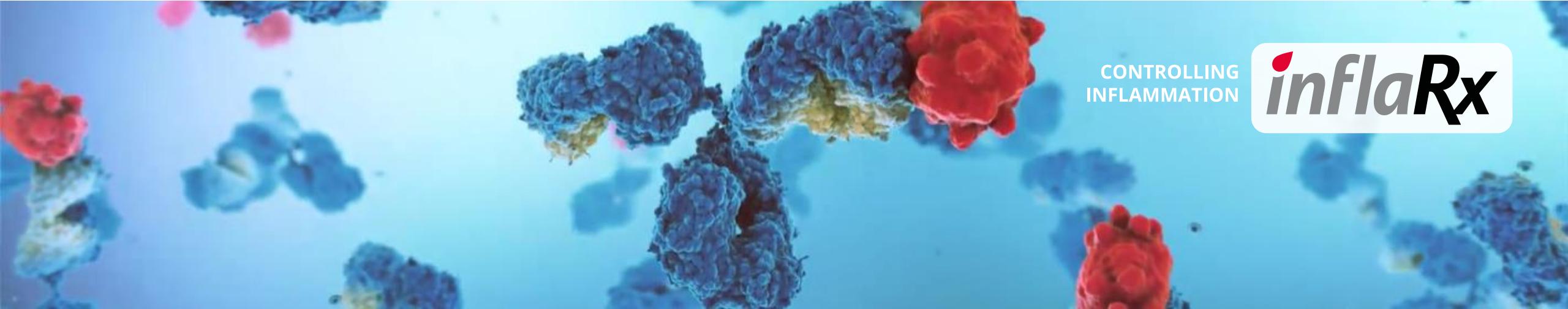


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INF904: TOPLINE RESULTS FROM PHASE I SAD STUDY

SEPTEMBER 2023



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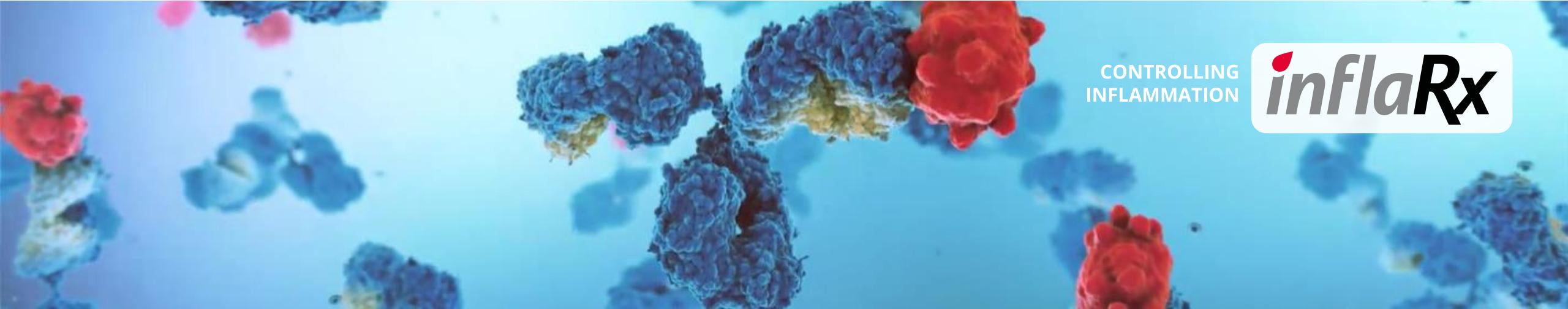
**inflaRx**

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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, our ability to 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 emergency use authorization (EUA) and in the future if approved for commercial use in the United States or elsewhere; the success of our future clinical trials for vilobelimab and any other product candidates 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, including the MAD part of the Phase 1 trial with C5aR inhibitor INF904, 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 (MAA) submission for vilobelimab and our biologics license application (BLA) submission for Gohibic (vilobelimab), and our ability to obtain and maintain full regulatory approval of vilobelimab or Gohibic (vilobelimab) for any indication; whether the U.S. Food and Drug Administration (FDA), the European Medicines Agency (EMA) or any comparable foreign regulatory authority will accept or agree with the number, design, size, conduct or implementation of our clinical trials, including any proposed primary or secondary endpoints for such trials; our expectations regarding the scope of any approved indication for vilobelimab; our ability to leverage our proprietary anti-C5a and C5aR technologies to discover and develop therapies to treat complement-mediated autoimmune and inflammatory diseases; our ability to protect, maintain and enforce our intellectual property protection for vilobelimab and any other product candidates, and the scope of such protection; our manufacturing capabilities and strategy, including the scalability and cost of our manufacturing methods and processes and the optimization of our manufacturing methods and processes, and our ability to continue to rely on our existing third-party manufacturers and our ability to engage additional third-party manufacturers for our planned future clinical trials and for commercial supply of vilobelimab and for the finished product Gohibic (vilobelimab); our estimates of our expenses, ongoing losses, future revenue, capital requirements and our needs for or ability to obtain additional financing; our ability to defend against liability claims resulting from the testing of our product candidates in the clinic or, if approved, any commercial sales; if any of our product candidates obtain regulatory approval, our ability to comply with and satisfy ongoing obligations and continued regulatory oversight; 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 U.S. Securities and Exchange Commission (SEC). These statements speak only as of the date of this press release and involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. Given these risks, uncertainties and other factors, you should not place undue reliance on these forward-looking statements, and we assume no obligation to update these forward-looking statements, even if new information becomes available in the future, except as required by law.



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### Avacopan Data

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

### About InflaRx

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

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

# Speakers

## Speaker



**PROF. NIELS RIEDEMANN, M.D., PH.D.**  
Chief Executive Officer, Founder, InflaRx

## Q&A



**PROF. RENFENG GUO, M.D.**  
Chief Scientific Officer, Founder, InflaRx



**THOMAS TAAPKEN, PH.D.**  
Chief Financial Officer, InflaRx



**CAMILLA CHONG, M.D.**  
Chief Medical Officer, InflaRx

# INF904: Topline Results from Phase I Single Ascending Dose (SAD) Part

## AGENDA

BACKGROUND AND PRE-CLINICAL WORK

STUDY DESIGN

SAFETY OUTCOME

PK OUTCOME

PD OUTCOME

SUMMARY

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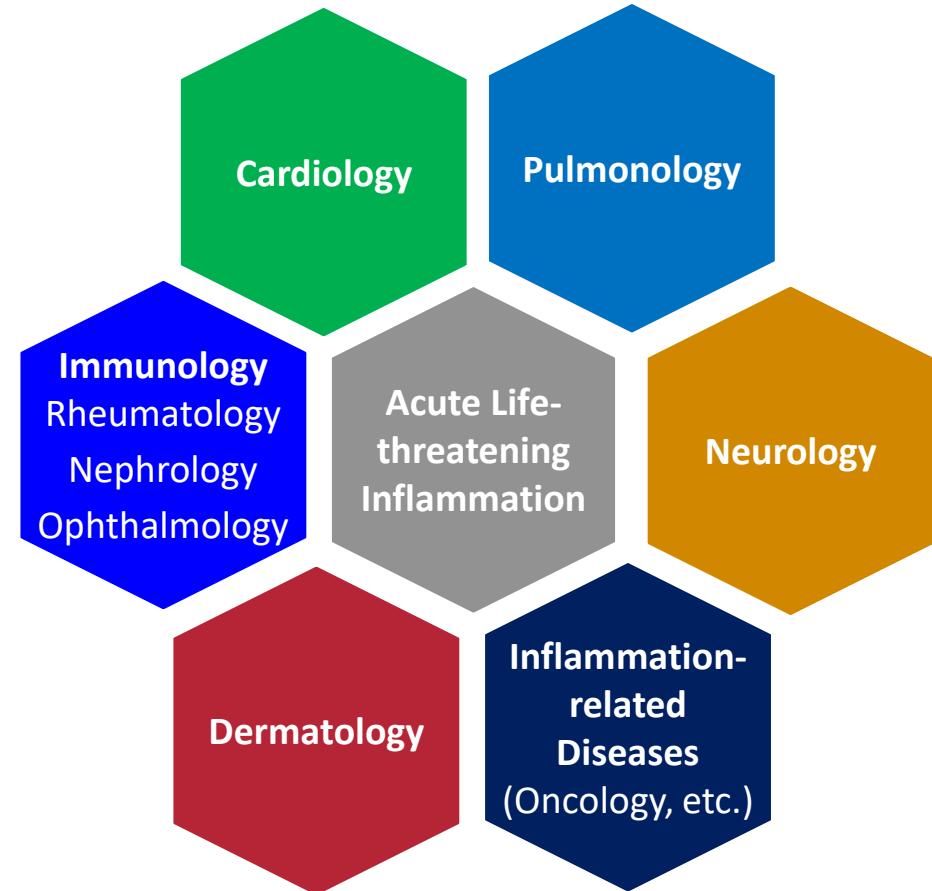
SUMMARY

# C5aR is a Validated Drug Target for Inflammation

## C5aR: a promising drug target

- C5aR, a G-protein-coupled receptor highly expressed on granulocytes and in epithelial cells, mediates the major pathophysiological effects of C5a.
- Blockade of C5aR showed strong anti-inflammatory effects in numerous pre-clinical disease models in both acute and chronic settings and in clinical research.
- C5aR antagonists are expected to be stand-alone or adjunct treatments for a variety of inflammatory disorders.
- Currently, one marketed CSaR inhibitor exists, which could be improved based on the published data of the Phase I clinical trial\*.

## Role of C5aR in pre-clinical and clinical R&D

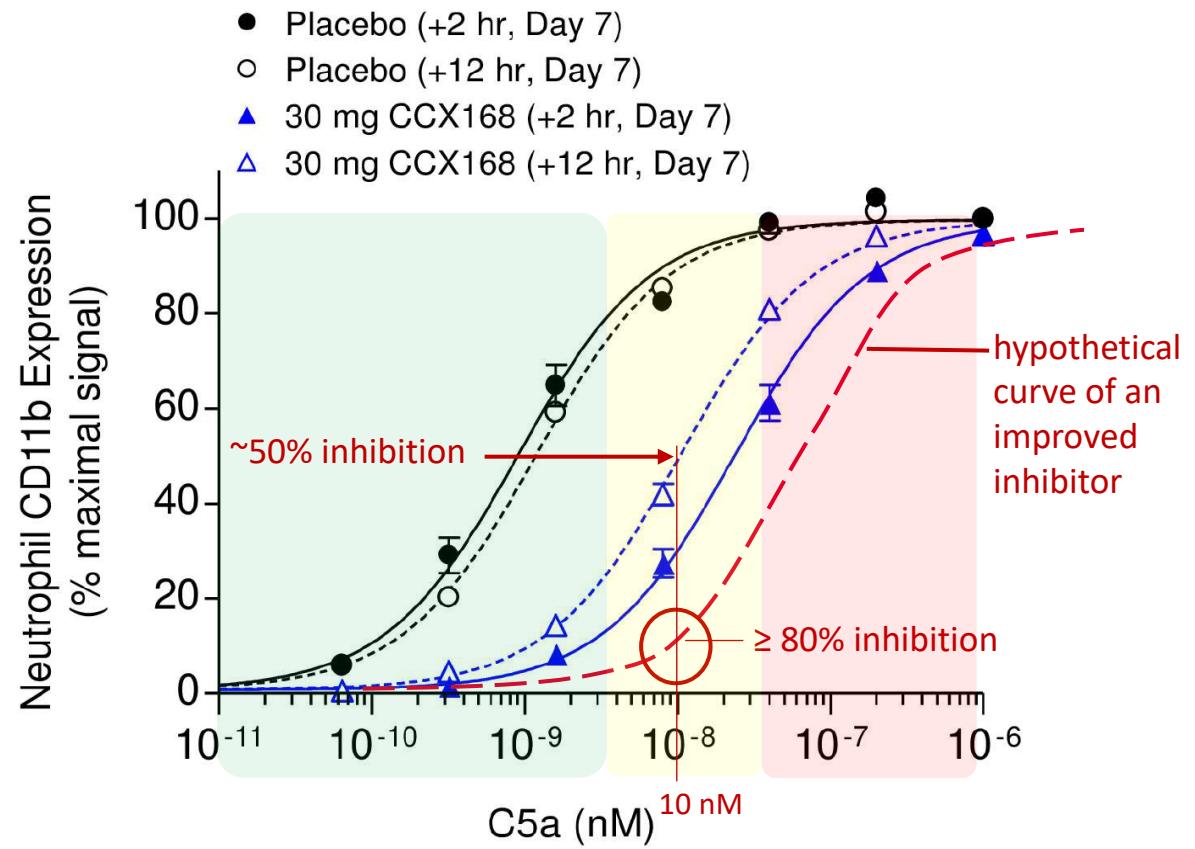


\*Bekker et al. 2016, PLOS ONE 11(10): E0164646

# Improvement of C5aR Inhibition

## Properties of a best-in-class C5aR antagonist

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



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

# INF904: Pre-clinical Summary



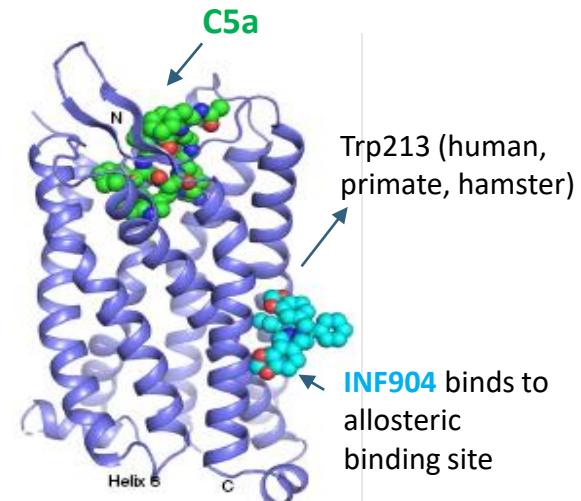
## INF904 Facts

- Chemistry: Amorphous solid (high exposure potential).
- INF904 binds to a well-defined allosteric site in C5aR.
- INF904 has a novel Markush structure.
- US patent was issued in October 2021; national phases for other select countries (PCT).

## Pre-clinical Findings

- **No obvious toxicity findings** even in the highest dose groups (rat and monkey; up to 300 mg/kg).
- **High in vitro potency** with a desired IC<sub>50</sub> (<1nM) in calcium mobilization assay.
- **Higher plasma exposures** in several in vivo models when compared to Avacopan\*.
- **Increased efficacy** in hamster neutropenia model when tested at equivalent dose with an Avacopan-like molecule\*.
- **Therapeutic effects** in pre-clinical disease models (renal / peritonitis).

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



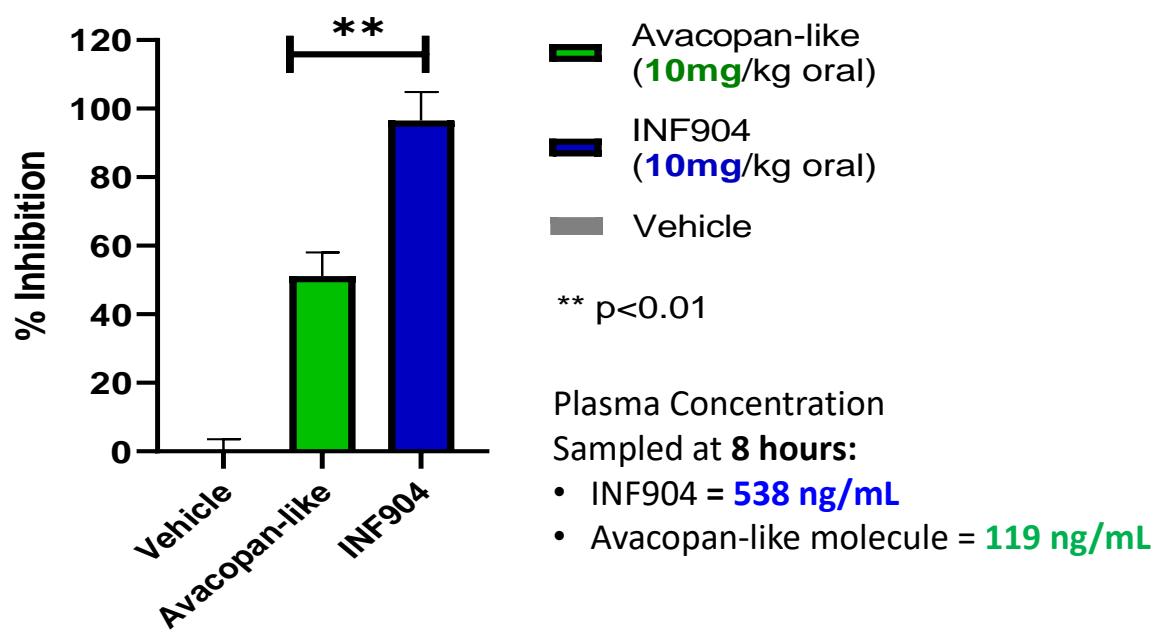
C5aR structure  
(Source: Nathan Robertson, 2018 Nature)

# INF904: Potential Best-in-Class C5aR Inhibitor



## PRE-CLINICAL IN VIVO EFFICACY COMPARISON OF INF904 to AVACOPAN\*

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



**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.

**Outcome:** INF904 is significantly superior to an identical dose of the Avacopan-like molecule\* in blocking C5aR, leading to an approximate doubling of neutrophil inhibition in vivo in this rodent model.

**Note:** INF904 dosing within this experiment exerts an approx. 4.5-fold higher plasma level 8 h after dosing when compared to the identical dosing with the Avacopan-like molecule\*.

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

# INF904: Topline Results from Phase I Single Ascending Dose (SAD) Part

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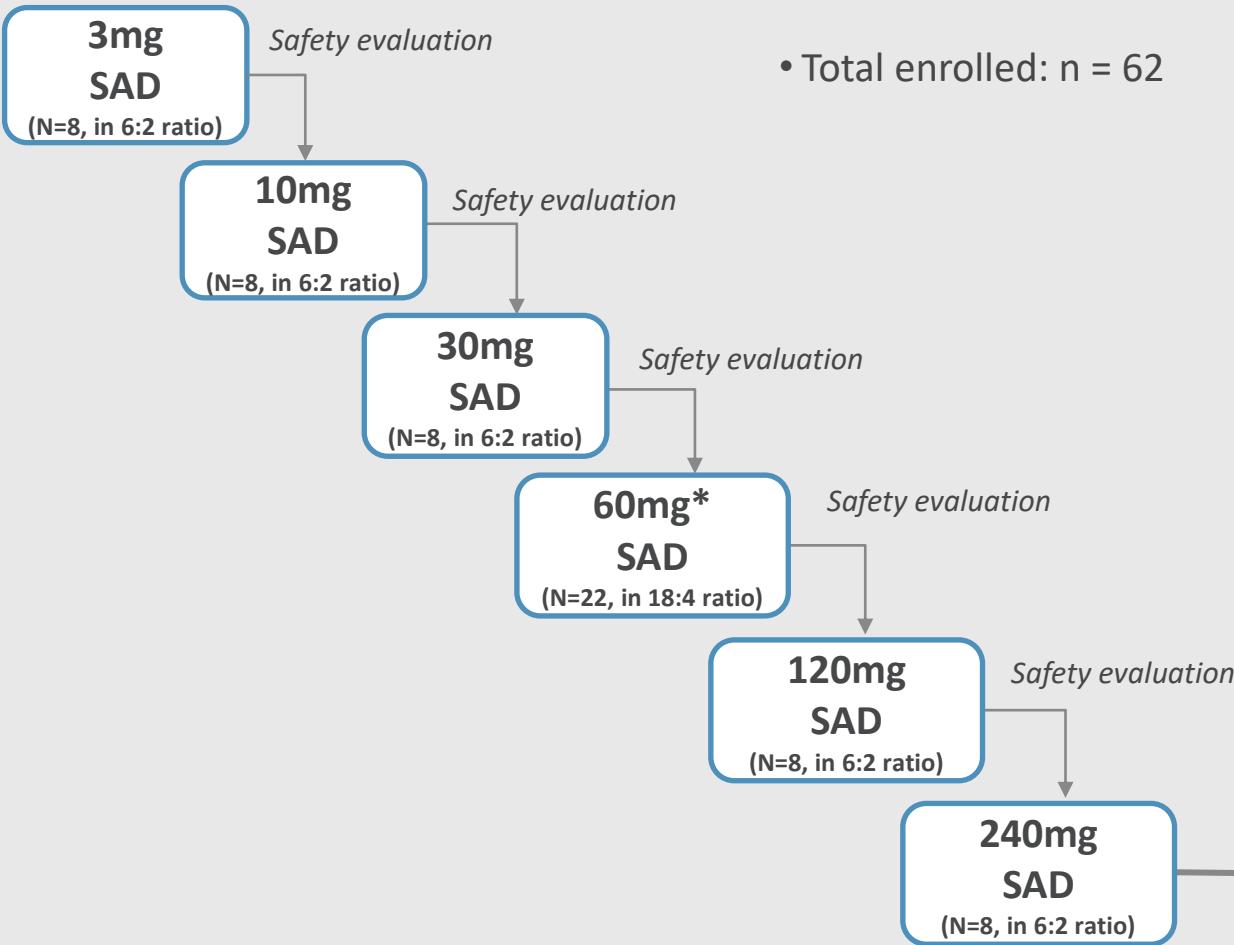
PD OUTCOME

SUMMARY

# INF904 Phase I Study Design



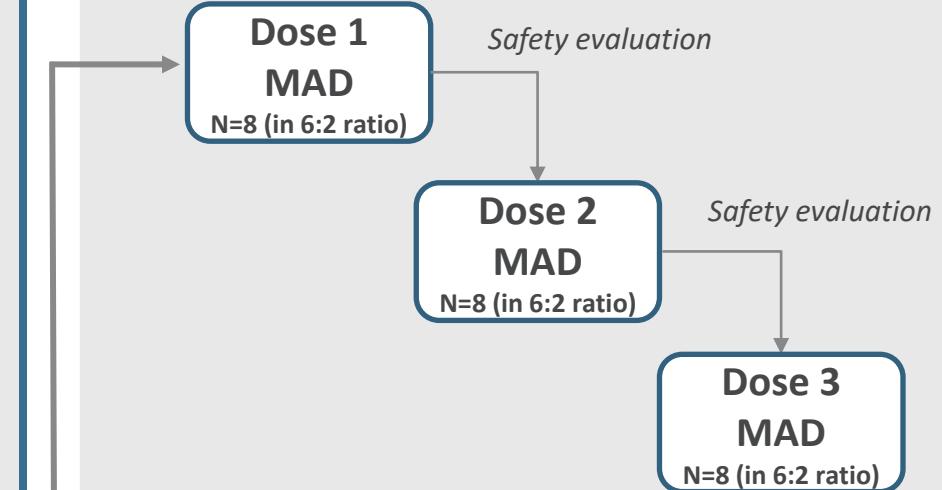
## Single Ascending Dose (SAD)



- Total enrolled: n = 62

## Multiple Ascending Dose (MAD)

- Enrollment ongoing



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

# INF904: Topline Results from Phase I Single Ascending Dose (SAD) Part

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# INF904 Phase I Study: Safety Results from SAD Part

## HIGHLIGHTS

- INF904 was well tolerated in treated healthy volunteers and resulted in no safety signals of concern in single ascending doses ranging from 3mg to 240mg.
- Overall percentage of adverse events (AEs) in placebo group was higher than in active treated subjects.
- AE severity:
  - Mild: 81
  - Moderate: 9
  - Severe: 0
- No serious AE (SAE) reported at any dosing level.
- 1 moderate AE rated as possibly related to study drug (headache), but subject had received placebo.
- 1 withdrawn subject in cohort 1.4 (60 mg) for unrelated AE.

# INF904: Topline Results from Phase I Single Ascending Dose (SAD) Part

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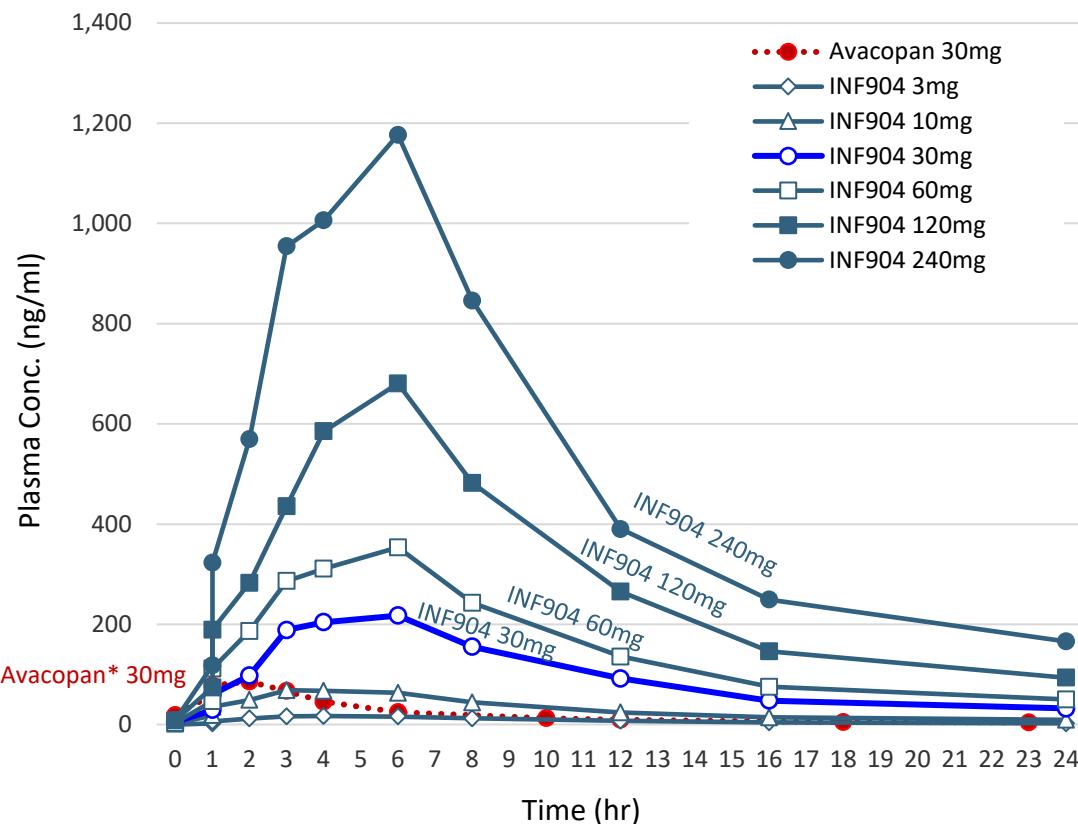
SAFETY OUTCOME

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SUMMARY

# INF904 Phase I Study: PK Results from SAD Part



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

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

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

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

# INF904 Phase I Study: PK Summary from SAD Part

## HIGHLIGHTS

- Exposure to INF904 is sustained with a time to maximum concentration ( $t_{max}$ ) at 6 hours.
- INF904 is dose proportional for systemic exposure over the dose range 3 – 240 mg ( $AUC_{last}$ ) and nearly dose proportional for maximum concentration ( $C_{max}$ ).
- 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).

# INF904: Topline Results from Phase I Single Ascending Dose (SAD) Part

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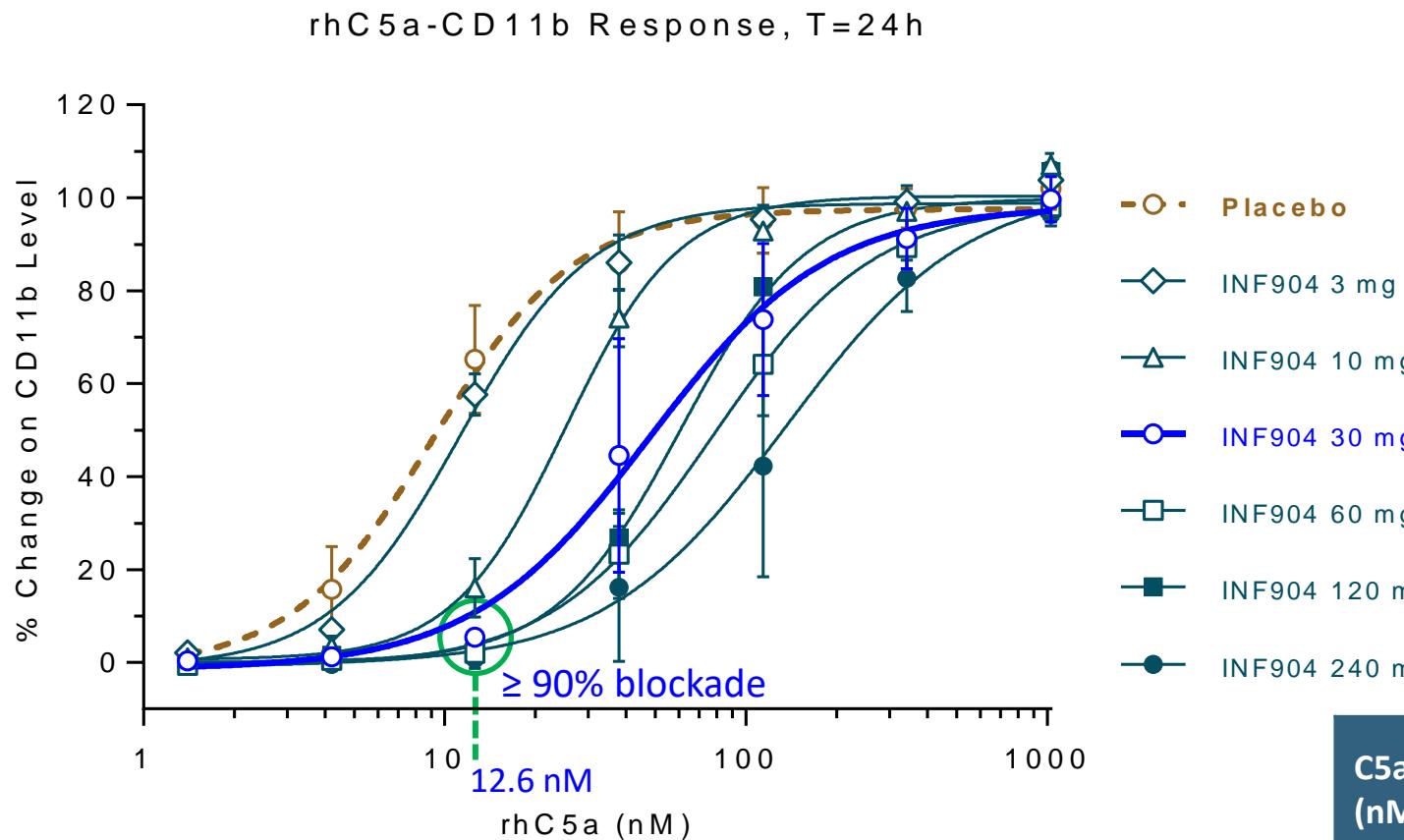
PK OUTCOME

PD OUTCOME

SUMMARY

# INF904 Phase I Study: PD Results from SAD Part

## *C5a-mediated CD11b up-regulation on neutrophils ex vivo at 24h post dosing*



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

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

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

# INF904: Topline Results from Phase I Single Ascending Dose (SAD) Part

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# Summary

## *Topline Results from INF904 Phase I SAD Study*



### KEY OUTCOMES

- ▶ INF904 was well tolerated in treated healthy volunteers and resulted in no safety signals of concern in single ascending doses ranging from 3mg to 240mg.
- ▶ INF904 demonstrated a favorable PK profile.
- ▶ INF904 demonstrated a strong C5a blocking potential at C5a concentrations observed in human diseases.
- ▶ INF904 can be formulated with a higher drug strength of 30mg per capsule vs. 10mg per capsule for the marketed comparator.

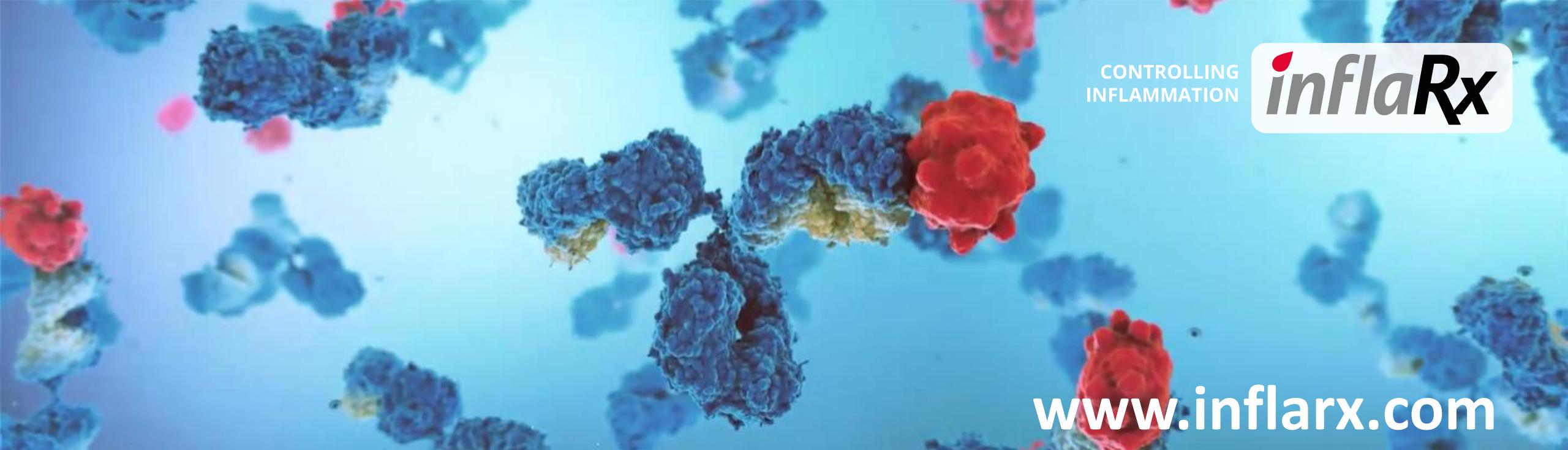


**INF904 confirms its best-in-class C5aR inhibitor potential within this Phase I single ascending dose study**

## Next Steps for INF904

- ▶ Phase I MAD study ongoing; data available to be presented at the end of Q4 2023 / early Q1 2024.
- ▶ Ongoing scientific and strategic work within select disease areas for future development.
- ▶ Work ongoing to optimize formulation for ideal manufacturing and dosing convenience.
- ▶ Work ongoing to prepare for next clinical development steps, including chronic toxicology study.

➤ Positive results from Phase I SAD study support further development of INF904



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