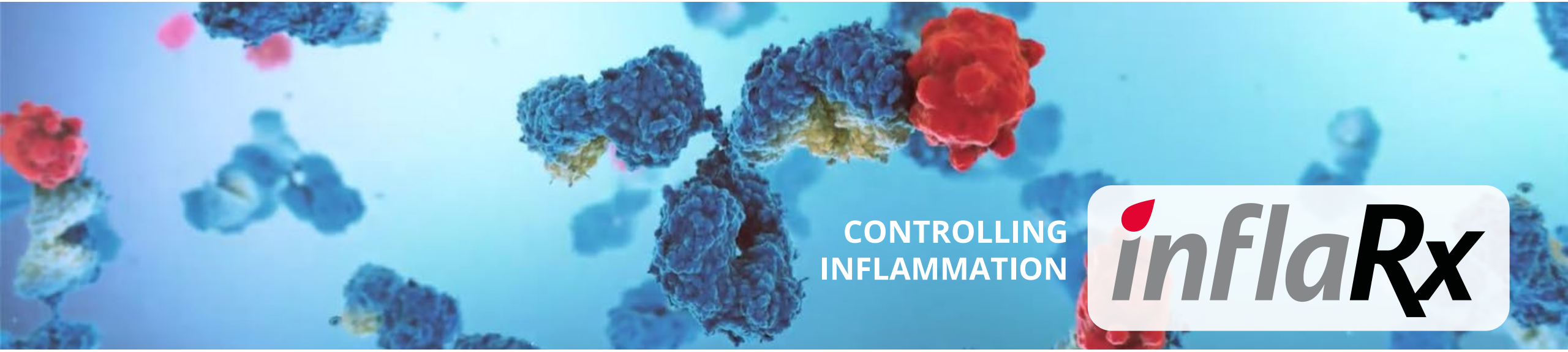
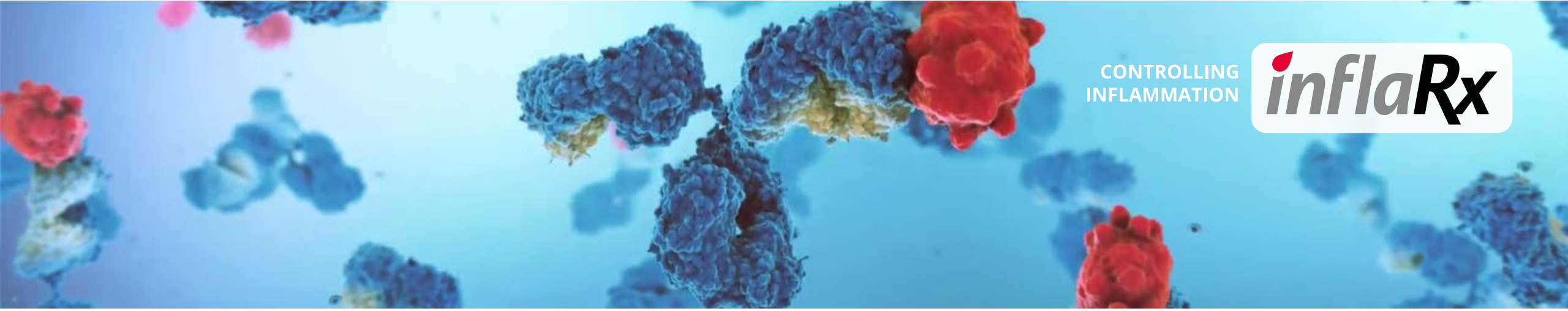


CORPORATE PRESENTATION

SEPTEMBER 2023



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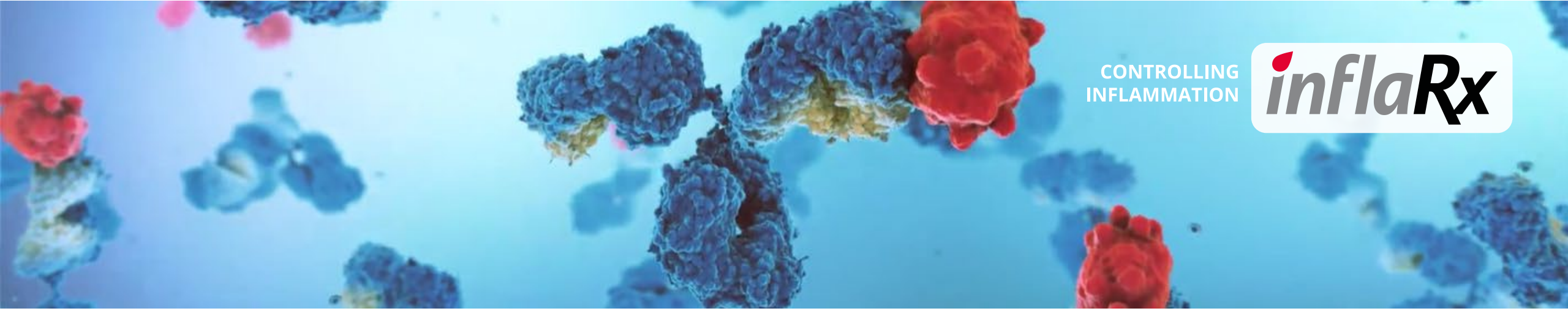
Forward-Looking Statements

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 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 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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Information and Sources

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

Avacopan Data

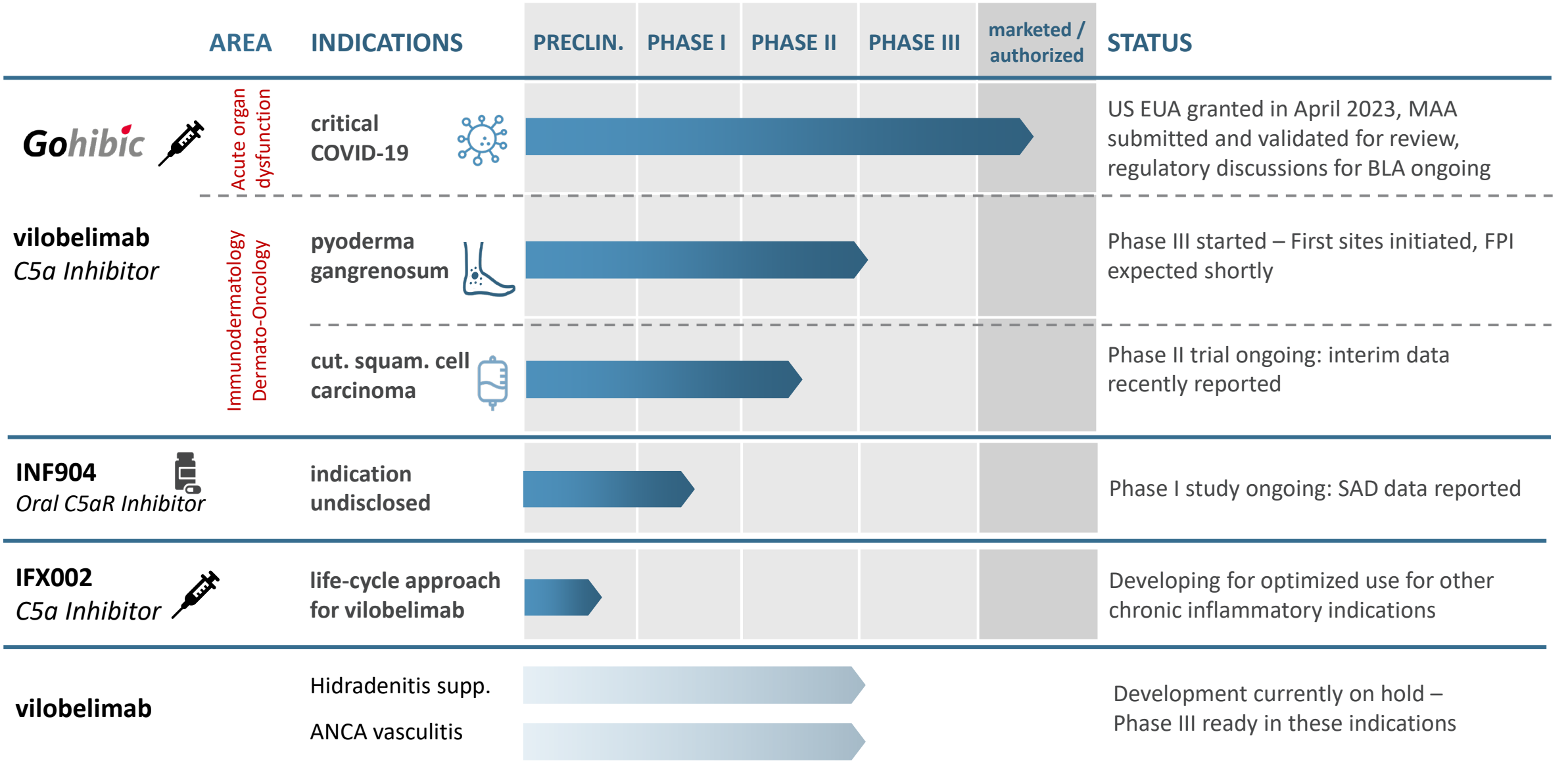
We have not conducted a head-to-head comparison of Avacopan to INF904 in a clinical trial but have compared the published data for Avacopan to data from our Phase 1 clinical trial of INF904. For the purpose of conducting pre-clinical studies (hamster neutropenia study), we synthesized Avacopan and did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

About InflaRx

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

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

Pipeline and Company Status Development Update



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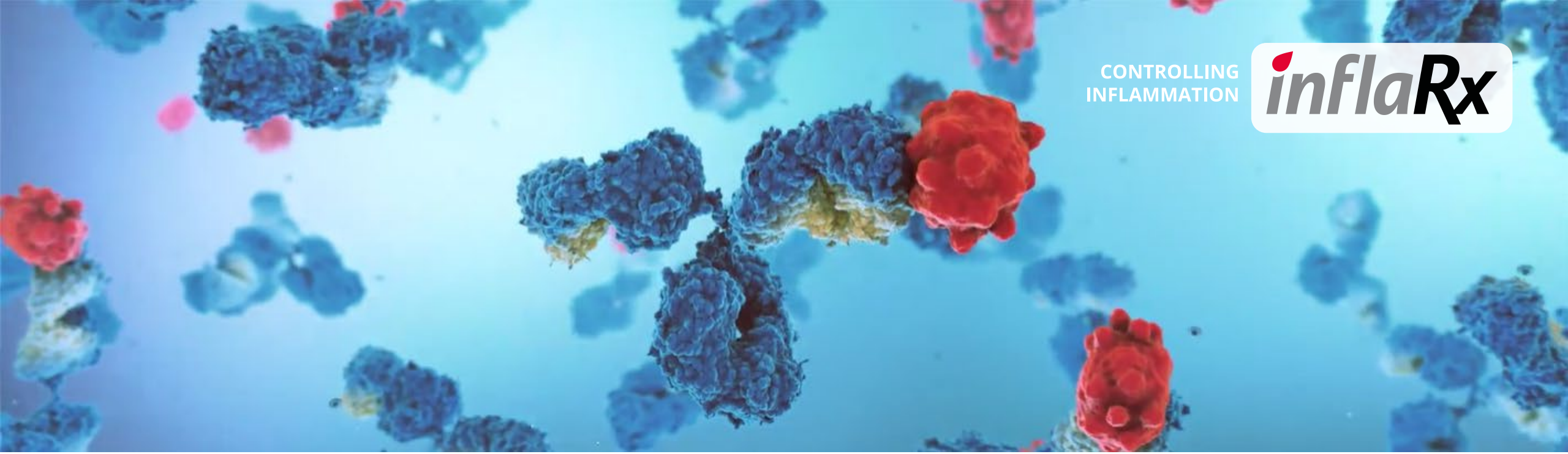
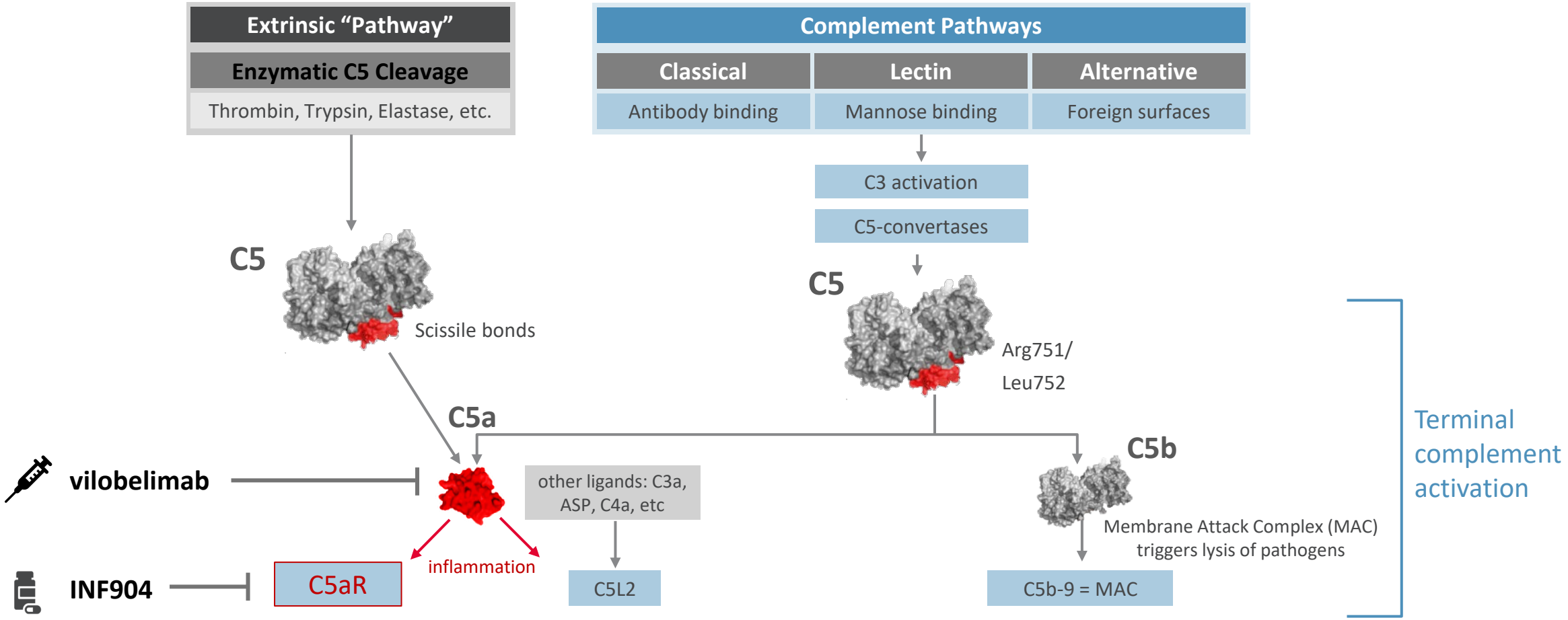


The Complement System and Our Targets

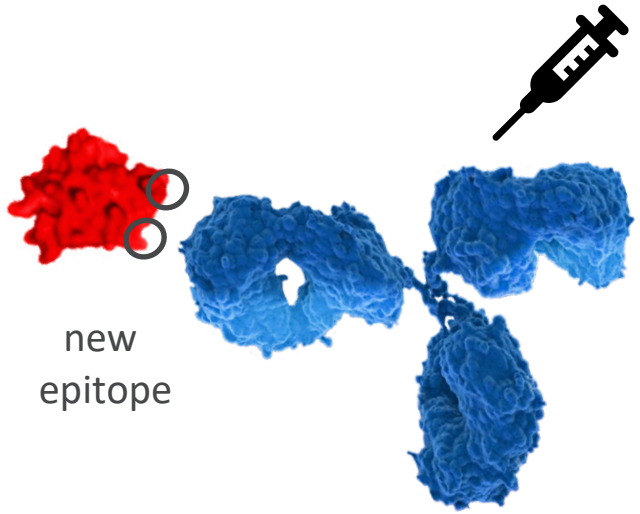
The Complement System and C5a Activation



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 Monoclonal Antibody directed against C5a

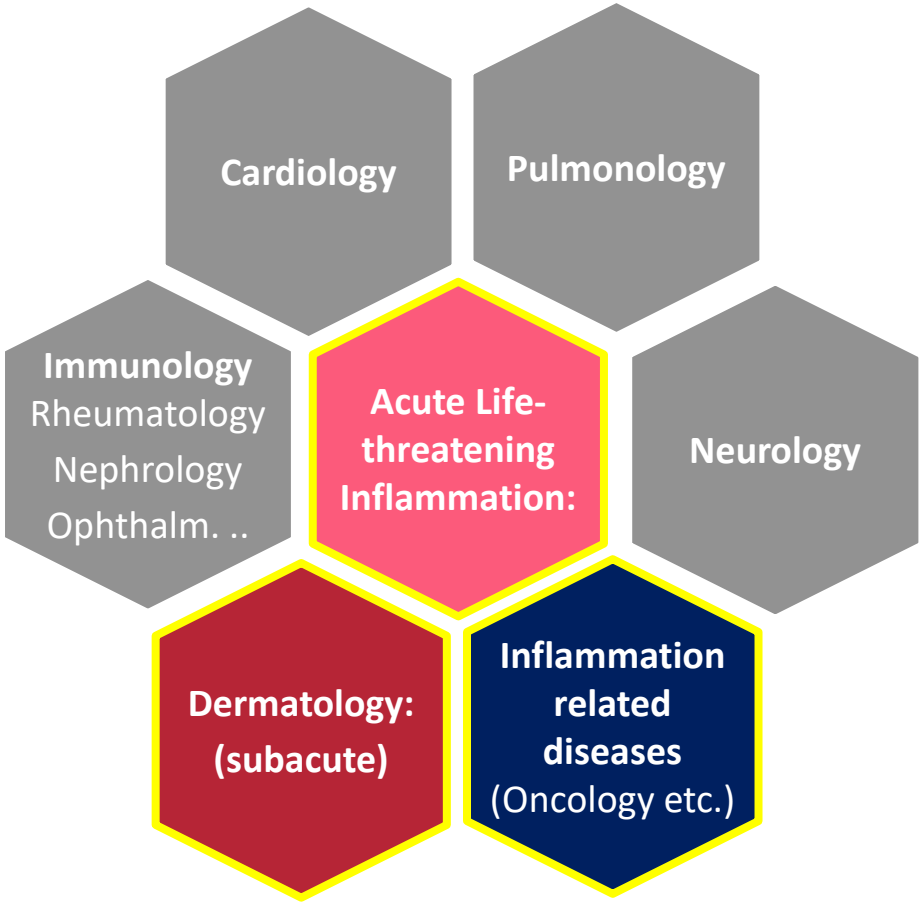
VILOBELIMAB (IFX-1)



Key Features vilobelimab:

- Blocks C5a biological effects up to 100% in human blood
- Highly selective – 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

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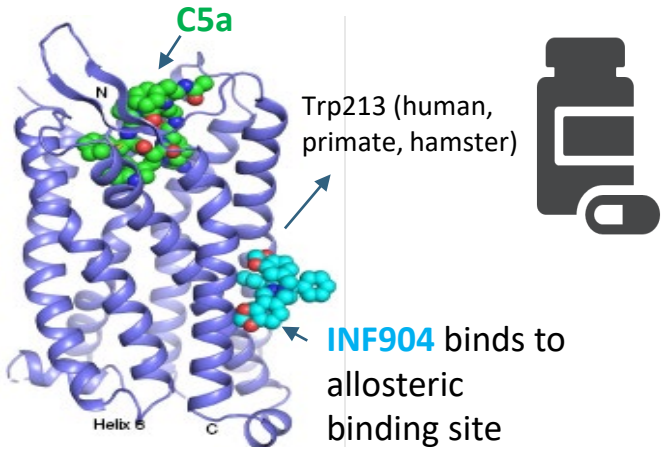
INF904 is a new Chemical Oral Inhibitor of C5aR

INF904

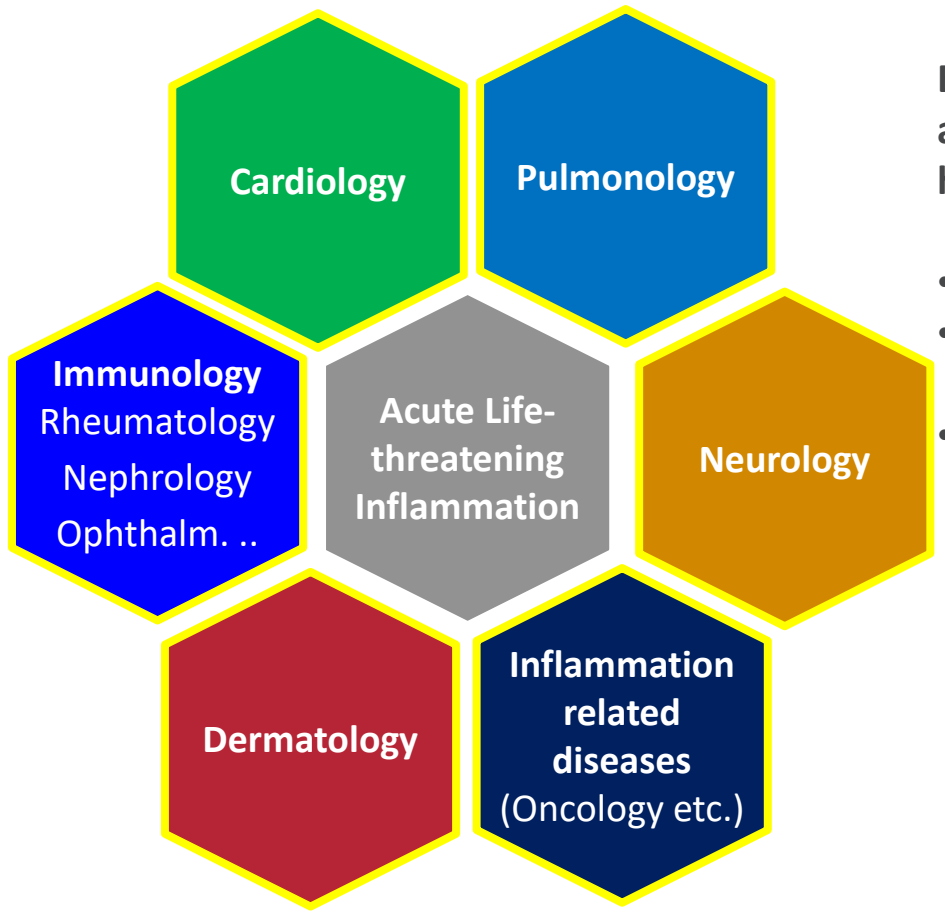


C5aR structure (Nathan Robertson, 2018 Nature)

Key Features INF904:

- Oral chemical inhibitor with new structure
- Best in class potential based on PK / PD profile observed in initial phase I single ascending dose study
- No obvious safety or tolerability concerns in initial phase I SAD study

POTENTIAL DEVELOPMENT AREAS IN CHRONIC INFLAMMATION



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

- Good tissue presence
- C5aR inhibition in various epithelial cells
- Deliver a sustained long-term control over C5a/C5aR activation in a variety of chronic inflammatory diseases in different tissues

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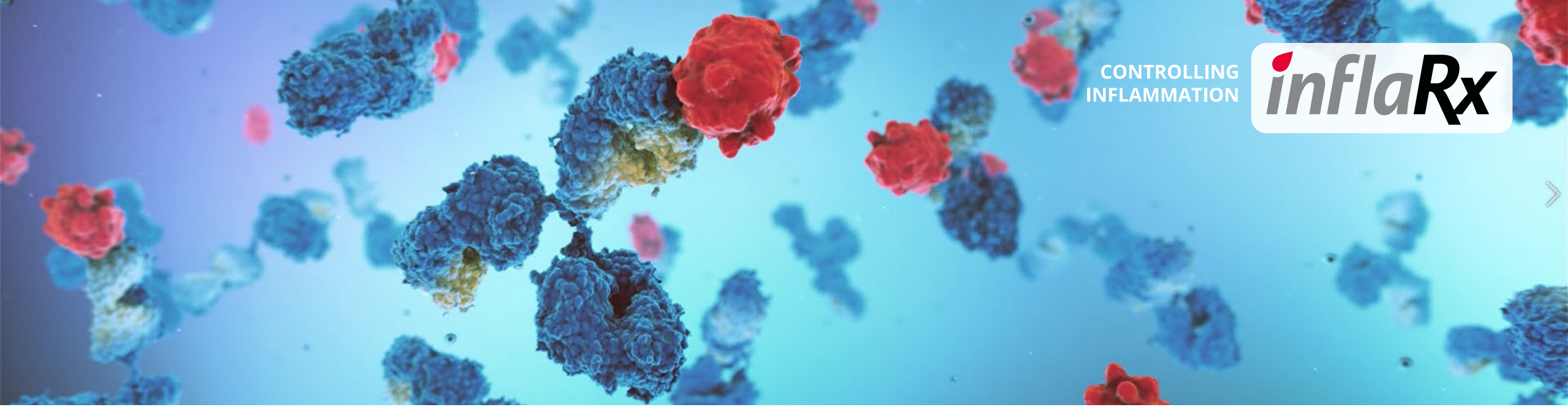
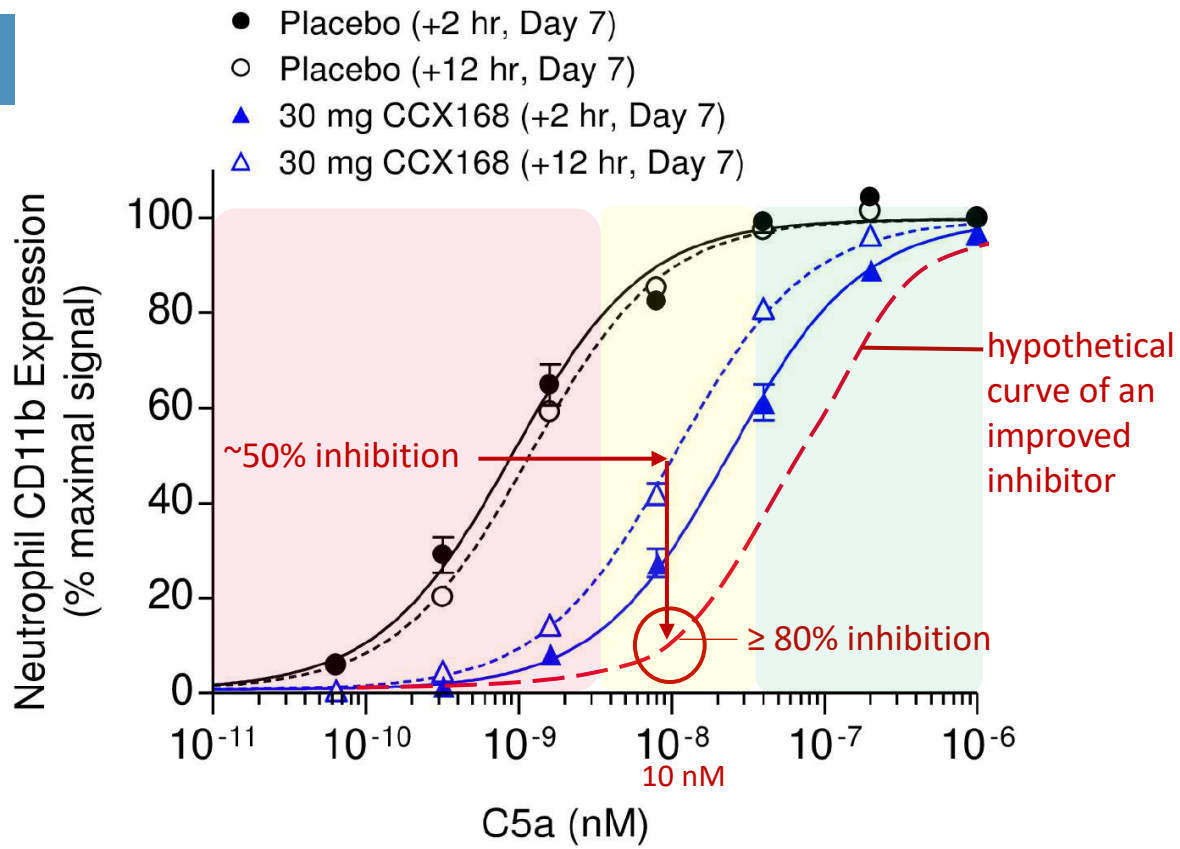


INF904
Orally Administered Low Molecular Weight C5aR Inhibitor

Improvement of C5aR Inhibition

PROPERTIES OF A BEST-IN-CLASS C5AR ANTAGONIST

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

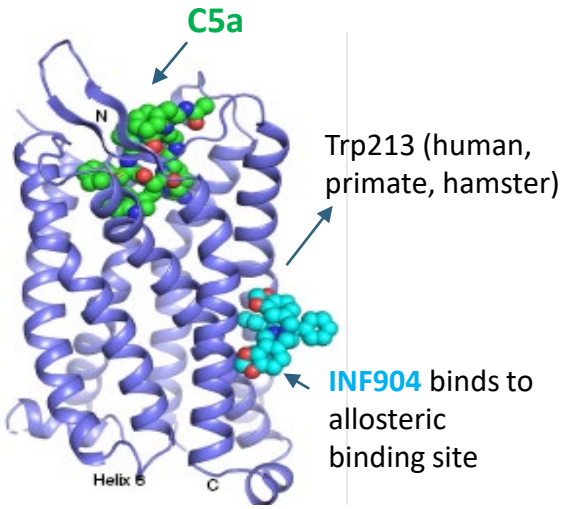


modified from Bekker et al. (2016, PLoS One; 11(10): e0164646); CCX168 = Avacopan; Whole blood ex vivo assay upon 7 days of 2 x qd dosing with Avacopan measuring up-regulation of CD11b on blood neutrophils upon challenge with addition of different levels of recombinant C5a. CD11b is a marker of neutrophil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

INF904: Pre-clinical Summary

INF904 FACTS

- INF904 binds to a well-defined allosteric site in C5aR
- INF904 has a novel molecular structure
- US patent was issued in October 2021;
  national phases for other select countries (PCT)



C5aR structure
(Source: Nathan Robertson, 2018 Nature)

PRE-CLINICAL FINDINGS

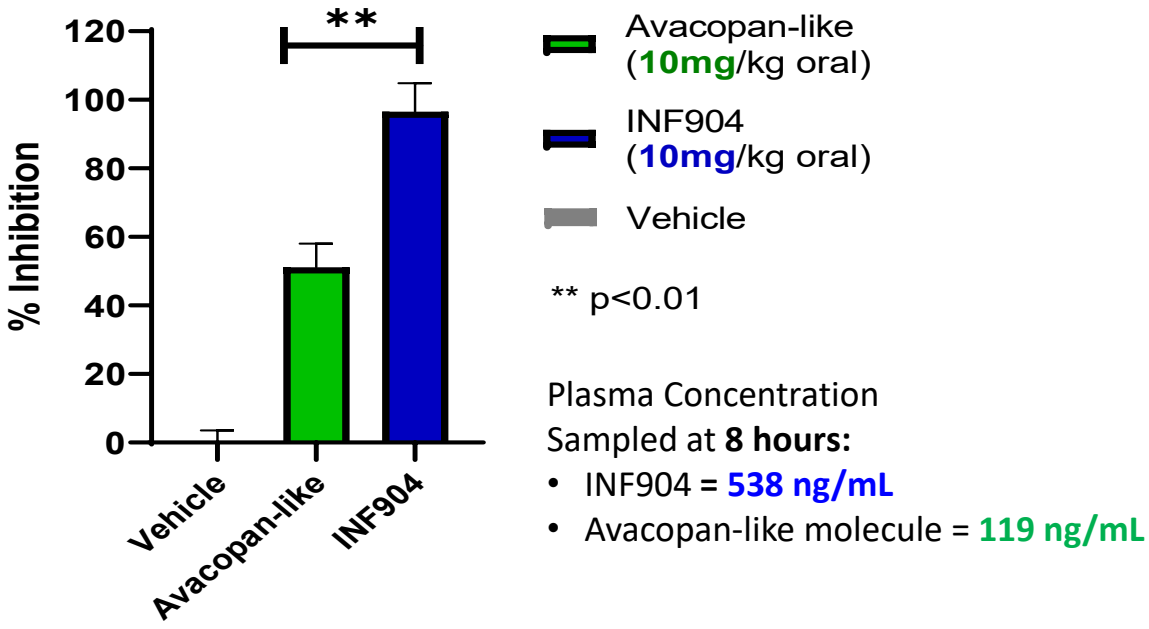
- **No toxicity findings** even in the highest dose groups (rat and monkey; up to 300 mg/kg)
- **High in vitro potency** with a desired IC50 (<1nM) in calcium mobilization assay
- **Higher plasma exposures** in several in vivo models when compared to Avacopan*
- **Increased efficacy** in hamster neutropenia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

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

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*



Plasma Concentration Sampled at **8 hours:**

- INF904 = **538 ng/mL**
- Avacopan-like molecule = **119 ng/mL**

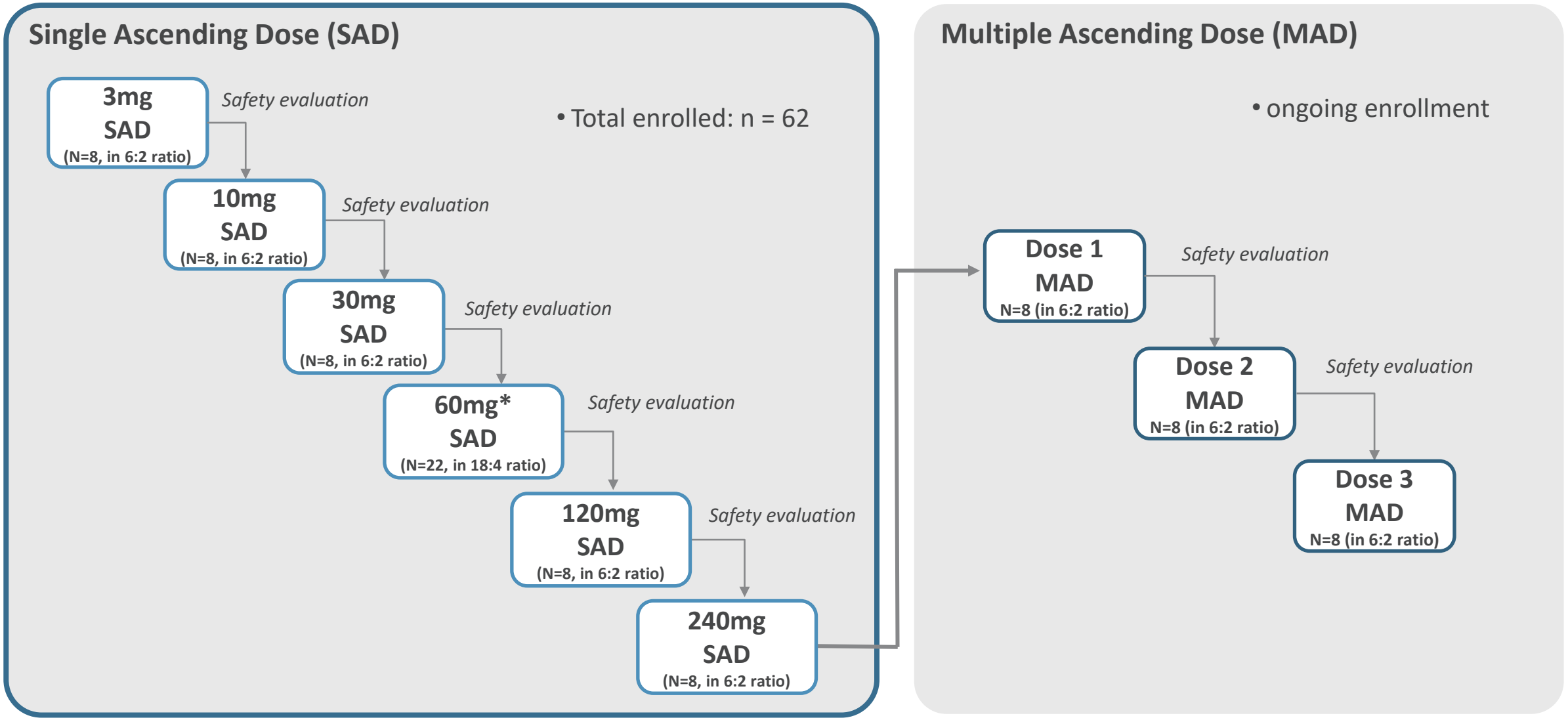
Experiment: Challenge of rodents with C5a leads to neutrophil activation and consequent adherence (sticking) of neutrophils to the endothelial cell wall of vessels = mimicking a neutropenia (vehicle). This effect can be completely inhibited when C5aR activation is blocked.

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*

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

INF904 Phase I Study Design



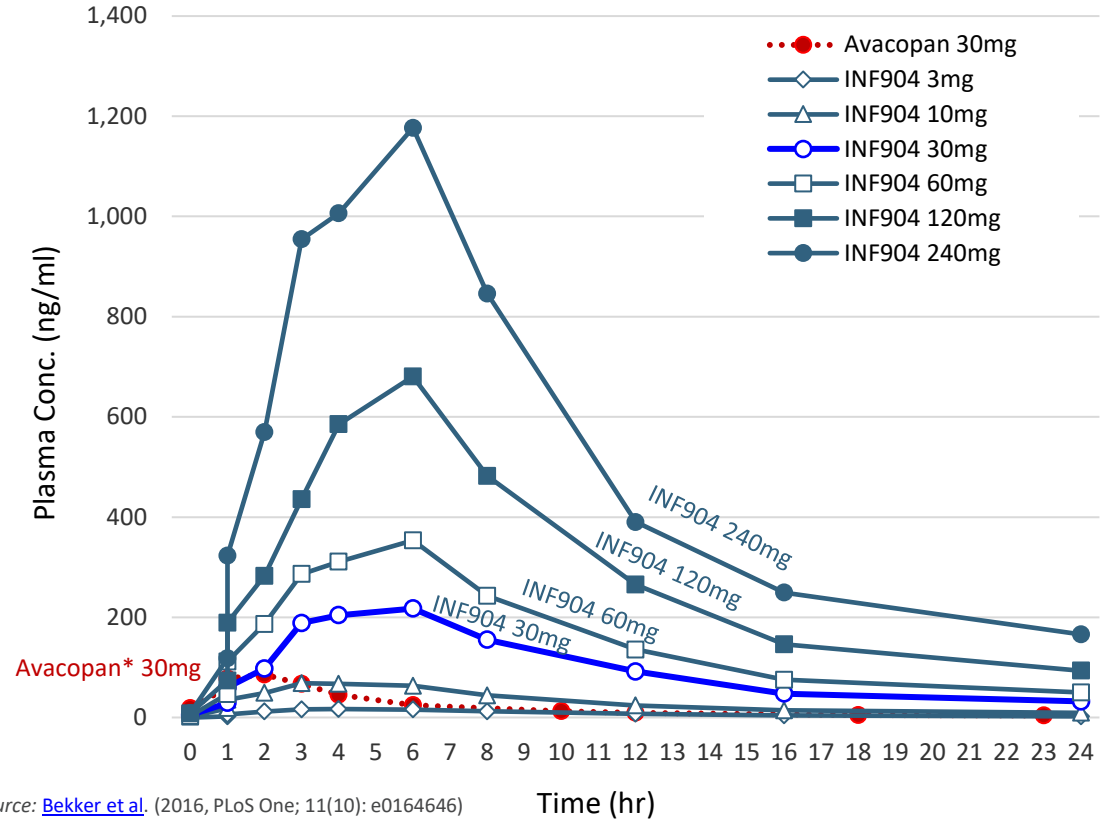
Footnotes: N= Subjects per dosing cohort, Ratio for randomization is shown for INF904 : matching Placebo; *3 different capsule strengths tested in a cross-over design

INF904 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 Phase I Study: PK Results from SAD Part



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)

***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_{max} and 10-fold higher in systemic exposure (AUC_{last}) for comparable doses (3, 10, 30 mg)

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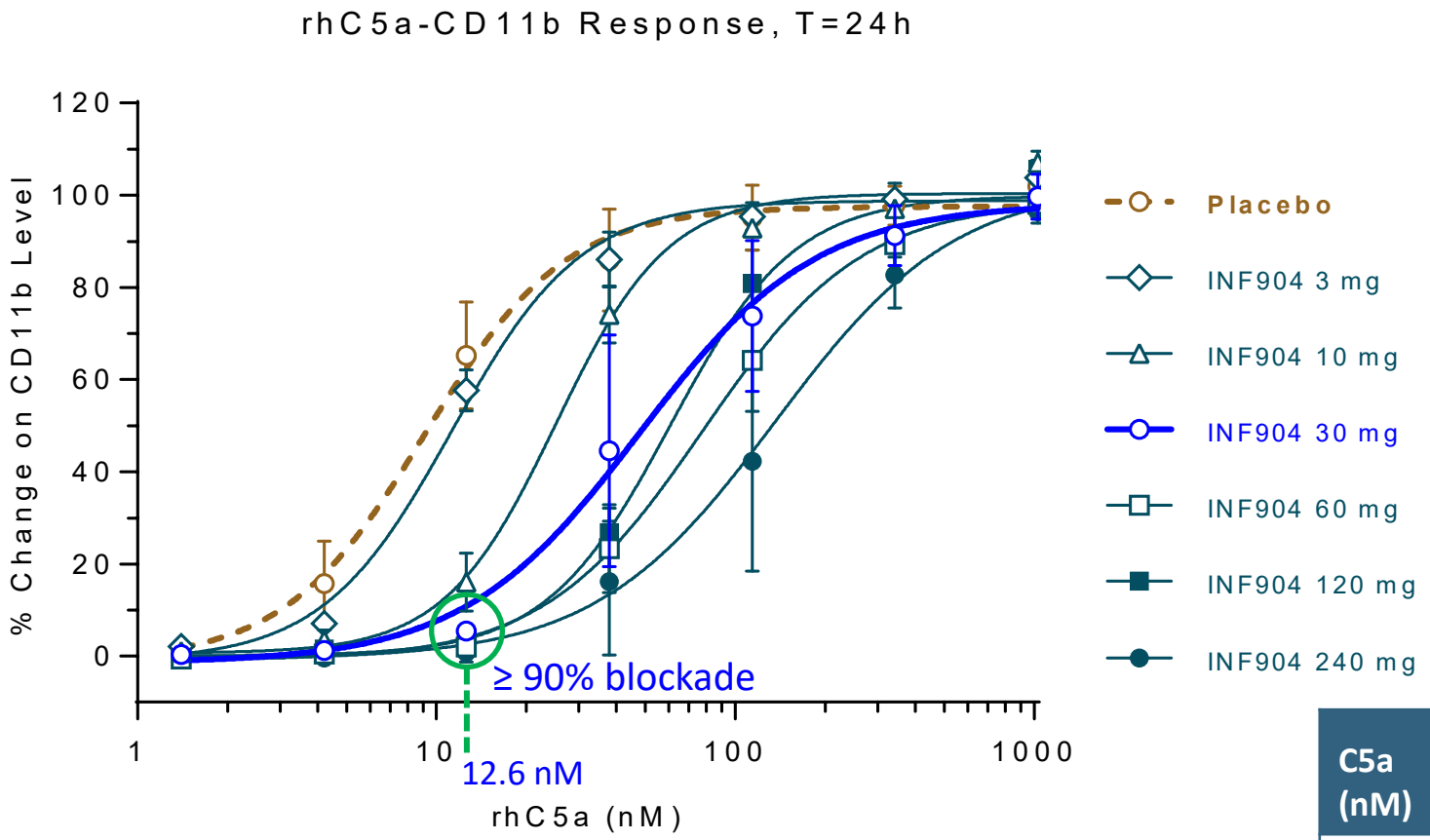
INF904 Phase I Study: PD Results from SAD Part

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



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

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

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

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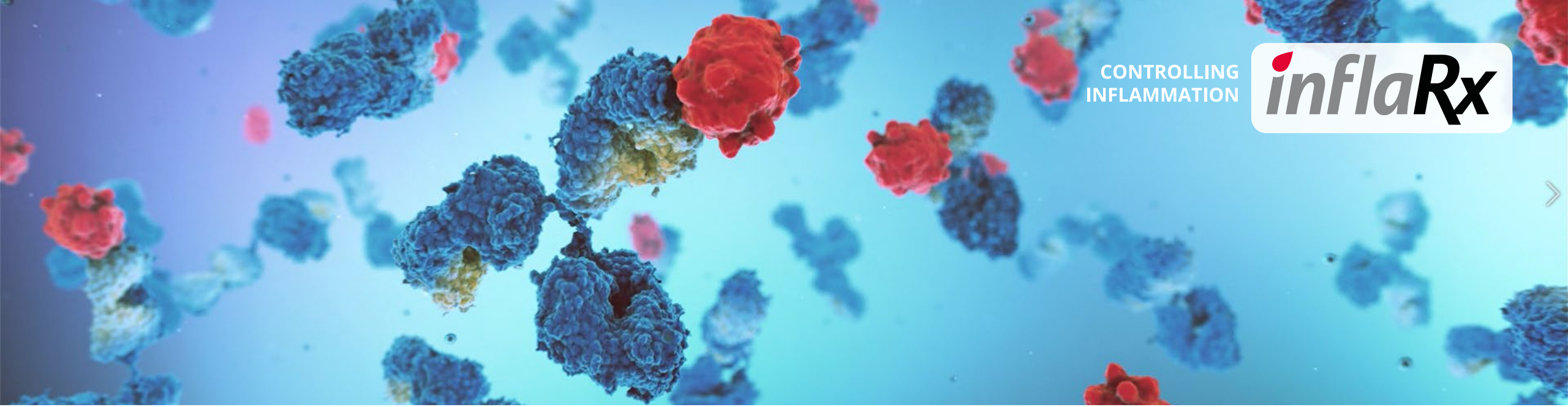
Summary

Topline Results from INF904 Phase I SAD Study

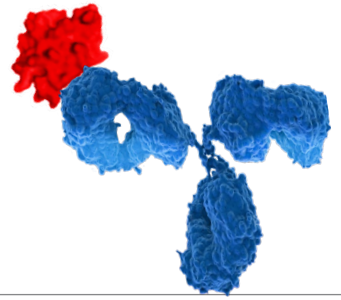
KEY OUTCOMES

- 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

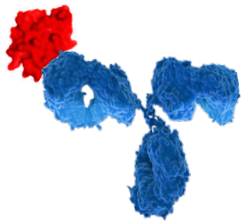


Gohibic (vilobelimab)
Critical COVID-19



Gohibic

Emergency Use Authorization (EUA) granted for Gohibic (vilobelimab)



Gohibic

- Gohibic 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

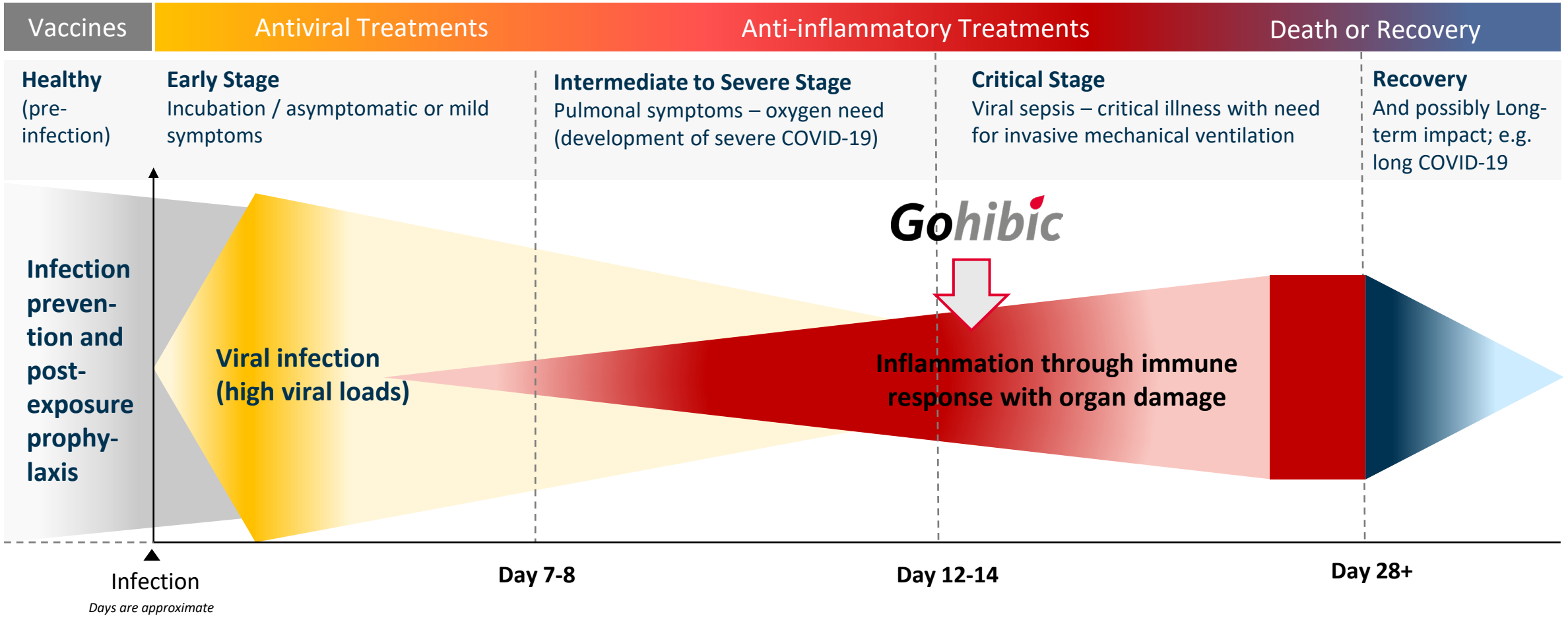
For additional and important safety information, please visit www.gohibic.com.

** 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

Gohibic



Gohibic is authorized for the treatment of COVID-19 in hospitalized adults when initiated within 48h of receiving IMV or ECMO

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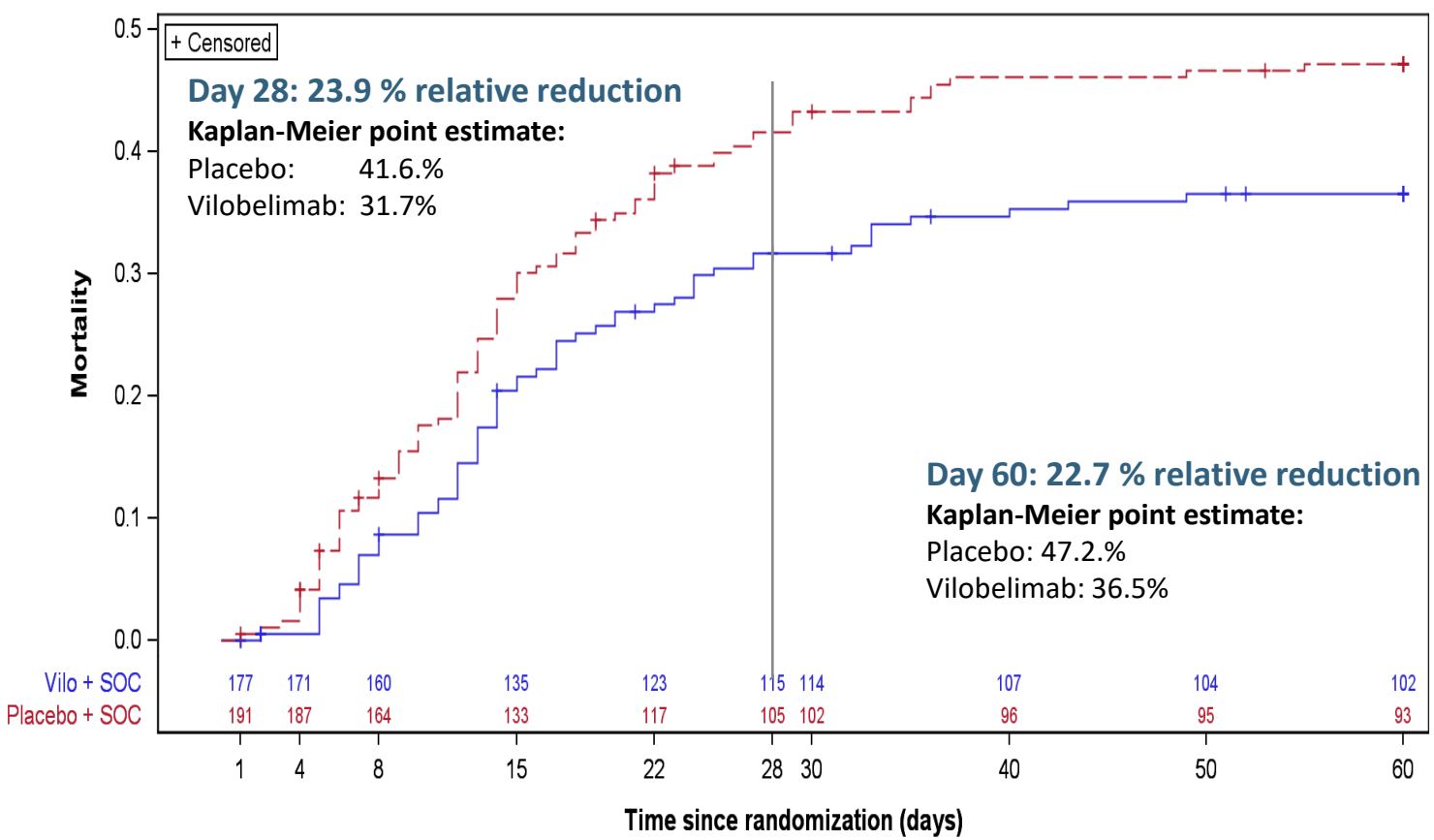
PANAMO Trial: Phase III – Primary Endpoint – *1:1 randomized, double-blind, placebo-controlled, multi-national trial in (n=369) invasive mechanically ventilated COVID-19 patients*

All-cause mortality: Overall



Placebo + SOC

Vilobelimab + SOC

SOC included concomitant use:

- Corticosteroid use: 97%
- Anti-coagulant use: 98%
- Biologics & other (tocilizumab > baricitinimb): 20% (prior or concomitant)

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

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PANAMO Trial: Phase III – Secondary Endpoint

Need for Renal Replacement

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

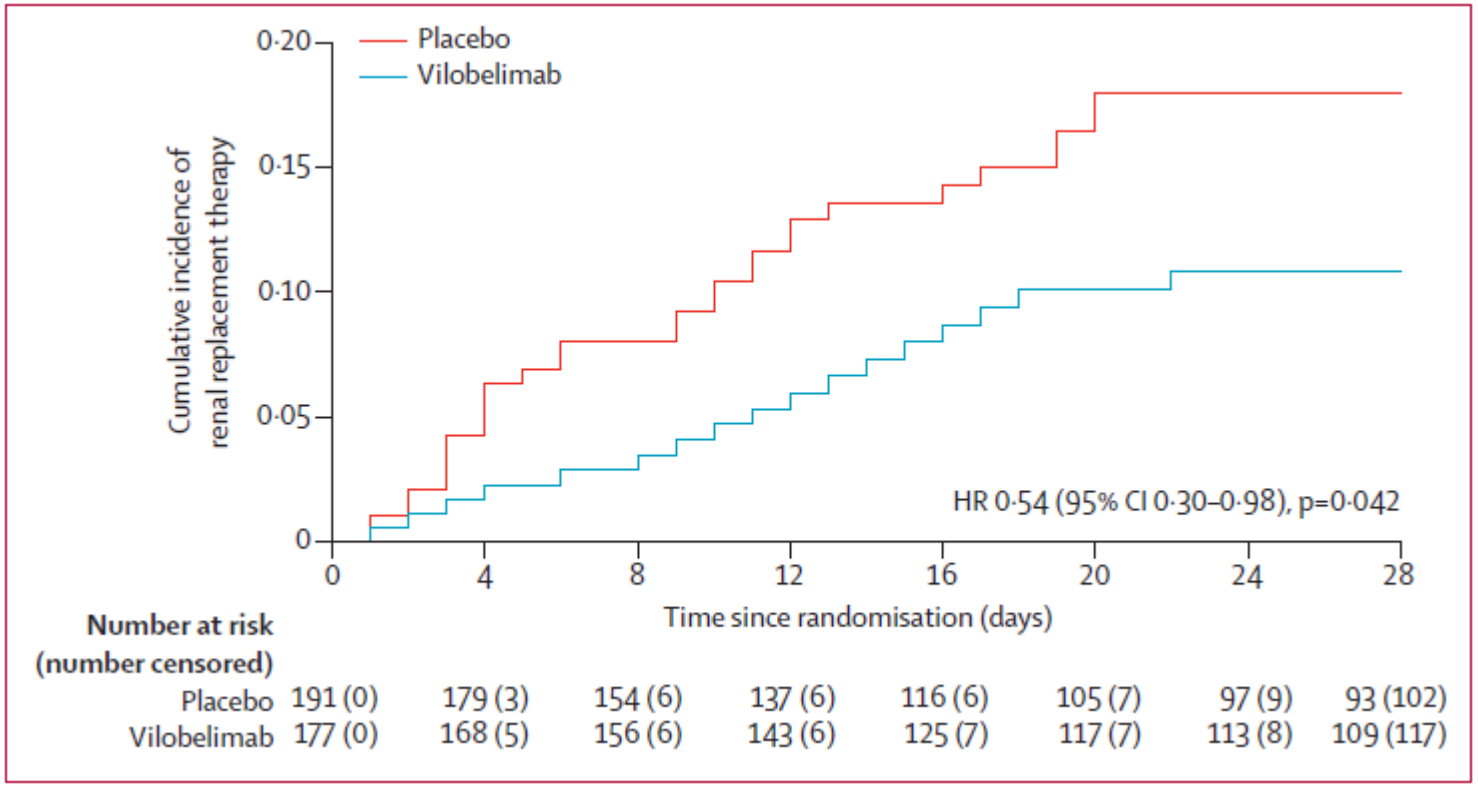

Figure from Vlaar, A et al. Lancet Resp Med 2022. https://doi.org/10.1016/ S2213-2600(22)00297-1

More patients were free of renal replacement need within 28 days in vilobelimab arm compared to placebo

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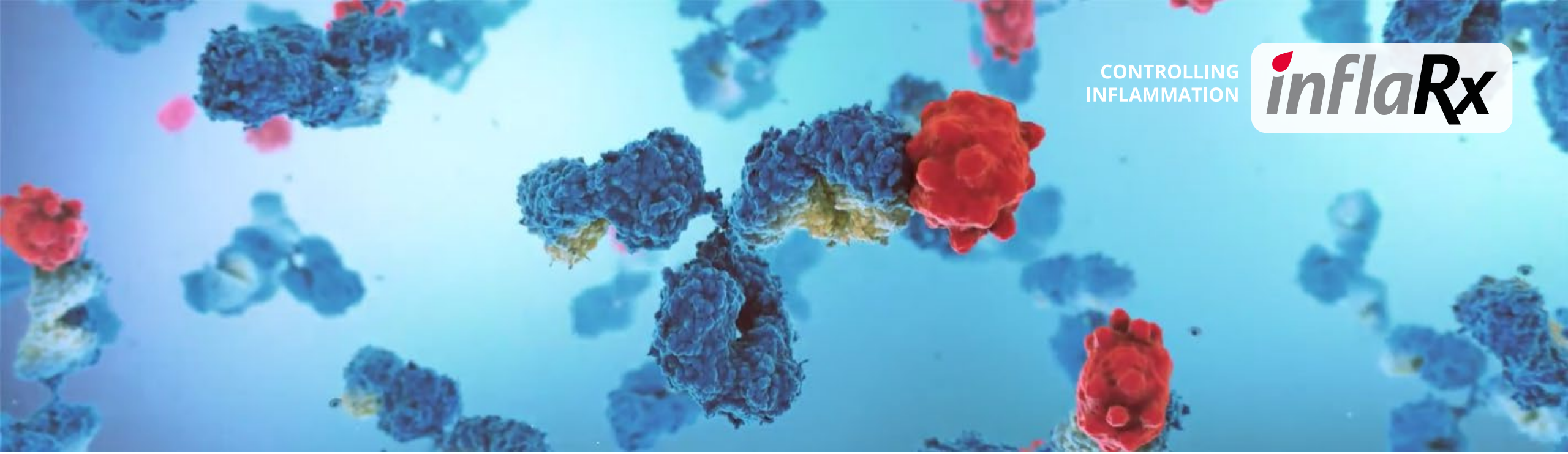
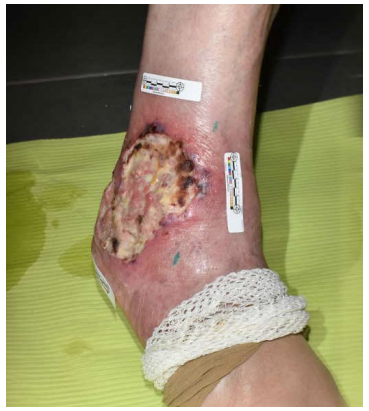
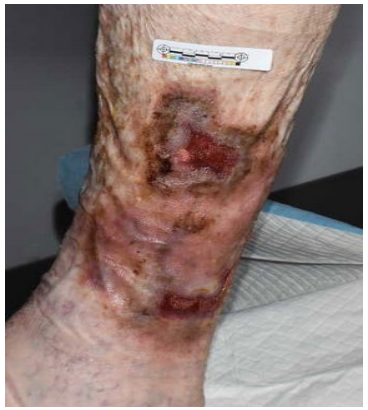


Vilobelimab
Pyoderma Gangrenosum (PG)

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

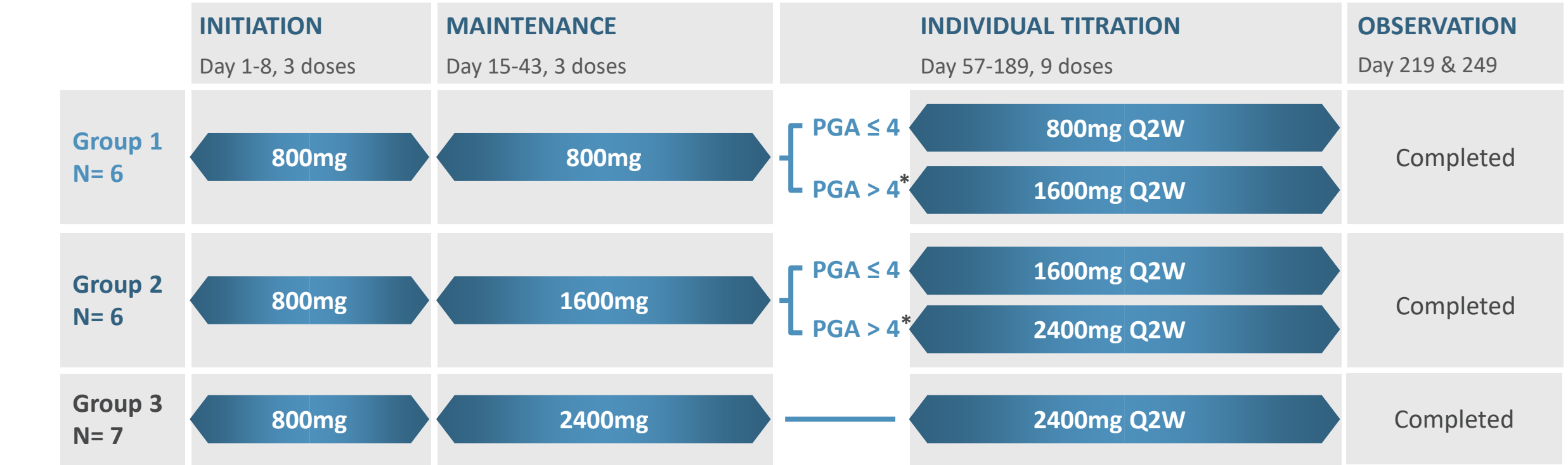
Strong rationale for treatment with vilobelimab:
PG associated with neutrophilic skin infiltration in affected areas and lesions, potentially triggered by C5a

Photo Source: InflaRx study

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

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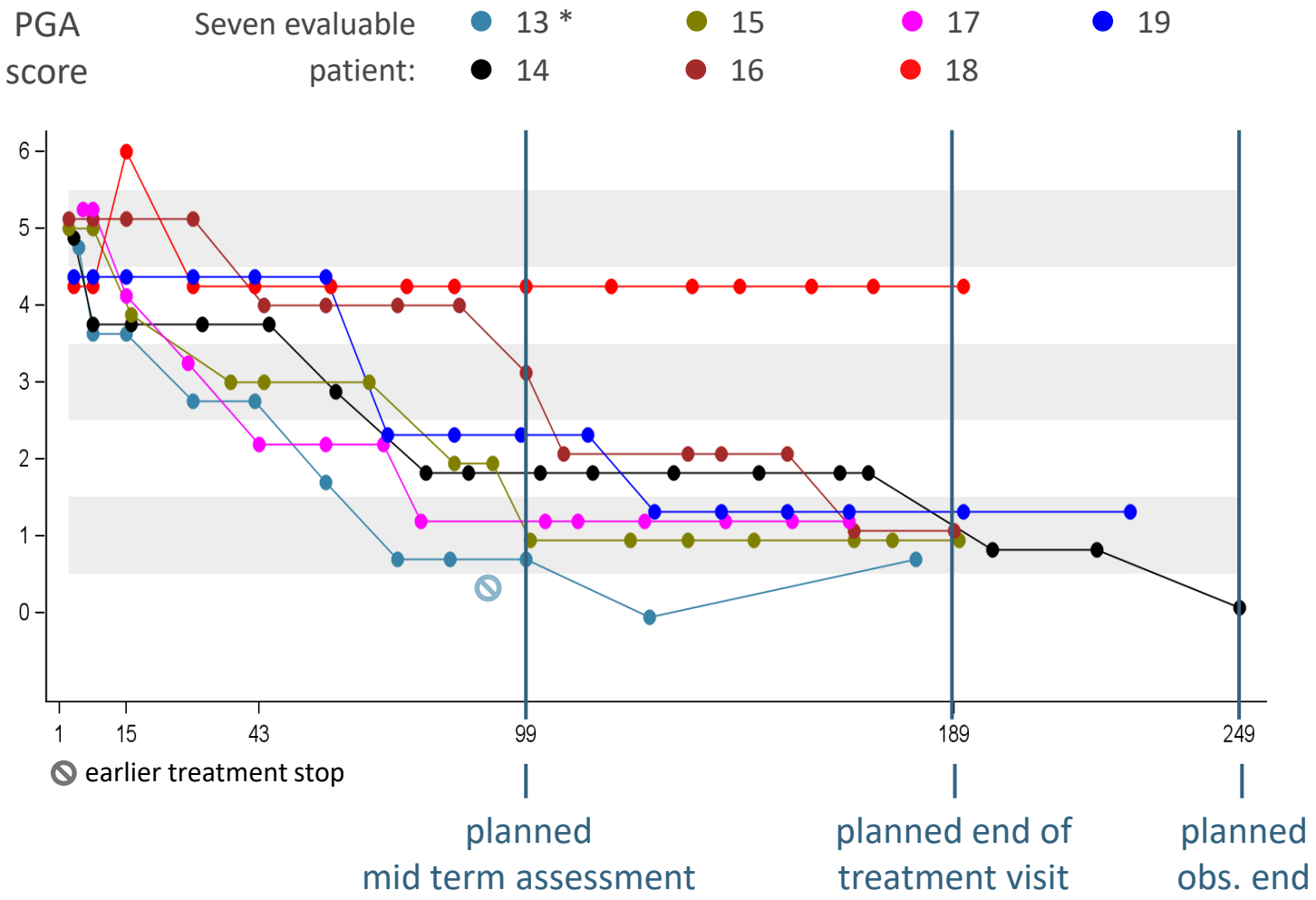
Phase IIa Study Results – Group 3 (High Dose)



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)

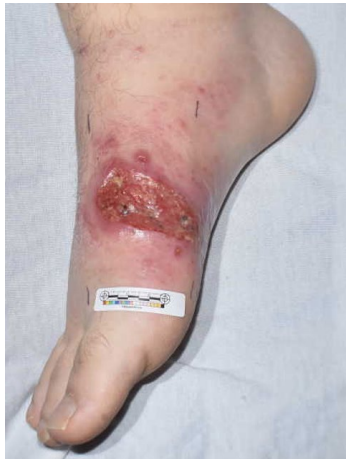
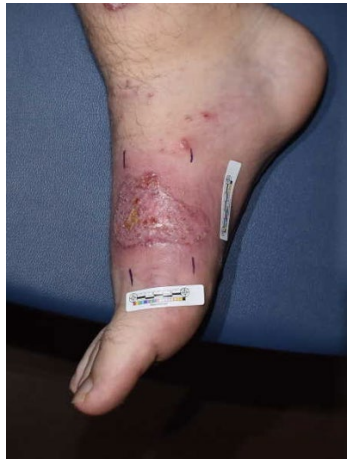
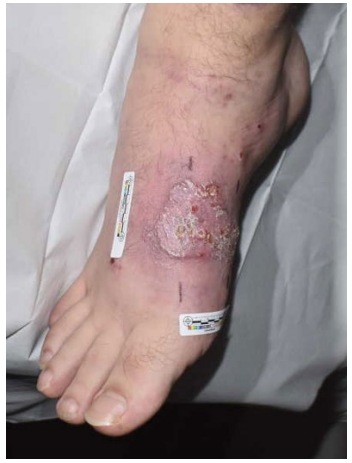
PGA line plot of absolute values over time by patient (Actual days displayed acc. to visit windows)
* Patient 13 was discontinued from treatment after day 71 due to delayed availability of pos. baseline TB testing result (exclusion criterion). No re-activation of TB reported up to end of observation.

PG Phase IIa

Patient Case Studies

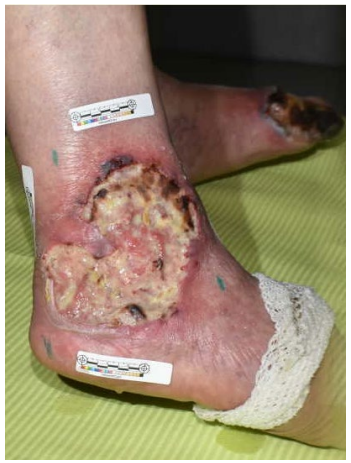
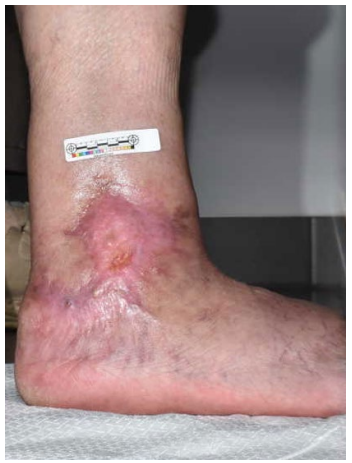
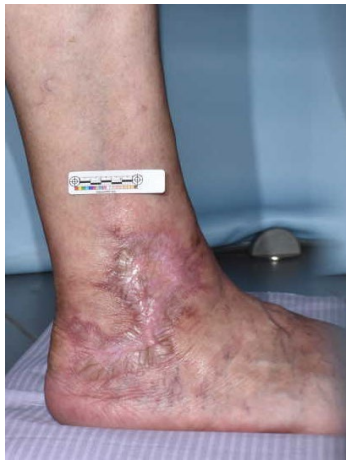
TARGET ULCER DEVELOPED WHILE ON ADALIMUMAB

- **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

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 2020

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

PG Phase 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 AEs 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%)**

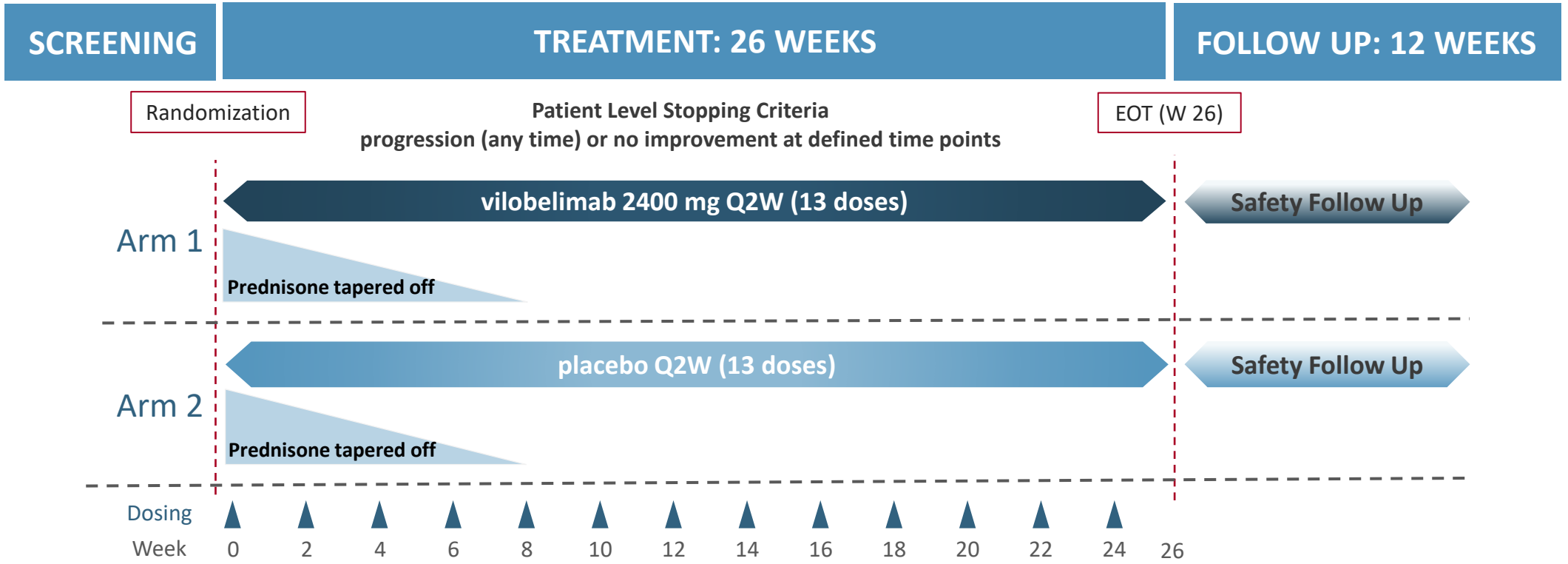
PHASE III INITIATED BASED ON FEEDBACK FROM FDA

ORPHAN DRUG AND FAST TRACK STATUS US FDA

ORPHAN DRUG STATUS EMA

Vilobelimab Q2W resulted in no safety or tolerability concerns and showed evidence of dose-dependent drug activity in PG

PG Phase III Study Design



Adaptive Design

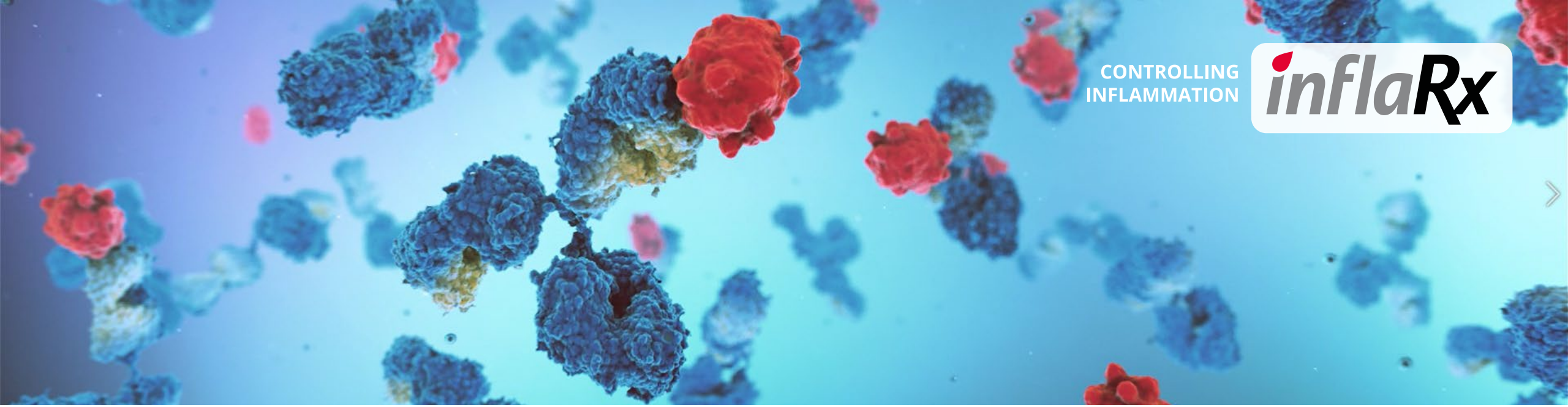
Arm 1 n approx. = 15
Arm 2 n approx. = 15

Interim Analysis*
set rules for adaptation or stop for futility by IDMC

n adapted to appr. 25 – 50 patients
n adapted to appr. 25 – 50 patients

Primary endpoint: complete target ulcer closure

* Blinded except for independent data safety monitoring committee



Vilobelimab
Oncology

Cutaneous Squamous Cell Carcinoma (cSCC)

Cutaneous Squamous Cell Carcinoma (cSCC)

Phase II Study Underway

POTENTIAL ROLE OF C5A IN ONCOLOGY & cSCC

- **C5a induces an immunosuppressive tumor microenvironment**
- **C5a promotes metastases**
- **C5a is readily available in the tumor environment and may promote tumor growth directly**

PRIMARY ENDPOINTS

- Arm A: Assess safety and antitumor activity of vilobelimab
- Arm B: Determine maximum tolerated dose (MTD) or recommended Phase II dose (RP2D); assess antitumor activity and safety profile of vilobelimab + pembrolizumab

DISEASE INFORMATION cSCC

- **Estimated incidence: 15-35 per 100,000 people**; expected to increase 2-4% per year; **metastasizes in approximately 2-5%** of cases[1,2,4]
- Advanced SCC 10-year survival rates <**20%** with regional lymph node involvement and **<10%** with distant metastases; distant metastases have median survival of <2 years[1,3]
- PD-1 checkpoint inhibitors pembrolizumab or cemiplimab are FDA approved for locally advanced, recurrent or metastatic cSCC (see FDA labeling)
- No treatment is available for PD-1 or PD-L1 resistant or refractory patients

1. Stratigos et al., 2015; 2. Burton et al., 2016; 3. Hillen et al., 2018; 4. Li et al, 2015

Inhibition of C5a signaling in the tumor microenvironment may decrease disease progression
Combination of C5a with PD-1 checkpoint inhibition could reverse resistance to PD-1 or PD-L1 inhibitor therapy

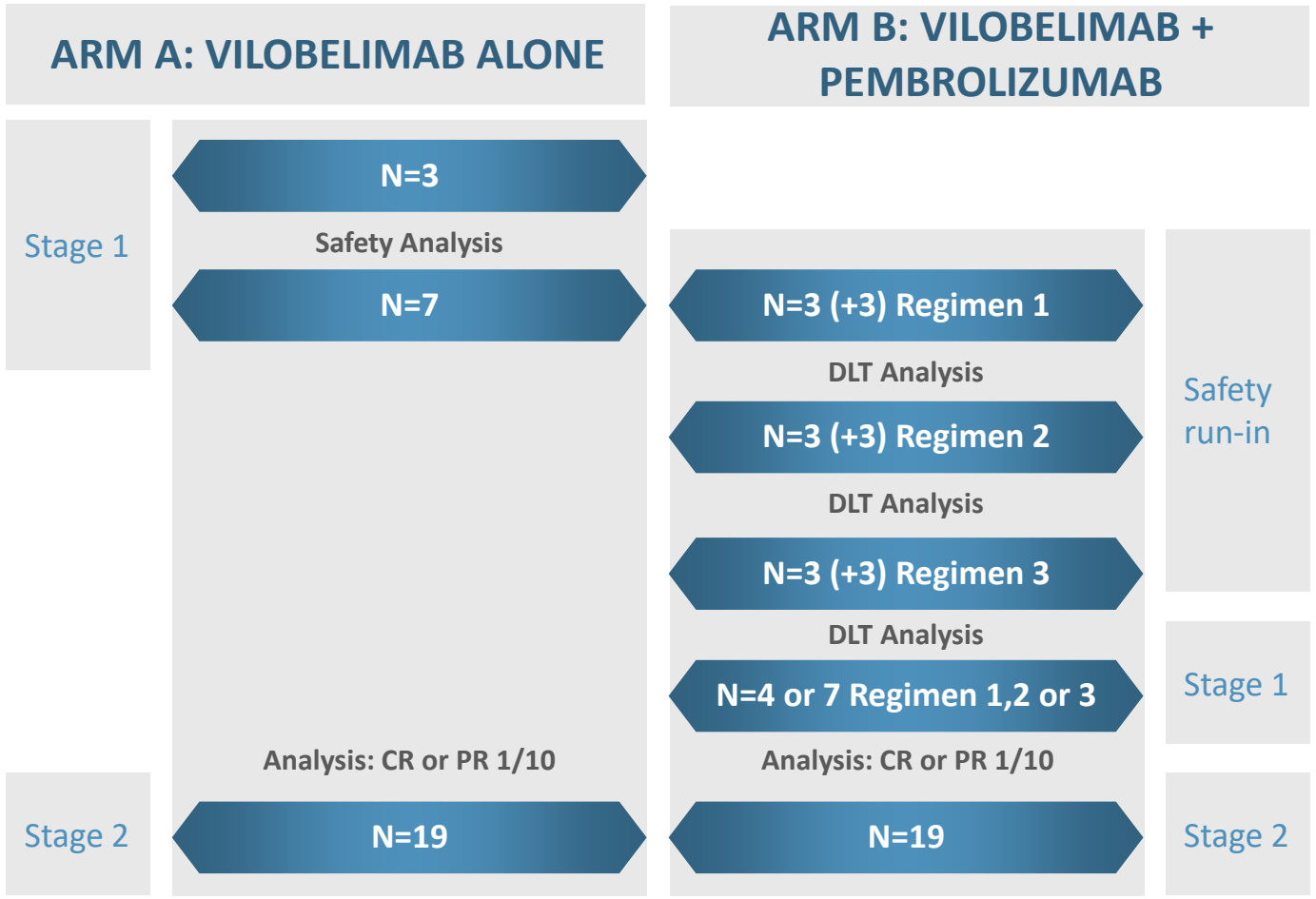
Cutaneous Squamous Cell Carcinoma (cSCC): Phase II Study Underway

INCLUSION CRITERIA

- Locally advanced or metastatic cSCC
- Refractory or resistant to PD-1 or PD-L1 inhibitor
- Locally advanced cSCC not amenable to curative treatment
- Metastatic cSCC resistant to all approved therapies

PRIMARY ENDPOINTS

- **Arm A:**
  Assess antitumor activity of vilobelimab as stand-alone therapy
- **Arm B:**
  Determine maximum tolerated dose (MTD) or recommended Phase II dose (RP2D)
  Assess antitumor activity and safety profile of vilobelimab + pembrolizumab



DLT: Dose limiting toxicity; CR: complete response; PR: partial response

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cSCC: Interim Data from the Mono-Therapy Group in Arm A

Summary

SAFETY CONCLUSION

- 10 patients enrolled in the monotherapy arm (1600 mg dose)
- No major safety issues identified

CLINICAL RESPONSE CONCLUSION

- Initial signs of responses detected in 2/10 patients (who are elderly and refractory to previous PD-1 inhibitor treatments)
  1. Complete Response, n=1 (patient ongoing in study)
  2. Stable Disease, n=1 (patient ongoing in study)

- Note: an additional patient had a definition of Stable Disease (SD) according to the RECIST* criteria

*RECIST: Response Evaluation Criteria in Solid Tumors

InflaRx N.V. - Summary

Targeting Complement to Control Inflammatory Diseases

INVESTMENT HIGHLIGHTS

InflaRx aims to be the leading biotechnology company in the terminal complement C5a / C5aR pathway and has developed highly active drug candidates:

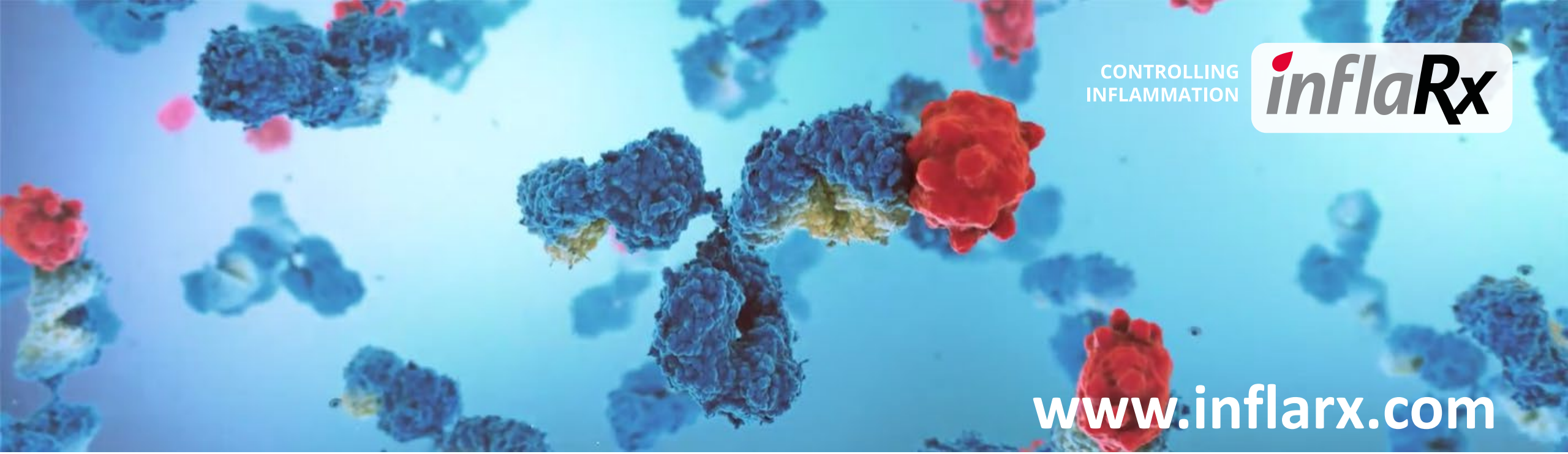
- **Vilobelimab is a first-in-class highly active fast acting anti-C5a antibody delivered i.v. with first commercial access**
- **INF904 is a new chemical oral anti-C5aR inhibitor with best-in-class potential**

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 €115.2 M as of June 30, 2023 (Q2)

- **Cash balance includes cash, cash equivalents and marketable securities**



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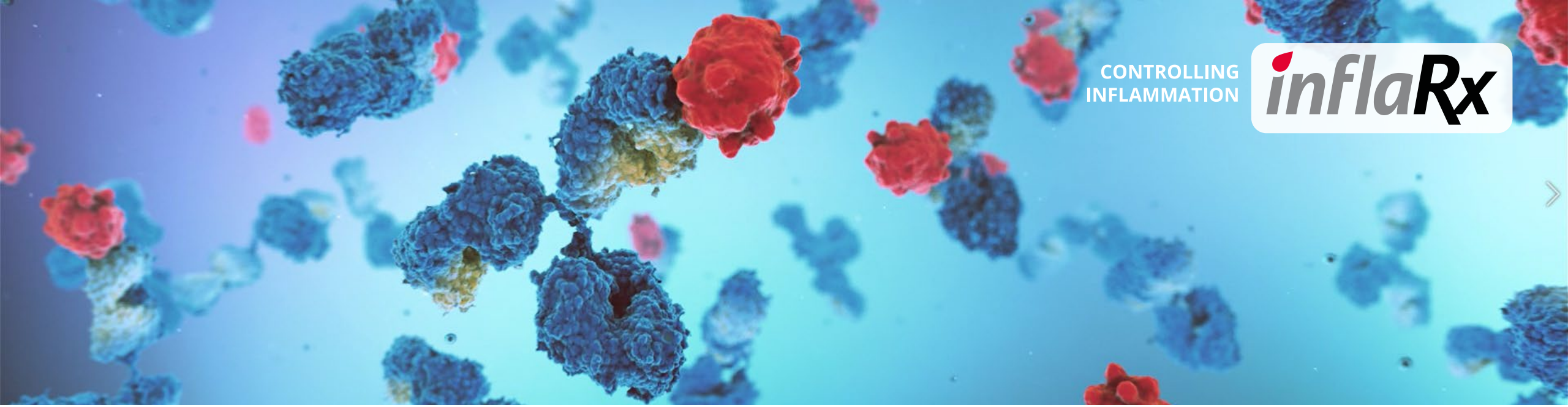


Appendix

Vilobelimab Key Clinical Data: Evidence for an Active Drug

PYODERMA GANGRENOSUM (PG)

Phase IIa trial:

- High Dose Group shows highest rate of target ulcer closure and clinical remission (85.7%)
- Evidence of dose-dependent drug activity in PG
- Vilobelimab 2400mg Q2W shows good safety and tolerability (no dose-related AE detected)
- Advancing into pivotal Phase III following detailed discussions with FDA

HIDRADENITIS SUPPURATIVA (HS)

Phase II SHINE trial:

- Significant difference between the total inflammatory lesion count (ANdT) reduction in the highest dose cohort compared with placebo (no signal on primary endpoint HiSCR with high placebo response rate)
- Increased dose required
- Vilobelimab treatment was well-tolerated (up to 44 weeks)

COVID-19 EUA GRANTED IN 04/2023

PANAMO Phase II/III trial – Phase III results:

- Significant 28-day mortality reduction detected (23.9% relative reduction) in global data set
- Vilobelimab treatment was well-tolerated

ANCA-ASSOCIATED VASCULITIS

Phase II IXCHANGE:

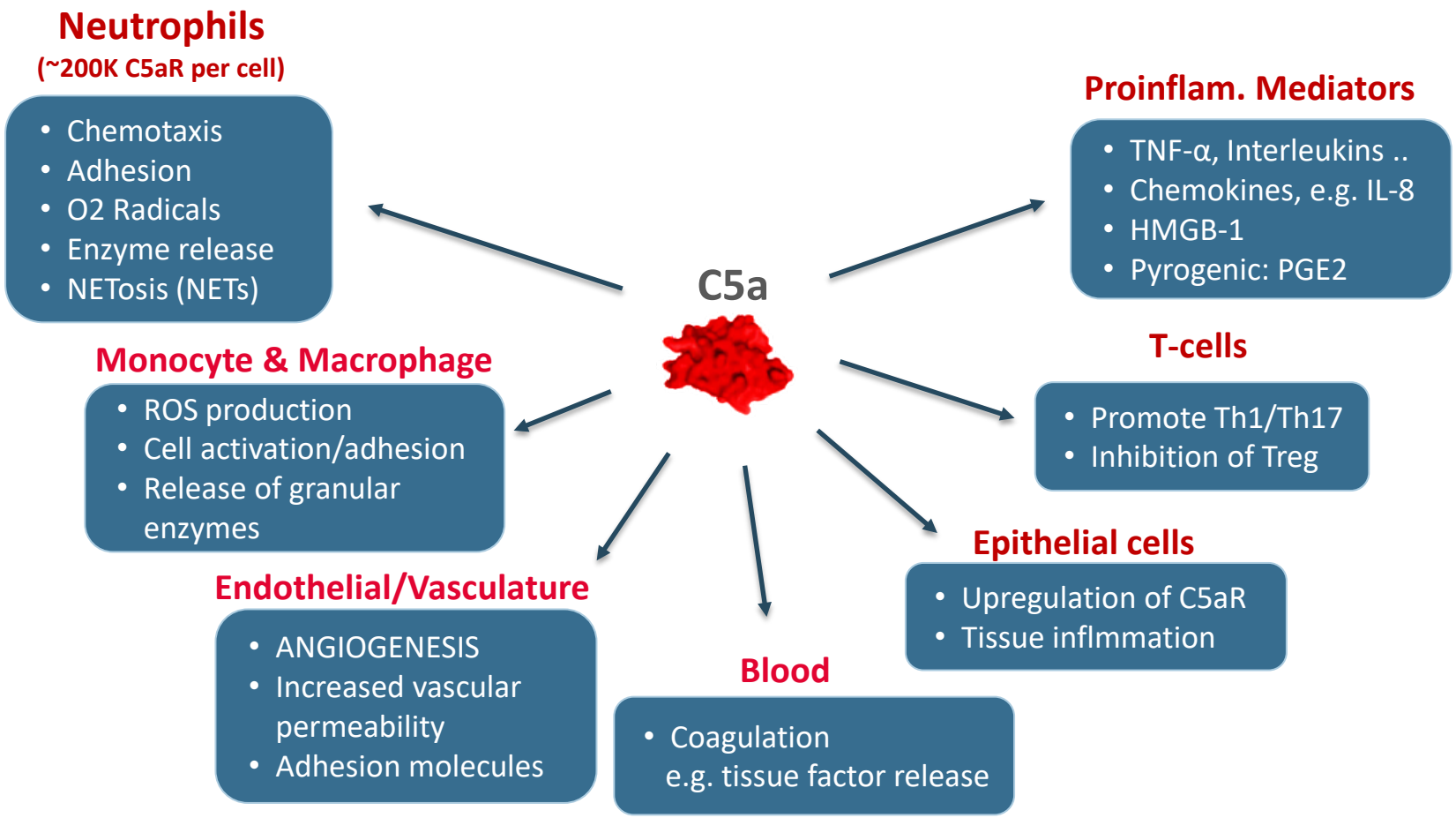
- Proof-of-concept for vilobelimab to reduce use of GC
- Lowest VDI total score at week 16 in vilobelimab-only group

Phase II IXPLORE:

- Primary endpoint met: safe and well-tolerated
- Strong clinical response (50% reduction in BVAS*) at week 16

BVAS: Birmingham Vasculitis Activity Score, **VDI**: vasculitis damage index, **ANdT** count: total # of abscesses, inflammatory nodules and draining tunnels

C5a / C5aR Signalling: A key Role in Inflammation



C5a is a key inflammatory mediator within the inflammatory response mainly through interaction with C5aR – Over 5000 publications on the role of the C5a / C5aR signalling axis

C5a / C5aR: Activation of Neutrophils as Major Tissue Damaging Event

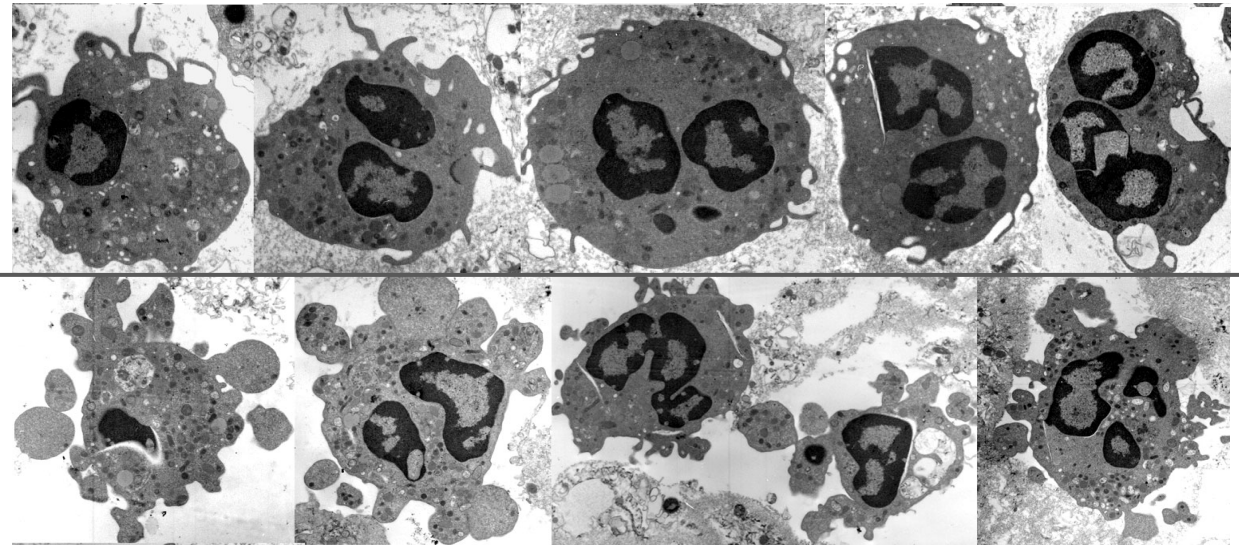
C5A /C5AR MODE OF ACTION ON NEUTROPHILS

- chemotaxis of neutrophils
  (Shin et al 1968, *Science* 162,361-3)
- enzyme release
  (Goldstein et al 1974, *J. Immunol.* 113, 1583-8)
- production of O^2-radicals in neutrophils
  (Sacks et al 1978, *J Clin Invest* 161, 1161-7)
- Netosis induction
  (Skendros et al. *J Clin Invest*. 2020;130(11):6151–6157)

NEUTROPHILS (4´600 X ENLARGED)

control

+ C5a



Data Source: Prof. Peter Ward, University of Michigan