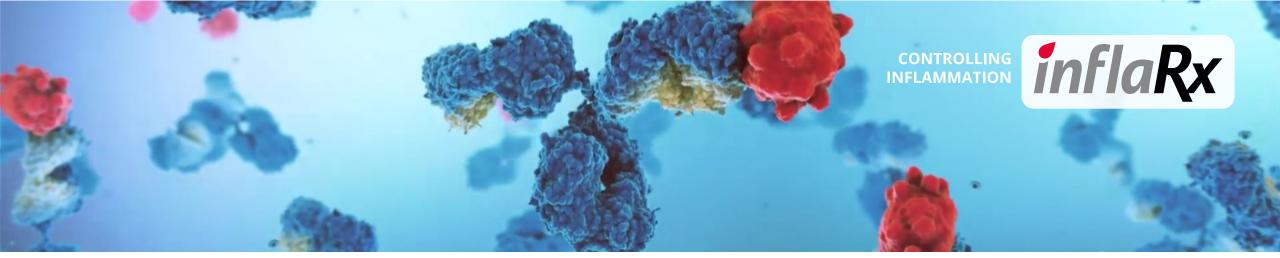


CORPORATE PRESENTATION

JANUARY 2024

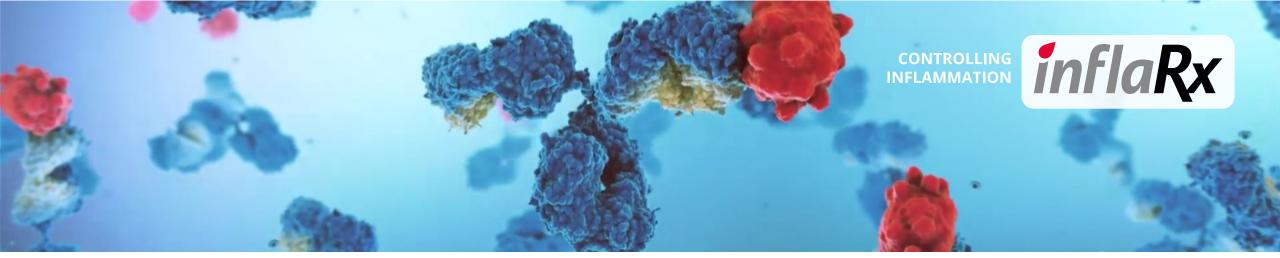


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Forward-Looking Statements

This presentation 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 presentation 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 preclinical 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 Medicine 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 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 U.S. Securities and Exchange Commission (SEC). These statements speak only as of the date of this presentation 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 preclinical 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

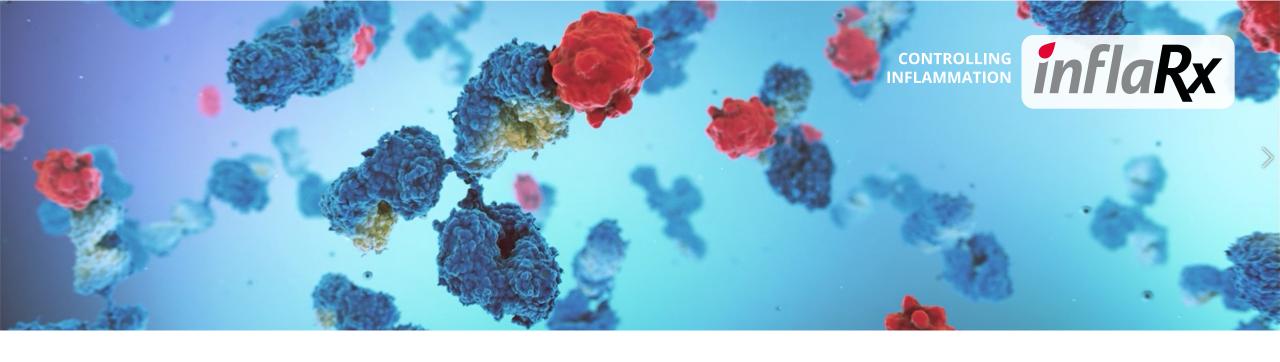
InflaRx GmbH (Germany) and InflaRx Pharmaceuticals Inc. (USA) are wholly owned subsidiaries of InflaRx N.V. (together, "InflaRx").

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.

PIPELINE AND COMPANY STATUS DEVELOPMENT UPDATE

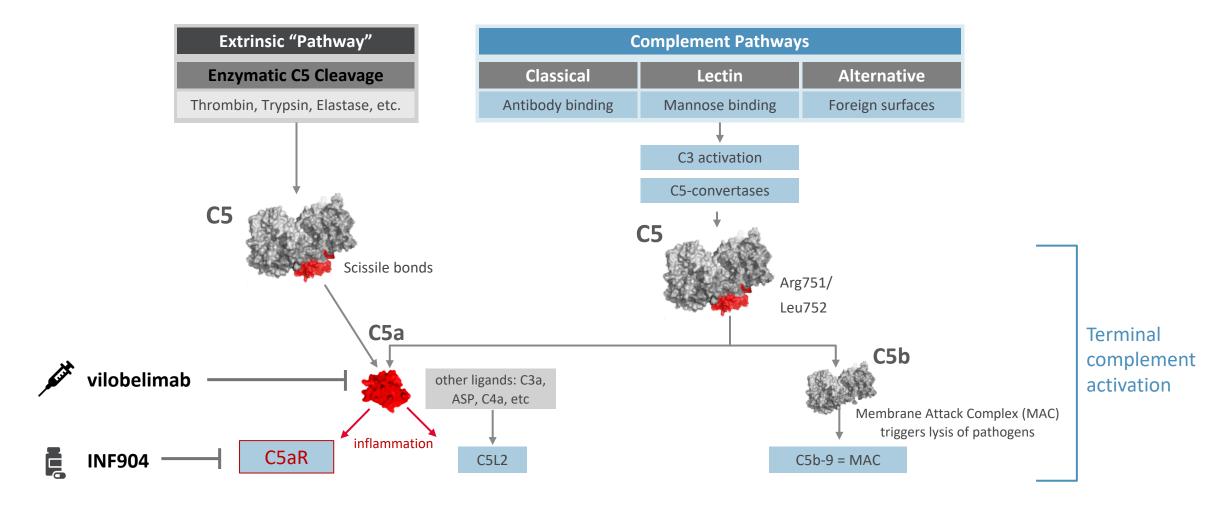
	AREA	INDICATIONS	PRECLIN.	PHASE I	PHASE II	PHASE III	MARKET	STATUS
Gohibic vilobelimab	Acute organ dysfunction	critical COVID-19 broader ARDS						US EUA granted in April 2023, EU MAA under review, possibility to develop towards broader ARDS label discussed with FDA (Phase III ready)
C5a Inhibitor	Immuno- dermatology	pyoderma gangrenosum						Phase III started – enrollment ongoing
INF904 Oral C5aR Inhibitor	Ę	indication undisclosed						Phase I study completed: Phase II initiation planned for end of 2024
IFX002 C5a Inhibitor	K	life-cycle approach for vilobelimab						Developing for optimized use for other chronic inflammatory indications
vilobelimab development opportunities		Hidradenitis supp. ANCA vasculitis						Development currently on hold – Phase III ready in these indications
in several other indications		cut. squam. cell				>		Development on hold due to shift in strategic priorities





THE COMPLEMENT SYSTEM AND OUR C5a TARGETED APPROACH

THE COMPLEMENT SYSTEM AND C5a ACTIVATION



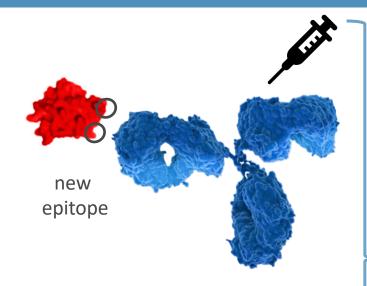


THE EXTRINSIC PATHWAY REPRESENTS AN ADDITIONAL ROUTE, OUTSIDE OF THE KNOWN COMPLEMENT PATHWAYS, TO CLEAVE C5A FROM C5 --- C5A ACTS PRIMARILY THROUGH C5AR



VILOBELIMAB: A FIRST-IN-CLASS ANTI-C5a MONOCLONAL ANTIBODY

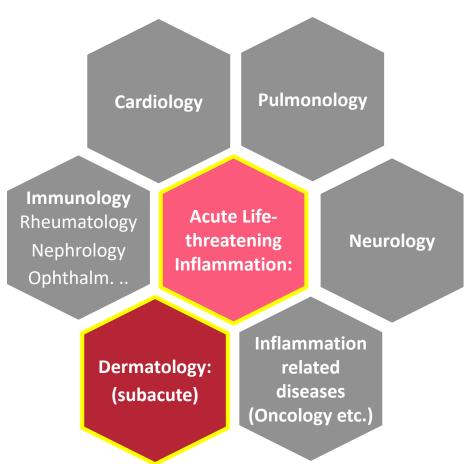
VILOBELIMAB (IFX-1)



Key features of vilobelimab:

- 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

DEVELOPMENT AREAS IN ACUTE AND SUB-ACUTE INFLAMMATION



Being a fast acting highly specific monoclonal antibody infused i.v., vilobelimab therapy delivers:

- Strong and immediate C5a inhibition in blood
- Fast onset of inhibition of neutrophil activation in human blood
- Potential disease modifying activity in which C5a signaling (potentially through both receptors C5aR and C5L2) may play a key role



INF904: ORAL INHIBITOR OF THE C5a-RECEPTOR

Trp213 (human, primate, hamster)

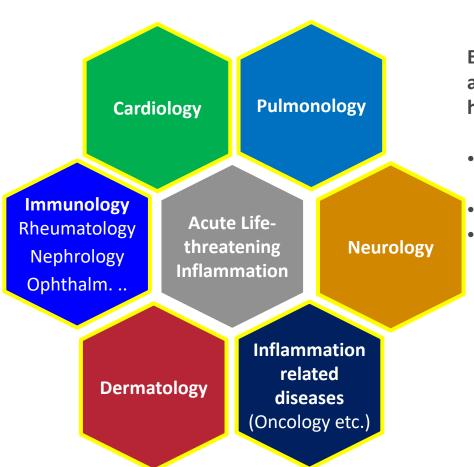
INF904 binds to allosteric binding site

C5aR structure (Nathan Robertson, 2018 Nature)

Key features of INF904:

- Oral low molecular weight inhibitor with proprietary structure
- Best in class potential based on PK / PD profile observed in phase I study
- No safety or tolerability concerns in phase I study

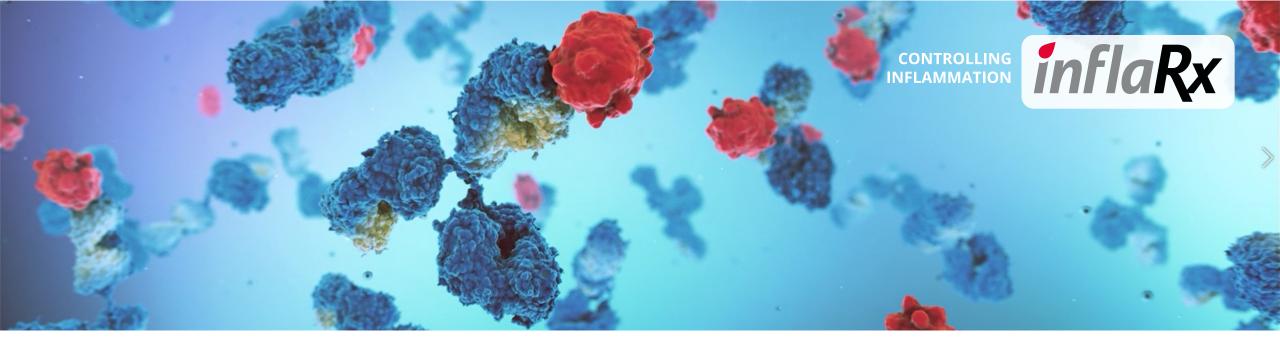
POTENTIAL DEVELOPMENT AREAS IN CHRONIC INFLAMMATION



Being a small molecule orally available C5aR inhibitor, INF904 has the potential to deliver:

- Favorable tissue and plasma concentrations
- Favorable dosing regimen
- Sustained long-term control over C5a/C5aR activation in a variety of chronic inflammatory diseases in different tissues





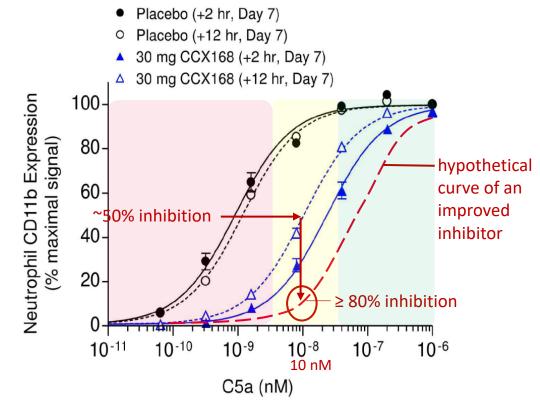
INF904: AN ORALLY ADMINISTERED, HIGHLY SELECTIVE LOW MOLECULAR WEIGHT C5aR INHIBITOR



TARGETED IMPROVEMENT OF C5aR-INHIBITION

PROPERTIES OF A BEST-IN-CLASS C5aR ANTAGONIST

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



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



INF904: PRE-CLINICAL SUMMARY

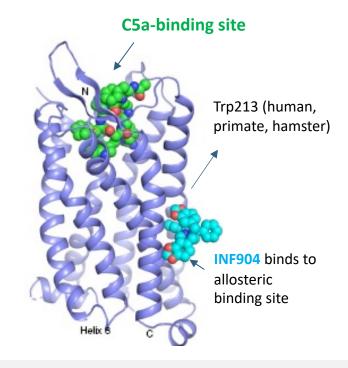


INF904 FACTS

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

PRE-CLINICAL FINDINGS

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



C5aR structure (Source: Nathan Robertson, 2018 Nature)

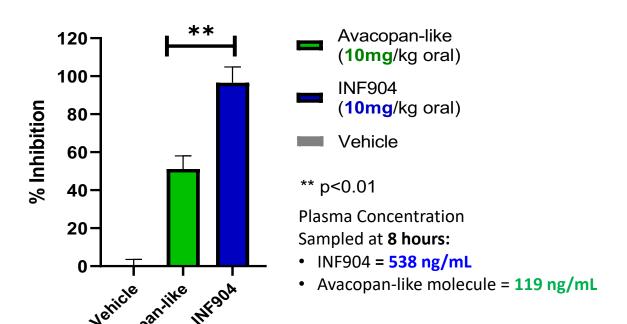


INF904: POTENTIAL FOR BECOMING A BEST-IN-CLASS C5aR INHIBITOR



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

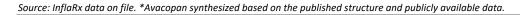
Inhibition of in vivo neutrophil activation by INF904 compared to Avacopan-like molecule*



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.

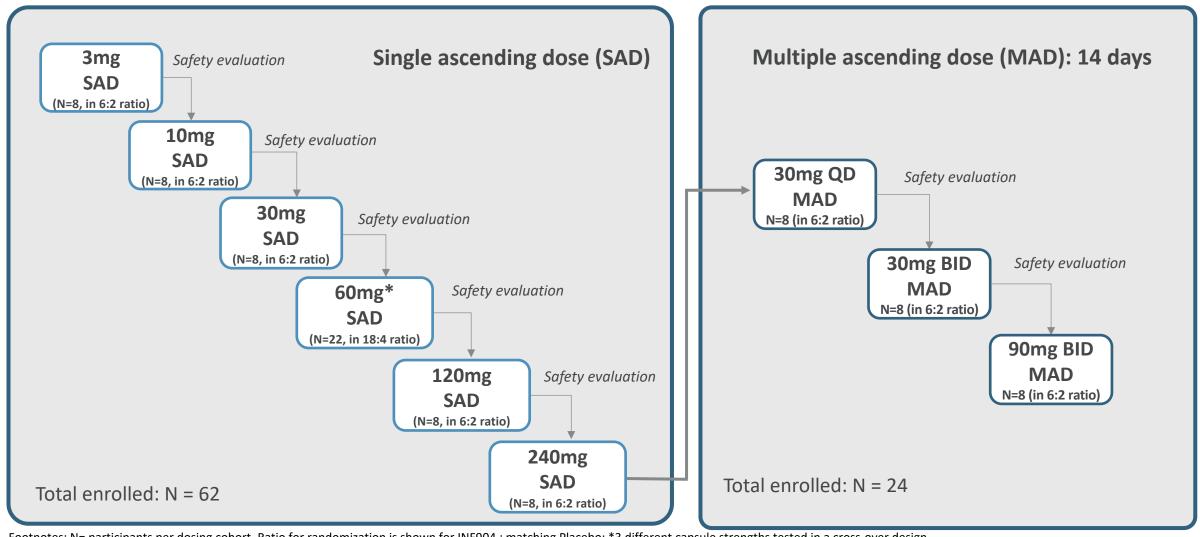
Outcome: INF904 is significantly superior to an identical dose of Avacopan* in blocking C5aR, leading to an approximate doubling of neutrophil inhibition in vivo in this rodent model.

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 Avacopan*



INF904: PHASE I STUDY DESIGN





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



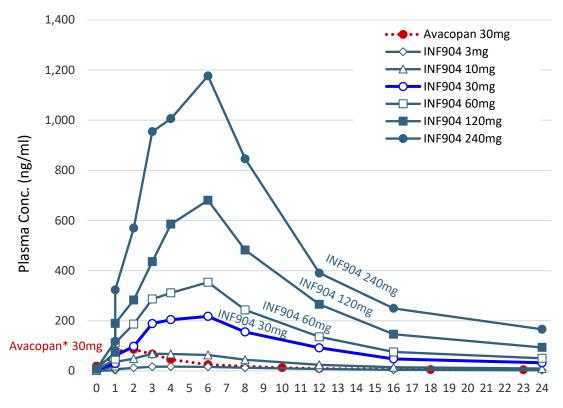
INF904 PHASE I STUDY: SAFETY RESULTS COMBINED (SAD AND MAD PART)

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
- No serious AE (SAE) reported at any dosing level (SAD and MAD)
- No subject withdrawn in MAD part, 1 subject withdrawn in cohort 1.4 (60 mg) in SAD part for unrelated AE



INF904: PK RESULTS FROM SINGLE ASCENDING DOSE (SAD) PHASE I



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

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

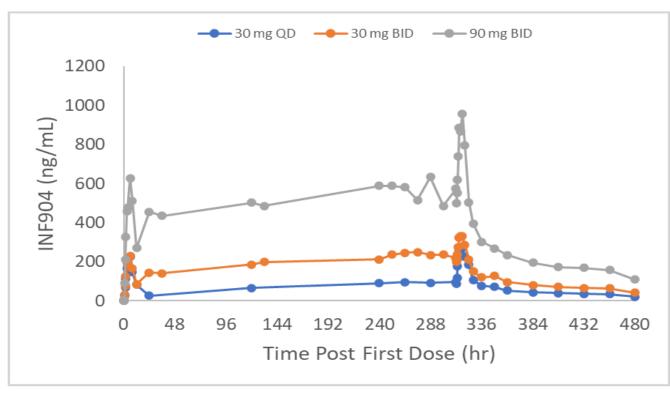
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IN COMPARISON TO PUBLISHED DATA FOR AVACOPAN, INF904 IS APPROXIMATELY 3-FOLD HIGHER IN C_{MAX} AND 10-FOLD HIGHER IN SYSTEMIC EXPOSURE (AUC_{LAST}) FOR COMPARABLE DOSES (3, 10, 30 MG)



^{*}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.

INF904: PK RESULTS FROM MULTIPLE ASCENDING DOSE (MAD) PHASE I



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

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

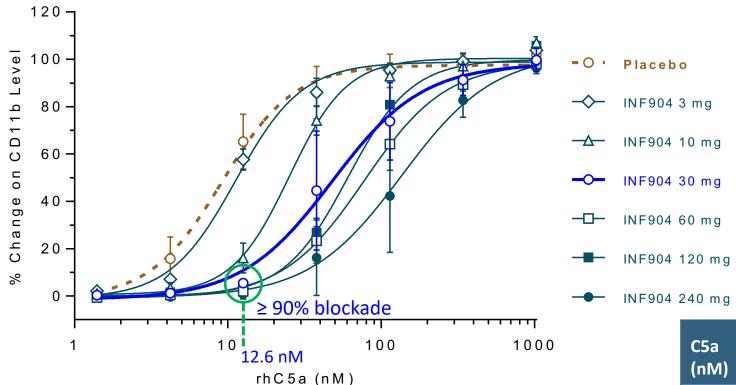
- INF904 dosing either once daily (QD) or twice daily (BID) exhibits favorable concentration-time profiles (after 14 days dosing)
- INF904 exposure is directly proportional to dose when comparing 30 mg BID versus the 90 mg BID regimens
- In BID regimen, accumulation observed (Day 1 to 14) for C_{max}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



INF904 PHASE I STUDY: PD RESULTS FROM SAD PART

C5a-mediated CD11b upregulation on neutrophils ex vivo at 24h post dosing





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

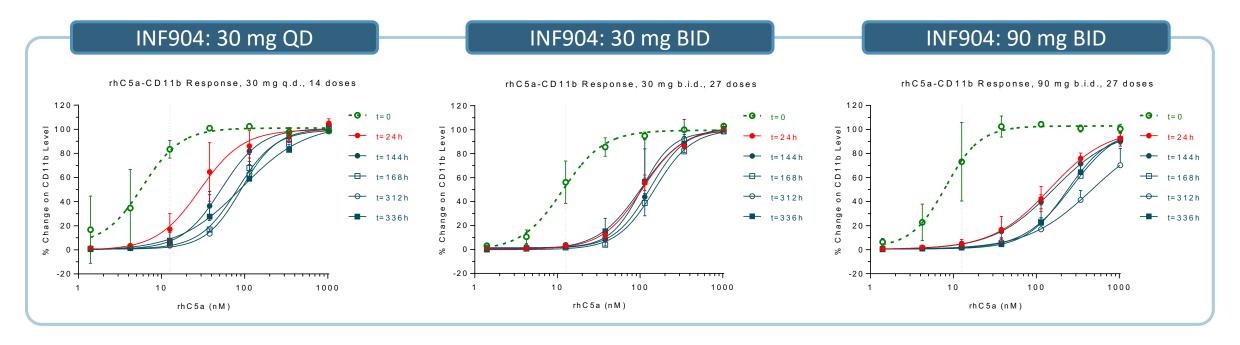
C5a		NF904 (B	locking A	ctivity % v	s Placebo)
(nM)	3mg	10mg	30mg	60mg	120mg	240mg
12.6	9.6	74.7	91.5	95.7	95.2	98.7

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 PHASE I STUDY: PD RESULTS FROM MAD PART

C5a-mediated CD11b upregulation on neutrophils ex vivo up to 14 day dosing



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



^{*}EC₅₀ (nM) is the half maximal effective C5a concentration

SUMMARY

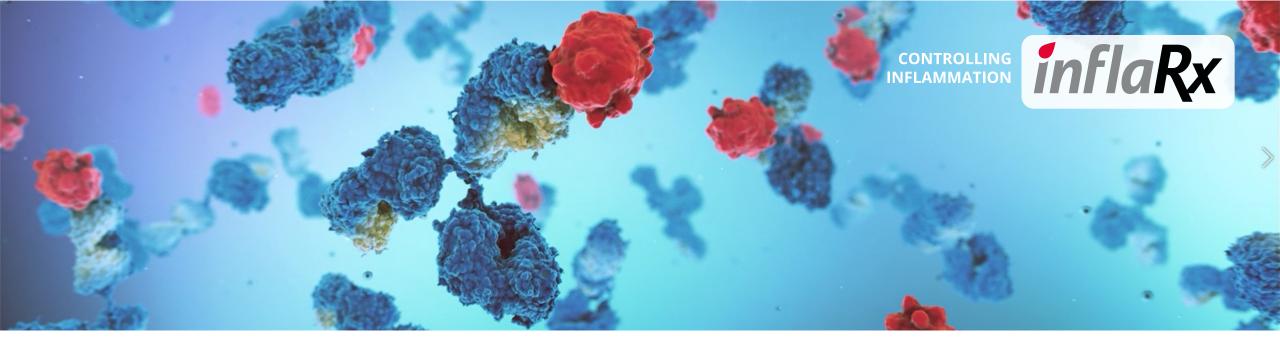
Topline Results from INF904 Phase I Study

KEY OUTCOMES

- INF904 was well tolerated in treated healthy volunteers and resulted in no safety signals of concerns over entire tested dose ranges and regimens.
- INF904 demonstrated a favorable PK profile with potential to achieve therapeutic exposures (AUC & Cmax) required in chronic immuno-inflammatory diseases.
- INF904 demonstrated a \geq 90% blocking potential for C5a induced neutrophil activation at C5a concentrations observed in human diseases for dosing regimens of 30mg or higher.
- 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 STUDY

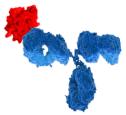


GOHIBIC (VILOBELIMAB)
CRITICAL COVID-19



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 is currently being launched by InflaRx in the US under the EUA:
 - building an experienced and highly focused commercial team and creating awareness with different healthcare players
 - Building a robust supply chain to allow for uninterrupted supply of Gohibic to US hospitals

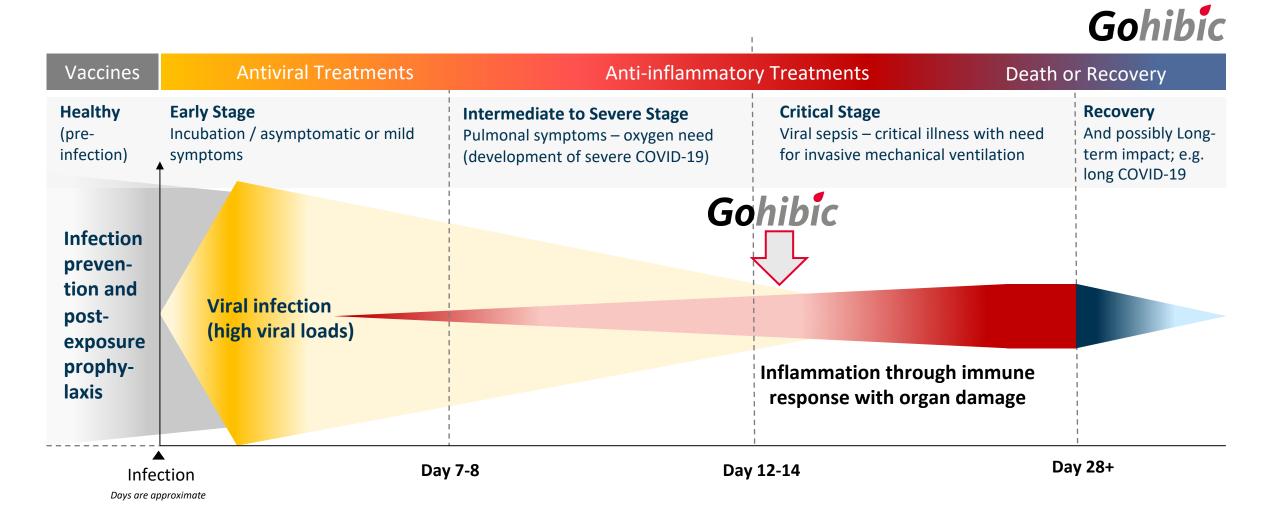
For additional and important safety information, please visit www.gohibic.com



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

^{**} IMV = invasive mechanical ventilation, ***ECMO = extracorporal membrane oxygenation

COVID-19: DISEASE PROGRESSION AND THERAPEUTIC INTERVENTIONS

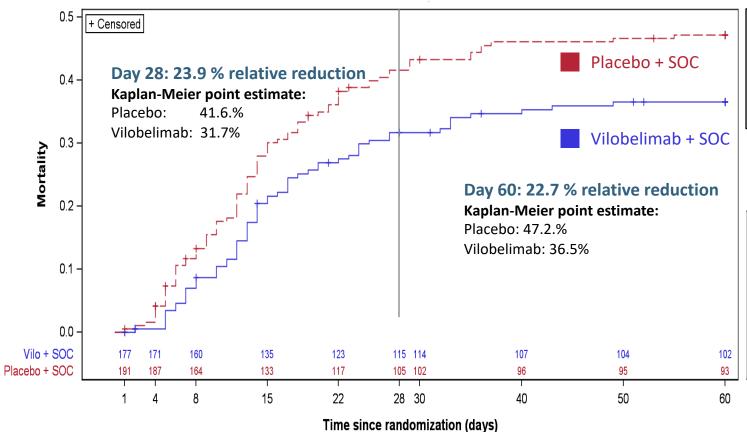






PANAMO PHASE III TRIAL – PRIMARY ENDPOINT

28 day all-cause mortality



PANAMO Phase III trial:

1:1 randomized, double-blind, placebo-controlled, multi-national trial in (n=369) invasive mechanically ventilated COVID-19 patients

SOC included concomitant use:

• Corticosteroid use: 97%

• Anti-coagulant use: 98%

prior or concomitant use of other immunomodulators

(tocilizumab > baricitinimb):

Data published in Vlaar, A et al. Lancet Resp Med 2022. https://doi.org/10.1016/ S2213-2600(22)00297-1



NUMBER OF PATIENTS NEEDED TO TREAT FOR SAVING ONE ADDITIONAL LIFE = 9



PANAMO PHASE III TRIAL – SECONDARY ENDPOINT

Need for Renal Replacement



SECONDARY ENDPOINT: PROPORTION OF PATIENTS FREE OF RENAL REPLACEMENT WITHIN 28 DAYS

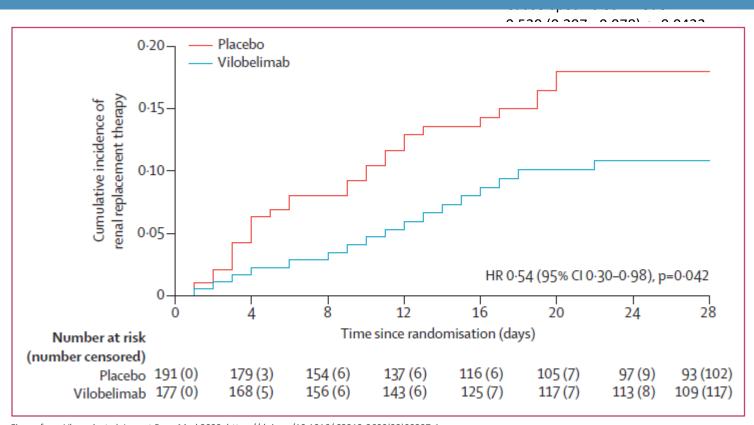
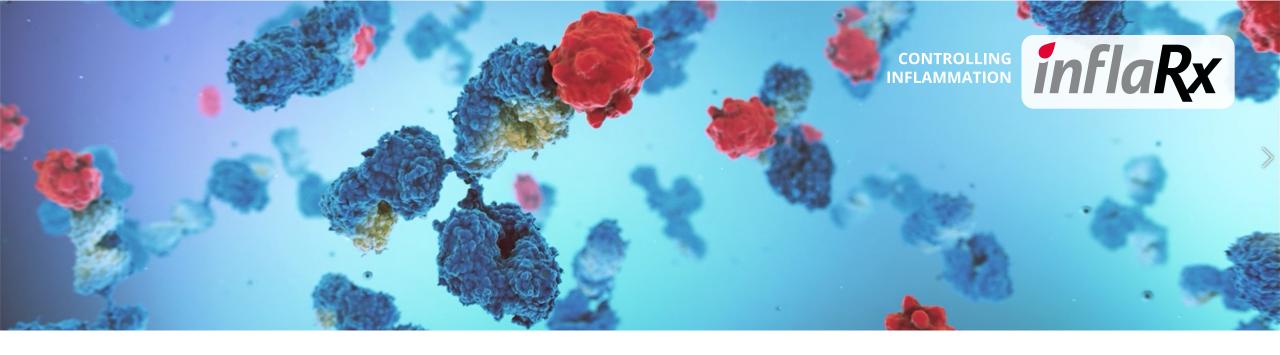


Figure from Vlaar, A et al. Lancet Resp Med 2022. https://doi.org/10.1016/ S2213-2600(22)00297-1



MORE PATIENTS WERE FREE OF RENAL REPLACEMENT NEED WITHIN 28 DAYS IN VILOBELIMAB ARM COMPARED TO PLACEBO





VILOBELIMAB FOR THE TREATMENT OF ULCERATIVE PYODERMA GANGRENOSUM (PG)



PYODERMA GANGRENOSUM (PG)



AN AUTOIMMUNE CONDITION WITH HIGH 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

Current Treatment – Medical Need

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

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



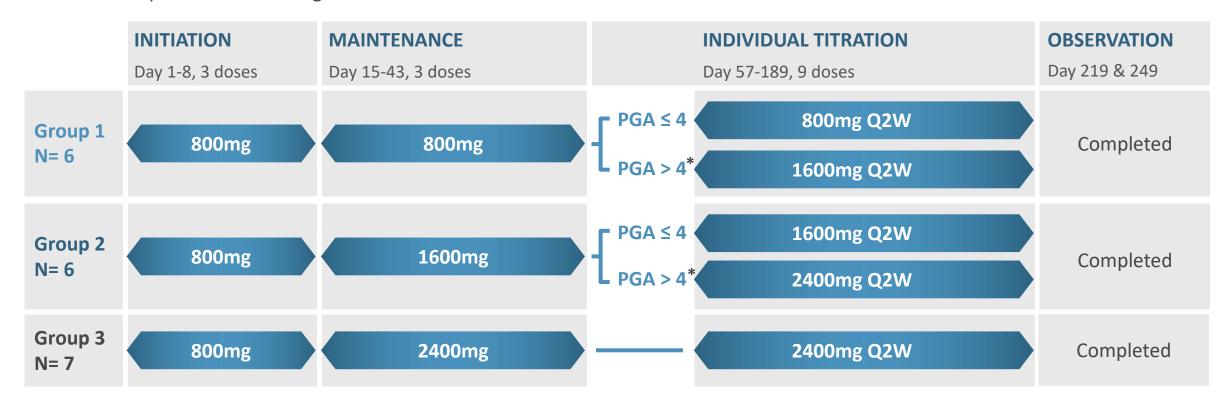
STRONG RATIONALE FOR TREATMENT WITH VILOBELIMAB:
PG ASSOCIATED WITH NEUTROPHILIC SKIN INFILTRATION IN AFFECTED AREAS AND LESIONS, POTENTIALLY TRIGGERED BY C5A



PG PHASE IIA TRIAL

Study Design

- 19 patients enrolled in the study
- **Primary endpoint:** Safety
- **Key secondary endpoints:** Responder rate defined as PGA ≤3 (PGA of ≤1 is considered clinical remission and closure of target ulcer); time to complete closure of target ulcer

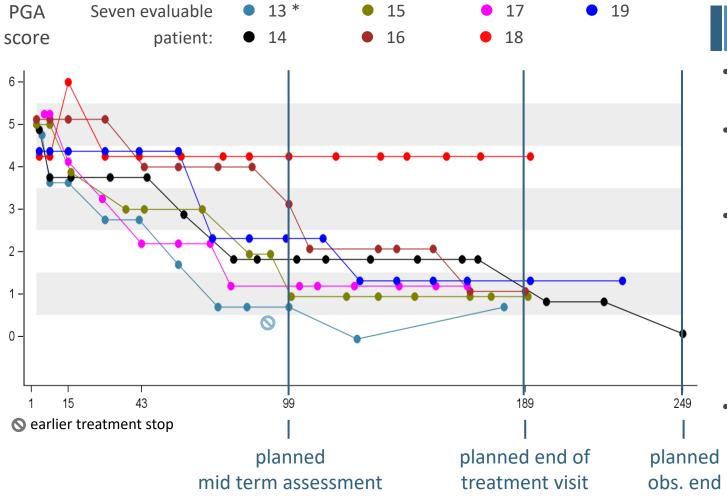


^{*} Up-titration to the next dose on day 57 if PGA > 4 and at least 5 patients treated with the current dose showed no safety issues



PHASE IIA STUDY RESULTS – GROUP 3 (HIGH DOSE)





GROUP 3 RESULTS

- Six out of seven patients achieved PGA score of ≤ 1 (remission)
- One patient (18) experienced target ulcer area decrease > 50%; however, new PG lesions developed
- Patient 19 with complicated disease course
 - Pre-existing diabetes and wound infection in target ulcer area on day 1 with start of antibiotics
 - Wound infection and local progression in target ulcer area on day 50
 - Broad spectrum antibiotics and cyclosporin A starting day 50
 - Closure of target ulcer on day 127
- Patient 16 started 10 mg/d prednisone on day 72 (allowed per protocol)

PGA line plot of absolute values over time by patient (Actual days displayed acc. to visit windows)



^{*} Patient 13 was discontinued from treatment after day 71 due to delayed availability of pos. baseline TB testing result (exclusion criterion). No re-activation of TB reported up to end of observation.

PG PHASE IIA

Patient Case Studies



TARGET ULCER DEVELOPED WHILE ON ADALIMUMAB

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

Day 85

PGA = 1

Area	: 1136 mm ²	28
	View Town	1
		1
	1	
	1	

Baseline



Day 89 PGA = 1 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

Area: 3695 mm²



Day 99

PGA = 1

Area: 0.00 mm²



Day 189

PGA = 1

Area: 0.00 mm²





PG PHASE IIA STUDY RESULTS



Summary and Conclusion



SAFETY CONCLUSION

- 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 AFs detected.



CLINICAL RESPONSE CONCLUSION

- Out of 17 evaluable patients at end of treatment visit or day of last drug administration
 - Clinical Remission (PGA ≤ 1): 9 patients (53%)
 - Clinical Response (PGA ≤ 3): 1 additional patient (6%)
 - Slight Improvement (PGA = 4): 7 patients (41%)
- High Dose Group shows highest rate of target ulcer closure and clinical remission (85.7%)

BASED ON
FEEDBACK FROM
FDA

ORPHAN DRUG AND FAST TRACK STATUS US FDA

ORPHAN DRUG
STATUS EMA



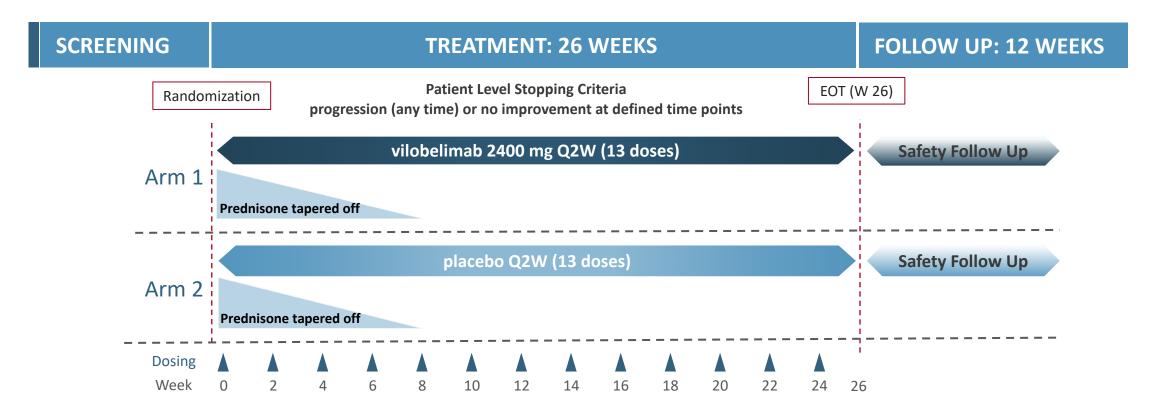
NO SAFETY OR TOLERABILITY CONCERNS AND DEMONSTRATED EVIDENCE OF DOSE-DEPENDENT DRUG ACTIVITY IN PG



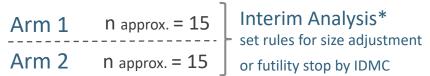
PG PHASE III STUDY DESIGN



Summary



ADAPTIVE DESIGN



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

Primary endpoint: complete target ulcer closure



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

INFLARX – SUMMARY

Targeting Complement to Control Inflammatory Diseases

INVESTMENT HIGHLIGHTS

InflaRx is a biotechnology company pioneering anti-inflammatory drug development in the terminal complement C5a / C5aR pathway and has developed highly active drug candidates for the target and the receptor:

- Vilobelimab is a first-in-class highly active fast acting anti-C5a antibody delivered i.v. with first commercial access
- INF904 is a new orally administered small molecule anti-C5aR inhibitor with best-in-class potential



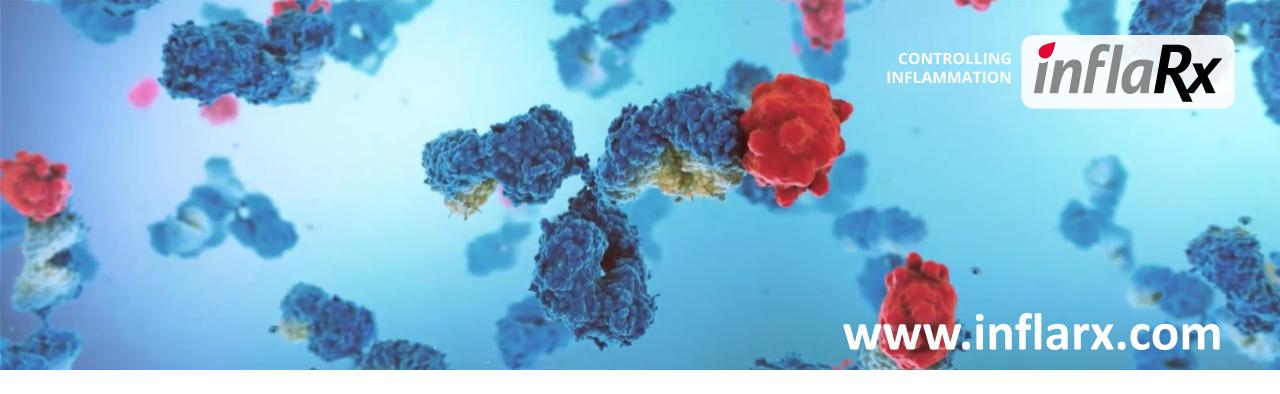
With these drugs, InflaRx targets various life-threatening and debilitating inflammatory diseases with a new mechanism, both in the acute/sub-acute as well as in the chronic disease setting

The developments are supported by strong core IP and medical use IP coverage for both molecules

Strong Cash Balance of € 113 M as of Q3 2023

Cash runway to fund development programs well into 2026





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