CONTROLLING INFLAMMATION

CORPORATE PRESENTATION

January 2021

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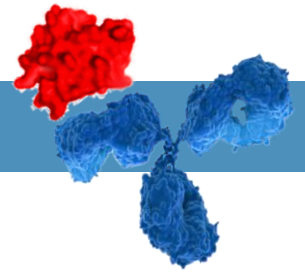
Investment Highlights



LEADING PROPRIETARY ANTI-C5A TECHNOLOGY

- **Complete and selective blockade** of the biological activity of C5a in vitro and in vivo
- **Strong patent coverage** on anti-C5a technology until end of 2030 / 2035 with extension

ESTABLISHED CLINICAL EFFICACY FOR VILOBELIMAB (IFX-1):

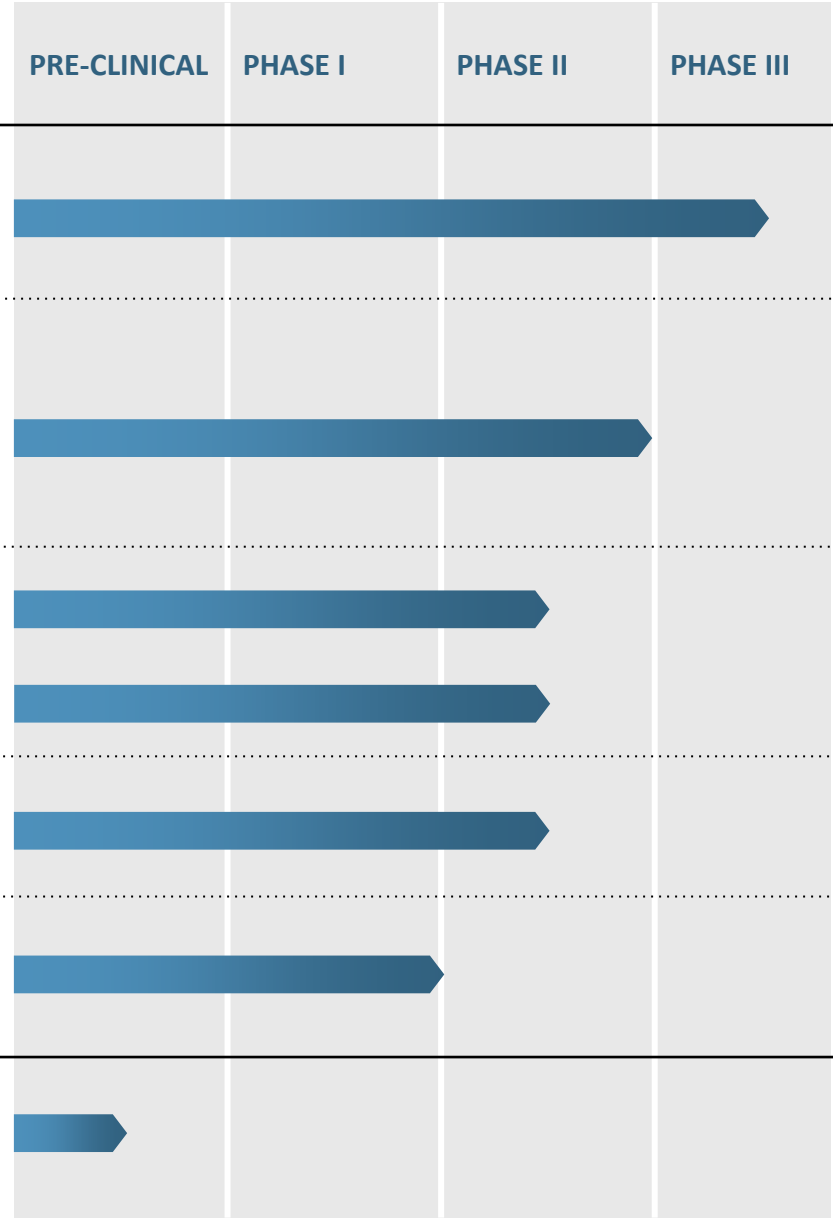
- **Proven anti-inflammatory effect** in multiple Phase II studies; favorable safety profile & excellent tolerability in >300 patients
- Statistically significant reduction of inflammatory lesions and impressive long-term efficacy in Hidradenitis Suppurativa (HS)
- **Encouraging Phase II data in patients with Severe COVID-19**

MULTIPLE ONGOING STUDIES

- **Severe COVID-19**: Phase III part of study is enrolling
- **HS**: Plan to submit Special Protocol Assessment (SPA) to the FDA for the Phase III in Q1 2021; positive scientific advice from European Medicines Agency (EMA)
- **ANCA-associated vasculitis (AAV) & Pyoderma Gangraenosum (PG)**: clinical data readouts expected in 2021

Pipeline with Multiple Opportunities

	PROPOSED INDICATIONS	PREVALENCE	UPDATE
Vilobelimab (IFX-1) ***C5a Inhibitor***	Severe COVID-19	• Currently unknown	• Phase II/III study: Phase II part: results published; Phase III part is enrolling
	Hidradenitis Suppurativa	• Up to 200,000 patients in the US • Over 200,000 patients in Europe	• Phase IIb completed • Positive EMA advice on pivotal program with new primary endpoint; SPA for the Phase III to be submitted to the FDA in Q1 '21
	ANCA-Associated Vasculitis	• ~40,000 patients in the US • ~75,000 patients in Europe	• Phase II: treatment completed in US; fully enrolled in Europe
	Pyoderma Gangraenosum	• ~50,000 patients in the US and Europe are affected	• Phase IIa open label; enrollment ongoing
	Cutaneous Squamous Cell Carcinoma (cSCC)	• PD-1 or PD-L1 Resistant/Refractory Locally Advanced or Metastatic	• Phase II to be initiated in FH 2021
IFX-2 ***C5a Inhibitor***	Undisclosed Chronic Inflammatory and Autoimmune Diseases	• Not applicable	• Developing for optimized use for other chronic inflammatory indications



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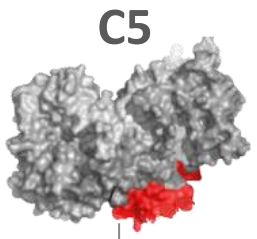
The Terminal Complement Pathway



C5

C5 concentration in blood:
~75 µg/ml (~400 nM)

C5a concentration in blood:
10 ~ 30 ng/ml
(~1-2.5 nM)

C5a

other ligands:
C3a, ASP, C4a etc

C5b

Membrane Attack Complex (MAC) triggers lysis of pathogens

C5b-9 = MAC

strong amplifier of inflammation

upregulated in many tissues during inflammation

C5aR

C5L2

C5L2 has a different binding pocket for C5a compared to other ligands like C3a, ASP, etc. and this causes different cell signaling.*
The C5a signaling has been shown to be pro-inflammatory.**

other signalling involved e.g. in triglyceride synthesis, etc.

- cell activation
- cytokine generation
- PKC-signaling
- **HMGB-1 induction*** (Inflammasome)

INFLAMMATION

* Kalant D. et. al. J. Biol. Chem. 2003, 278 (13) 11123–11129

**Rittirsch et al. Nat Med. 2008 May ; 14(5): 551; Colley et al. MABS. 2018,10 (1), 104Songlin et. al. J. Biol. Chem. 2019; 294(21) 8384–839 Muenstermann et al.. Virulence, 2020; 10(1) 677-694

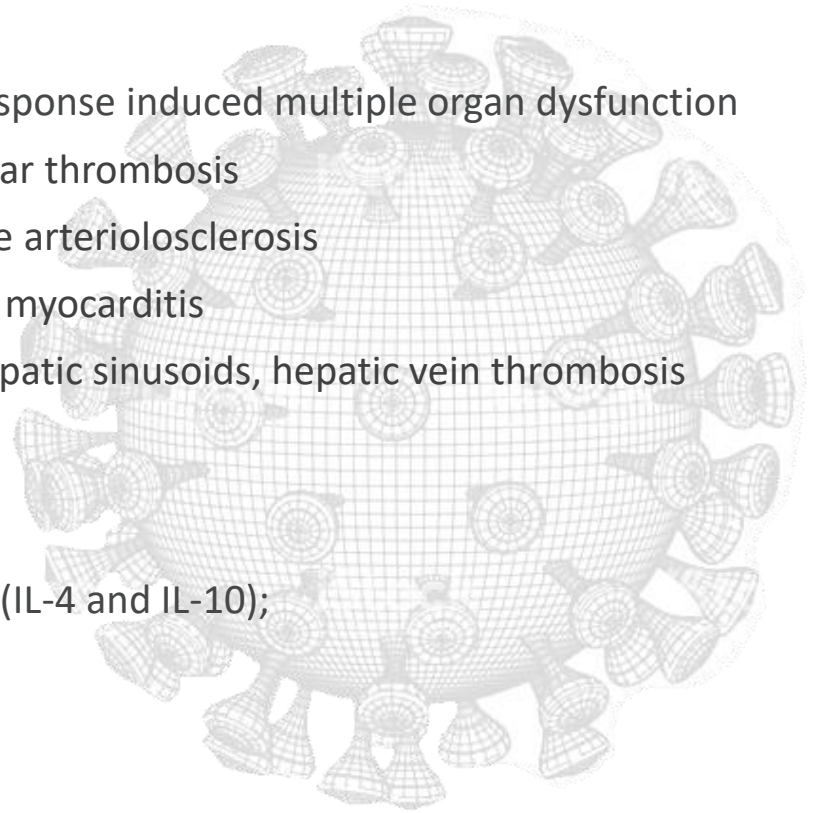
VILOBELIMAB (IFX-1) FOR SEVERE COVID-19

Coronavirus Disease 2019 (COVID-19)

A VIRAL PNEUMONIA WITH A BROAD SPECTRUM OF IMMUNE-MEDIATED INJURY

CLINICAL & PATHOLOGY FEATURES

- Death is typically caused by respiratory failure and viral sepsis in presence of immune-response induced multiple organ dysfunction
- Pathology in lung: extensive inflammation, diffuse alveolar damage, marked microvascular thrombosis
  in kidney: thrombotic microangiopathy within the glomeruli; mild to moderate arteriolosclerosis
  in heart: scattered individual cell myocyte necrosis, not sufficient sign of viral myocarditis
  in liver: macro-vesicular steatosis, cirrhosis, platelet-fibrin microthrombi in hepatic sinusoids, hepatic vein thrombosis



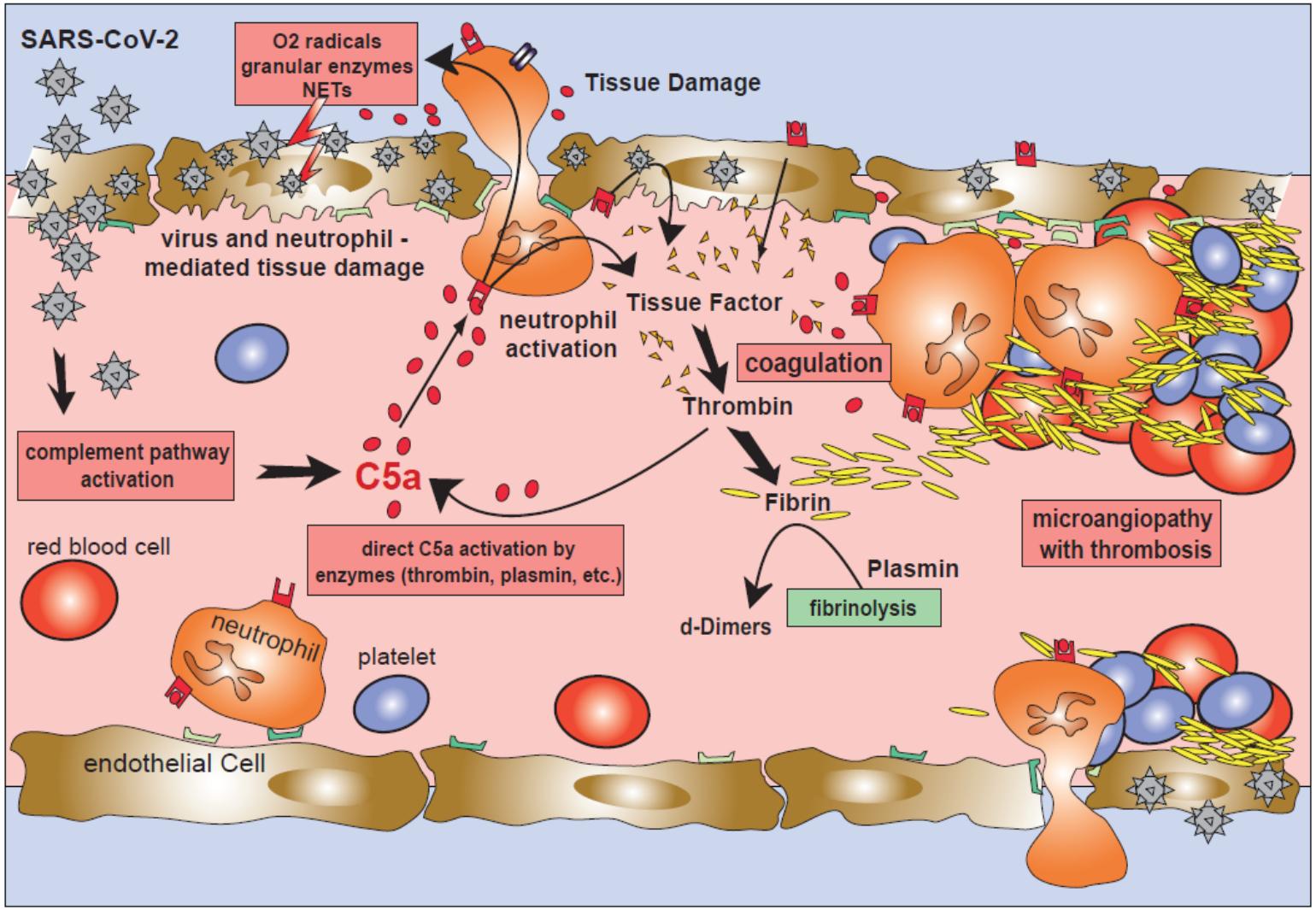
LABORATORY FINDINGS

- Systemic inflammation: lymphocytopenia (>80%) + elevated CRP (>60%) at admission
- Moderately elevated levels of both Th1 cytokines (IL-6, TNF-α, IFN-Y) and TH2 cytokines (IL-4 and IL-10);
- Other frequently increased markers: LDH, AST, ALT, troponin-I, ESR, serum ferritin et al.
- Coagulopathy markers: increased levels of D-dimer, fibrinogen, VWF, Factor VIII et al.
- Complement activation markers: C5a, sC5b-9

Source: https://www.chinalawtranslate.com/en/coronavirus-treatment-plan-7/;
Rapkiewicz et all, EclinicalMedicine (2020) 100434; Goshua et al., Lancet Haematol 2020 June 30; Cugno et al., J Allergy Clin Immunol July 2020:215;

COVID-19 induced Vascular Injury – Potential Role of C5a

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



- 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 as well as 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 viscous circle leading to microangiopathy with thrombosis

Source: InflaRx GmbH

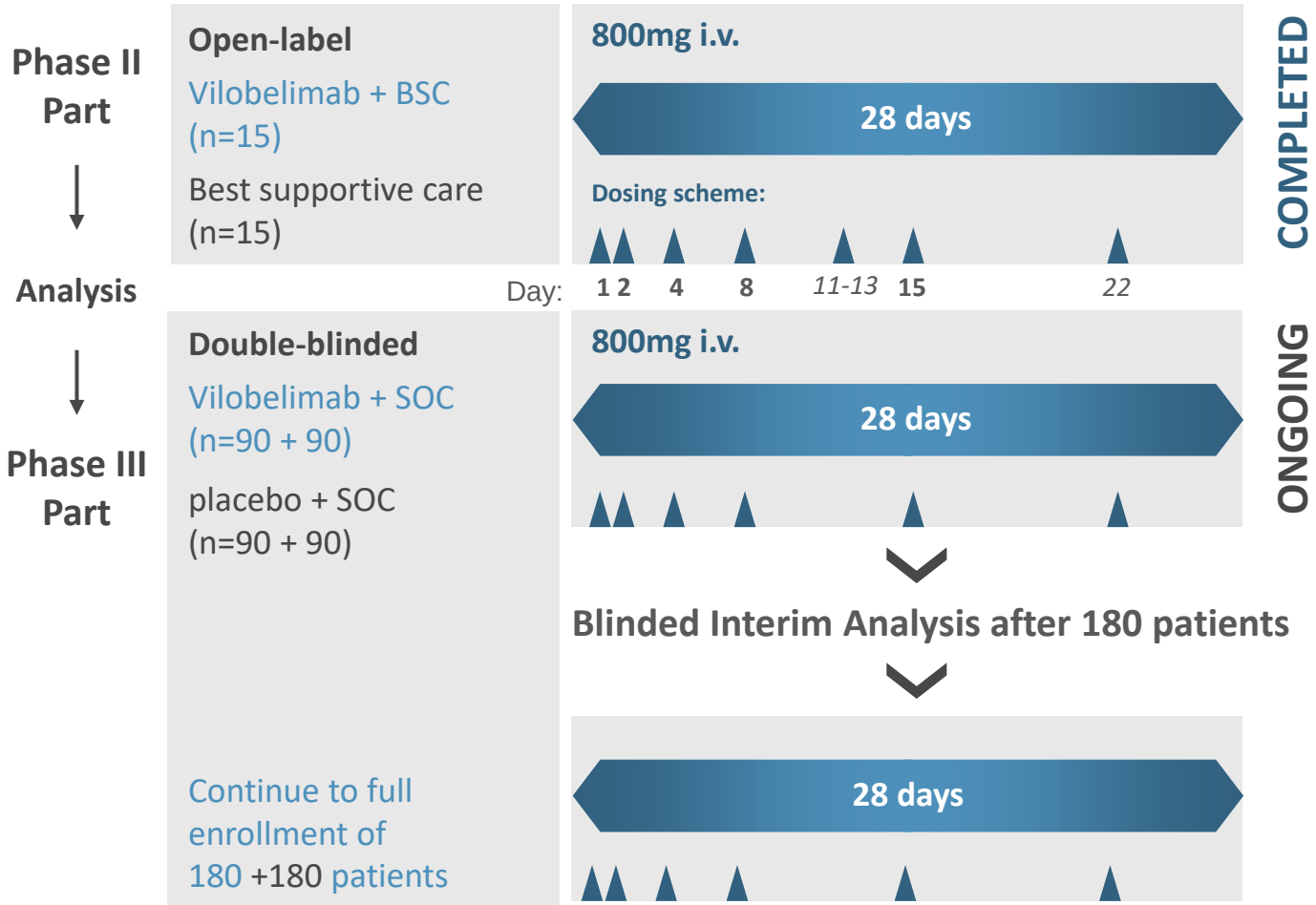
Design of Phase II/III study with Vilobelimab in Severe COVID-19

STUDY DESIGN

- Critically ill intubated* patients with COVID-19 induced pneumonia
- Phase III primary endpoint: 28-day all-cause mortality
- Other key endpoints include assessments of organ support, assessment of disease improvement on the ordinal scale

STATUS

- **Phase 2 part completed:**
  Encouraging Topline Results published
- **Phase 3 part ongoing:**
  Blinded Interim Analysis after 180 patients
  Potential for an early stop for efficacy or futility



SOC: Standard of Care
SOC includes venous thromboembolism prophylaxis at a minimum and may include other specific recommended treatments for COVID-19 per the locally adopted treatment recommendations.
* in phase III part eligible patients must be early intubated, in the phase II part, patients were enrolled if either being early intubated or dependent on oxygen delivery

Phase II Part Results: Overview

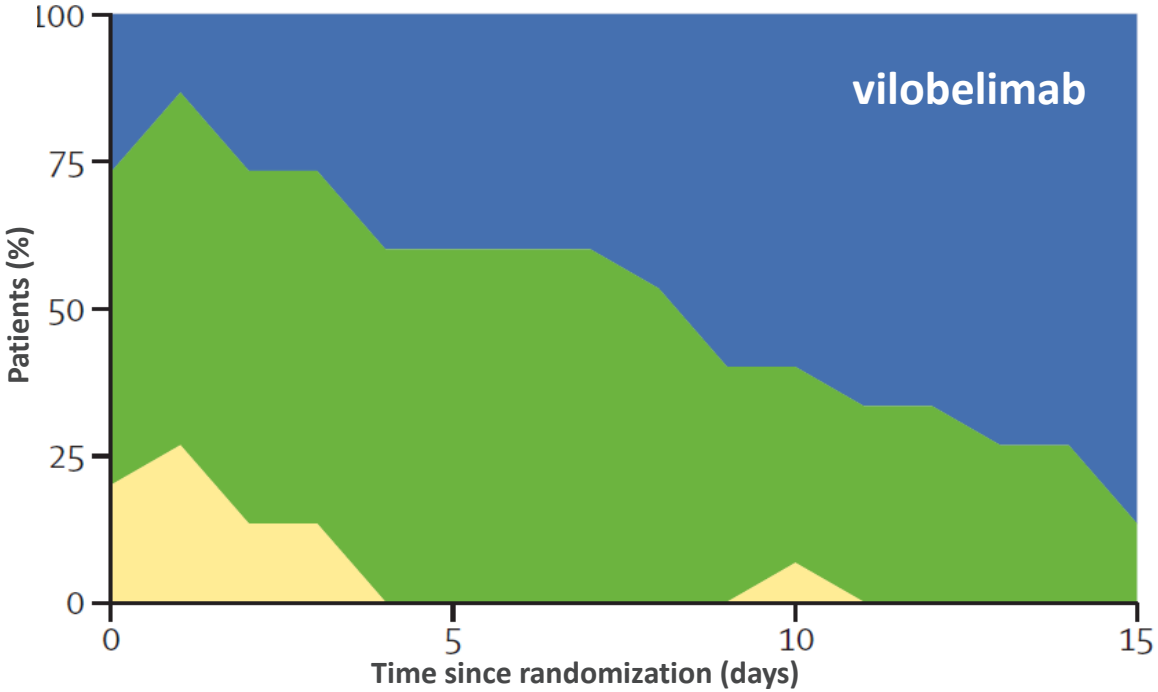
PHASE II STUDY RESULTS*

- **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 in vilobelimab treatment arm compared to best standard of care arm:**
  - **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 of D-dimer levels** in first days after vilobelimab administration - **potential signal of induction of blood clot lysis**

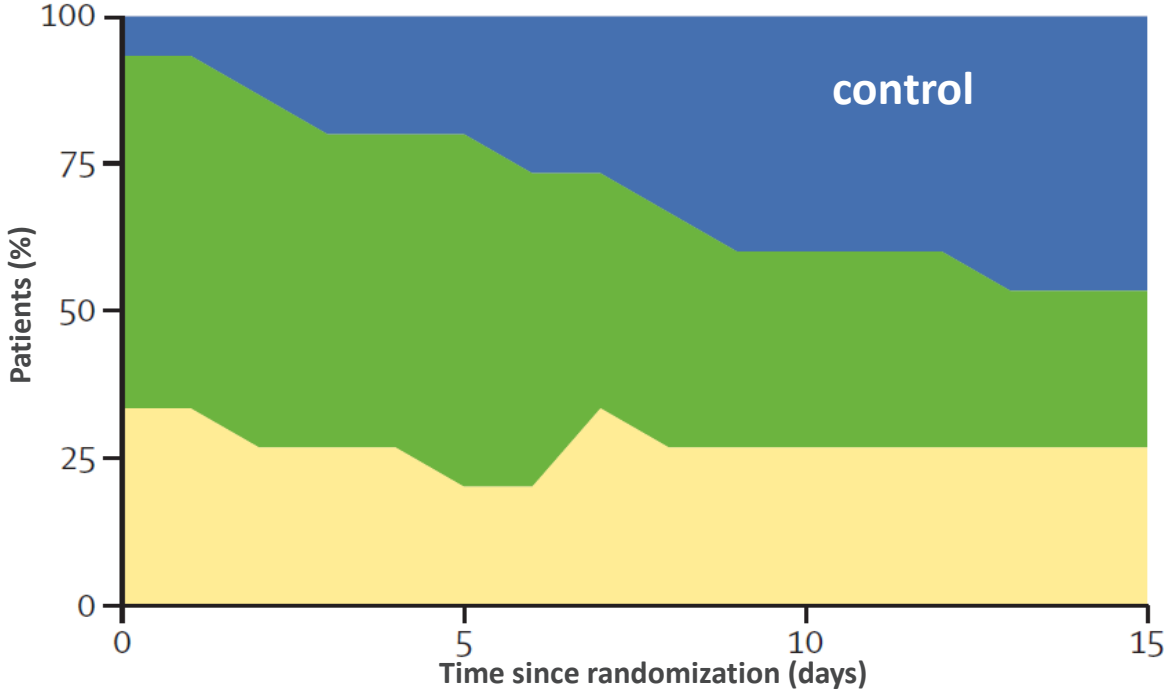
* Vlaar, A et al. *Lancet Rheumatol 2020*. https://doi.org/10.1016/S2665-9913(20)30341-6

Phase II Part Results: Lymphocyte Count Normalization

LYMPHOCYTES - VILOBELIMAB+BEST SUPPORTIVE CARE*



LYMPHOCYTES - BEST SUPPORTIVE CARE ONLY*



Normal (1.5 to 4.0 x 10^9 per L) Low (0.75 to 1.5 x 10^9 per L) Very low (<0.75 x 10^9 per L)

87% of vilobelimab treated patients showed normalized lymphocytes counts vs 47 % in control group (p=0.050)

* Shift Plot Lymphocytes – all randomized, n=15 per arm

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

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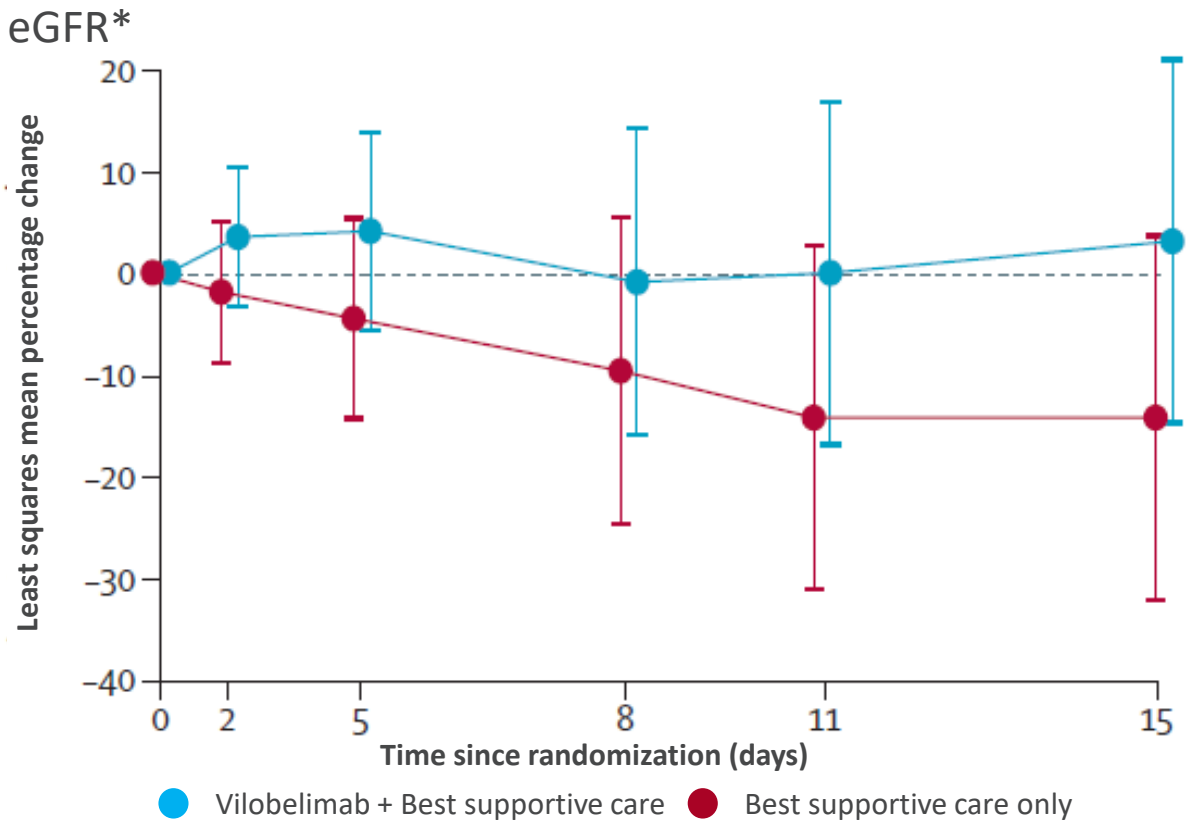
Phase II Part Results: eGFR Level Stability

eGFR LEVEL

- At day 15, mean eGFR showed a 3% change from baseline in the vilobelimab group versus -14% in the best supportive care group
- Only 1 patient developed a kidney injury in the vilobelimab group vs 4 patients in the control group developed acute renal failure with moderately to severely decreased eGFR (7% vs. 27%)
- **eGFR higher and mean unchanged in vilobelimab-treated patients** while a trend to worsening could be detected in the best supportive care group



Kidney function in vilobelimab treated patients remained mostly within normal limits or declined only mildly

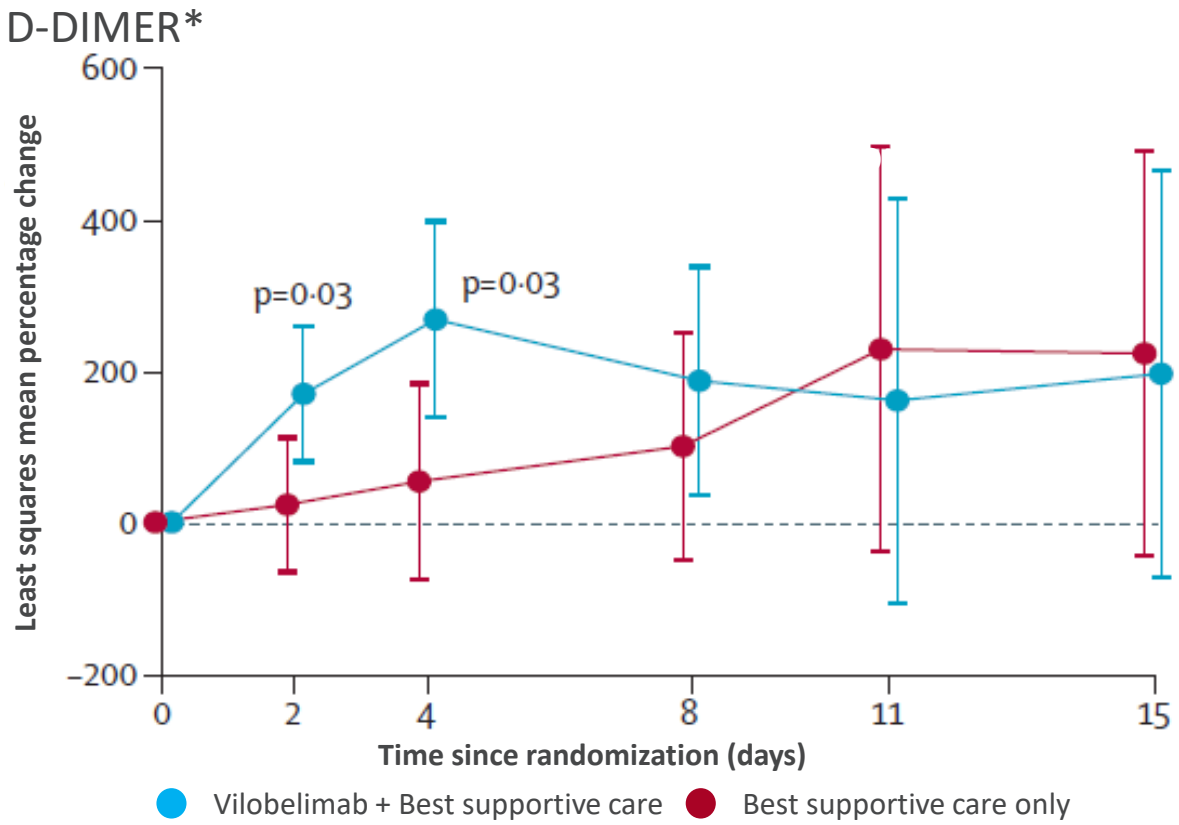
eGFR: estimated glomerular filtration rate

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

Phase II Part Results: Vilobelimab Treatment Associated Significant Increase in D-dimer Levels

LEVELS OF D-DIMER

- **Significant temporary D-dimer increase** observed directly upon initiation of vilobelimab therapy (day 2)
- Possible sign of induction of a direct or indirect pro-fibrinolytic effect
- In line with observed 3-fold lower rate in pulmonary embolisms reported as SAE´s in vilobelimab treatment arm and may be mechanistically linked to observed lower death rate
- Hypothesis: Inhibition of C5a by vilobelimab may lead to a **decrease in C5a-induced coagulation** and **directly or indirectly fostered thrombolysis**



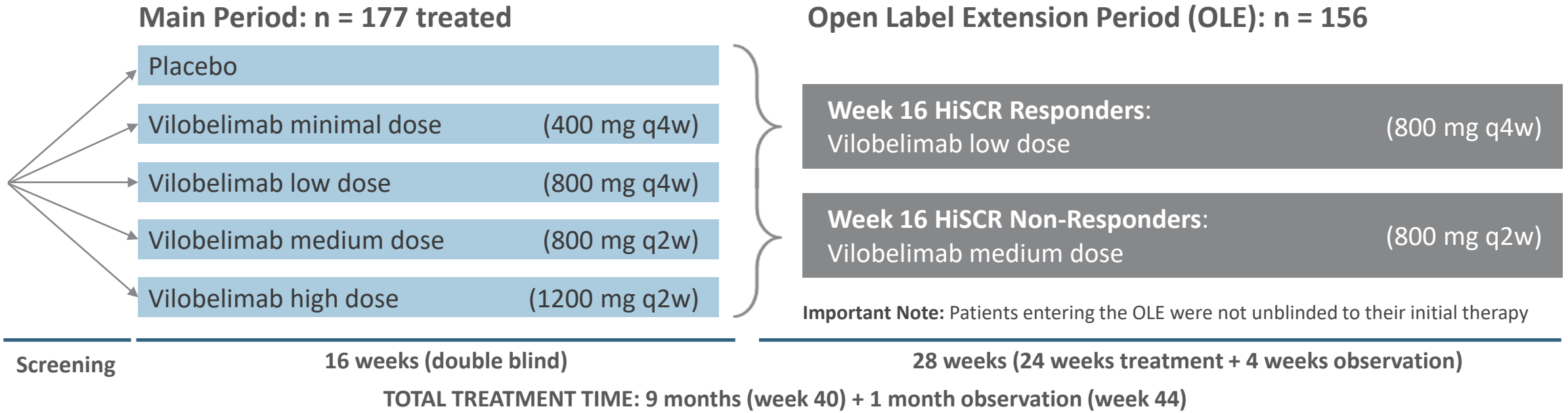
Vilobelimab treatment was associated with significant increase of D-dimer levels, suggesting potential pro-fibrinolytic activity of anti-C5a treatment

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

VILOBELIMAB (IFX-1) FOR HIDRADENITIS SUPPURATIVA

Hidradenitis Suppurativa (HS)
Phase IIb SHINE Study Details

HS: A chronic severely debilitating C5a-driven inflammatory skin condition with high unmet need



MAIN GOALS

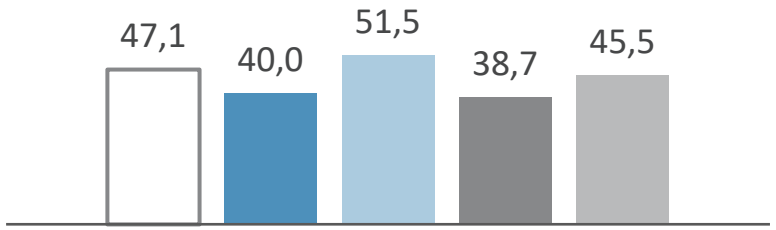
- Test a dose-dependent effect of vilobelimab on HiSCR* response at week 16 (primary endpoint)
- Assess long-term safety of vilobelimab
- Test durability of response with lower maintenance therapy in open label extension period

*HiSCR response defined as: At least 50% reduction in total AN count (abscesses & inflammatory nodules) with no increase in the number of abscesses from baseline and no increase in the number of draining fistulas from baseline

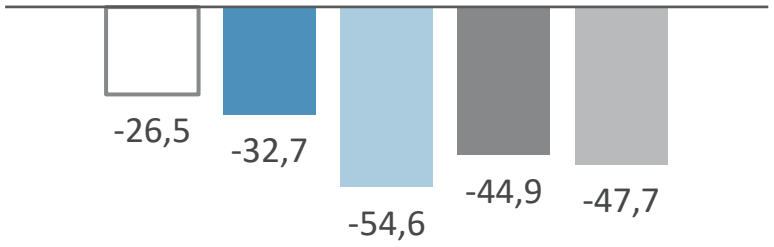
SHINE Study:
Outcome at Week 16

HISCR RESPONSE RATE (%) WEEK 16*

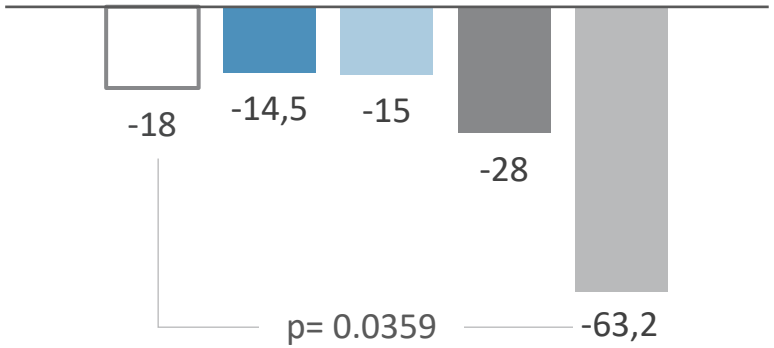
n = approx. 35/ group



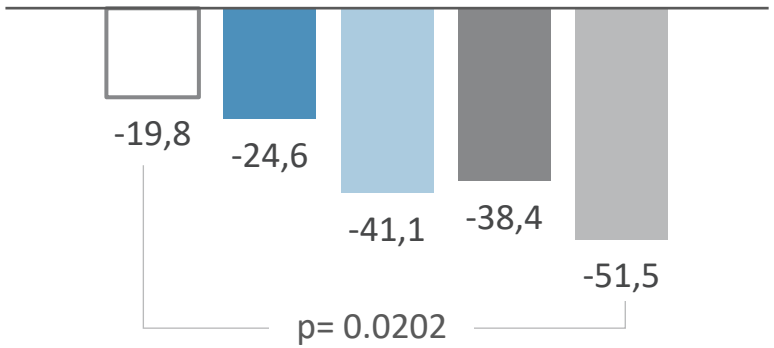
AN COUNT SCORE CHANGE (MEAN %) WEEK 16*



DRAINING FISTULA CHANGE (MEAN %)**



IHS-4 SCORE CHANGE (MEAN %)**



Treatment group:

- placebo
- minimum
- low
- medium
- high

› Primary Endpoint HiSCR Dose Response Signal not met but Signal towards Improved AN count
Statistically significant change in DF and in IHS-4 scores detected

* Full analysis set

** Full analysis set – baseline adjusted

IHS-4 Points = Sum of the number of inflammatory nodules (x1), number of abscesses (x2) and number of draining fistulas (x4)
AN Count = Total number of combined inflammatory nodules and abscesses

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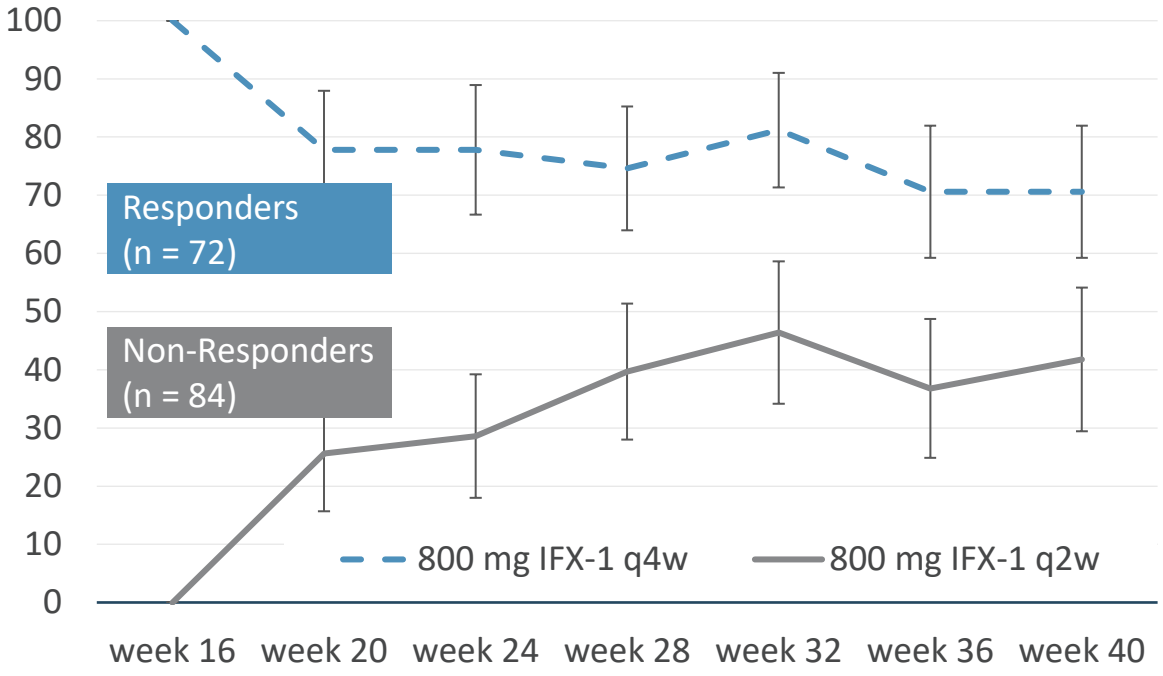
Responder versus Non-responder OLE Week 16 to Week 40

HISCR RESPONSE RATE (%) PER VISIT* (OLE) WITH 95% CI

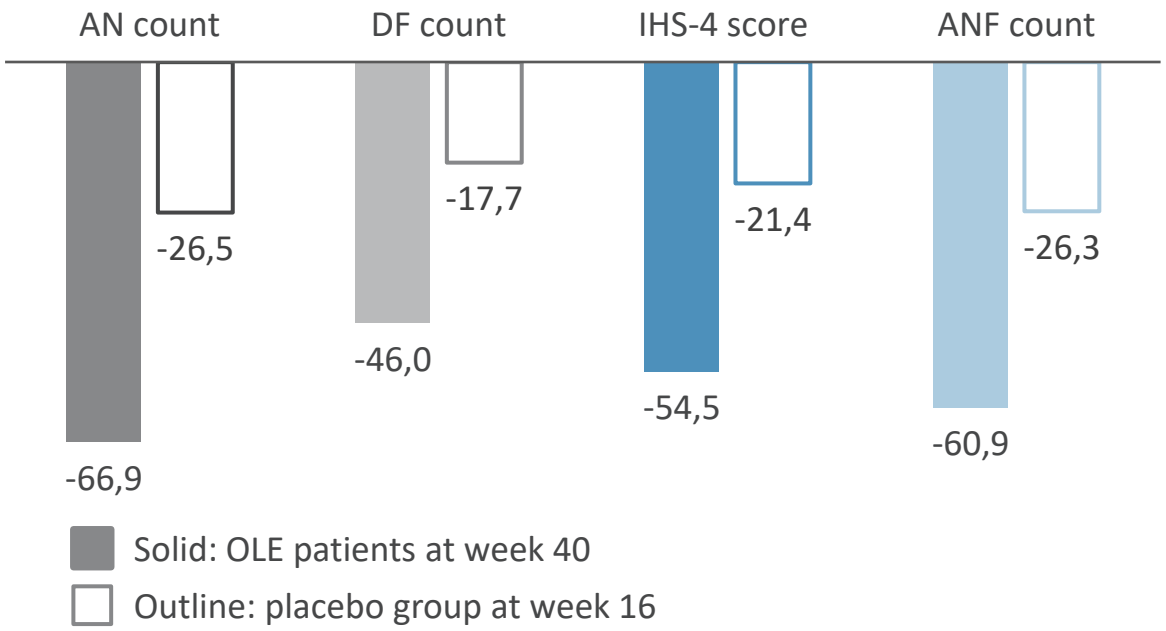


› Responders: 71 % maintain HiSCR response
Non-responders: 42 % become HiSCR responders

* Full analysis set; OLE: open label extension

Inflammatory Lesion Reductions: OLE Patients (w40) Compared to Placebo (w16)

RELATIVE REDUCTION (% MEAN)
COMPARED TO RESPECTIVE BASELINE (DAY1)*



› Marked improvement of all inflammatory lesions over time – not explainable by placebo effect

* Full analysis set (unadjusted)

SHINE Study and Next Steps in HS Development

OUR CONCLUSIONS

- HiSCR is burdened by high variability (driven by AN count variability) and by a lack of capturing reduction of draining fistulas
- Evidence for a high C5a turnover rate in HS, leading to increased dose requirements of vilobelimab
- Vilobelimab leads to a marked reduction of all inflammatory lesions in HS with a durable long-term effect detected even at non-optimal doses
- Vilobelimab long-term treatment was well tolerated, no drug related SAEs* in the open label extension phase

CURRENT STATUS & NEXT STEPS

- **Scientific Advice received from EMA in July 2020**
  - EMA agreed to key proposals for pivotal program** including change of primary endpoint to support MAA submission
  - Acknowledged that HiSCR response does not account for the clinical relevance of a reduction in draining fistulas.
  - Agreed that **IHS4 could be an appropriate tool to evaluate the efficacy of a novel compound in HS as primary endpoint**
- **End-of-Phase II meeting with FDA held in June 2020**
  - FDA agreed to key proposals** to support BLA submission
  - FDA did not agree that IHS4 score is fit for purpose as a primary efficacy endpoint tool to support labeling
    - Recommended that IFRX obtain HS patient input to help determine validity
- **Plan to submit Special Protocol Assessment (SPA) to the FDA for the Phase III in Q1 2021**

* Serious adverse events
** including aspects of the Ph III design, vilobelimab dosing, target study population, nonclinical & clinical pharmacology packages

VILOBELIMAB (IFX-1) FOR ANCA-ASSOCIATED VASCULITIS

AAV, Life-threatening Autoimmune Condition
Clinical PoC established for Role of C5a / C5aR Pathway in AAV

LEADING PROPRIETARY ANTI-C5A TECHNOLOGY

- C5a is essential for development of MPA-ANCA crescentic glomerulonephritis in a mouse model
- Complement activation in active AAV patients is significant
- Evidence for role of C5a / C5aR pathway through recent Phase III success of an oral C5aR inhibitor*

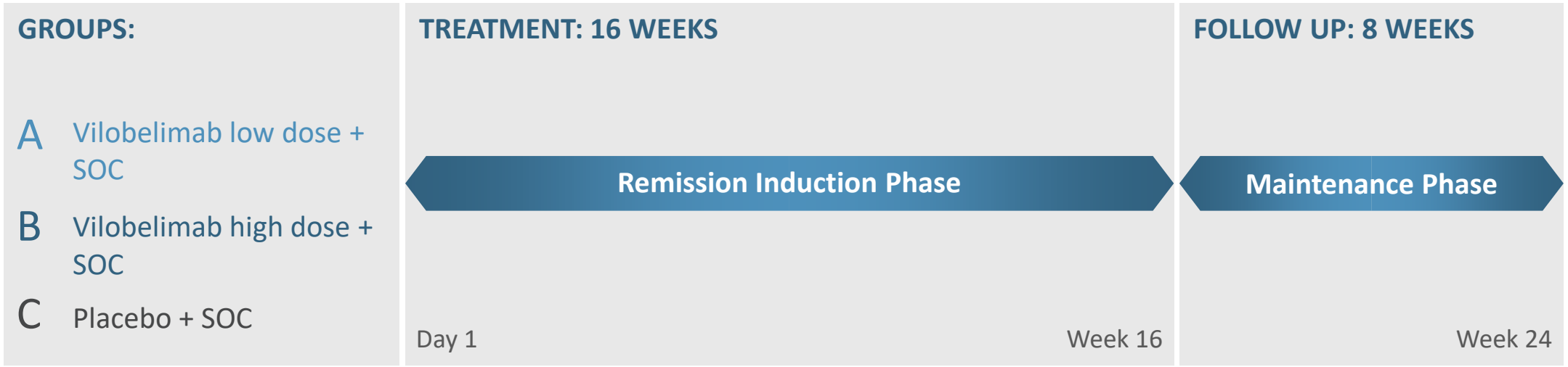
POTENTIAL ADVANTAGES OF VILOBELIMAB (IFX-1) FOR AAV

- **Rapid onset of action:** intravenous administration with fast onset of action, inhibiting C5a signaling completely protecting from C5a induced priming and activation of neutrophils → potentially quicker induction of remission compared to the SOC
- **Potential potency difference:** by blocking upstream ligand C5a, which inhibits signaling through both receptors, C5aR and C5L2; C5a pro-inflammatory MoA through both C5aR and C5L2 has been shown to be important for ANCA-primed and C5a-induced neutrophil degranulation as key disease-driving mechanism in AAV.**

* Chemocentryx. (25 November 2019). ChemoCentryx and VFMCRP Announce Positive Topline Data from Pivotal Phase III ADVOCATE Trial Demonstrating Avacopan's Superiority Over Standard of Care in ANCA-Associated Vasculitis; (9 July 2020) ChemoCentryx Submits New Drug Application to the U.S. FDA for Avacopan in ANCA-Associated Vasculitis

** Hao & Wang et al 2013, PLoS ONE, 8(6)

Phase II Study in AAV in the US (IXPLORE)
Study Design



Randomization 1:1:1

SOC = Rituximab or Cyclophosphamide + glucocorticoids

Maintenance Therapy:
remain on SOC or transition to guideline maintenance therapy*

Study objectives (total enrolled: n=19)

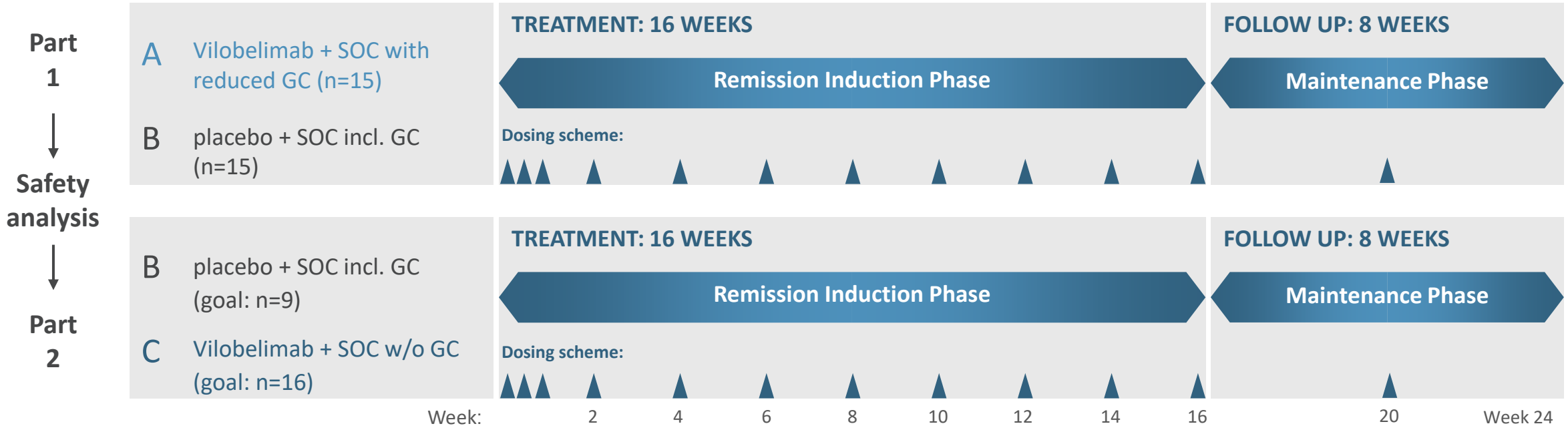
- Assess safety and efficacy of vilobelimab in AAV
- Primary objective: Safety
- Secondary objectives: Efficacy (Response rate based on the Birmingham Vasculitis Score (BVAS), various other)

Status:

- Blinded interim analysis completed
- Enrollment finalized early following assessment of interim analysis and of potential impact of COVID-19 pandemic; patients have finished treatment and data is expected in the first half of 2021

* Guideline maintenance allows for: azathioprin / methotrexate / mycophenolate mofetil / low dose corticosteroids

Phase II study in AAV in Europe (IXCHANGE)
Study Design



SOC = rituximab or cyclophosphamide
GC = glucocorticoids

Maintenance Therapy:
remain on SOC or transition to guideline maintenance therapy*

Study (total enrolled: n=57)**

- Primary objective: Proof of Concept for efficacy of vilobelimab as replacement for glucocorticoid (GC) therapy in GPA and MPA
- Secondary objectives: To assess safety and tolerability of vilobelimab & compare toxicity of standard-dose GC with vilobelimab
- Status: Blinded interim analysis of Part 1 completed. Part 2: enrollment finalized. Final results expected in 2021

- Guideline maintenance allows for: azathioprin / methotrexate / mycophenolate mofetil / low dose corticosteroids

** Enrolled two more patients on Part 2 over the goal

VILOBELIMAB (IFX-1) FOR PYODERMA GANGRAENOSUM

Pyoderma Gangraenosum (PG)
Rationale and Phase IIa Study Overview

STUDY OBJECTIVE

- Assessing safety and efficacy of vilobelimab in PG
- **Rationale:**
  Pyoderma Gangraenosum (PG) is a rare ulcerative skin disease with a high unmet medical need. PG is associated with a neutrophilic leukocytosis, which is likely to be triggered by C5a. Pyoderma Gangraenosum lesions have pronounced neutrophilic infiltration and the expression of interleukin (IL)-1β, IL-17, tumor necrosis factor (TNF)-alpha, and their receptors are significantly elevated, indicating auto-inflammatory conditions.
- **Primary endpoint:**
  Safety
- **Key secondary endpoints:**
  Responder rate defined as Physicians Global Assessment ≤3 of target ulcer at visits V4, V6, V10, and V16 (end of treatment); Time to complete closure of Pyoderma Gangraenosum target ulcer (investigator assessment)

STUDY DESIGN

- Open label
- Multicenter
- Target enrollment – **18 patients**
- First patient dosed – June 2019

TREATMENT

- The first 5 patients were dosed at 800mg biweekly and have finished treatment
- Two additional biweekly higher dose groups have been added; enrollment is ongoing, and patients are under treatment
- **Additional data in the higher dose groups expected in 2021**

Status and Next Steps

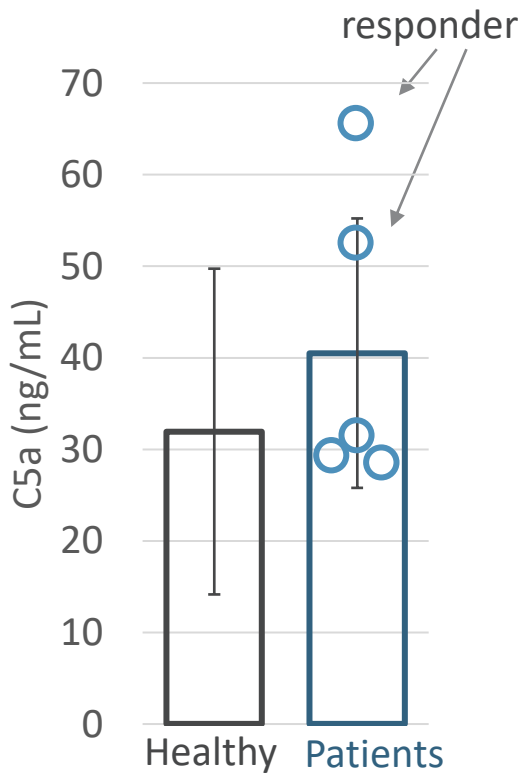
PHASE IIA STUDY UPDATE

Data reported on first 5 patients treated in Feb 2020

- 2 of 5 pts: **complete closure of target ulcer; full disease remission;** remained healed even after finishing the study
- Additional patient: **initial wound healing activity** in first 2-3 weeks of treatment; no wound size decrease or closure detected
- Remaining 2 pts had extensive disease*: target ulcer did not heal, were still under treatment
- The “responders” showed higher baseline C5a levels
- PD analysis (C5a levels) warranted higher dosing
- Two additional higher dose groups are enrolling, and patients are undergoing treatment
- **Additional data with higher doses expected in 2021**

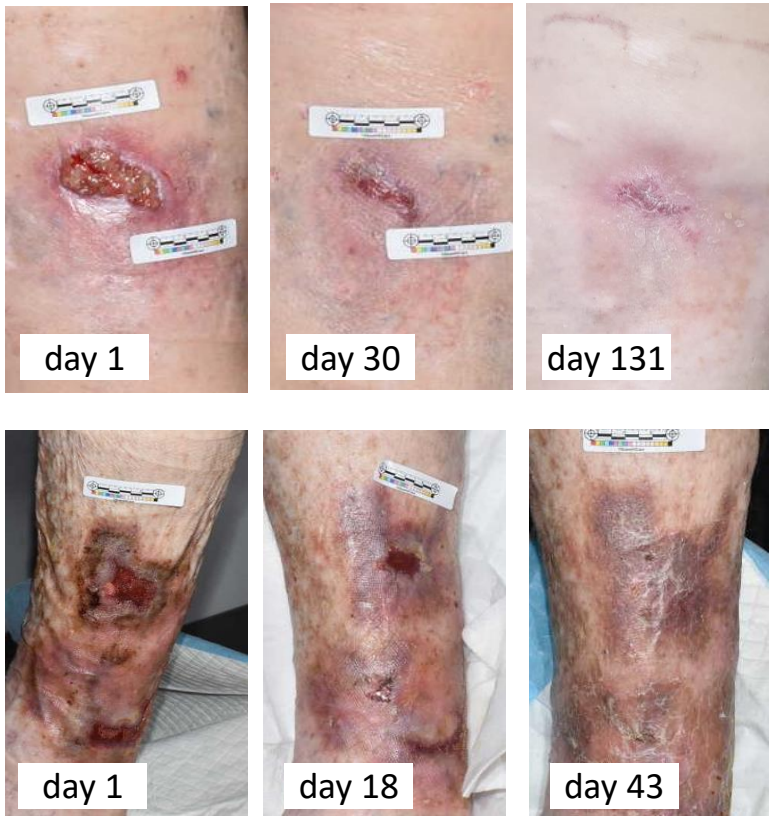
* very large ulceration / ulcer reaching entire circumference of leg

C5A LEVELS AT BASELINE



TREATMENTS

Two Patients Show Complete Wound Closure with Vilobelimab Treatment



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VILOBELIMAB (IFX-1) IN ONCOLOGY

Cutaneous Squamous Cell Carcinoma (cSCC)

PD-1 or PD-L1 Inhibitor Resistant/Refractory Locally Advanced or Metastatic Patients

POTENTIAL ROLE OF C5A IN ONCOLOGY & cSCC

- **C5a induces an immunosuppressive tumor microenvironment**
  - Accumulation of immunosuppressive MDSC and M2 macrophages[1]
  - Induction of PD-L1 expression on tumor-associated macrophages (TAM)[1,2]
  - PD-L1+ TAM are predictive for worse outcome of PD-1 inhibitor treatment[3]
- **C5a promotes metastases**
  - Increase of EMT, tumor cell motility and vascular permeability[4]
- **C5a is readily available in the tumor environment and may promote tumor growth directly**
  - Tumor cells, immune cells and coagulation pathway generate C5a[5]
  - Tumor cells inhibit complement deactivation[2]
  - C5aR expression increased in many epithelial tumors, incl. cSCC[1]

DISEASE INFORMATION cSCC

- Risk factors include Hidradenitis Suppurativa, cumulative UV radiation, irradiation, chronic inflammatory processes, immunosuppression, β-HPV infection, BRAF-inhibitor treatment (e.g., vemurafinib, dabrafenib)[6]
- **Incidence is estimated at 15-35 per 100,000 people**; expected to increase 2-4% per year; **Metastasizes in approximately 2-5%** of cases overall[7,8,10]
- Advanced SCC 10-year survival rates are **less than 20%** with regional lymph node involvement and **less than 10%** with distant metastases; Distant metastases have median survival of less than 2 years[7,9]
- 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 tumor growth
Combination of C5a with PD-1 checkpoint inhibition could reverse resistance to PD-1 or PD-L1 inhibitor therapy

EMT: epithelial–mesenchymal transition; MDSC: myeloid-derived suppressor cell

1. Medler et al., 2018; 2. Bonavita, 2015; 3. Liu, et al., 2019 ; 4. Hu et al., 2016; 5. Corrales et al., 2012; 6. Que et al., 2018; 7. Stratigos et al., 2015; 8. Burton et al., 2016; 9. Hillen et al., 2018; 10. Li et al, 2015

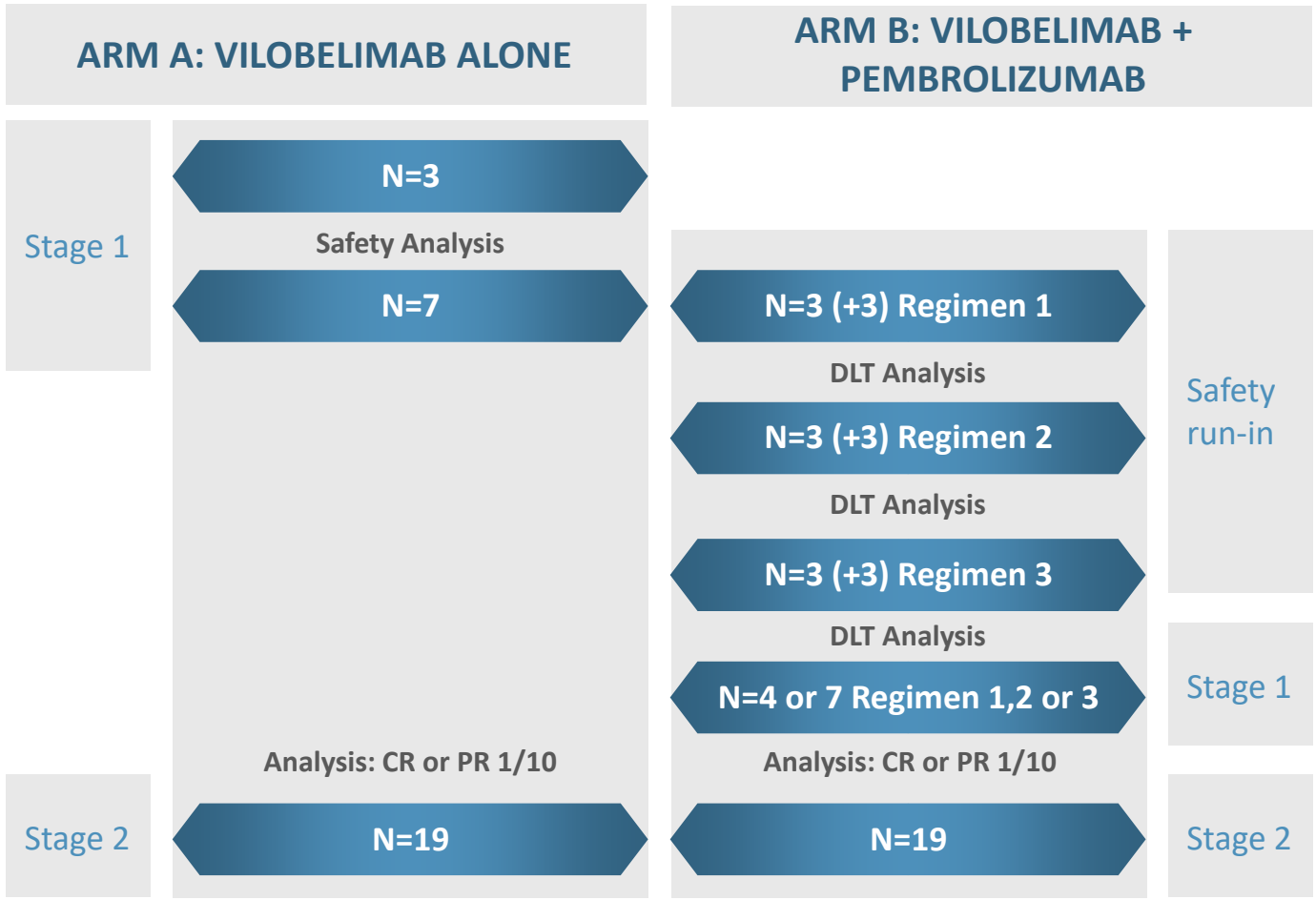
Study in Cutaneous Squamous Cell Carcinoma (cSCC)

INCLUSION CRITERIA

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

Primary Endpoints

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



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

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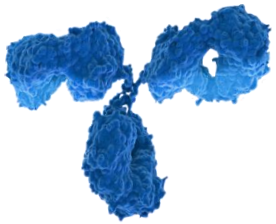
STRATEGY AND OUTLOOK

Medium Term Deliverables and Strategic Objectives



GOALS AND STRATEGY

Complete **Phase III development of lead program vilobelimab** in **Severe COVID-19; submit for approval** if results positive

Advance vilobelimab in **HS towards Phase III and ultimate approval based on regulatory guidance**

Explore application of vilobelimab for **AAV, PG and oncology in clinical development**

Explore extension of pipeline with initiation of clinical development of vilobelimab in **other complement-mediated autoimmune / inflammatory diseases**

Pursue development of early-stage pipeline and continue to expand the breadth of our anti-C5a technology

Continue to explore broadening the R&D pipeline beyond anti-C5a technology as part of diversification strategy

We have a strong cash balance to pursue these activities (€95.7 million as of September 30, 2020)

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