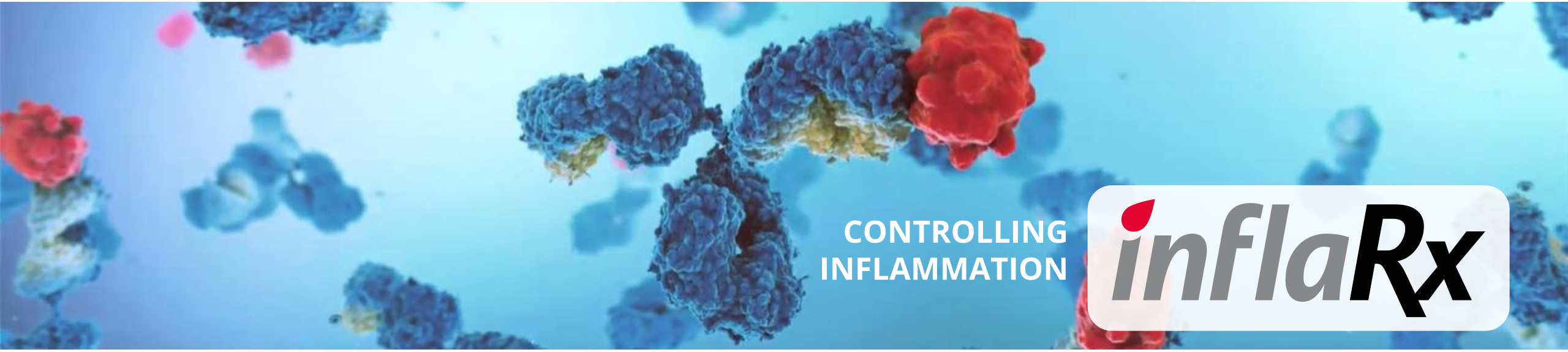
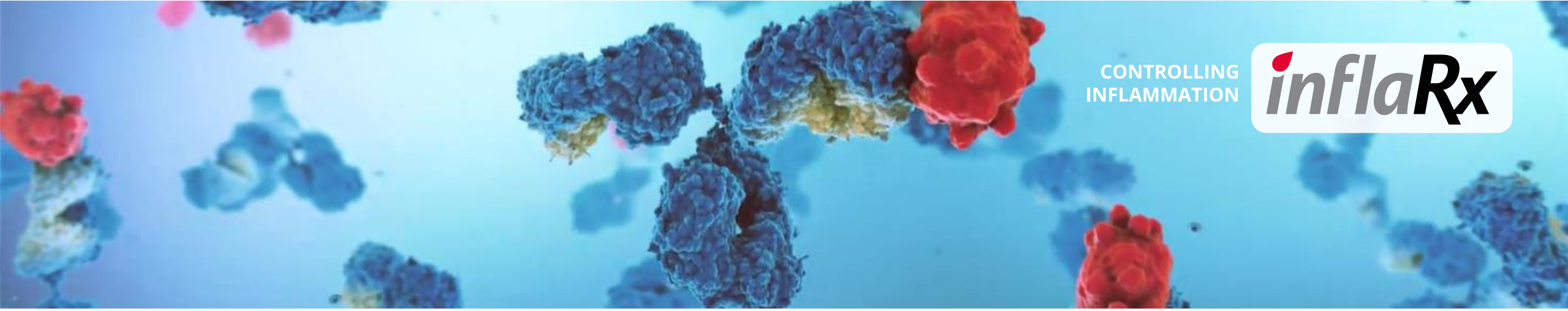


CORPORATE PRESENTATION

MAY 2022



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About InflaRx

InflaRx (Nasdaq: IFRX) is a clinical-stage biopharmaceutical company focused on applying its proprietary technology to discover and develop first-in-class or best-in-class, potent and specific inhibitors of C5a and C5aR. Complement C5a and C5aR are powerful inflammatory mediators involved in the progression of a wide variety of autoimmune and other inflammatory diseases. 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.

InflaRx N.V.

Targeting Complement to Control Inflammatory Diseases

Focused late-stage clinical development of lead asset and promising, earlier-stage development pipeline

Vilobelimab - Efficacy Signals and Clean Safety Profile in Clinical Testing

- **Pyoderma gangrenosum:** Positive Phase IIa data – seeking regulatory advice on next steps for pivotal program
- **Critical COVID-19:** Encouraging Phase III topline data in Q1 2022 – approaching regulatory authorities for next steps
- **Cutaneous squamous cell carcinoma**: Phase II study ongoing
- Following strategic review, decision to **halt hidradenitis suppurativa & ANCA-associated vasculitis programs** to better focus resources

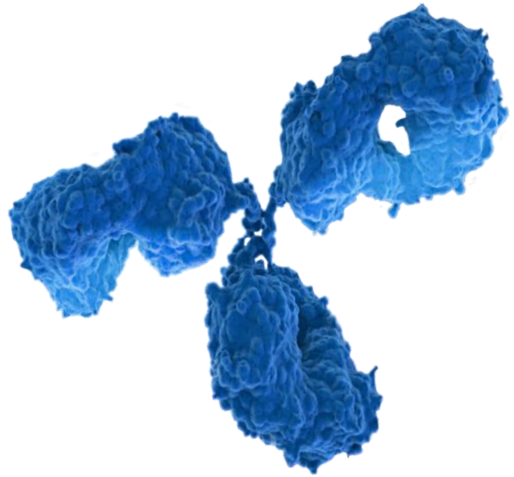
Proprietary Anti-C5a Technology has Strong Patent Coverage

- Patent protection until at least the end of 2030 / 2035 with an extension

New Program INF904: Oral C5aR Inhibitor to Enter Clinic in H2 2022

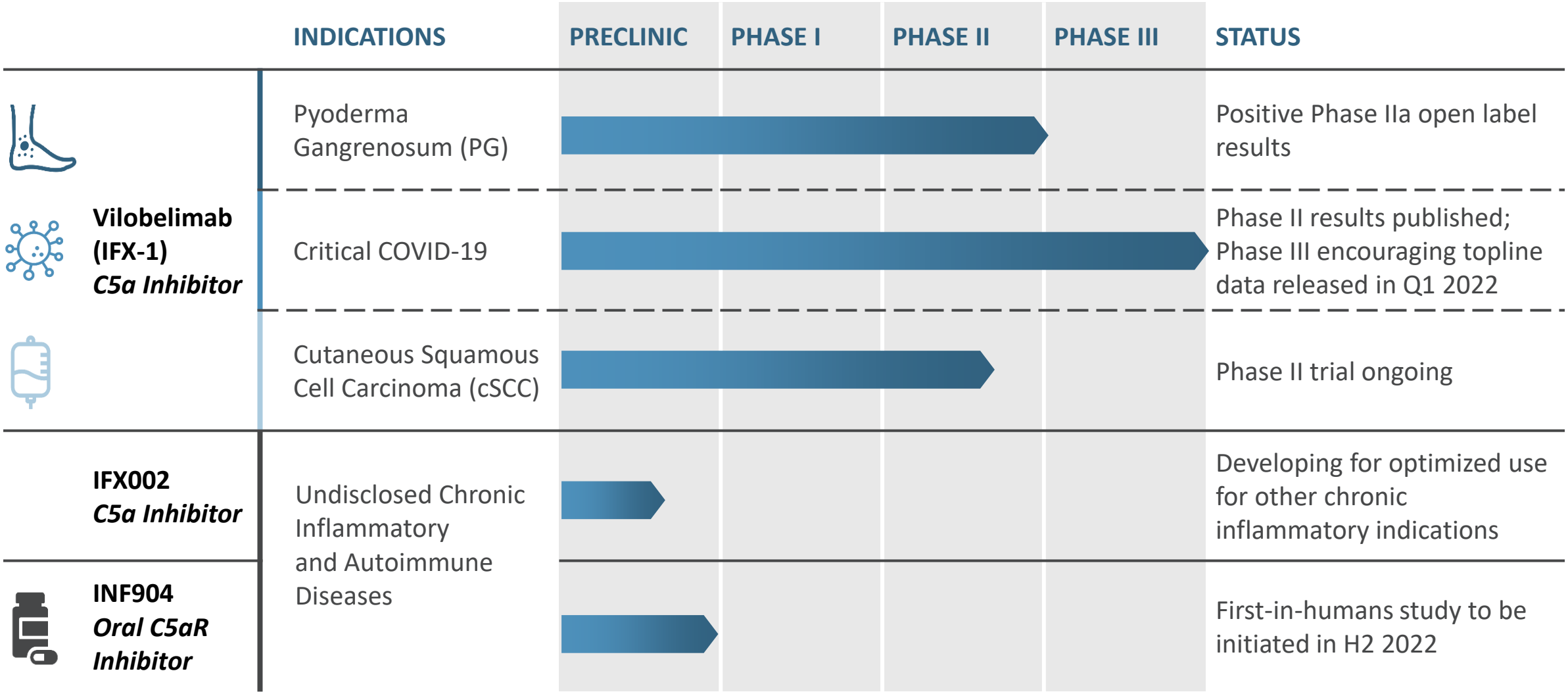
- Promising activity and clean safety profile in pre-clinical models

Strong Cash Balance of €99.3 Million – Cash runway into H2 2024



Cash balance includes cash, cash equivalents and financial assets as of March 31, 2022

Pipeline with Multiple Opportunities



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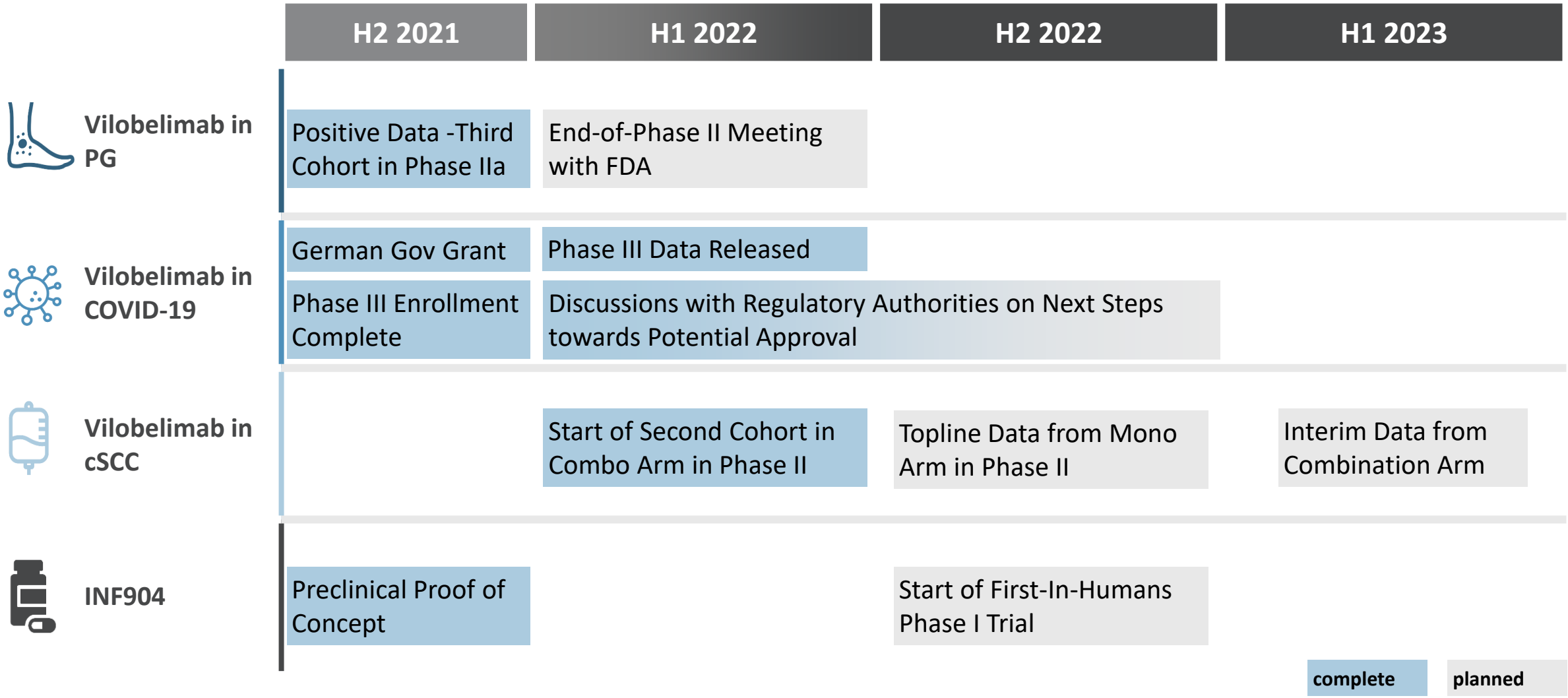
Important Expected Catalysts



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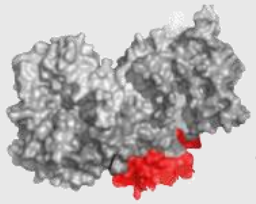
Vilobelimab Mode of Action

C5

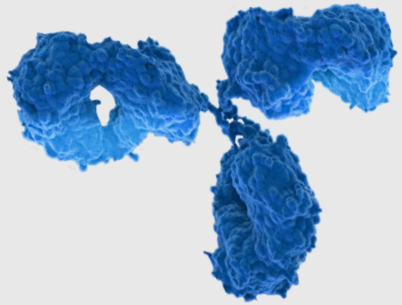


C5a

Cleavage and Conformational Change

New Epitope

Vilobelimab (IFX-1)



Cleavage of C5 through:

- complement pathway activation; or
- directly through enzymes via “extrinsic” pathway.

C5a is a key chemo-attractant and a strong activator of neutrophils leading to Neutrophil Extracellular Traps (NET) which are believed to be a disease driver in HS; Vilobelimab is believed to target this key mechanism.

Key Features:

- Blocks C5a biological effects up to 100% in human blood
- Fully selective – leaves MAC formation intact
- Binds with high affinity to the newly discovered epitope

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)

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
- Long-term vilobelimab treatment was well-tolerated

COVID-19

PANAMO Phase II/III trial – Phase III results:

- 23.9% relative reduction in 28-day mortality detected in global data set
- 43% relative mortality reduction in pre-specified W. Europe subgroup
- Significant improvement in mortality in all three pre-specified more severe patient subpopulations
- Drug 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

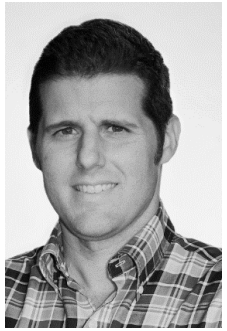
Leadership Team

NIELS RIEDEMANN, M.D., PH.D.
CHIEF EXECUTIVE OFFICER

- Founder of InflaRx
- 15+ years in biotech industry
- 20+ years in immunology research
- Vice Director of ICU at University Hospital Jena

THOMAS TAAPKEN, PH.D.
CHIEF FINANCIAL OFFICER

- 25+ years in senior management positions
- CFO of Medigene AG
- CEO & CFO of Epigenomics AG
- PH.D. in organic chemistry

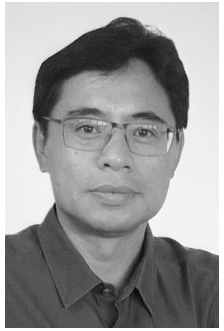


JORDAN ZWICK
CHIEF STRATEGY OFFICER

- 10+ years of industry operational experience
- Head of Strategy at Salix Pharmaceuticals
- M.B.A., University of San Francisco

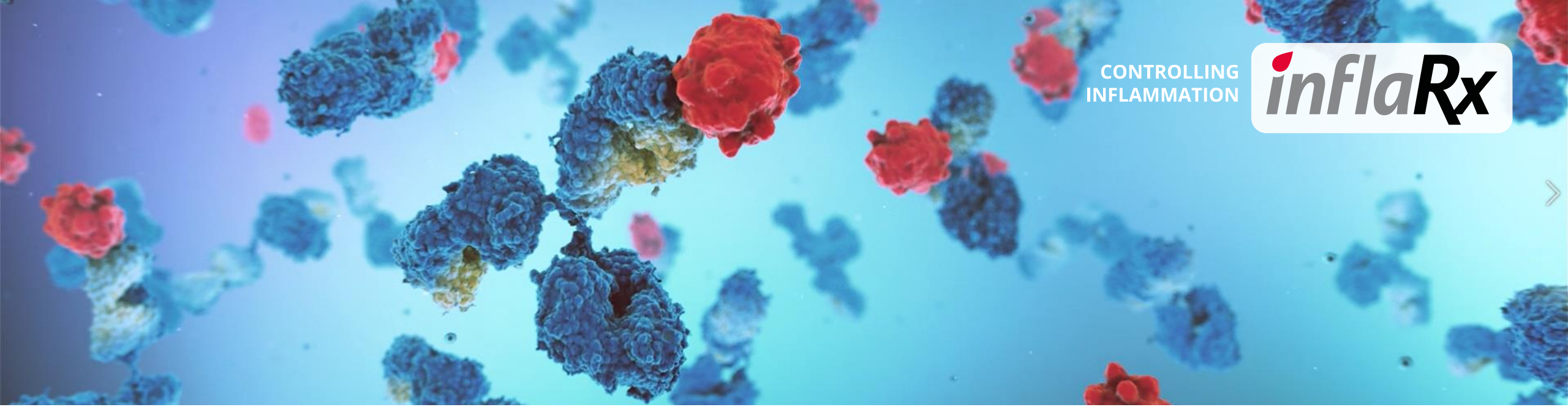
KORINNA PILZ, M.D., M.SC.
CHIEF CLINICAL DEVELOPMENT OFFICER

- 20+ years in academia and industry
- Innate Pharma, Boehringer Ingelheim, Roche, Merck KGaA and Bayer
- M.D., Dipl. Biology, University of Düsseldorf



RENFENG GUO, M.D.
CHIEF SCIENTIFIC OFFICER

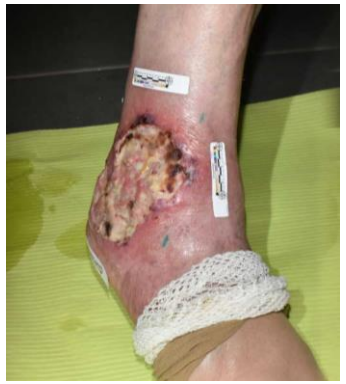
- Founder of InflaRx
- Professor at University of Michigan
- 80+ peer-reviewed publications in cancer, infectious disease, and inflammation research

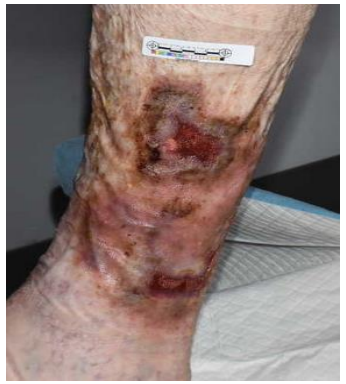


Vilobelimab
Pyoderma Gangrenosum (PG)

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
- Many PG patients also suffer from other autoimmune disorders, such as ulcerative colitis, rheumatoid arthritis and hematological diseases
- Patients suffer from severe pain, long healing times and frequent relapses

Incidence

- Rare – estimated that up to 50,000 patients in the US and Europe are affected

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

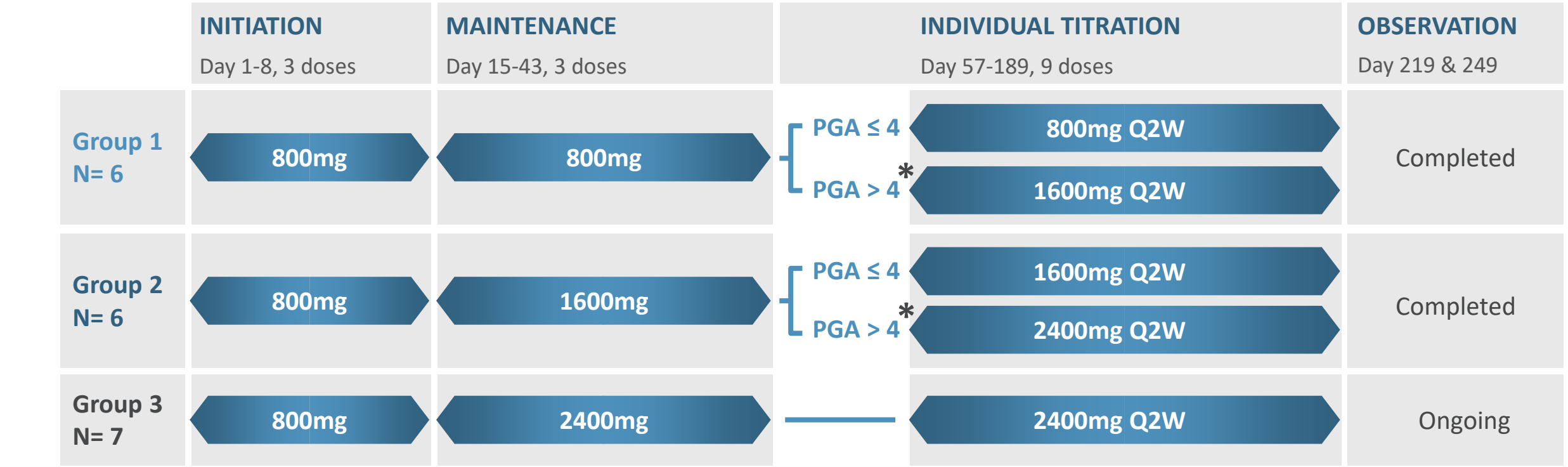
Strong rationale for treatment with vilobelimab: PG associated with neutrophilic skin infiltration in affected areas and lesions, potentially triggered by C5a

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



* Uptitration 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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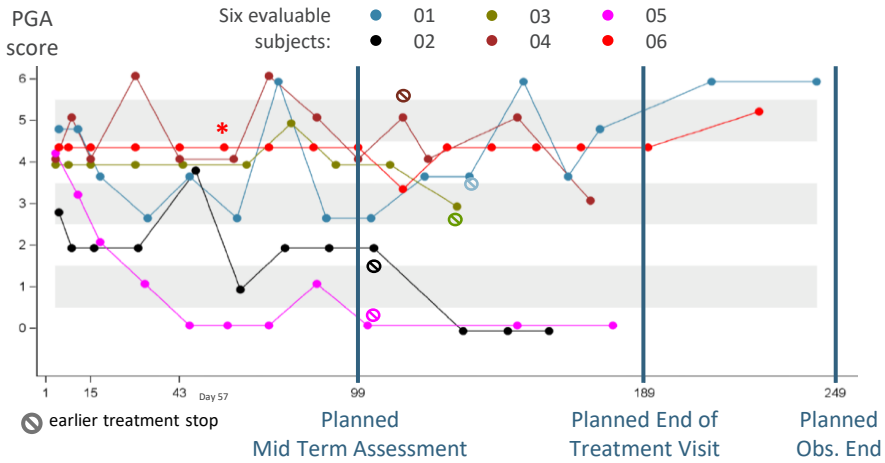
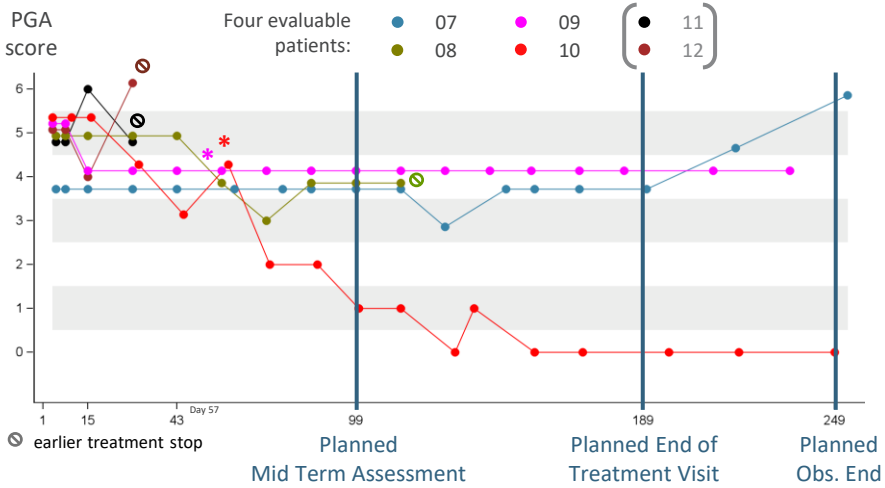
Phase IIa Study Results – Group 1 & 2 (Low & Medium Dose)



PGA line plot of absolute values over time by patient (Actual days displayed acc. to visit windows)
*Uptitration to 1600mg on day 57 **if** PGA > 4 and at least 5 patients treated with 800mg show no safety issues. Applied to patient 06

GROUP 1 RESULTS

- Two patients (02 and 05) achieved **complete remission of target ulcer**.
- One patient (01) with initial response and fluctuating PGA.
- Patients 02 and 05 stopped treatment before Day 189 based on investigator decision because of complete disease remission.
- Patient 03 dosed until Day 130 but stopped treatment due to the COVID-19 pandemic. No follow up.

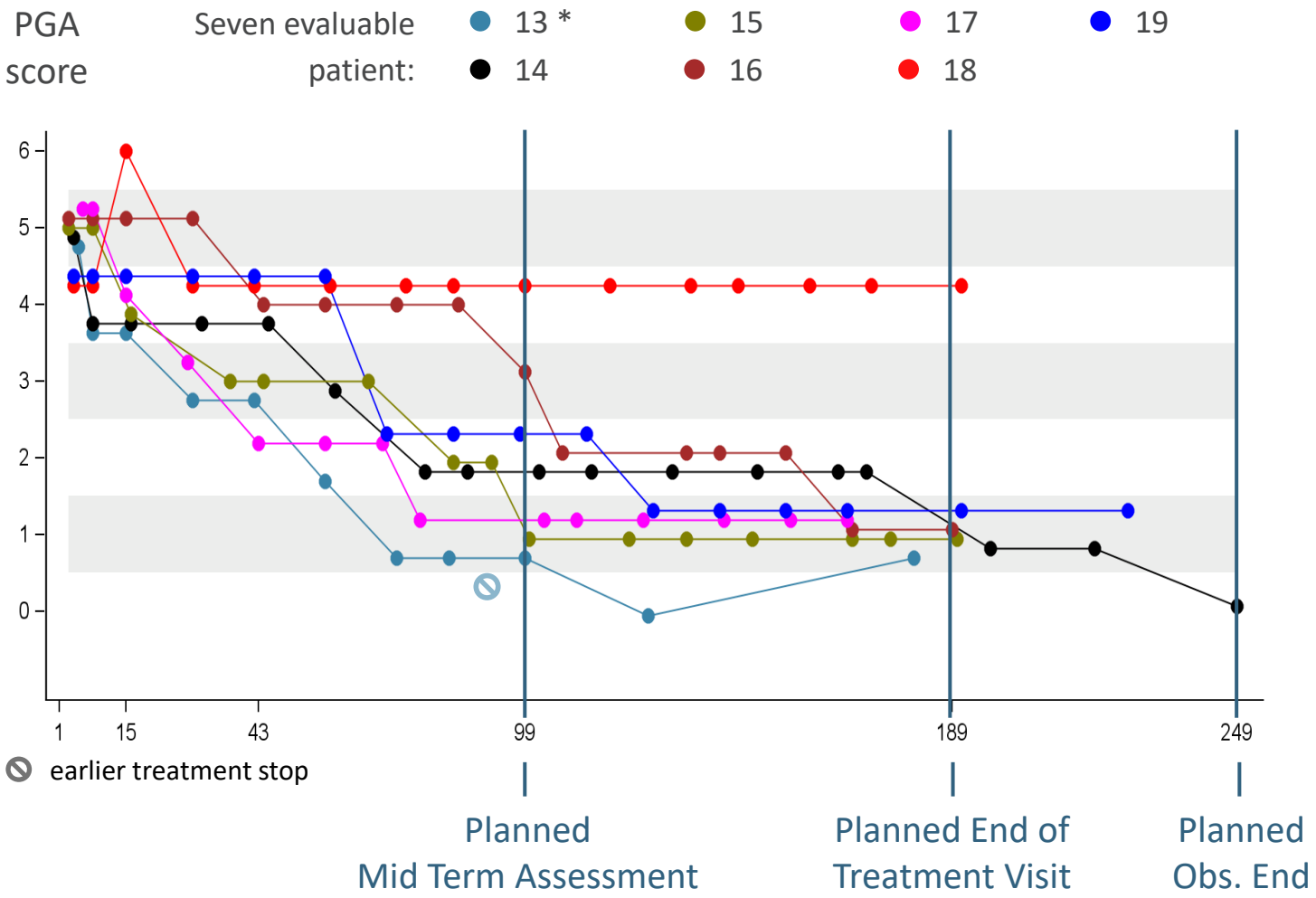


PGA line plot of absolute values over time by patient (Actual days displayed acc. to visit windows)
*Uptitration to 2400mg on day 57 **if** PGA > 4 and at least 5 patients treated with 1600mg show no safety issues. Applied to patients 09 and 10

GROUP 2 RESULTS

- One patient (10) out of four **healed upon up-titration to 2400mg group on day 57** with PGA = 0 since visit 12 (closure of large target ulcer area).
- Two patients (08, 09) showed temporary response, not considered responder.
- Two patients (11, 12) discontinued early in study and were non-evaluable.

Phase IIa Study Results – Group 3 (High Dose)

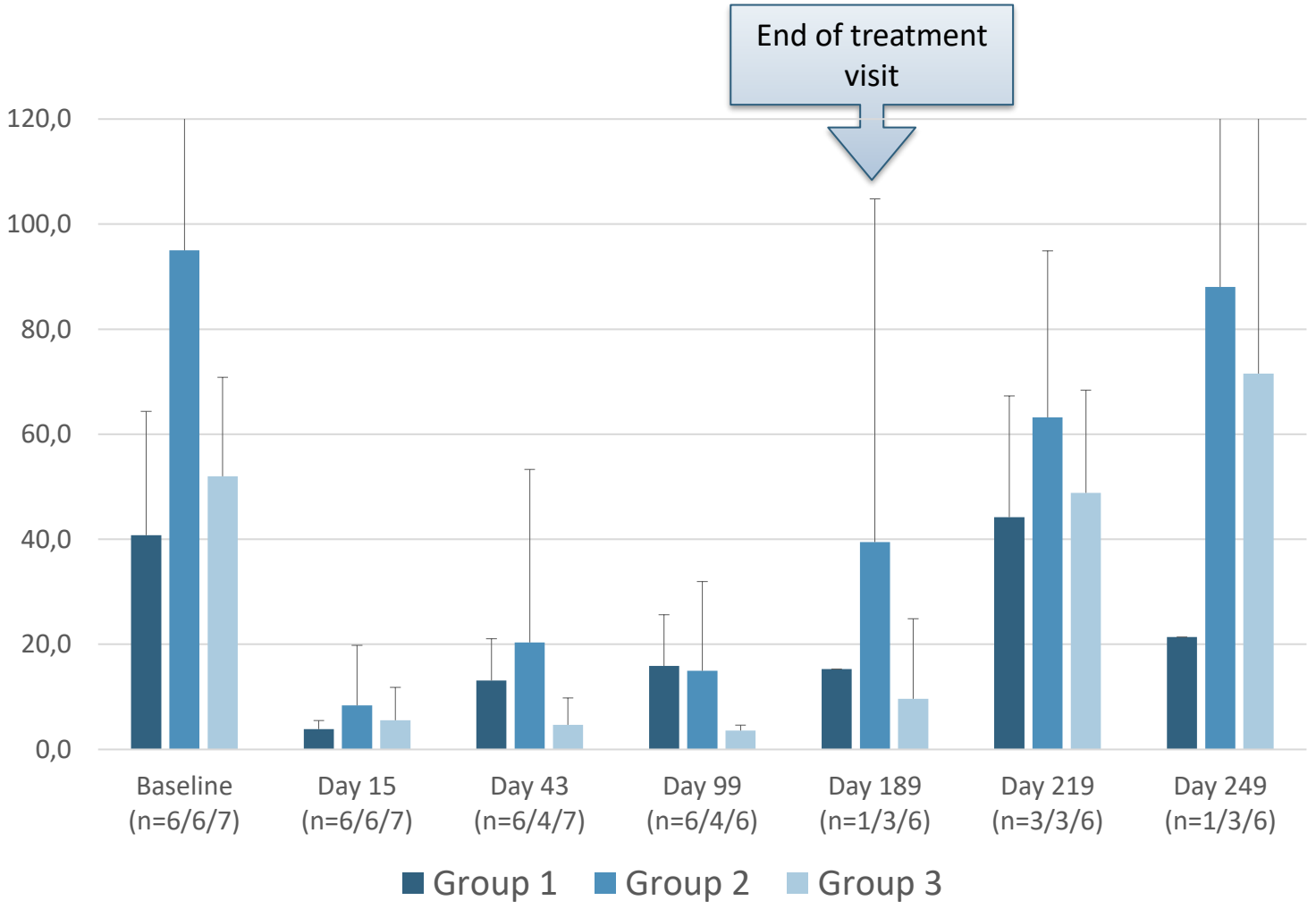


GROUP 3 RESULTS

- 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.

C5a Plasma Concentration per Dosing Group



Full Analysis Set

CLINICAL OBSERVATION

- C5a sustained suppression for 26 weeks in Group 3
- Six out of seven patients in the High Dose Group reached PGA ≤ 1 (clinical remission)

PG Phase IIa

Patient Case Studies

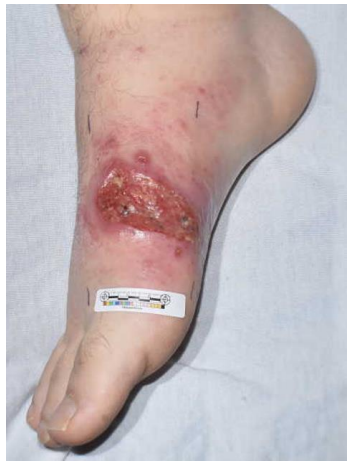
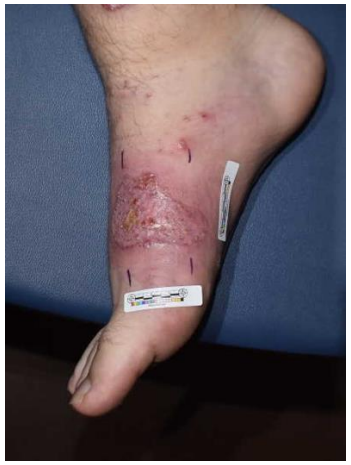
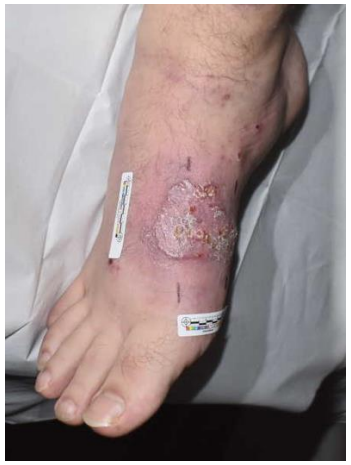
TARGET ULCER OPENED (NOV 2020) WHILE ON STABLE ADALIMUMAB

MH: PG since August 2020, Psoriasis since 2017
Treatment Start: March 2021
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 40mgQD 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 (AUG 2020)

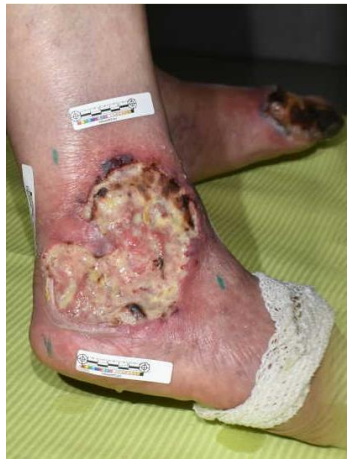
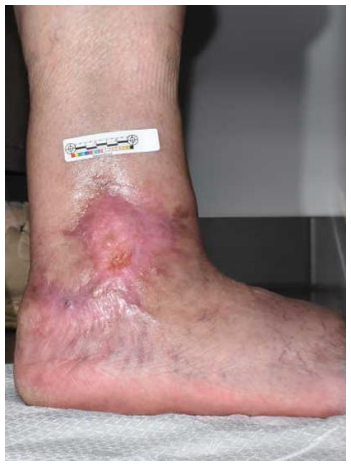
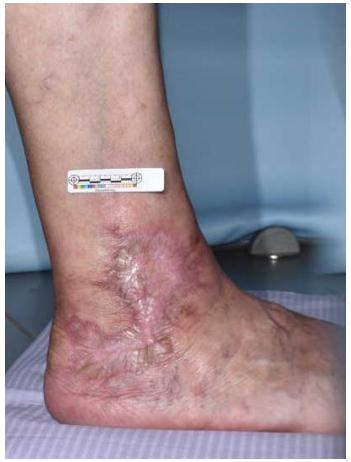
MH: PG since 2019, Hypertension since 1998; **Study Day 1:** Feb 2021

Cohort 2: 1600 mg Q2W, individual uptitration to 2400 mg at D57, treatment completed

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

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

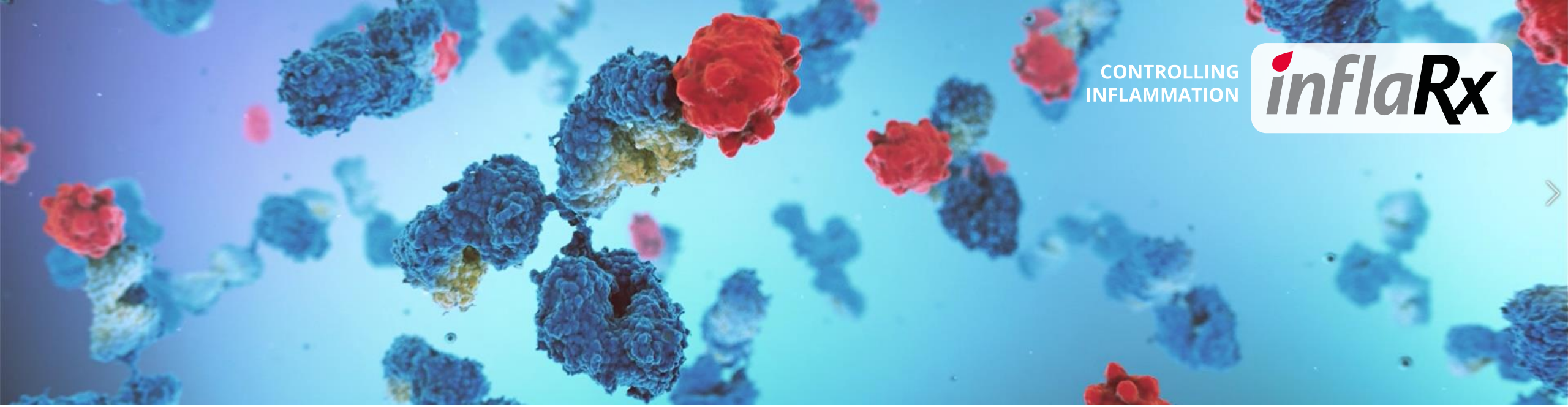
- No infusion-related reactions observed
- For 2 patients, related SAEs were reported:
  - Erysipelas leading to hospitalization (judged as non-related by sponsor)
  - Rash due to delayed hypersensitivity reaction
- Observed AE profile in line with patients' underlying diseases
- No dose-related AEs 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%)**

END-OF-PHASE II FDA MEETING SCHEDULED MID-YEAR 2022 TO DISCUSS NEXT STEPS

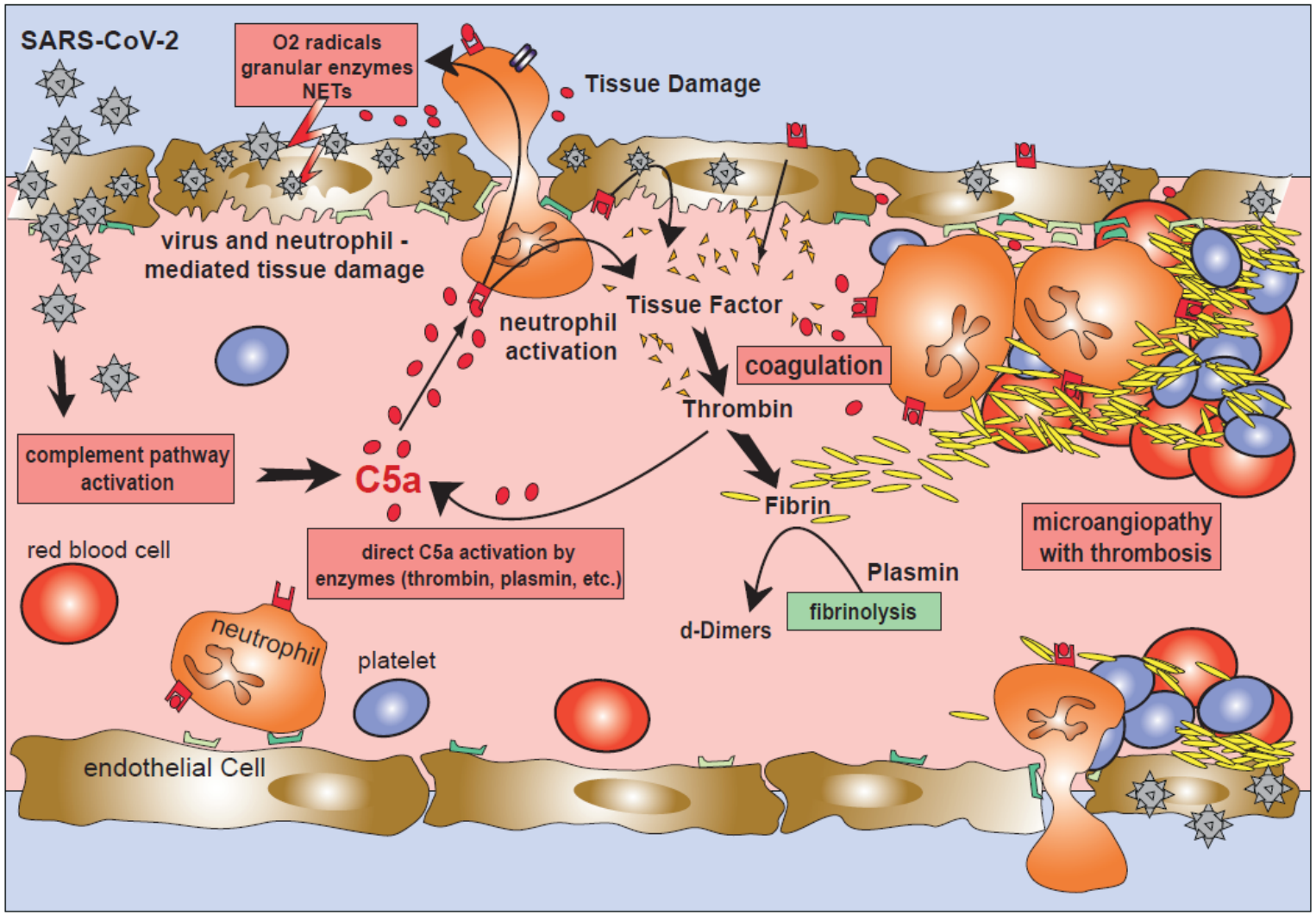
Vilobelimab Q2W shows good safety and tolerability
Evidence of dose-dependent drug activity in PG



Vilobelimab
Critical COVID-19

COVID-19 induced Vascular Injury – Potential Role of C5a

Model for Proposed Mode of Action of C5a in COVID-19 induced vascular injury



Source: InflaRx GmbH

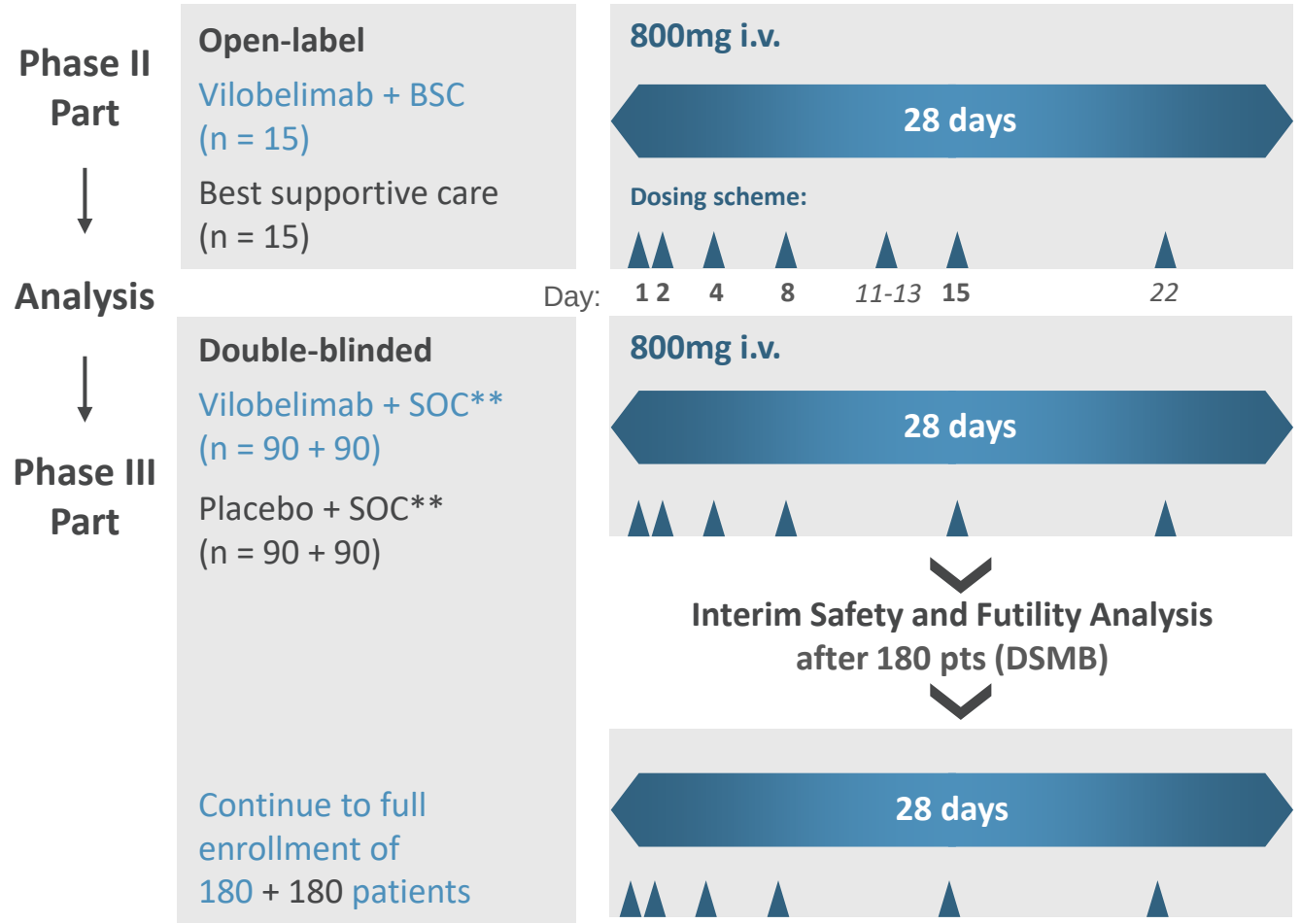
OUR HYPOTHESIS

- Endothelial damage is induced by SARS-CoV-2 infection which also activates the complement system leading to C5a generation.
- C5a activates neutrophils via C5aR, leading to increased adherence to endothelial cells and damage through generation of oxidative radicals, granular enzyme release and neutrophil extracellular traps (NETs).
- C5a induces release of tissue factor from neutrophils & endothelial cells, which promotes coagulation, leading to Fibrin formation.
- Thrombin, plasmin and other enzymes can further induce direct C5a activation (through direct cleavage of C5), which may establish a vicious circle leading to microangiopathy with thrombosis.

Design of PANAMO Phase II/III Study with Vilobelimab in Severe COVID-19

STUDY DESIGN

- Critically ill intubated* patients with COVID-19 induced pneumonia
- Phase III primary endpoint:
  28-day all-cause mortality
- Key secondary endpoints
  - 60-day all-cause mortality (proportion of patients deceased until Day 60)
  - Proportion of patients with an improvement in the 8-point ordinal scale (Day 15, Day 28)
  - Proportion of patients developing acute kidney failure during ICU stay and at Day 28
  - Proportion of patients free of any renal replacement therapy within 28 days upon randomization
  - Frequency, severity, and relatedness to study drug of serious and non-serious TEAEs



* In Phase III part, eligible patients were required to have been early intubated. In the Phase II part, patients were enrolled if either early intubated or dependent on oxygen delivery.
** SOC includes venous thromboembolism prophylaxis at a minimum and may include other specific recommended treatments for COVID-19 per the locally adopted treatment recommendation. 97% of patients received corticosteroids.

PANAMO Phase II/III Trial: Phase II Results

PHASE II STUDY RESULTS

Primary endpoint:

- No difference detected in improvements between groups in PaO2/FiO2 ratio
- High variability between patients
- Conclusion: Endpoint not suitable as response parameter

Key secondary and other endpoints – observed effects with vilobelimab compared to best standard of care:

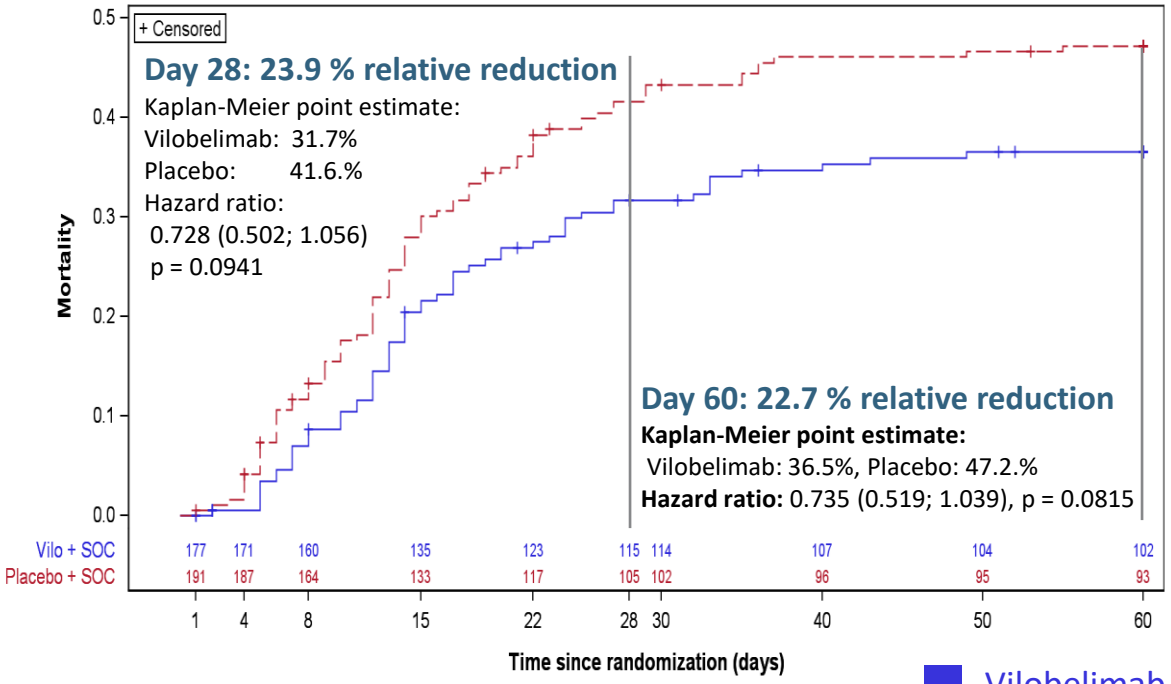
- **50% lower all-cause mortality rate** (13% in vilobelimab group vs. 27% for control group)
- **Fewer patients experienced renal impairment** assessed by estimated glomerular filtration rates
- **Faster reversal of blood lymphocytopenia**
- **Reduction in tissue damage**: Greater lowering of lactate dehydrogenase concentrations
- Temporary but **statistically significant increase in D-dimer levels** in first days after vilobelimab administration – **potential signal of induction of blood clot lysis**

Source: Vlaar, A et al. *Lancet Rheumatol 2020*. https://doi.org/10.1016/S2665-9913(20)30341-6

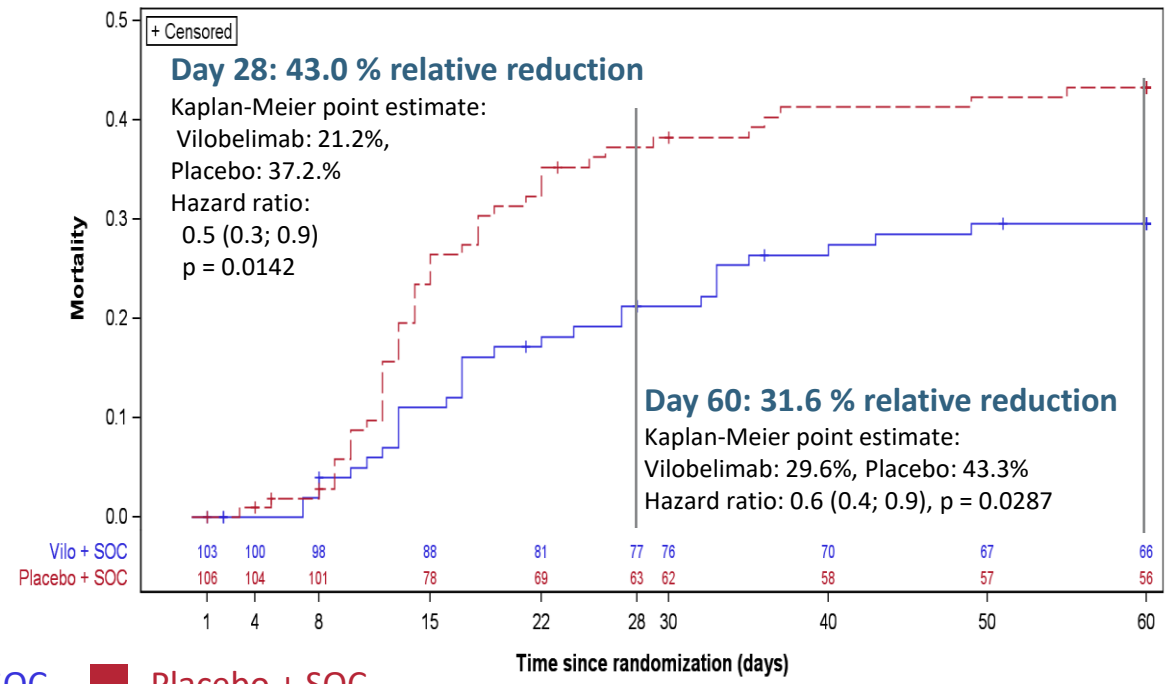
PANAMO Trial: Phase III Topline Results

MORTALITY – OVERALL & WESTERN EUROPE AT DAY 28 & 60

All-cause mortality: Overall



All-cause mortality: Western Europe



Vilobelimab + SOC Placebo + SOC

One additional life was saved for every 10 (Overall) vilobelimab-treated participants at day 28

One additional life was saved for every 9 (Overall) / 7 (Western Europe) vilobelimab-treated patients at day 60

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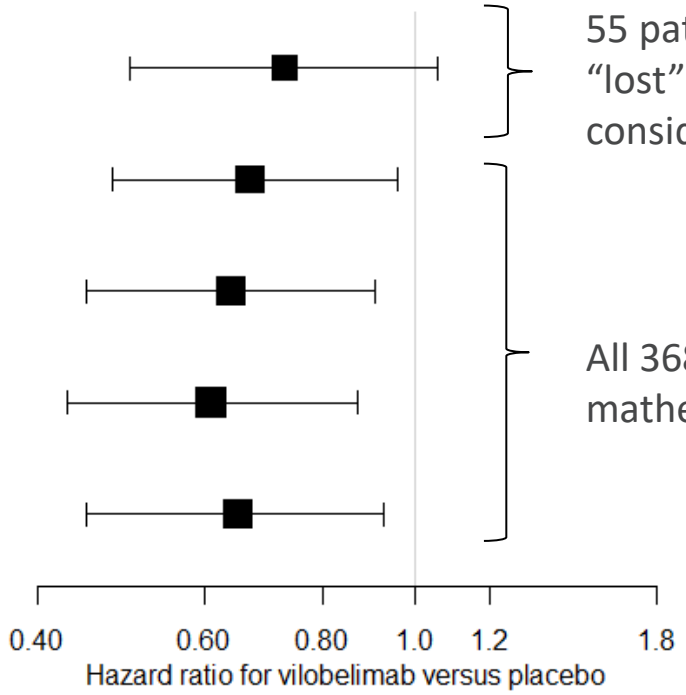
PANAMO Trial: Analyzing the Primary Outcome (28-day Mortality)

Cox Regression Analysis

Model	p-value	HR
Stratified by site (primary per SAP)	0.094	0.73
Unstratified (primary per protocol)	0.027	0.67
Stratified by region	0.014	0.64
Stratified by country	0.007	0.61
Random effect for site (Frailty)	0.018	0.65



55 patients from 17 sites with no events (death) are "lost" in this mathematical analysis = model only considers 313 patients instead of 368

All 368 treated patients are considered for these mathematical analyses

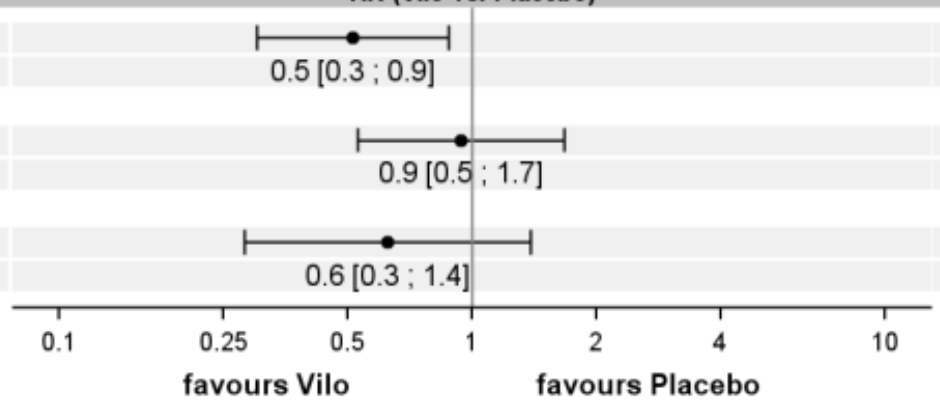
The same observation is made for 60-day mortality data (secondary endpoint)

Vilobelimab shows a robust and significant treatment effect in all Cox regression analysis models, which consider all available patient data – but fails statistical significance when adjusting for site stratification (losing 55 patients)

PANAMO Trial: Phase III Topline Results

PRE-SPECIFIED SUBGROUP ANALYSES BY REGION

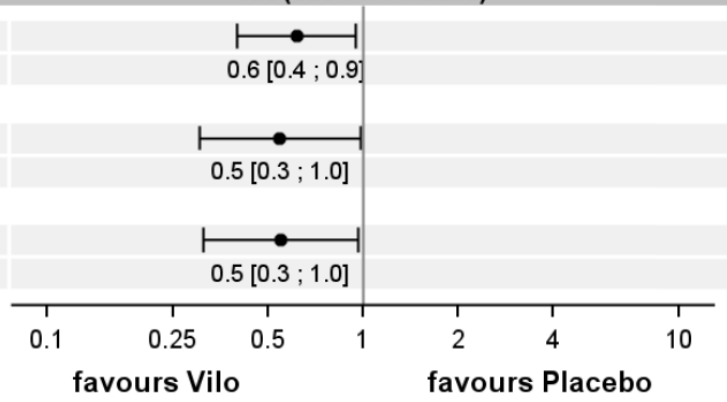
Region	Treatment	n	Mortality (KM-Estimate)	HR (Vilo vs. Placebo) *	p-value
Western Europe *	Vilo + SOC	103	21.2% (14.4% - 30.7%)		0.0142
	Placebo + SOC	106	37.2% (28.7% - 47.4%)	0.5 [0.3 ; 0.9]	
South America **	Vilo + SOC	58	39.6% (28.1% - 53.7%)		0.8337
	Placebo + SOC	68	37.3% (26.9% - 50.0%)	0.9 [0.5 ; 1.7]	
South Africa/Russian Federation	Vilo + SOC	16	68.8% (46.4% - 88.6%)		0.2450
	Placebo + SOC	17	87.3% (66.7% - 97.9%)	0.6 [0.3 ; 1.4]	



*Belgium, France, Germany, Netherlands
**Brazil, Mexico, Peru

These subgroup analyses point **towards a stronger treatment effect on 28-day all-cause mortality in more severe patients**:

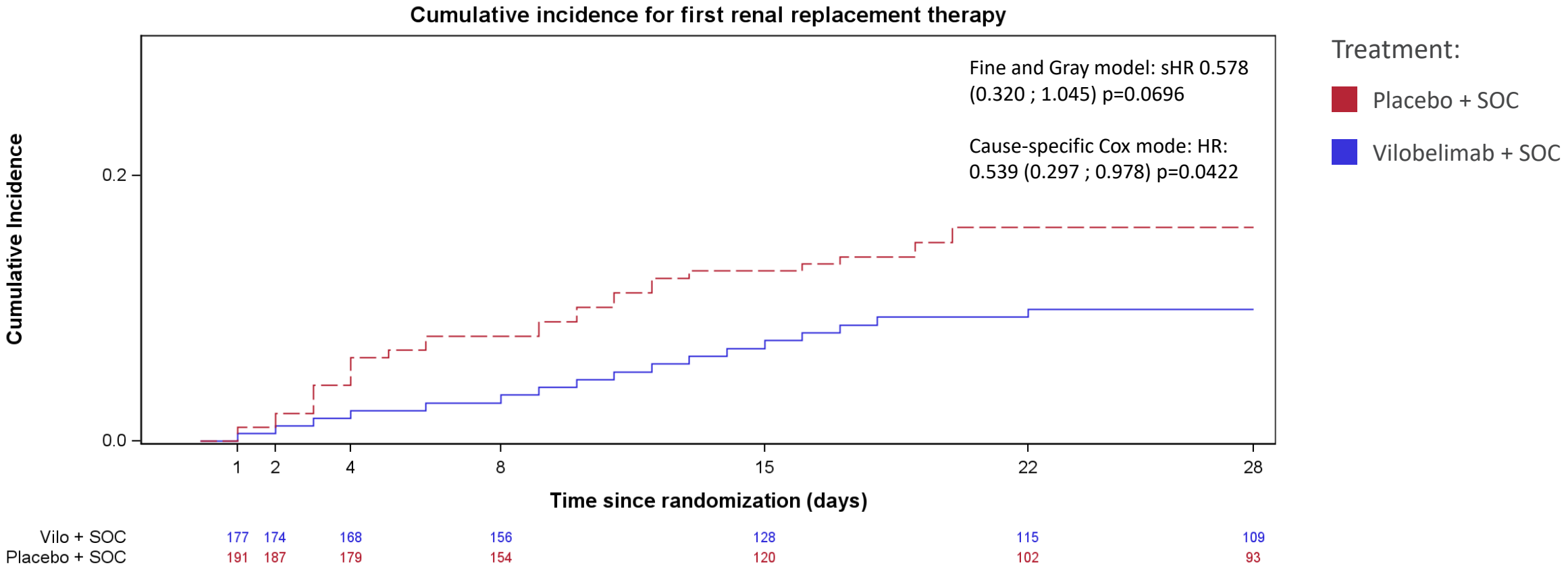
Subgroup	Treatment	n	Mortality (KM-Estimate)	HR (Vilo vs. Placebo) *	p-value
Baseline Ordinal Scale = 7	Vilo + SOC	105	32.1% (24.0% - 42.1%)		0.0279
	Placebo + SOC	132	43.7% (35.6% - 52.7%)	0.6 [0.4 ; 0.9]	
Baseline ARDS: Severe (PaO2/FiO2 <= 100 mmHg)	Vilo + SOC	43	40.0% (27.1% - 56.2%)		0.0441
	Placebo + SOC	55	59.5% (46.8% - 72.6%)	0.5 [0.3 ; 1.0]	
Baseline eGFR < 60 mL/min/1.73m²	Vilo + SOC	47	41.5% (28.8% - 57.1%)		0.0358
	Placebo + SOC	61	59.4% (47.0% - 72.1%)	0.5 [0.3 ; 1.0]	



Phase III Part Top Line Results

SECONDARY ENDPOINT

Proportion of patients free of any renal replacement therapy within 28 days of randomization



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Phase III Safety Results

SAFETY OVERVIEW

Adverse event category	Vilobelimab (N=175)	Placebo (N=189)
Any TEAE	159 (90.9%)	172 (91.0%)
Any related TEAE	20 (11.4%)	16 (8.5%)
Any serious TEAE	103 (58.9%)	120 (63.5%)
Any serious related TEAE	8 (4.6%)	9 (4.8%)
Any fatal TEAE*	62 (35.4%)	85 (45.0%)

INFECTIONS

MedDRA High Level Group Term	Vilobelimab (N=175)	Placebo (N=189)
Any TEAE in SOC infections and infestations	110 (62.9%)	112 (59.3%)
Infections – pathogen unspecified	91 (52.0%)	88 (46.6%)
Bacterial infectious disorders	68 (38.9%)	75 (39.7%)
Fungal infectious disorders	21 (12.0%)	15 (7.9%)
Viral infectious disorders	22 (12.6%)	14 (7.4%)
Ancillary infectious topics	4 (2.3%)	4 (2.1%)

*149 deaths were observed in all randomized patients, but 2 patients did not contribute to fatal TEAEs. One patient died before receiving the first IMP infusion, and one patient died on Day 4, but the fatal AE started before the first IMP infusion.

Phase III Safety Results

INFECTIONS AND INFESTATIONS BY PATIENT DAYS IN HOSPITAL

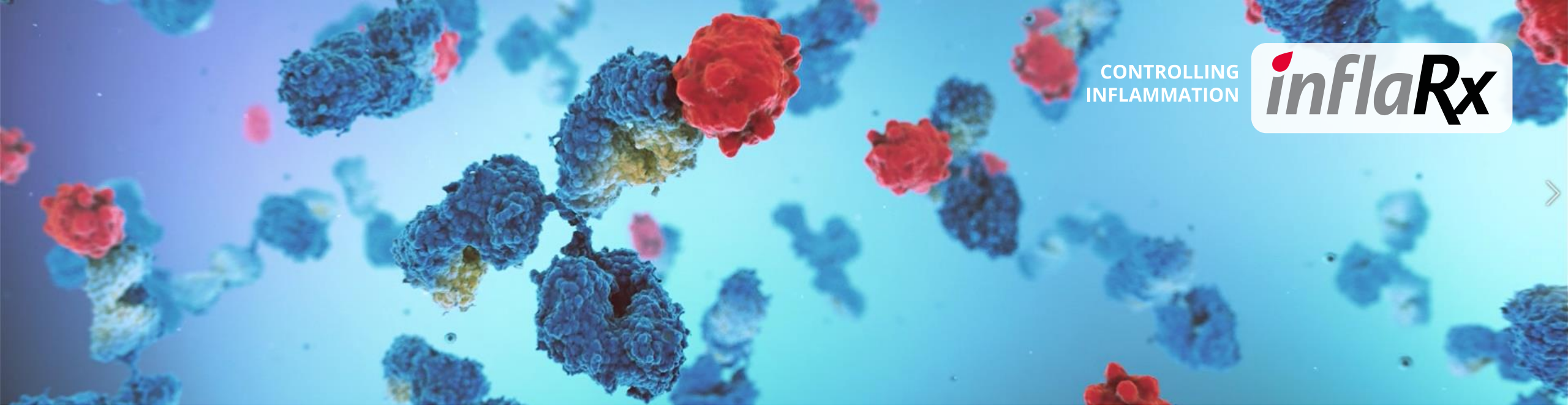
MedDRA High Level Group Term	Vilobelimab (N=175) n (%)		Placebo (N=189) n (%)	
	Incidence (patient days = 2384)		Incidence (patient days = 1736)	
	Patients per 100 patient days	Events per 100 patient days	Patients per 100 patient days	Events per 100 patient days
Any TEAE in SoC infections and infestations	4.57	15.69	6.45	18.72
Infections – pathogen unspecified	3.78	7.89	5.07	8.87
Bacterial infectious disorders	2.85	5.54	4.26	7.66
Fungal infectious disorders	0.88	0.96	0.86	0.98
Viral infectious disorders	0.92	1.09	0.81	0.98
Ancillary infectious topics	0.17	0.21	0.23	0.23

Summary

KEY LEARNINGS FROM PHASE III RESULTS

- Primary endpoint of 28-day all-cause mortality – **clinically meaningful benefit in vilobelimab arm** but missed statistical significance using pre-specified analysis method
- Pre-specified and post-hoc analyses suggest **robust reduction in mortality at day 28 and day 60** in vilobelimab treated patients compared to placebo
- **Significant treatment benefit (p=0.014)** in pre-defined analysis of Western European patient population with 43% relative reduction in 28-day all-cause mortality
- **Significant treatment benefit (p<0.05)** in all three pre-defined subgroup analyses of patients with higher disease severity at baseline
- **Favorable safety profile** for vilobelimab confirmed in this critically ill patient population

Company in discussions with regulatory authorities on next steps towards potential approval



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

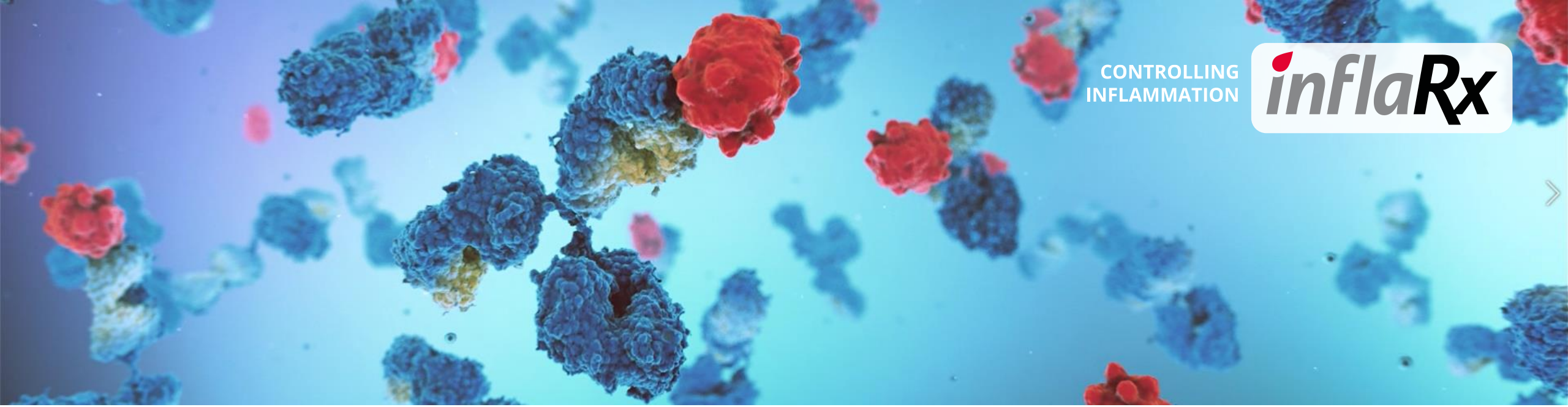
- Arm A: Assess antitumor activity of vilobelimab
- Arm B: Determine maximum tolerated dose (MTD) or recommended Phase II dose (RP2D); assess antitumor activity and safety profile of vilobelimab + pembrolizumab

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

› 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

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



INF904
New Pipeline Program

Allosteric Inhibitor INF904

Potential for Best-in-Class C5aR Inhibition

RATIONALE

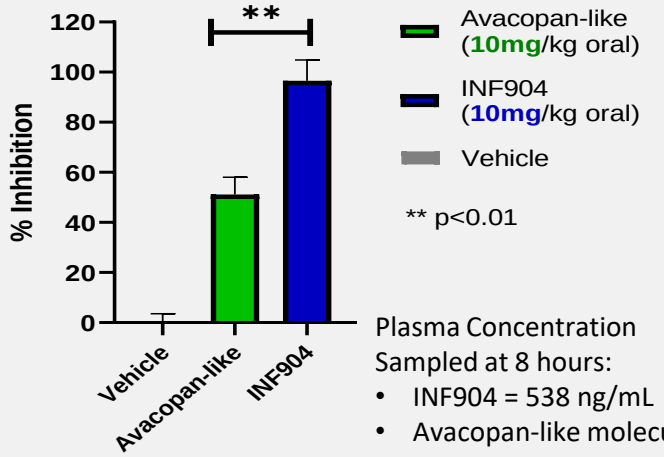
- INF904 binds to a well-known allosteric site in C5aR
- INF904 has a novel Markush structure
- US patent was issued in October 2021

PRE-CLINICAL FINDINGS

- **No obvious toxicity findings** even in the highest dose groups
- **High in vitro potency** with a desired IC50 (<1nM) in calcium mobilization assay
- **Significantly less CYP3A4/5 inhibition** in vitro vs. avacopan-like molecule; CYP3A4/5 plays important role in metabolic clearance of glucocorticoids
- **Higher plasma exposures** in several in vivo models vs. avacopan-like molecule
- **Better potency in in vivo neutropenia model** vs. avacopan-like molecule
- **Therapeutic effect** in renal disease models and peritonitis model

First-in-human trial expected to start in H2 2022

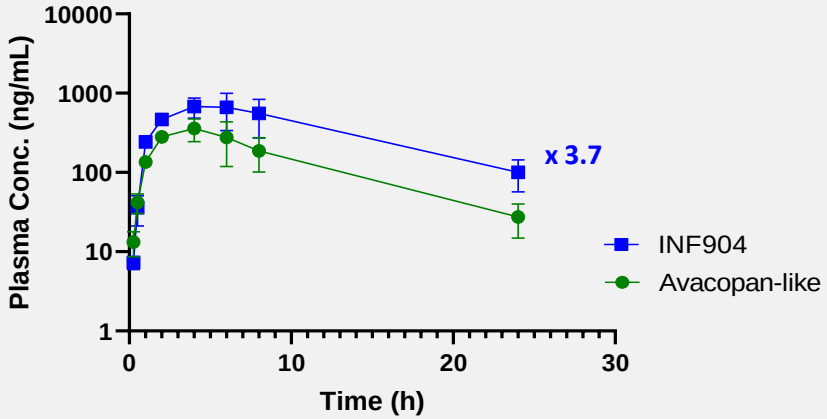
Neutropenia *in vivo* Model



Plasma Concentration Sampled at 8 hours:

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

Hamster PK – 10mg/kg oral



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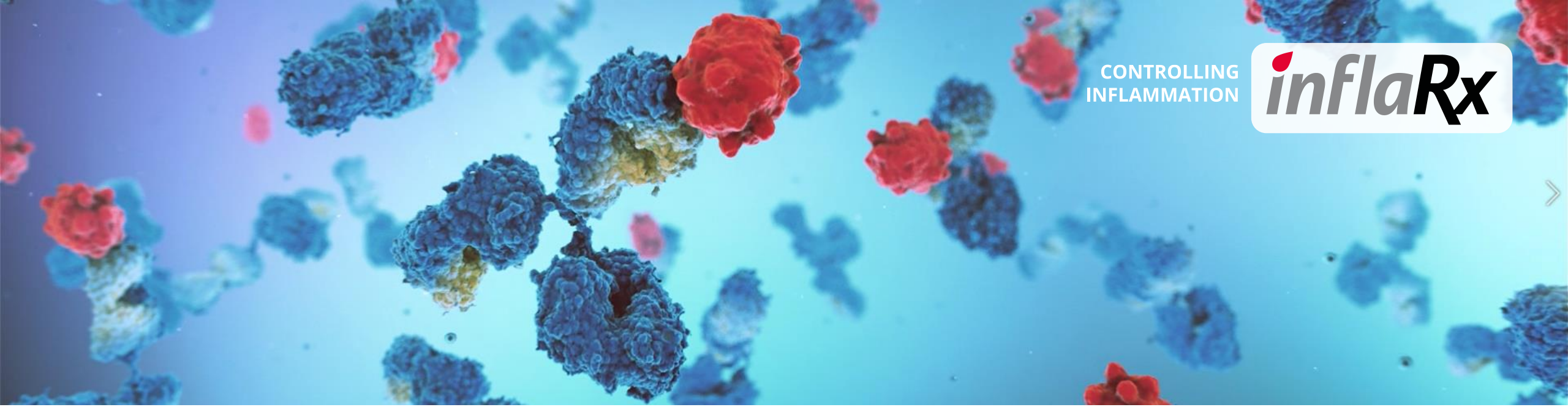
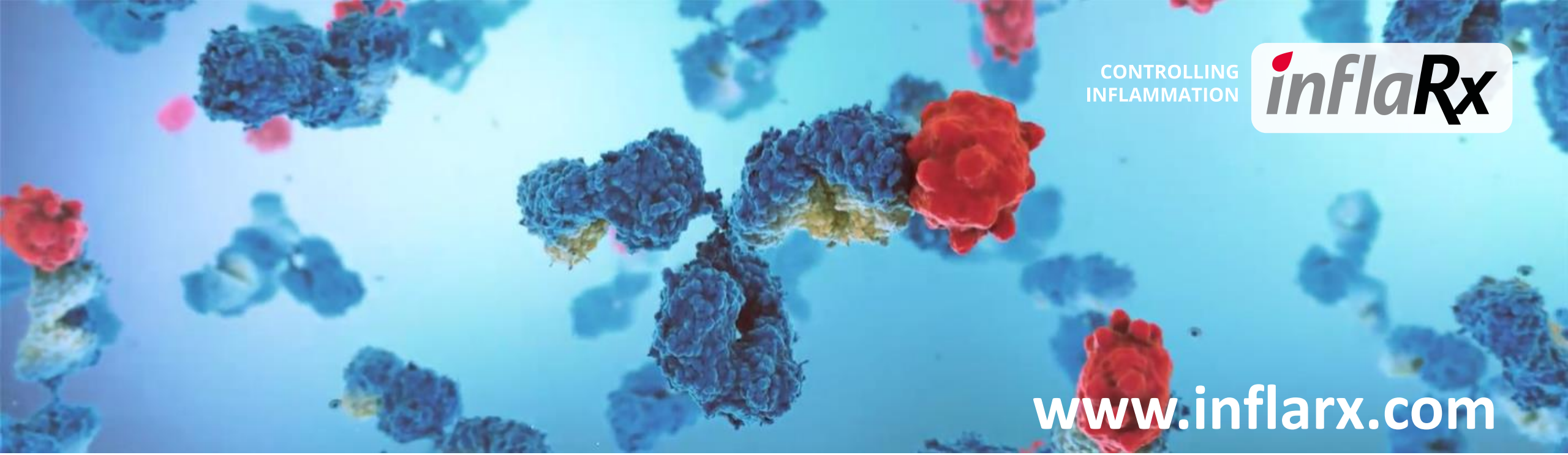


Strategy and Outlook

Strategy and Outlook

Vilobelimab in PG	• End-of-Phase II meeting with FDA in Q2 2022
Vilobelimab in COVID-19	• Discussions with regulatory authorities on next steps towards potential approval
Vilobelimab in cSCC	• Start of second cohort of combination arm in Phase II in H1 2022 • Topline data from monotherapy arm in Phase II in H2 2022 • Interim data from combination arm in H1 2023
INF904	• Preclinical proof of concept • Initiate first-in-human Phase I trial in H2 2022



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