UNITED STATES SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 6-K

REPORT OF FOREIGN PRIVATE ISSUER PURSUANT TO RULE 13A-16 OR 15D-16 UNDER THE SECURITIES EXCHANGE ACT OF 1934

For the month of March 2024

Commission File Number: 001-38283

InflaRx N.V.

(Translation of registrant's name into English)

Winzerlaer Str. 2 07745 Jena, Germany (Address of principal executive office)

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 \square Form 40-F \square

EXPLANATORY NOTE

As previously announced, on March 21, 2024, InflaRx N.V. (the "Company") issued a press release titled "InflaRx Reports Full Year 2023 Financial and Operating Results." In connection with such announcement, on March 21, 2024, the Company hosted a conference call and presented its corporate presentation on immuno-dermatology where the Company provided details and developments on its oral C5aR inhibitor INF904 and on the development of vilobelimab in pyoderma gangrenosum. A copy of the corporate presentation is attached hereto as Exhibit 99.1 and is incorporated by reference herein.

EXHIBIT INDEX

Exhibit No.	Description		
<u>99.1</u>	Corporate Presentation, dated March 21, 2024		
	3		

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.

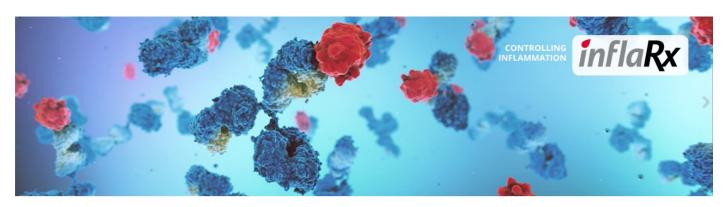
INFLARX N.V.

Date: March 21, 2024 By: /s/ Niels Riedemann

Name: Niels Riedemann
Title: Chief Executive Officer

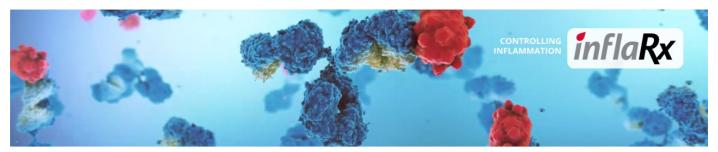
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Exhibit 99.1



CORPORATE PRESENTATION

MARCH 2024

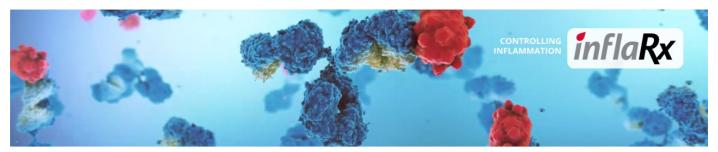


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This presentation has been prepared by InflaRx N.V. ("InflaRx" or the "Company"). This presentation is made for informational purposes only and does not constitute an offer to sell or a solicitation of an offer to buy securities. This presentation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

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 are grading our intentions, beliefs, projections, outlook, analyses and current expectations correnting, among other things, the receptiveness of GOHIBIC (vilobelimab) as a streatment 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 trails for vilobelimab and any other product candidates, including INPO and whether such clinical results seen in previously conducted pre-clinical studies and clinical trials; the timing, progress and results of the rials and over available, the costs of such trials and our product candidates in clinical trials of our product candidates in clinical trials and our subjects of the timination of previously conducted pre-clinical studies and clinical trials and our subjects of the subjects of the program subjects of the program subjects of th



IMPORTANT NOTICE AND DISCLAIMER

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 werified, 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.

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").

Inflack (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. Inflack's lead product candidate, vilobelimab, is a novel, intravenously delivered, first-in-class, anti-C5a monoclonal antibody that selections by binds to free C5s and has demonstrated disease-modifying clinical activity and tolerability in multiple clinical studies in different indications. Inflack 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.inflacx.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]. L&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

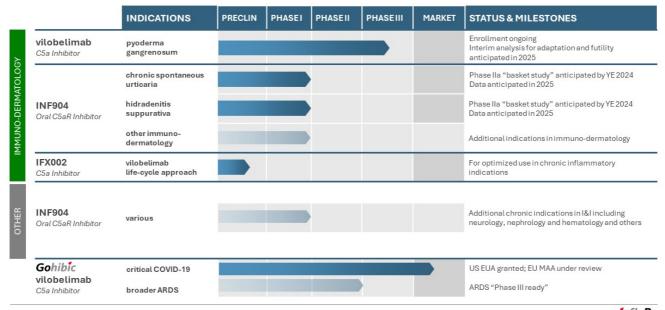
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Focus INF904 on Immuno-Dermatology: I&I Pipeline-in-a-Drug Potential

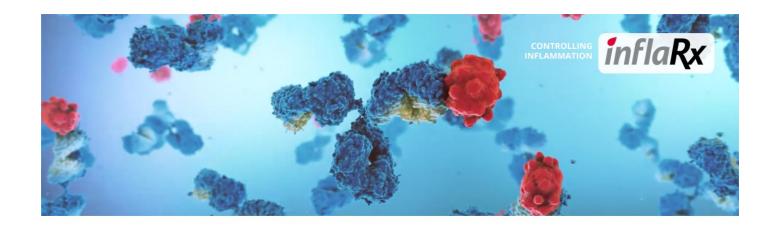
· Chronic spontaneous urticaria (CSU) Key initial development focus Immuno-· Hidradenitis suppurativa (HS) and area to demonstrate dermatology potential of INF904 others • Chronic inflammatory demyelinating polyneuropathy (CIDP) Neurology Dermatomyositis Potential for future development or development in collaboration with a partner • Anti-C3 glomerulopathy (C3G) Nephrology & • Atypical haemolytic uraemia syndrome (aHUS) Hematology • Immunoglobulin A nephropathy (IgAN) • ANCA-associated vasculitis (AAV)

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Late-Stage Pipeline Targets Multiple Sizable Markets



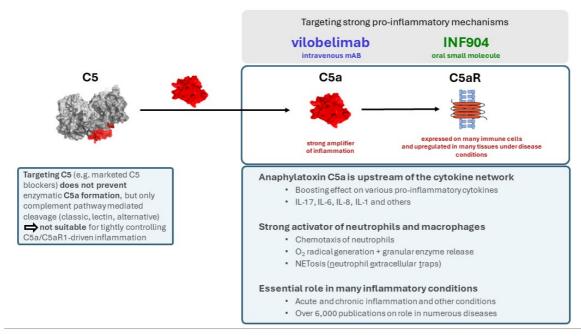
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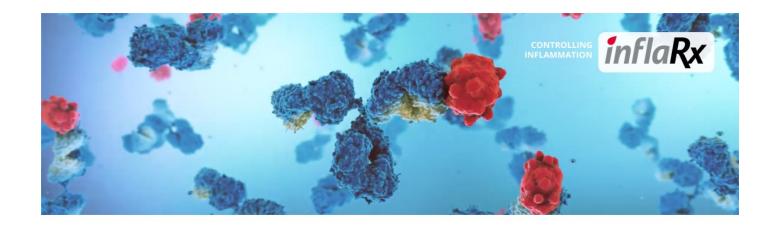
C5a/C5aR: A Strategic Position in the Inflammatory Cascade

Vilobelimab [C5a monoclonal antibody] INF904 [oral C5aR inhibitor]

C5a/C5aR are Validated Targets Promoting Inflammation



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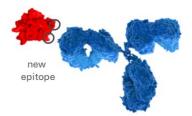


Vilobelimab for Ulcerative Pyoderma Gangrenosum (PG)

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

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PG: An Autoimmune Condition With High Unmet Need

PG Overview and Unmet Need

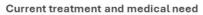


Clinical features

- · 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



- 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



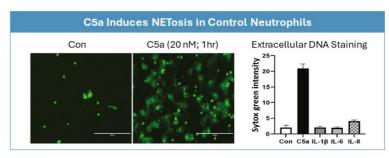
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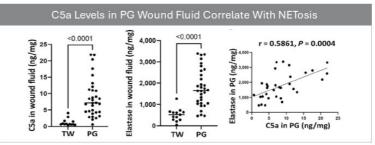
Strong rationale for treatment with vilobelimab:

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





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%)

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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 Q2Wup 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 40 mg 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

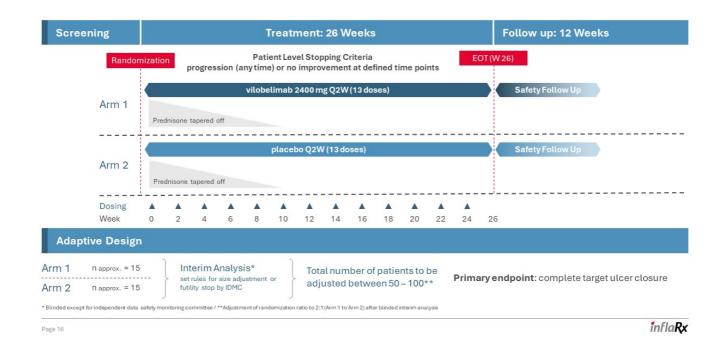
Target Ulcer Reappeared

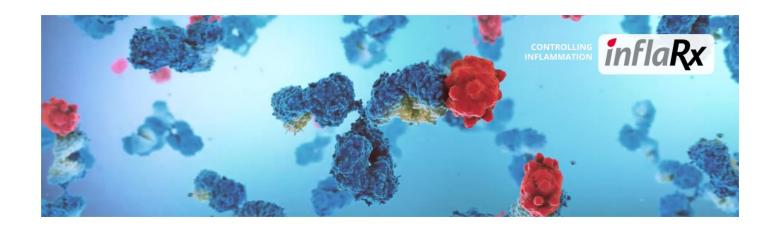
- MH: PG since 2019, Hypertension since 1998
- Previous PG medication: Methylprednisolone only in Jun 2019, Dapsone Jun 2019 Aug 2020, Cyclosporine Oct 2019 Aug 2020 -> ulcer healed and reappeared after discontinuation of immunosuppressants
- Cohort 2: 1600 mg Q2W, individual up-titration to 2400 mg 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

InflaRx

PG Phase III Study Design: Interim Analysis Expected in 2025





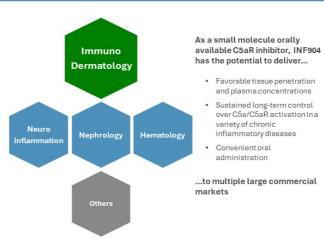
INF904: An Oral Highly Selective C5aR Inhibitor With Best-in-Class Potential

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)
 - o 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
 - o 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

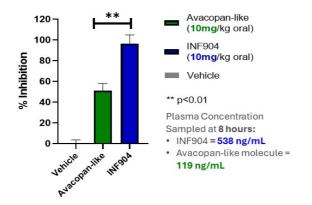


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INF904: Oral C5aR Antagonist With Best-in-Class Potential

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.

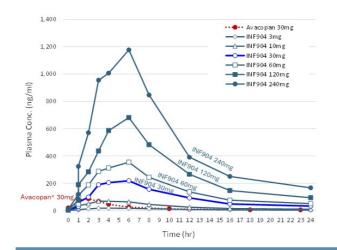
Experiment: Challenge of rodents with C5a leads to neutrophil activation and consequent adherence (sticking) of neutrophils to the endothelial cell wall of vessels = mimicking a neutropenia (vehicle). This effect can be completely inhibited when C5aR activation is blocked.

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*

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

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)

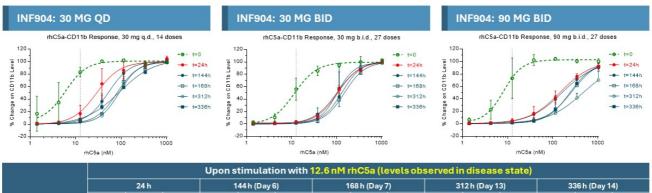
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Source: <u>Bekker et al.</u> 2016, PLoS One; 11(10): e0164646

*Please note: Avacopan data taken from <u>Bekker et al.</u> 2016, PLoS One; 11(10): e0164846 are superimposed in graph for orientation. Avacopan was not included as a comparator in INF904 Phase I study.

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

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



30BID 90BID 30BID 30BID 90BID 30BID 90BID 300D Blockade (%) 94 90 93 95 94 95 97 97 96 92 97 95 97 80 90 EC₅₀ (nM) 35.6 106.2 145.6 52.4 134.7 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)**

avacopaniesutts will cirriave shown approximate

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

Investing Into INF904 Development

Phase IIa Expected to Begin by EOY 2024

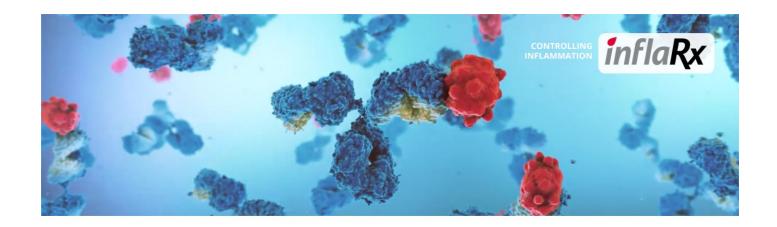
Initial Phase IIa – demonstrating pipeline-in-a-drug potential of INF904

- 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 Ila data anticipated in 2025
- Larger and longer-term Phase IIb study expected to begin in 2025

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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-lgE 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**

 $^{\star}\,\mathsf{Metz}\,\mathsf{et}\,\mathsf{al},\mathsf{Clin}\,\mathsf{Rev}\,\mathsf{Allergy}\,\mathsf{Immunol.}\,2020;59(1);38-45.\,^{\star\star}\,\mathsf{GlobalData}\,\mathsf{and}\,\mathsf{Leerink}\,\mathsf{analyst}\,\mathsf{report}$

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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

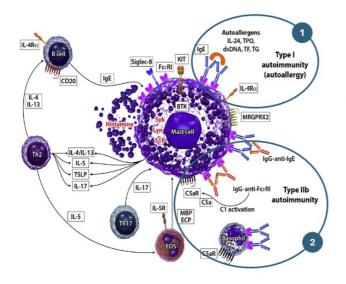
- Therapies such as 2nd-generation antihistamines are not effective in a significant number of patients
- Options such as anti Ig-E therapy and immunosuppressants also do not adequately serve the CSU population





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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 Ilb autoimmunity (IgG mediated; ~30% of CSU)
 - C5a is activated by the binding of IgG-anti-FccRI or IgG-anti-IgE to FccRI on mast cells and basophils

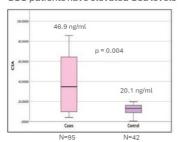
C5aR signaling is suggested to be involved in both, type I and type IIb endotypes

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

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

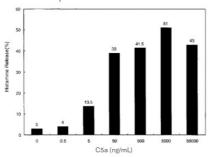
CSU patients have elevated C5a levels



CSU patients show evidence of complement activation in the skin



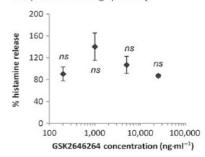
Bhatia et al. 2024 Asia Pacific Allergy 14; Aghdam et al. 2021 Clin Transl Allergy. 11 C5a induces histamine release from basophils in a dose-dependent manner



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 $\,$

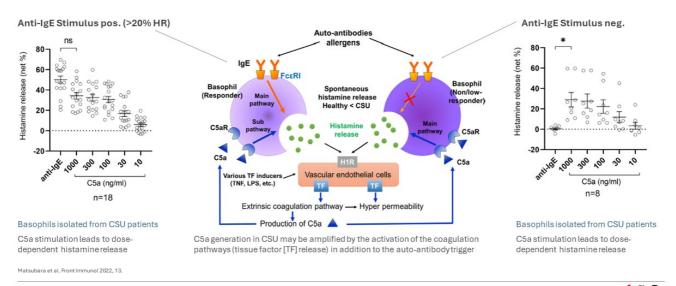
C5a stimulation of histamine releases is not affected by IgE pathway/ SYK inhibitor GSK2646264

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



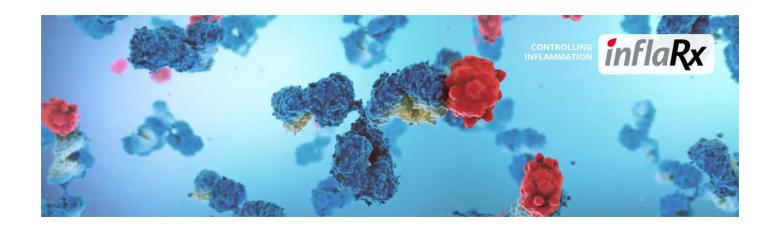
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INF904 Development in CSU

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

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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
- 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 $including induction of NETosis-mechanisms \ which have been suggested to be involved in HS progression and specifically in HS lesion$
 - 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)

** Global Data and Leerink / Guggenheim analystreports

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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
- $\bullet \quad \text{Associated with severe bacterial infections, tremendous QoL impairment and functional disability} \\$

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

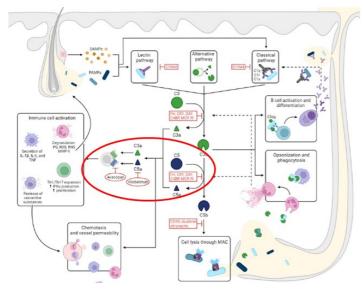
- · Current treatments including pain management, antibiotics, corticosteroids and biologics
- Current approved therapies have shown a waning of effect in a significant number of patients over time
- In addition, high-unmet medical need exists in affected patients with active draining disease





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



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

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

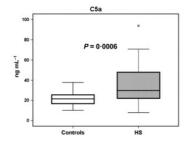
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Strong Rationale for Developing an Anti-C5a/C5aR in HS

(G)3

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

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${\tt C5a/C5aR}\ activation\ is\ a\ key\ neutrophil\ activator\ in\ {\tt HS}\ patient\ plasma$

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

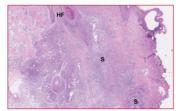
- Vilobelimab (anti-C5a antibody) and
- INF904 (anti-C5aR inhibitor)

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

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

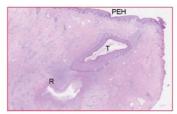
H&E Staining







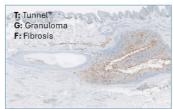
Hurley stage I patient with extensive deep dermal and subcutaneous suppurative abscessing inflammation, surrounding hair follicles with hyperkeratosis. C5aR1 staining positive – neutrophils





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

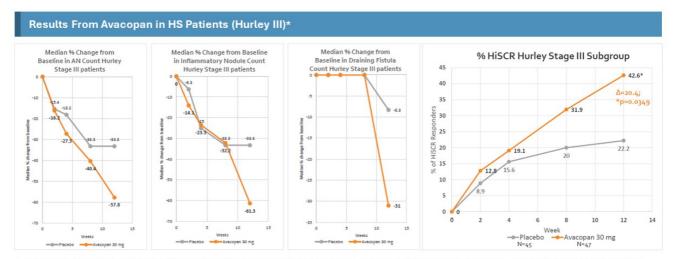
Avacopan (oral C5aR inhibitor)

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

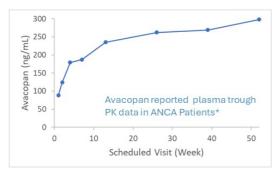


- Avacopan's efficacy (separation from placebo group) in HS only starts to emerge at W12 please note: steady state reported from ANCA patients was only reached at approx. 3 months due to prolonged drug accumulation (x4)
- Avacopan's 30mg BID dosing regimen may be too low to show adequate clinical efficacy 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_{0-12hr}) 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

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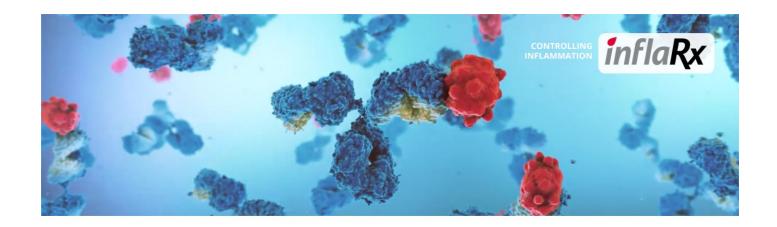
Vilobelimab* and Avacopan** Provide Evidence for Efficacy in HS Patients

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 dataon file. ** Source: Datafrom Chemocentryx presentation on AURORA trial results, October 28, 2020

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Gohibic (vilobelimab) Critical COVID-19 & ARDS



Emergency Use Authorization (EUA) Granted for Gohibic



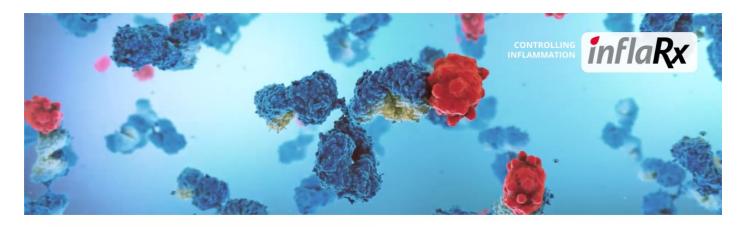


- Gohibic (vilobelimab) has not been approved, but has been authorized for emergency use by FDA under an EUA*, for the treatment of COVID-19 in hospitalized adults when initiated within 48 hours of receiving IMV**, or ECMO**
- Authorization granted based on results from a Phase III clinical trial in 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. 5 360bbb-5(b)(1), unless the declaration is terminated or authorization revoked sooner **IMV= invasive mechanical ventilation, **ECCMO= extraccoprom lemerbance oxigenation

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