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CONTROLLING INFLAMMATION

CORPORATE PRESENTATION

September 2021

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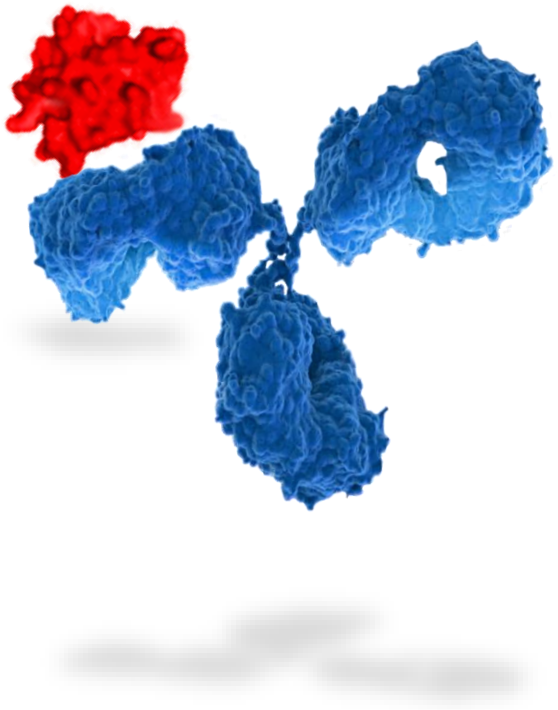
Investment Highlights

InflaRx's Approach: Targeting Complement to Control Autoimmune Diseases



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

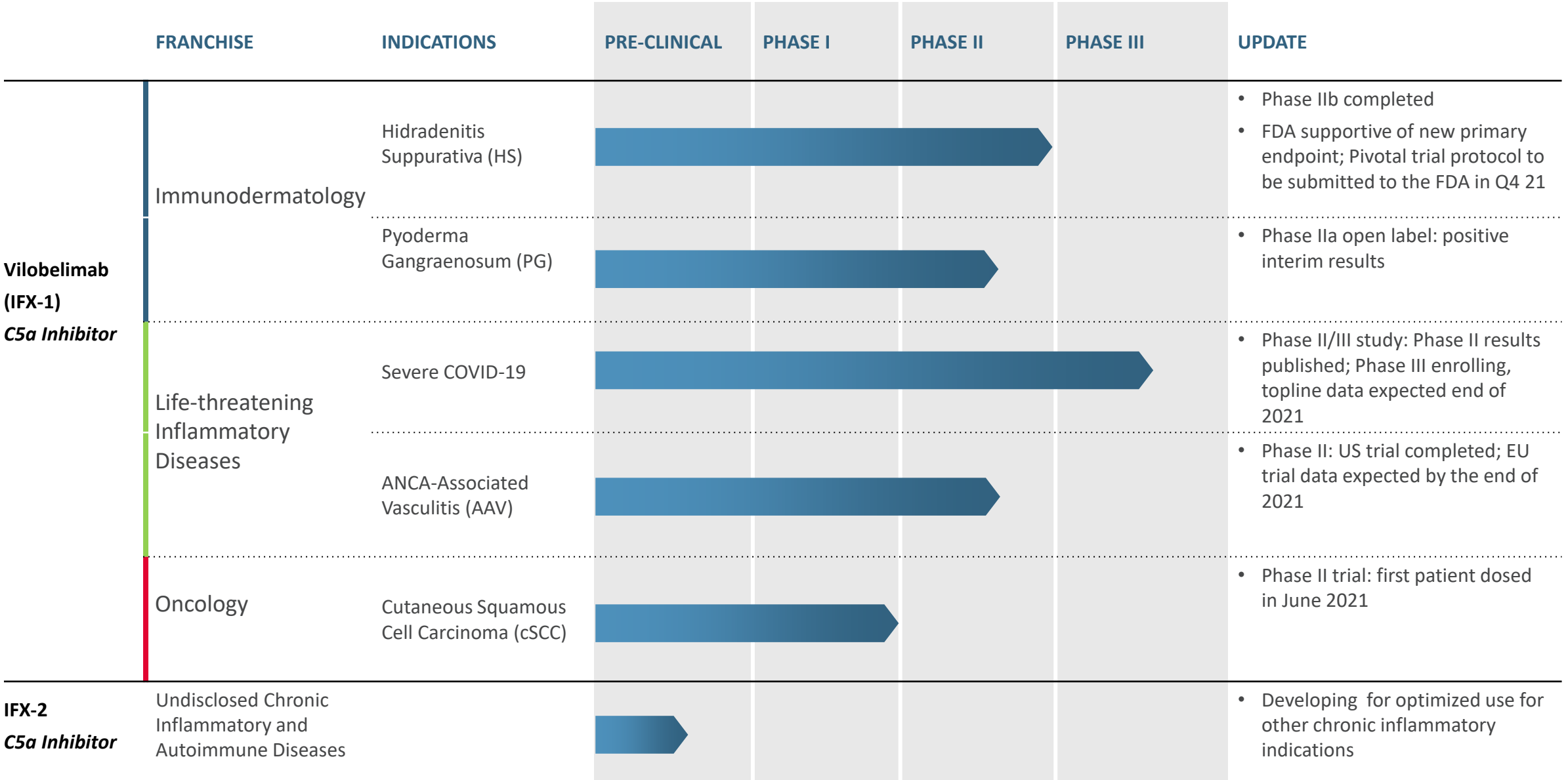
Established Clinical Efficacy for Vilobelimab (IFX-1):

- 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 and positive interim Phase IIa data in Pyoderma Gangraenosum (PG)

Multiple Clinical Programs and Near-Term Inflection Points

- **HS**: FDA supportive of pivotal study program that focuses on patients with active draining tunnels and a new primary endpoint that will measure the reduction of all three HS lesions - including draining tunnels
- **Severe COVID-19**: Phase III trial enrollment on track, topline data expected end of 2021
- **ANCA-associated vasculitis (AAV)** EU data readout in Q4 2021
- **Pyoderma Gangraenosum (PG):** Positive interim readouts - final Phase IIa data to readout in FH 22

Pipeline with Multiple Opportunities



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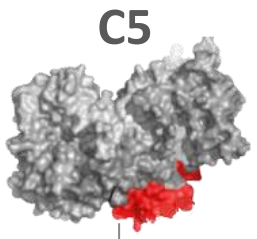
The Terminal Complement Pathway



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

Immunodermatology Focus

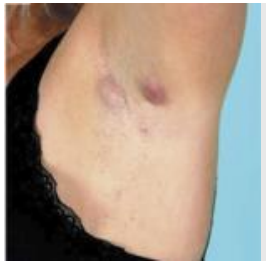
- Hidradenitis Suppurativa (HS)
- Pyoderma Gangraenosum (PG)

VILOBELIMAB (IFX-1) FOR HIDRADENITIS SUPPURATIVA

Hidradenitis Suppurativa (HS)

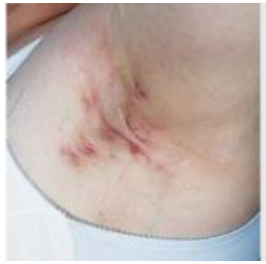
A chronic severely debilitating C5a-driven inflammatory skin condition with high unmet need

HURLEY STAGING FOR HS



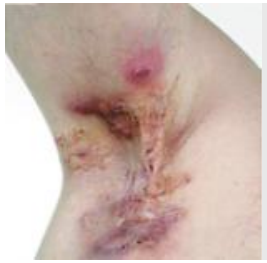
Stage I

Single / multiple abscesses but no sinus tracts or scarring



Stage II

Single or multiple separated, recurrent abscesses with tract formation and scarring



Stage III

Multiple interconnected tracts and abscesses involving an entire anatomic region

CLINICAL FEATURES

- Chronic, inflammatory, recurrent, debilitating skin disease of the hair follicles
- Most commonly in the armpit, groin and genital regions
- Extremely painful inflammatory nodules, boils or abscesses
- Draining fistulas leading to considerable scarring and functional disability
- Hurley staging system used to classify progression / chronicity (Stage I – III)

PREVALENCE

- Up to 200,000 moderate to severe (Hurley II+III) HS patients in US
- Higher prevalence in Europe with reports > 1% total HS prevalence

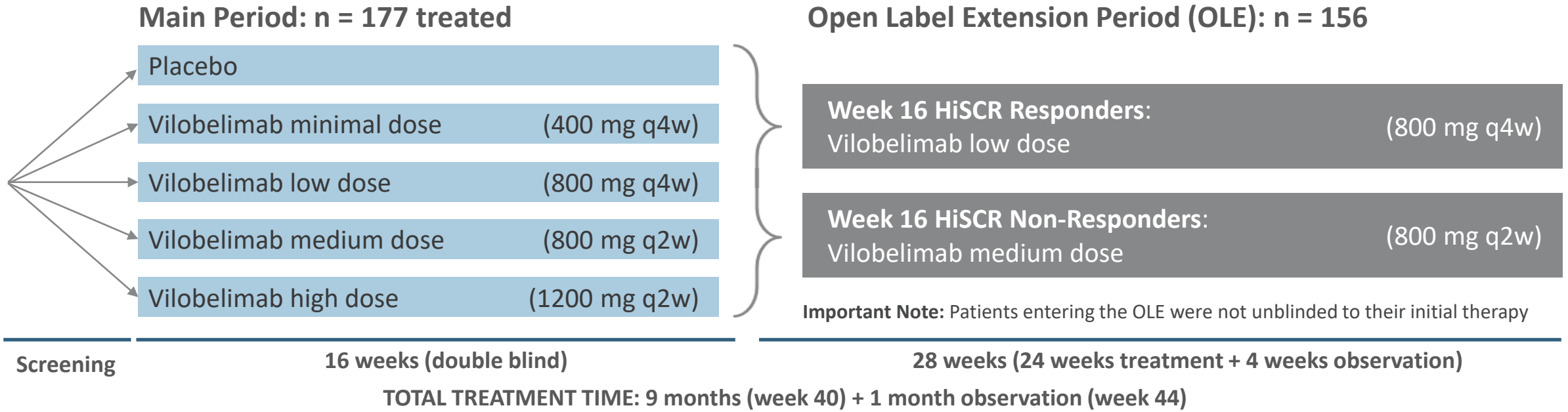
CURRENT TREATMENT – MEDICAL NEED

- Adalimumab (TNF-alpha inhibitor) from AbbVie is the only approved biological in US and Europe
- Accepted (but not approved) SOC includes topical, oral or i.v. antibiotics
- In some instances, surgery is required
- Approximately 50% of patients with moderate to severe HS do not respond and about 50% of responder patients lose response to Humira*

* Combined Phase III trial data for Humira: response measured by HiSCR 50 and KOL quotes in LifeSci Capital initiation report 12/2018

Hidradenitis Suppurativa (HS)
Phase IIb SHINE Study Details

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



MAIN GOALS

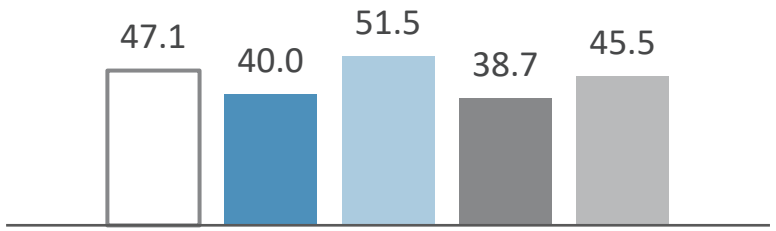
- Test a dose-dependent effect of vilobelimab on HiSCR* response at week 16 (primary endpoint)
- Assess long-term safety of vilobelimab
- Test durability of response with lower maintenance therapy in open label extension period

*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

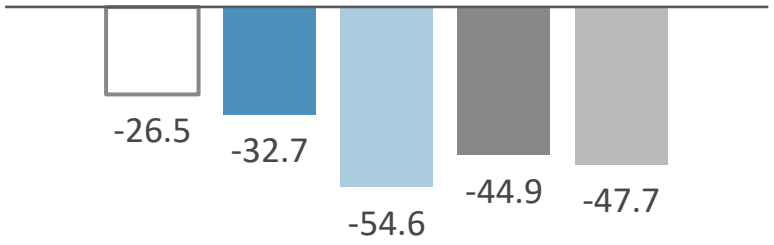
SHINE Study:
Outcome at Week 16

HISCR RESPONSE RATE (%) WEEK 16*

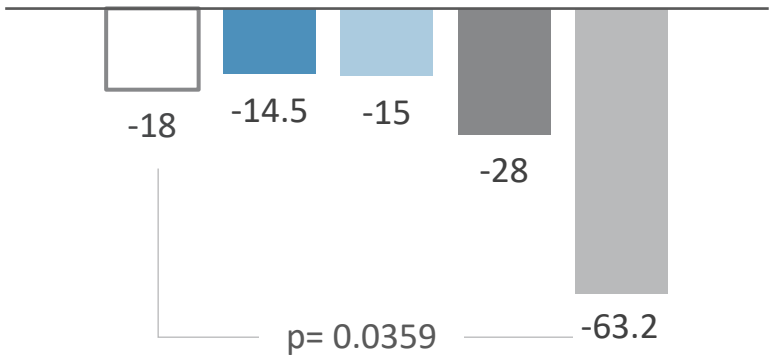
n = approx. 35/ group



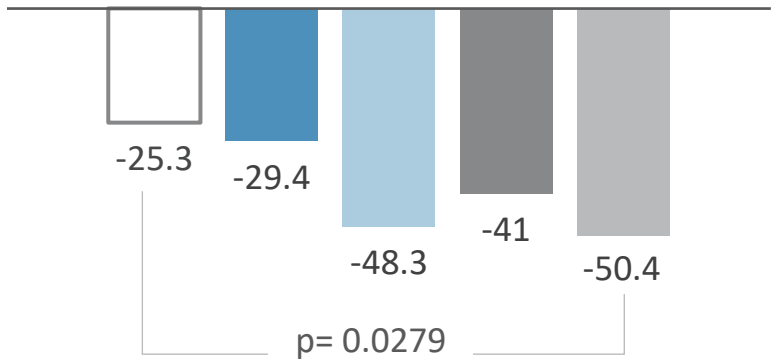
AN COUNT SCORE CHANGE (MEAN %) WEEK 16*



DRAINING TUNNEL CHANGE (MEAN %)**



ANDT COUNT 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 DT and in ANdT count detected

* Full analysis set

** Full analysis set – baseline adjusted

ANdT Count = Total number of combined inflammatory nodules, abscesses and draining tunnels
AN Count = Total number of combined inflammatory nodules and abscesses

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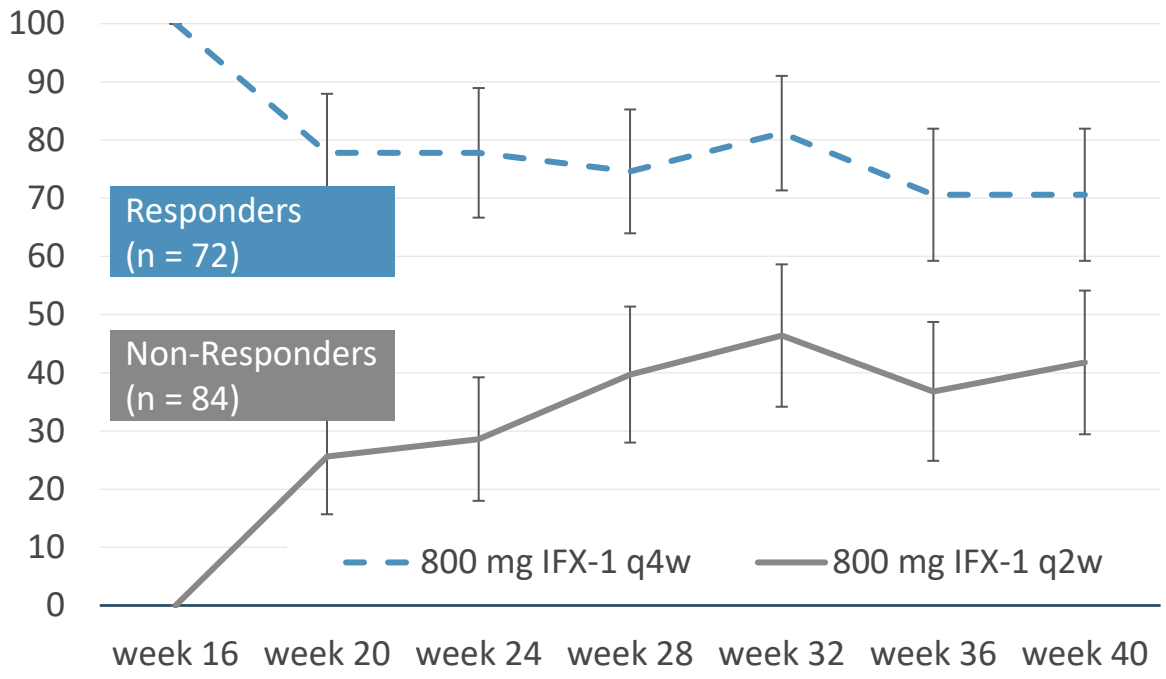
HiSCR Responder versus Non-responder - OLE Week 16 to Week 40

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

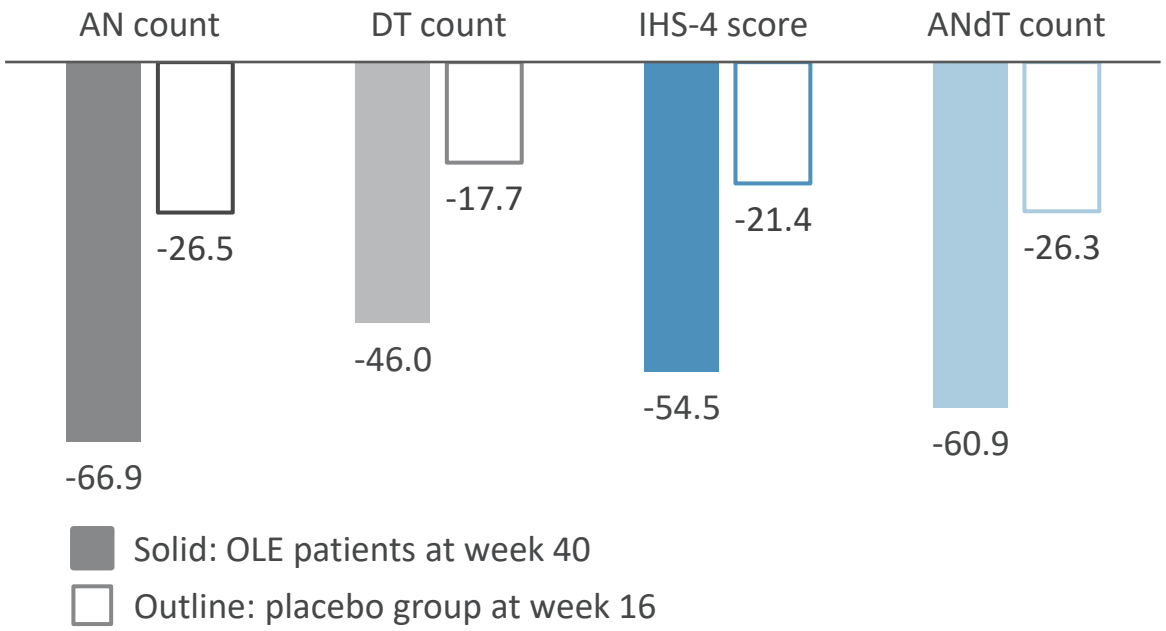


› Responders: 71 % maintain HiSCR response
Non-responders: 42 % become HiSCR responders

* 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)

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

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SHINE Study and Next Steps in HS Development

OUR CONCLUSIONS

- HiSCR is burdened by high variability (driven by AN count variability) and does not capture reduction of draining fistulas
- Evidence for a high C5a turnover rate in HS, leading to increased dose requirements of vilobelimab
- Vilobelimab leads to marked reduction in all inflammatory lesions, with a durable long-term effect detected even at sub-optimal doses
- Vilobelimab long-term treatment was well tolerated, no drug related SAEs* in OLE phase

CURRENT STATUS & NEXT STEPS

- **FDA Type A Meeting (August 2021)**
  - Received feedback from FDA within its Type A meeting which **is supportive of a new primary endpoint measuring reductions in all three inflammatory HS lesions** – including reductions of **draining tunnels** (previously referred to as draining fistulas)
  - The pivotal development program will focus on patients suffering from **moderate to severe HS with active draining disease**, as supported by the FDA
  - FDA feedback will be incorporated in the pivotal study protocol and will be **submitted in Q4 2021. Study activities will begin upon approval by FDA**

At the FDA end of Phase II meeting in June 2020, FDA has agreed to the Phase III dosing regimen which is a higher dose that what was studied in the Phase IIB SHINE Study

* Serious adverse events

VILOBELIMAB (IFX-1) FOR PYODERMA GANGRAENOSUM

Pyoderma Gangraenosum (PG)
Rationale and Phase IIa Study Overview

STUDY OBJECTIVE

- **Rationale:**
  Rare ulcerative skin disease with a high unmet medical need associated with a neutrophilic leukocytosis, likely to be triggered by C5a.
  PG 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.

STUDY DESIGN

- Open label, multicenter with 3 dose groups (800mg, 1600mg or 2400mg)
- Enrollment of **19 patients reached in April 2021**
- **Primary endpoint:** Safety
- **Key secondary endpoints:** Responder rate defined as Physicians Global Assessment ≤3; Time to complete closure of target ulcer
- **Final study data expected in the first half of 2022**

TOPLINE INTERIM RESULTS

- **Four** out of 10 evaluable **patients showed clinical response** (PGA score ≤ 3), **three of whom achieved complete closure of target lesion**
  - **The other six patients all showed slight improvement in their condition according to the PGA definition**
- **Treatment was well tolerated; no adverse events associated with dose escalation**
- **Treatment of the third cohort with 2400 mg dose is ongoing**

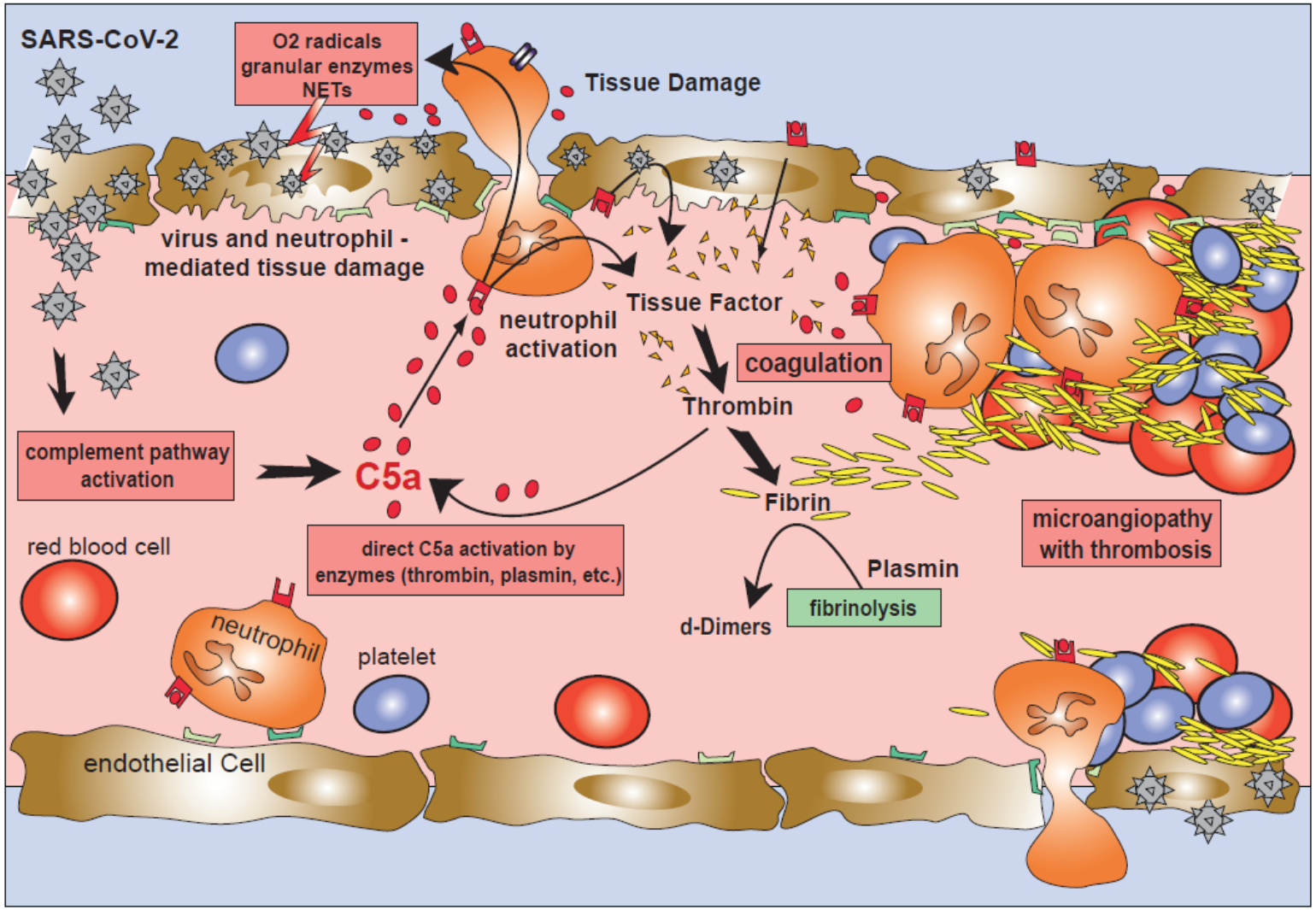
Life-threatening Inflammatory Diseases

- COVID-19
- ANCA Associated Vasculitis (AAV)

VILOBELIMAB (IFX-1) FOR SEVERE 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



- 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 viscous circle leading to microangiopathy with thrombosis

Source: InflaRx GmbH

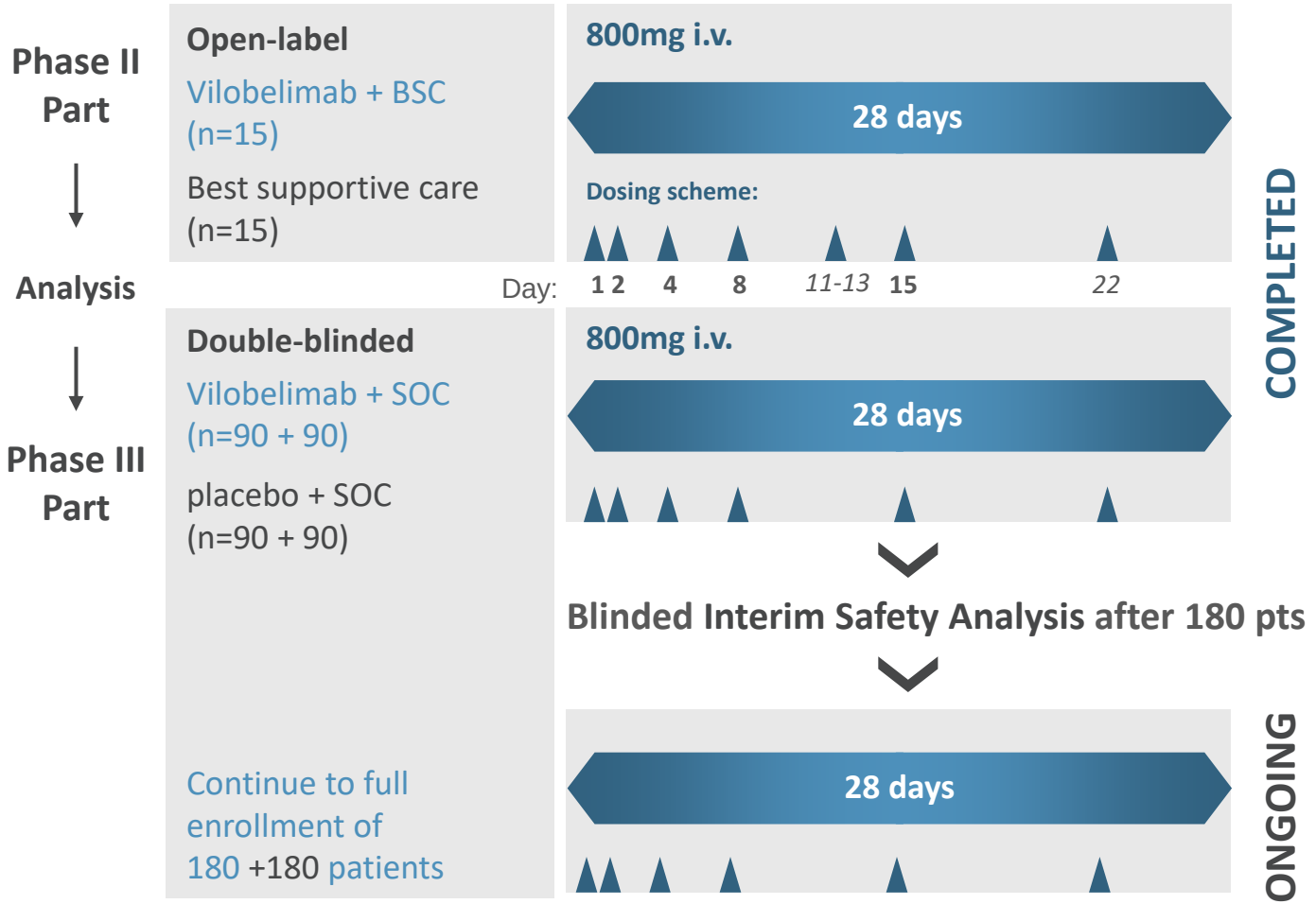
Design of Phase II/III study with Vilobelimab in Severe COVID-19

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, disease improvement on ordinal scale

STATUS

- **Phase 2 part completed:**
  Encouraging topline results published
- **Phase 3 part ongoing: *299 patients enrolled*****
  Topline data expected by end of 2021
- IDMC recommended continuing the trial at interim analysis (180 patients evaluated)



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 recommendation
* 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
** Enrollment as of Aug 5, 2021

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 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**

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

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.**

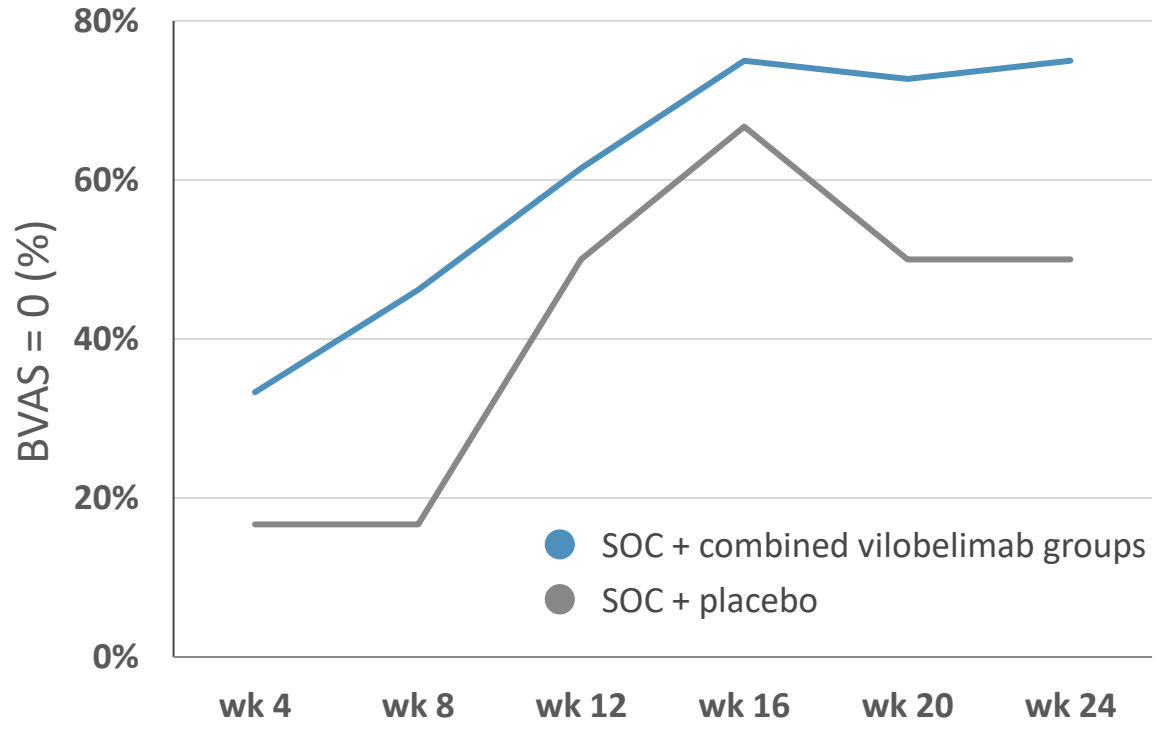
* Chemocentryx. (25 November 2019). ChemoCentryx and VFMCRP Announce Positive Topline Data from Pivotal Phase III ADVOCATE Trial Demonstrating Avacopan's Superiority Over Standard of Care in ANCA-Associated Vasculitis; (9 July 2020) ChemoCentryx Submits New Drug Application to the U.S. FDA for Avacopan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

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

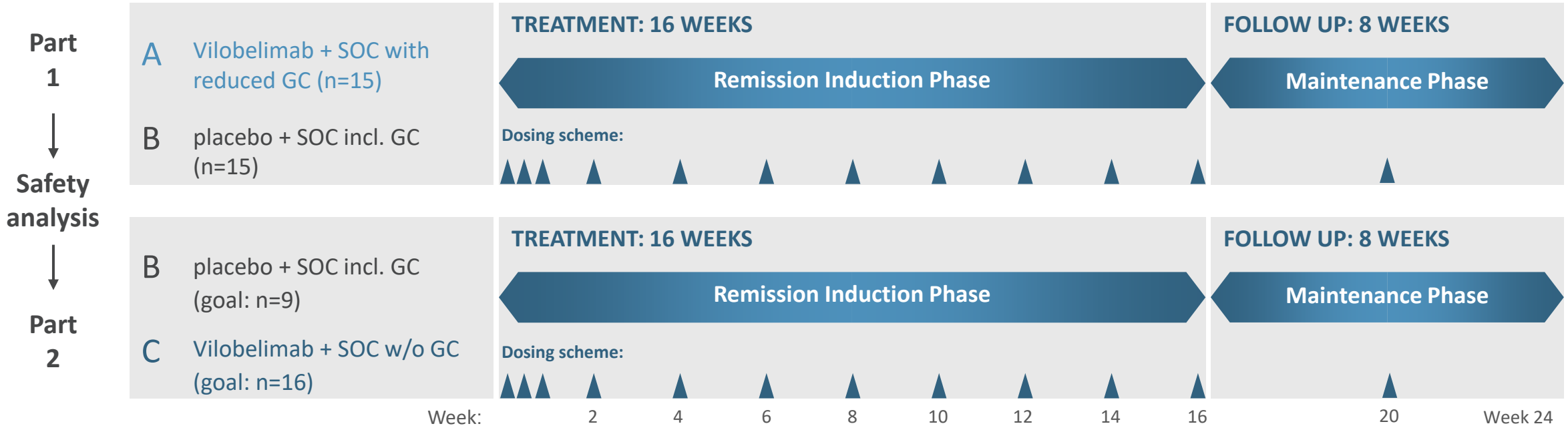
Phase II Study in AAV in the US (IXPLORE) Results

PHASE II TOPLINE RESULTS

- Three-arm, placebo-controlled trial with 16 weeks of treatment and 8 weeks follow-up (n=19)
- **Primary endpoint met: safe and well-tolerated** in patients with AAV when added to SOC;
  - Observed TEAEs are reflective of the disease and SOC treatment
- **Efficacy endpoints** (study was not powered for statistical significance)**:**
  - All three treatment groups showed **a strong clinical response** (50% reduction in BVAS) **at week 16**
  - **Clinical remission** (BVAS = 0): **higher number & percentage of patients in remission in vilobelimab groups at various timepoints** compared to SOC plus placebo



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: Part 1 - Blinded interim analysis completed. Part 2 - Enrollment completed. 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

Oncology

- Cutaneous Squamous Cell Carcinoma (cSCC)

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]
- **Estimated incidence: 15-35 per 100,000 people**; expected to increase 2-4% per year; **Metastasizes in approximately 2-5%** of cases[7,8,10]
- Advanced SCC 10-year survival rates <**20%** with regional lymph node involvement and **<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

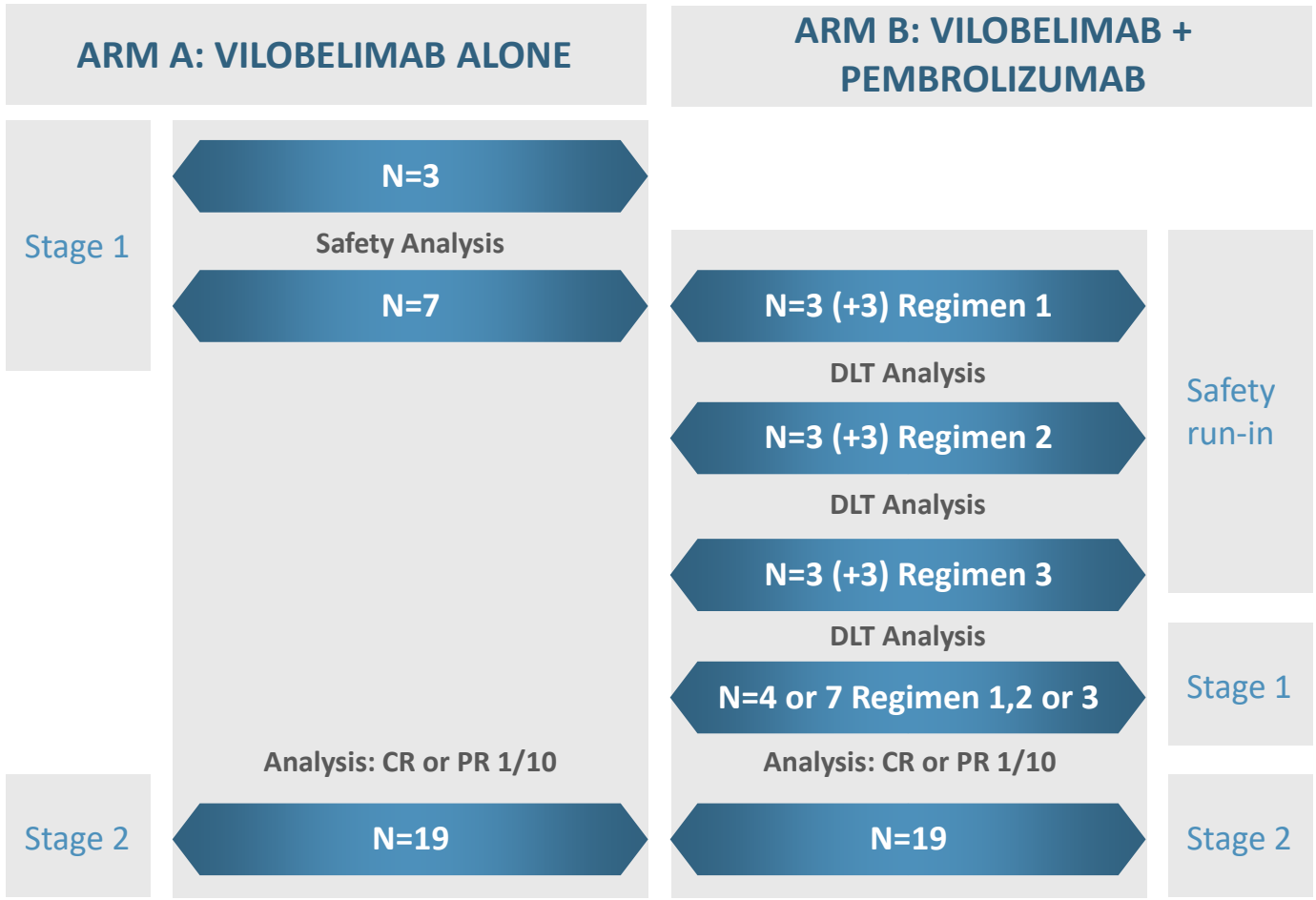
Cutaneous Squamous Cell Carcinoma (cSCC): Phase II Study Underway

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
- 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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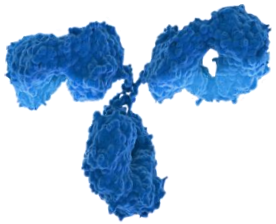
STRATEGY AND OUTLOOK

Medium Term Deliverables and Strategic Objectives



GOALS AND STRATEGY

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

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

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

Strong cash balance to pursue these activities: €127.5 million as of June 30, 2021

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