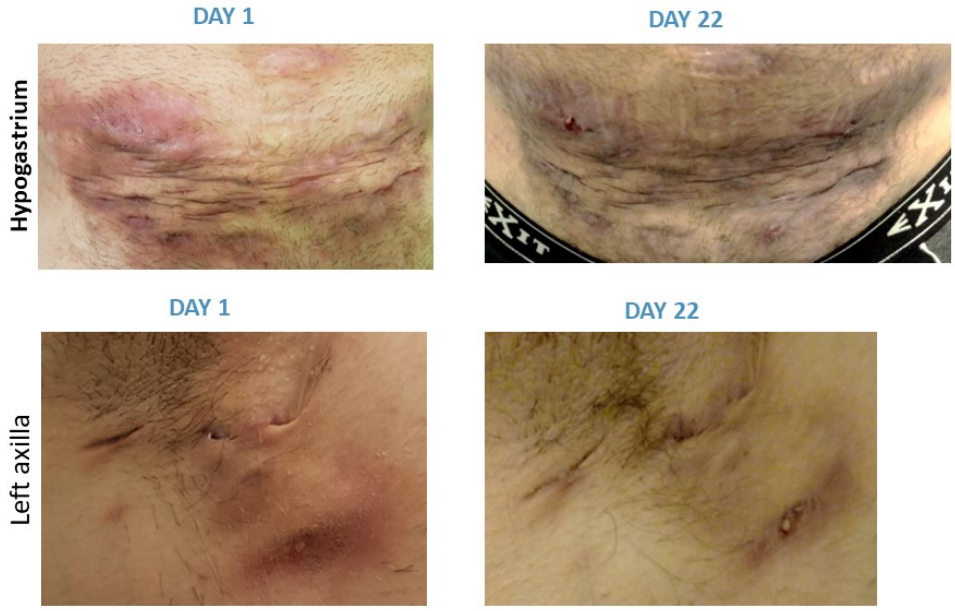
SAFETY / TOLERABILITY RESULTS

Repeated high dose i.v. administration of vilobelimab was well tolerated with a good safety profile

* Last vilobelimab administration

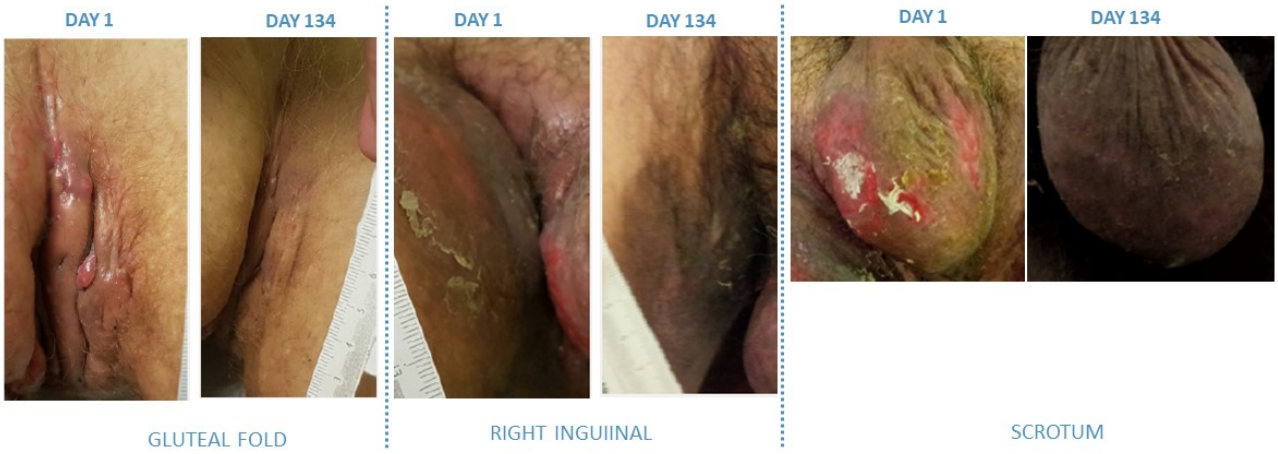
InflaRx data on file

Phase 2a open label study vilobelimab in HS: visual result examples



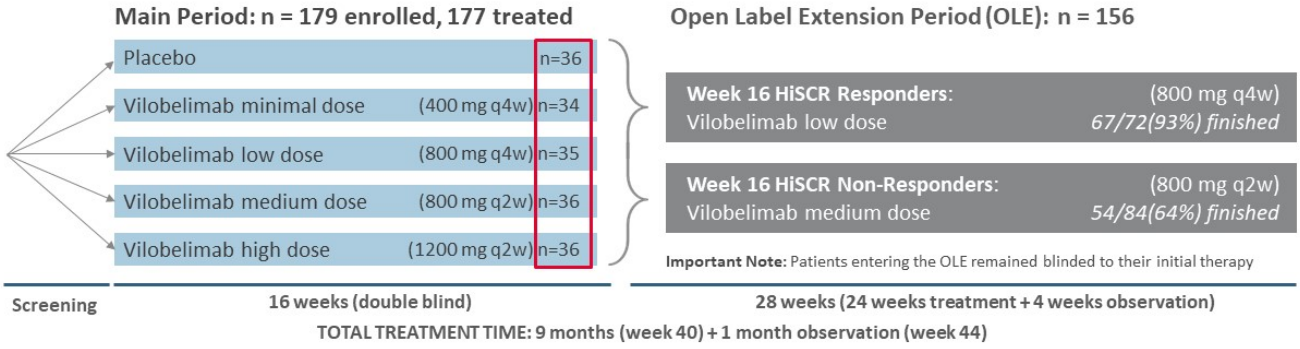
InflaRx data on file

Phase 2a open label study vilobelimab in HS: visual result examples



InflaRx data on file

Vilobelimab in HS: Phase 2b SHINE study details



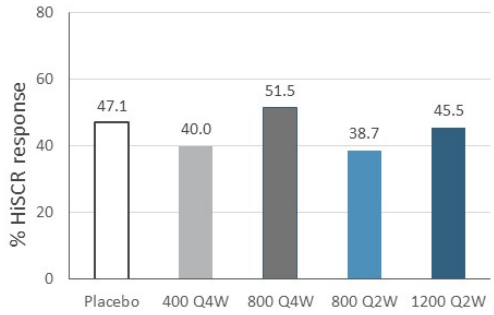
MAIN GOALS

- Test a dose-dependent effect of vilobelimab on HiSCR* response at week 16 (primary endpoint)
- Assess long-term safety of vilobelimab
- Test durability of response with lower maintenance therapy in OLE

*HiSCR response defined as: At least 50% reduction in total AN count (abscesses & inflammatory nodules) with no increase in the number of abscesses from baseline and no increase in the number of draining fistulas from baseline

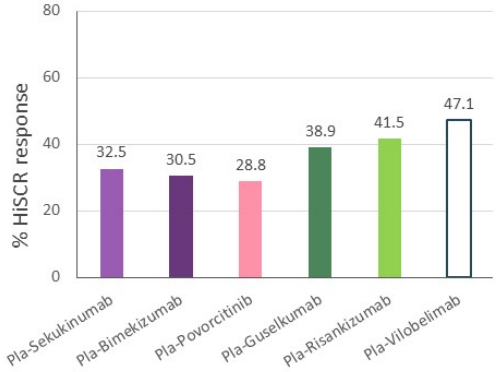
SHINE STUDY: primary endpoint HiSCR response not achieved with unusually high placebo response rate (week 16)

HiSCR 50 (week 16)



An unusually high placebo response rate

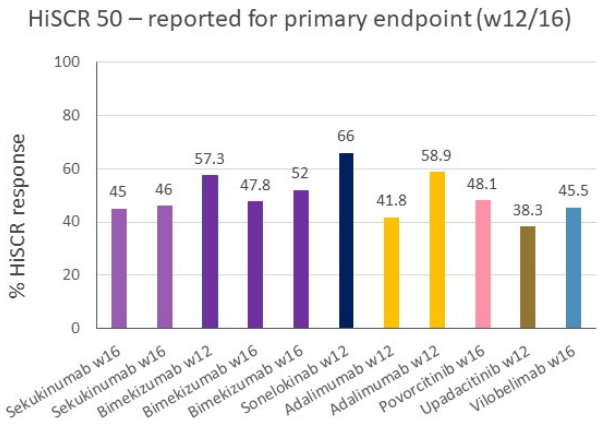
HiSCR 50 – reported placebo data week 16



- Secukinumab (anti-IL17A) 2 x phase 3 placebo n=180 -183 (mean in graph) / Kimball et al, Lancet 2023
- Bimekizumab (anti-IL17A&17F) 2 x phase 3 placebo n=72 – 74 (mean in graph) /Kimball et al, Lancet 2024
- Povorcitinib (oral JAK inhibitor) phase 2; placebo n=52 / Kirby et al, Acad Dermatol. 2024
- Guselkumab (IL-23 inhibitor) phase 2, placebo n=62 / Kimball et al, J Eur Acad Dermatol Venereol. 2023
- Risankizumab (IL-23 inhibitor) phase 2, placebo n=80 / Kimball et al, Dermatol Ther (Heidelb). 2023
- Vilobelimab (anti-C5a) phase 2, placebo n=36

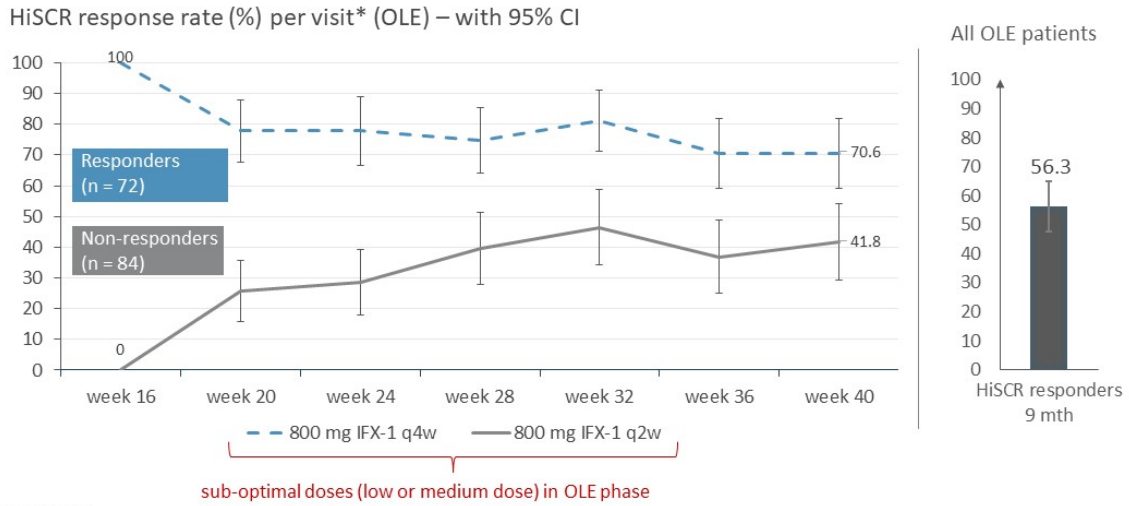
InflaRx data on file

HiSCR50 comparison from reported positive studies (week 16 or week 12) to vilobelimab outcome



Secukinumab (anti-IL17A) phase 3 Sunshine n=180 / Kimball et al, Lancet 2023
Secukinumab (anti-IL17A) phase 3 Sunrise n=180 / Kimball et al, Lancet 2023
Bimekizumab (anti-IL17A&17F) phase 2 n=44 / Glatt et al, JAMA Dermatol. 2021
Bimekizumab (anti-IL17A&17F) q2w phase 3 n=289 / Kimball et al, Lancet 2024
Bimekizumab (anti-IL17A&17F) q2w phase 3 n=291 / Kimball et al, Lancet 2024
Sonelokimab 120 (anti-IL17A&17F) n=66 phase 2 / Kimball et al. EADV 2023
Adalimumab (anti-TNFα) Phase 3 Pioneer 1 n= 153 / Kimball et al, N Engl J Med. 2016
Adalimumab (anti-TNF α) Phase 3 Pioneer 2 n= 163 / Kimball et al, N Engl J Med. 2016
Povorcitinib (oral JAK inhibitor) phase 2; n=52 / Kirby et al, Acad Dermatol. 2024
Upadacitinib (oral JAK inhibitor) phase 2; n=47 / Kimball et al; JAAD 89(3), Supplement, AB42
Vilobelimab (anti-C5a) 1200 mg n=36 phase 2

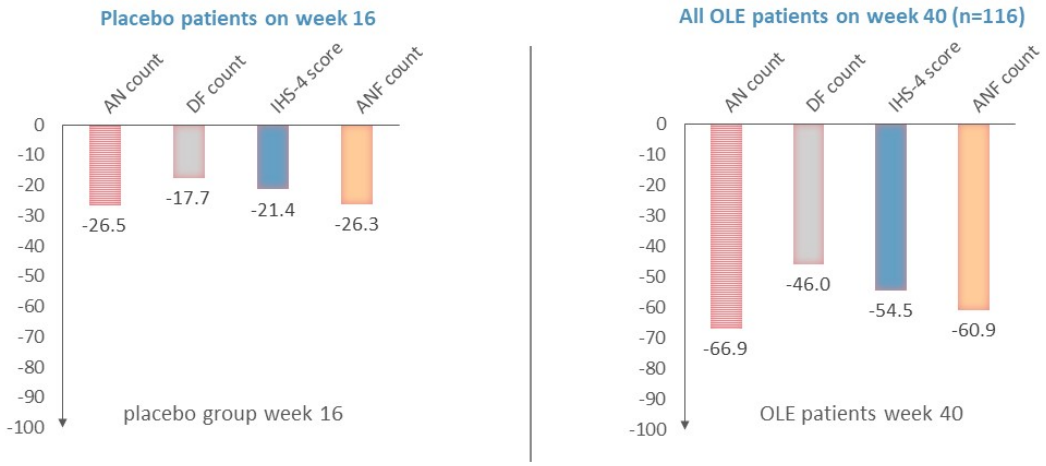
HiSCR 50% response in the OLE suggests that non-responders gain response (42%) on vilo and that 71% keep response on low dose vilo maintenance treatment



* Full analysis set
InflaRx data on file

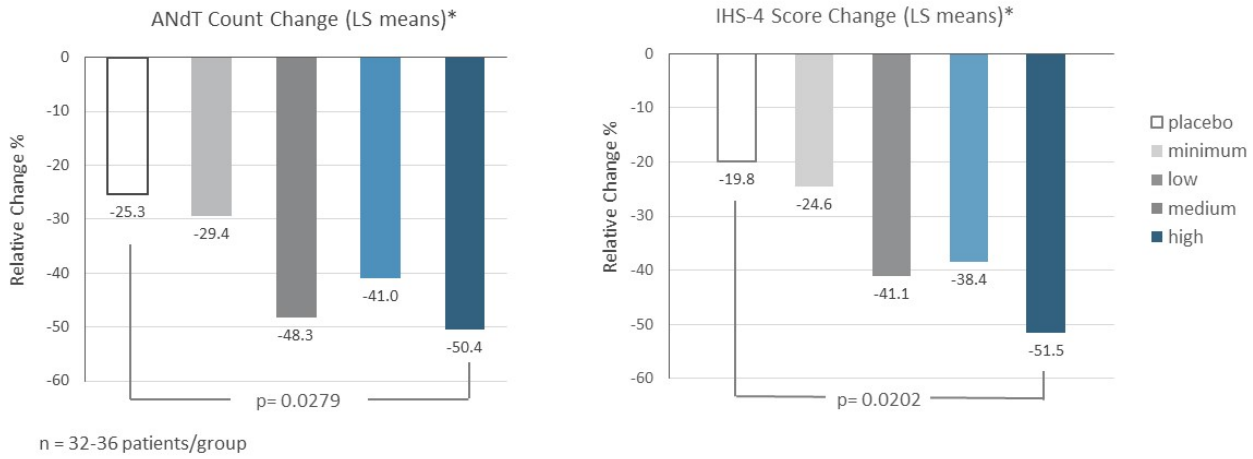
SHINE STUDY: Patients who completed the OLE phase (w 40) showed substantial inflammatory lesion reductions when compared to observed placebo count reductions from the double-blind main period (w 16)

Relative reduction (% mean) of counts / scores compared to respective baseline (Day1)*



* Full analysis set (unadjusted)
InflaRx data on file

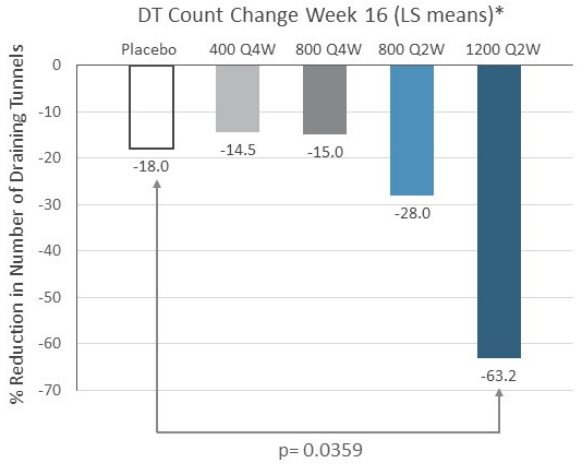
SHINE STUDY: outcome of ANdT count and IHS4 score (week 16)

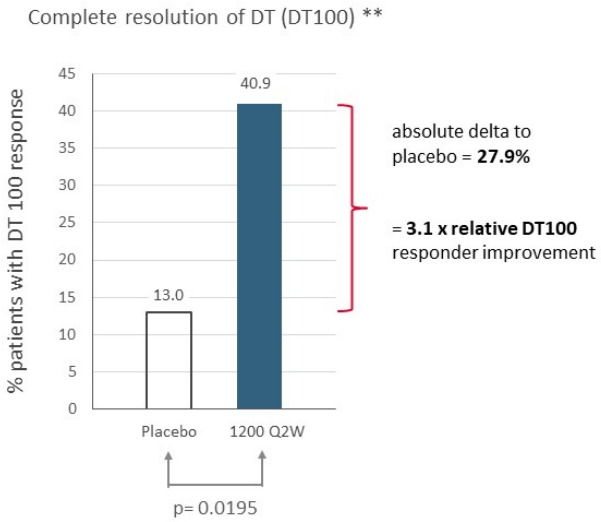


n = 32-36 patients/group

* Full analysis set baseline adjusted
InflaRx data on file

SHINE STUDY: evidence of inflammatory lesion reduction under C5a inhibition (w 16)

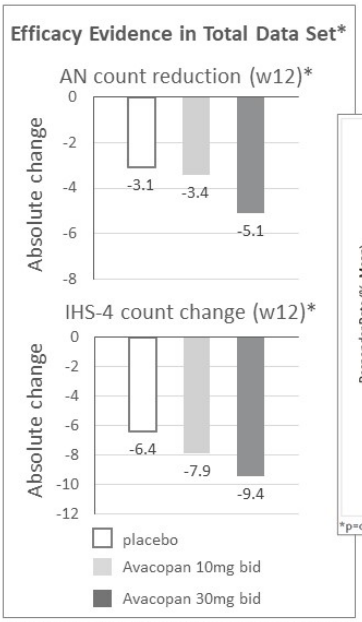


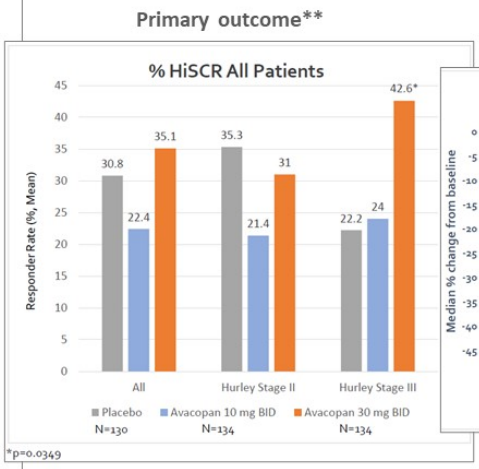


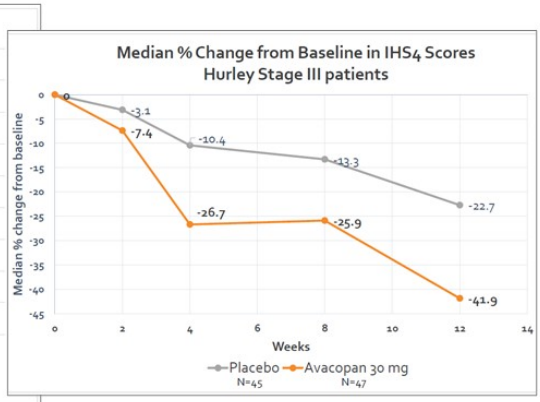
* Full analysis set baseline adjusted, LS Means
InflaRx data on file

** Patients with at least 1 DT at baseline, placebo n=23, vilobelimab n=22

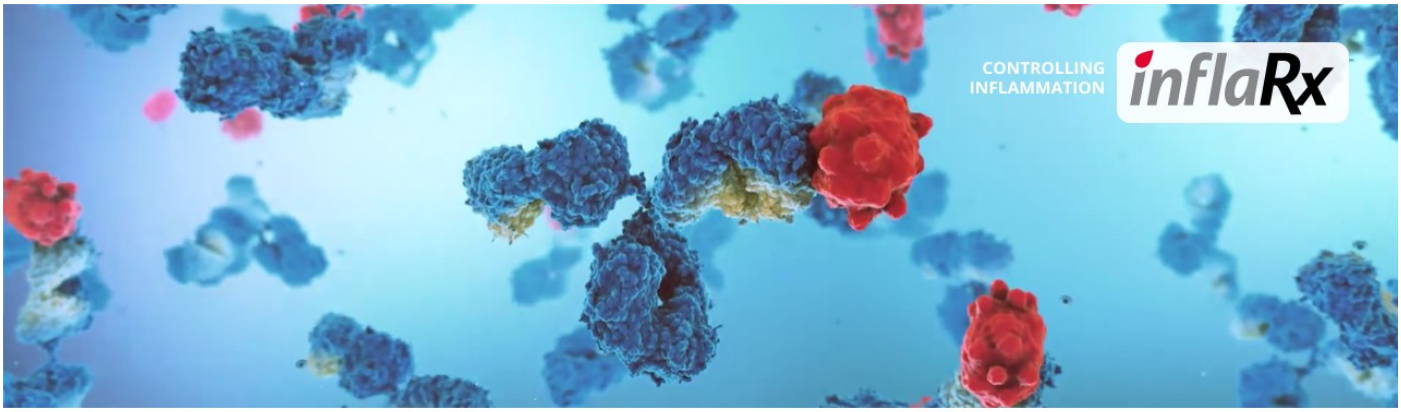
Evidence for efficacy of C5aR inhibition in HS: Avacopan data







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



What have we learned from C5a/C5aR inhibition in HS

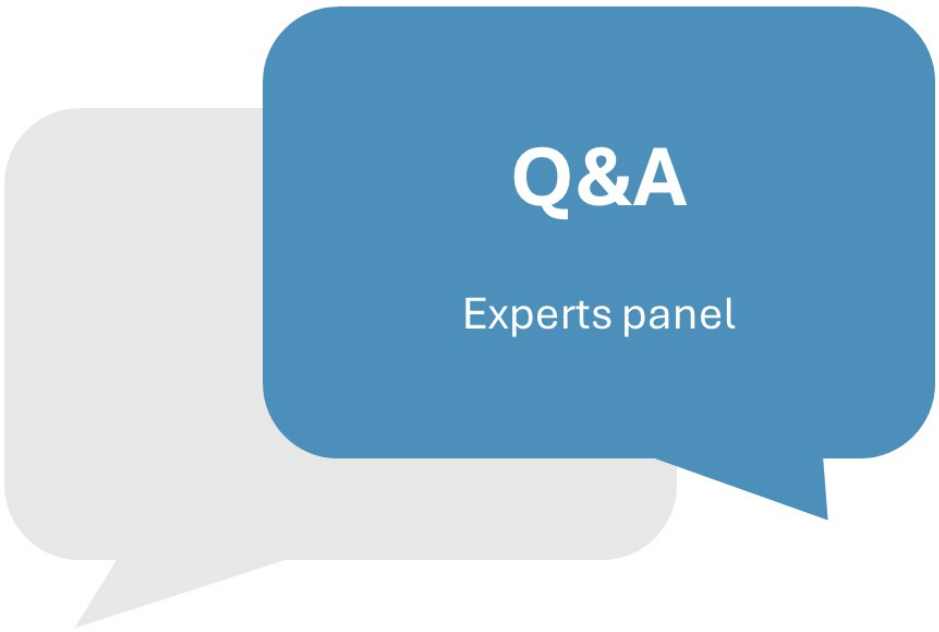
Dr. Chris Sayed, MD
Prof. of Dermatology

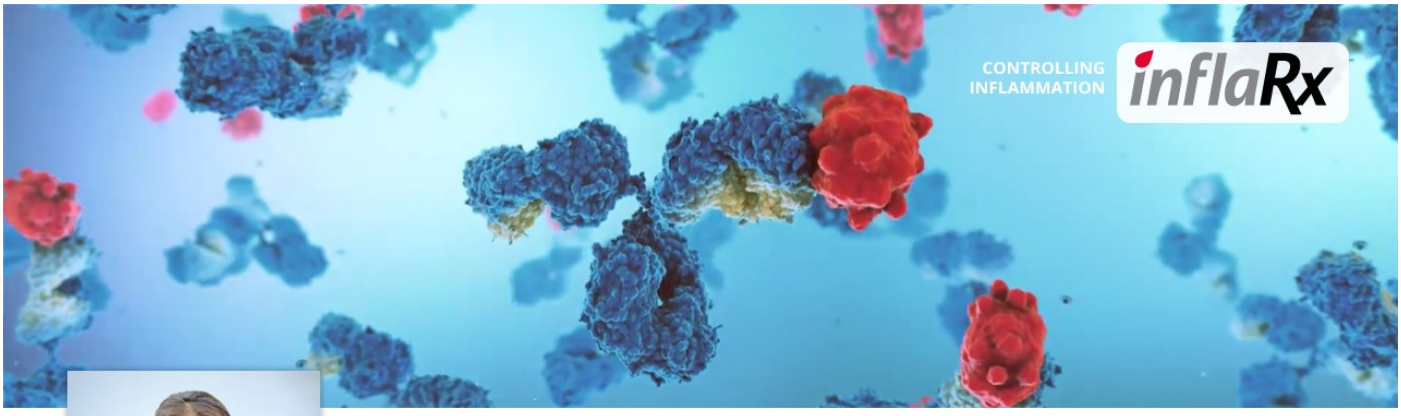
Summary and rationale for developing INF904 as potent oral C5aR inhibitor in HS

Learnings:

- Both C5a as well as C5aR signaling inhibition have resulted in clear signals of efficacy (reduction of inflammatory lesions + established scores) in moderate to severe HS patients
- Both treatment attempts (vilobelimab and avacopan) were likely underdosed. Of note: avacopan has been reported to have a long accumulation pattern, reaching steady state only at week 13*
- **INF904 is ideally positioned as an oral C5aR inhibitor with optimized PK / PD profile to address C5aR signaling in HS**

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


Q&A
Experts panel



Next clinical development steps for INF904

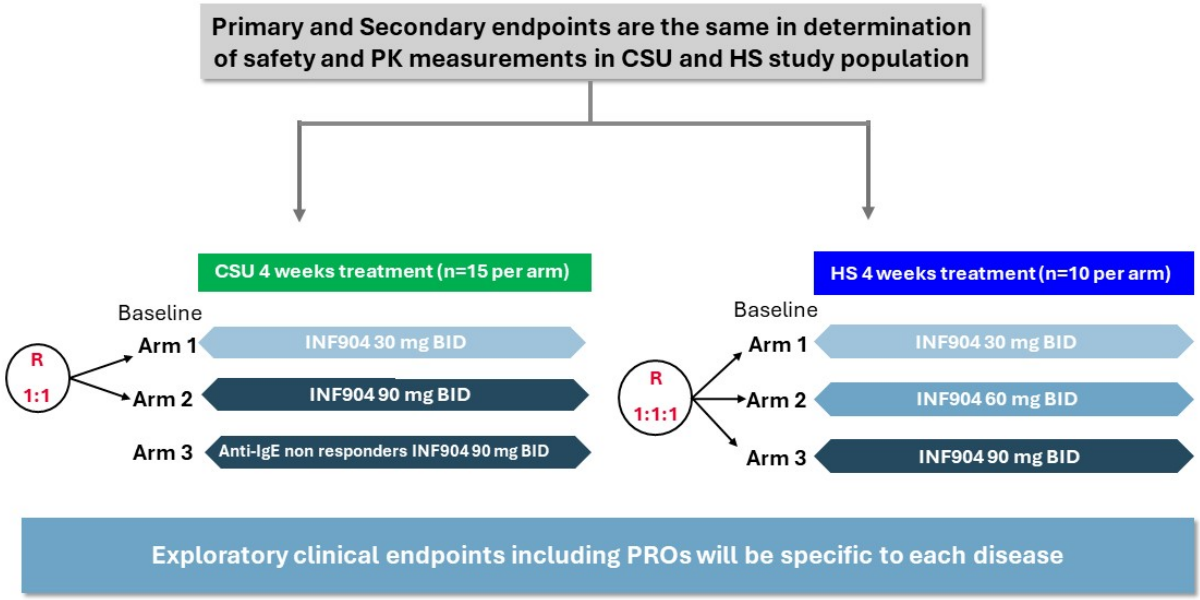
Dr. Camilla Chong, MD
CMO

Phase 2a open label basket study in CSU and HS

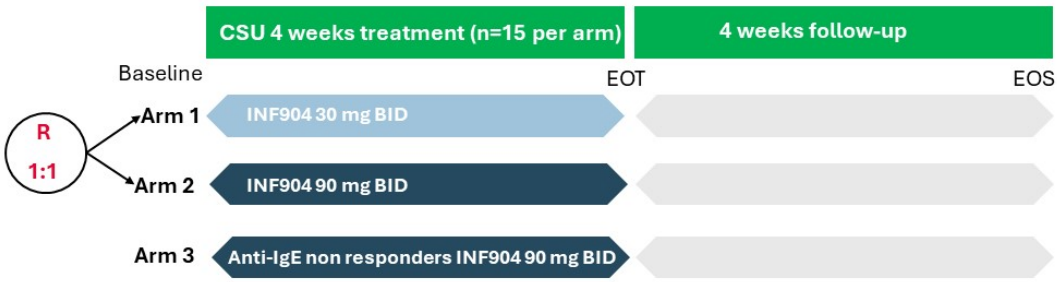
Clinical development strategy

- To determine the appropriate dosing regimen in CSU & HS with safety, PK and efficacy measurements in order to progress to a larger placebo-controlled Phase 2b clinical program.
- Basket study approach has been agreed with FDA for a single IND submission.
- FPI scheduled for Q4 2024 with preliminary results expected in Summer 2025.

Phase 2a open label basket study concept



Study design for CSU



Exploratory Endpoints

- Change of the weekly Urticaria Activity Score 7 (UAS7) , Hives Severity Score (HSS7) and Itch Severity Score (ISS7)
- Biomarkers : Tryptase, IgE, IgG, anti-TPO

Patient Reported Outcome (PRO) Endpoints:

- Urticaria Control Test (UCT7)
- Angioedema Activity Score (AAS 7)
- Chronic Urticaria Quality of Life Questionnaire (CU-Q2oL)

Primary Endpoint:

- Frequency, severity, and relatedness of treatment-emergent adverse events (TEAEs), serious adverse events (SAEs) using MedDRA classification.

Secondary Endpoints:

- Plasma PK parameters of INF904 will be calculated as appropriate from observed data for Cmax, Cmin, T max and systemic exposure $AUC_{0\text{-}24}$, $AUC_{0\text{-last}}$

Main inclusion criteria for CSU

Patients diagnosed with moderate to severe CSU and inadequately controlled by second generation H1-antihistamines at the time of randomization as defined in the following:

1. The presence of itch and hives for ≥6 consecutive weeks prior to screening in spite of use of non-sedating H1-antihistamines according to local treatment guidelines during this time period
2. UAS7 score (range 0-42) ≥16 and UCT7 <12 during 7 days prior to randomization (Day 1)
3. Arm 3: non-responder to Anti-IgE therapy as defined by previous treatment with at least 300 mg (q4w) anti-IgE therapy for at least 4 months (minimum of 4 injections) and who had an inadequate response resulting in anti-IgE therapy discontinuation, as confirmed by investigator assessment

CSU diagnosis for ≥ 6 months

Study design for HS

Exploratory Endpoints (all change from baseline, CFB):

- Total Abscess (A), Inflammatory Nodule (N) and draining tunnels (dT) Count (ANdT, AN and separate counts for each lesion), HS lesions related scores (HiSCR –various, modified-HiSCR, IHS-4)
- Clinician's Global Impression of Change (CGI-C)

Patient Reported Outcome (PRO) Endpoints (all CFB):

- Global Impression of Change in General Quality of Life related to HS (PGI-C QoL)
- Global Assessment of Skin Pain (NRS)
- Dermatology Life Quality Index (DLQI)

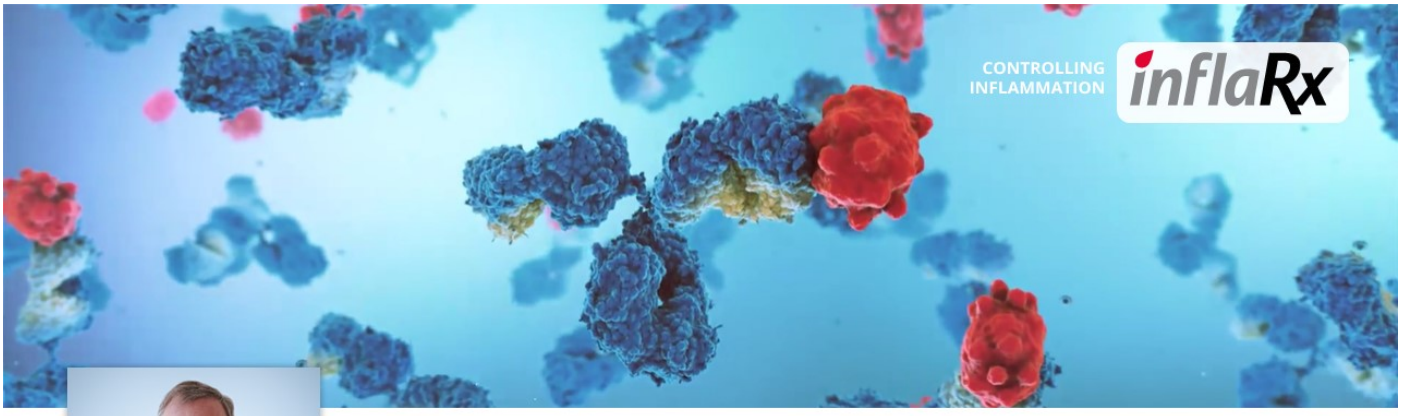
Primary and Secondary Endpoints on Safety and PK are similar to CSU

Main inclusion criteria for HS

Moderate or severe hidradenitis suppurativa (with Hurley Stage II or III), and an Abscess and Nodule (AN) count ≥ 5. Inflammatory lesions should affect at least 2 distinct anatomic areas

Diagnosis of HS based on clinical history and physical examination for at least 6 months prior to the Baseline visit; diagnosis must be verifiable through medical notes and documentation

Patients must have had an inadequate response to at least a 3-month (90 days) trial of oral antibiotics for treatment of HS (or demonstrated intolerance to or have a contraindication to oral antibiotics for treatment of their HS)



Commercial Opportunity

Dr. Thomas Taapken
CFO

Focus on immuno-dermatology – attractive and rapidly growing market opportunity

The immuno-dermatology market is witnessing robust growth, driven by the increased incidence of disease, emerging targeted therapeutics and improved diagnostic capabilities

Market growth rates are attracting several pharmaceutical companies to this area, with a noticeable increased focus on immuno-dermatology by companies active in the overall I&I field

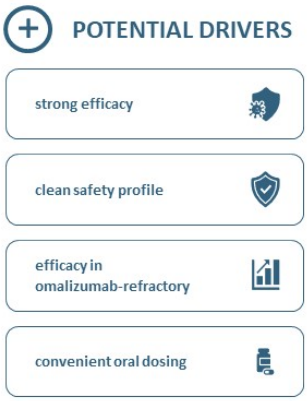
With the right product profile, there is the potential to address multiple multi-billion-dollar market opportunities in several diseases in this category

Strong commercial potential based on differentiated profile of INF904

- Oral availability provides for **ease of administration** and patient acceptance
- Favorable drug metabolism, PK and toxicology profile positions INF904 as a potential **strong alternative** to other drug classes
- INF904 could address areas of **high unmet medical need** given its strong emerging profile
  - CSU: mechanism of action that suggests impact on IgE-dependent and -independent disease phenotypes
  - CSU and HS: Maintenance / durability of response
  - HS: treatment of draining disease / draining tunnels in HS
  - CSU and HS: safe mechanism of action not associated with known serious side effects
- **MoA is highly relevant** in several immuno-dermatology indications, including most neutrophilic dermatoses
  - Unique mechanism could provide a strong alternative to biologic therapies and may offer advantages to currently developed oral approaches
- Differentiation also applies to other **inflammation & immunology** disease areas beyond immuno-dermatology

CSU market dynamic and INF904 commercial opportunity

- Overall CSU US market size* estimated to be US$ 1.1 Bn in 2024 and growing at 10% CAGR to US$ 3.1 Bn by 2035, mainly driven by new therapies for severe disease entering the market
  - incidence approx. 400k patients p.a. (US)
  - anti-histamine refractory; approx. 85k (US, eligible for treatment with biologics and other "advanced therapies") – numbers estimated for 2035
- Overall maximum market potential for INF904 in CSU **could exceed US$ 1 Bn p.a.** – based on primary market research conducted for IFRX, including physician interviews and additional research



*IFRX proprietary market research, Clarivate

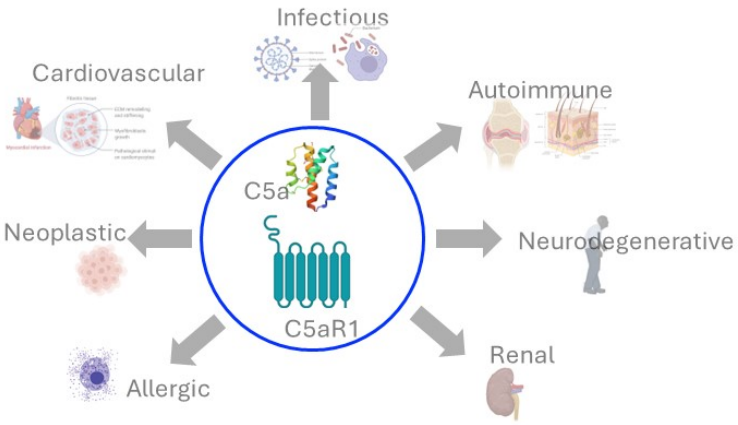
HS market dynamic and INF904 commercial opportunity

- Overall HS US market size* estimated to be US$ 1.3 Bn in 2024 and growing at 15% CAGR to US$ 6 Bn by 2035, mainly driven by new therapies for severe disease entering the market
  - incidence approx. 320k patients p.a. (US)
  - 2nd line treatment options benefiting approx. 115k patients p.a. (US, biologics and other advanced therapies) – numbers estimated for 2035
- Overall maximum market potential for INF904 in HS **could exceed US$ 1.5 Bn p.a.** – based on primary market research conducted for IFRX, including physician interviews and additional research

*IFRX proprietary market research, Clarivate

Role of C5aR inhibition spans well beyond immuno-dermatology

Possible disease areas in which C5aR has relevance



- "*Pipeline in a drug*" potential of INF904
- C5aR inhibition could be developed broadly in different I&I indications
- Initial focus on HS and CSU but other indications (e.g., renal diseases) are likely areas for development
- This opportunity could provide INF904 with multiple multi-billion-dollar market opportunities
- Partnering could provide upside potential

Conclusions and next steps

InflaRx has sufficient resources to complete Phase 2a basket study in HS and CSU

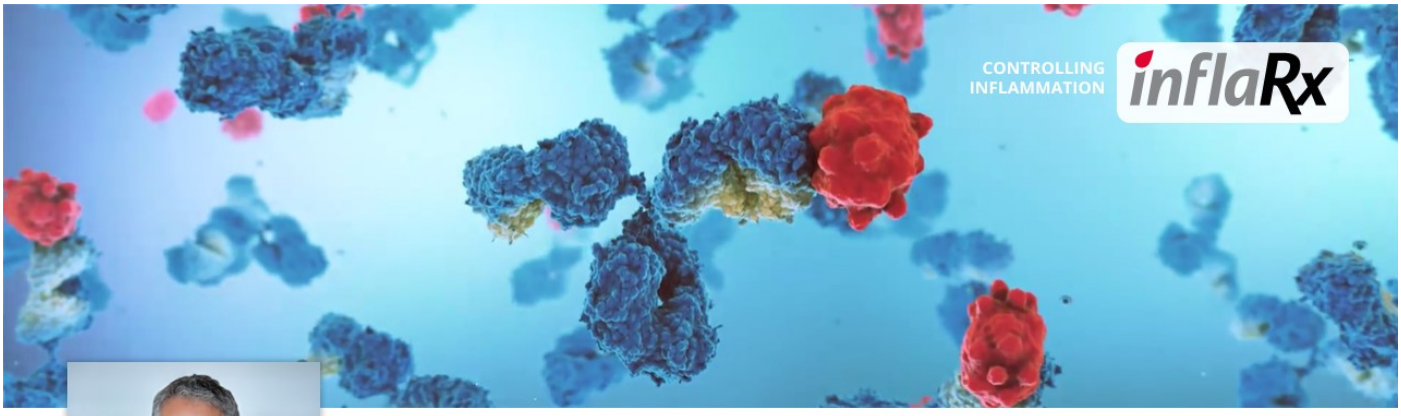
- Cash position as of Q1 2024 was EUR 85.8M (~$93M) – a runway into 2026

Expected start of Phase 2a open label study by year-end 2024, with data in summer 2025

- Goal is to generate additional safety and PK data, as well as show meaningful improvements in relevant disease activity measures in CSU and HS

Open label design may allow for interim read-out and to solidify planning for Phase 2b trials currently planned for 2025

- May accelerate development timelines and/or partnering discussions



Summary

Prof. Dr. Niels C. Riedemann
CEO

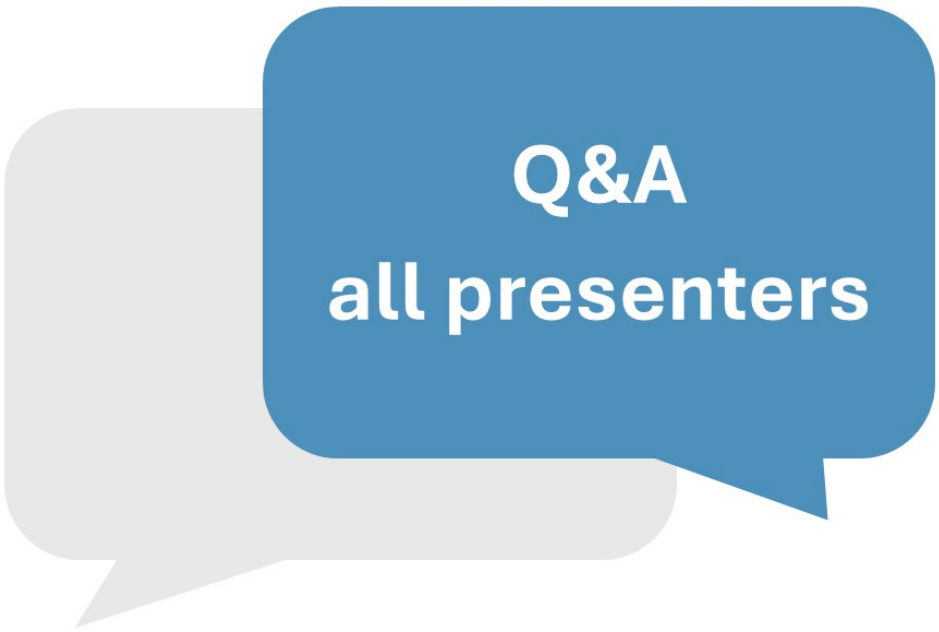
Summary and conclusions from today's presentation

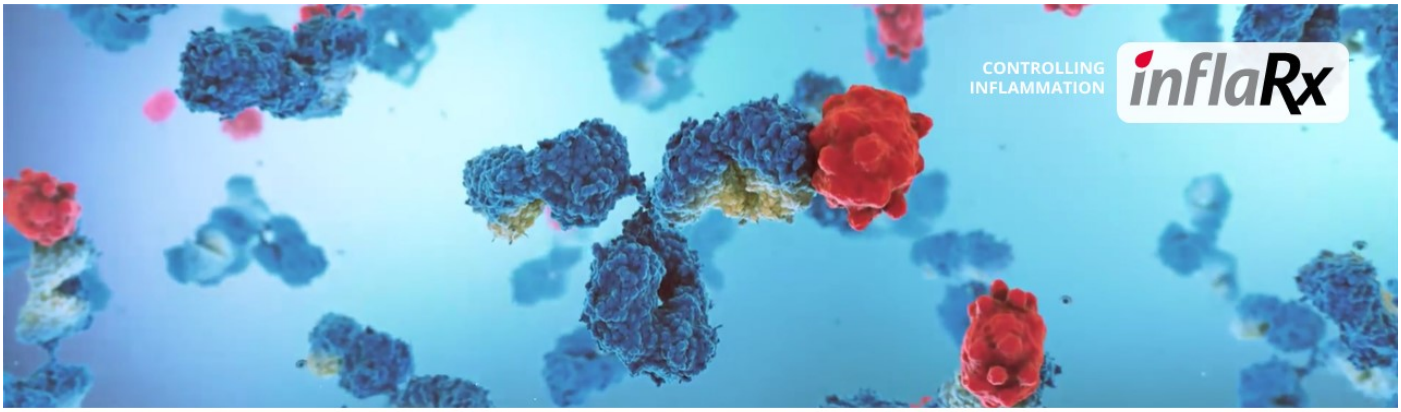
The new oral C5aR inhibitor INF904 has best-in-class potential and has been developed to become a pipeline-in-a-drug in the immuno-dermatology space and broader I&I space

Supported by cutting-edge science, there is significant market potential for INF904 in CSU, HS and beyond

Special thanks from the entire InflaRx team to our experts on the call and supporters:
Prof. Jörg Köhl, Prof. Marcus Maurer and Prof. Chris Sayed!

Thank you for your attention!


Q&A
all presenters



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