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LIFESCI CAPITAL ALPHA SERIES CONFERENCE 2020
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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 LEAD DRUG IFX-1**
- Proven anti-inflammatory effect in multiple Phase II studies
- Statistically significant reduction of inflammatory lesions in Phase IIb Hidradenitis Suppurativa study and impressive long-term efficacy
- Full data set analysis warrants continued development towards Phase III despite missing the primary endpoint (HiSCR) in Phase IIb study
- Favorable safety profile and excellent tolerability (n > 300 patients)

**MULTIPLE ONGOING STUDIES AND INDICATION + PIPELINE EXTENSION**
- Running phase II/III study in patients with COVID-19 Pneumonia in the EU
- Ongoing Phase IIb studies in ANCA-associated vasculitis in the US and EU
- Ongoing Phase IIa open label study in Pyoderma Gangraenosum in US and Canada
- Follow-on anti-C5a mAb IFX-2 in pipeline (pre-clinical stage)
- Pipeline extension of IFX-1 in other inflammatory diseases & oncology
## Pipeline with Multiple Opportunities

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<td><strong>IFX-1</strong>&lt;br&gt;<strong>C5a Inhibitor</strong></td>
<td><strong>Hidradenitis Suppurativa (HS)</strong>&lt;br&gt;• Up to 200,000 patients in the US&lt;br&gt;• Over 200,000 patients in Europe</td>
<td></td>
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<td>• Phase IIb completed&lt;br&gt;• Planning for next steps</td>
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<td><strong>ANCA-Associated Vasculitis</strong>&lt;br&gt;• ~40,000 patients in the US&lt;br&gt;• ~75,000 patients in Europe</td>
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<td>• Phase IIb enrollment ongoing in both Europe and US</td>
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<td><strong>COVID-19 Pneumonia</strong>&lt;br&gt;• Currently Unknown</td>
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<td>• Phase II/III ongoing in the EU</td>
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<td><strong>Pyoderma Gangraenosum</strong>&lt;br&gt;• ~50,000 patients in the US and Europe are affected</td>
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<td></td>
<td>• Phase IIa open label enrollment ongoing in US and Canada</td>
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<td><strong>Oncology</strong>&lt;br&gt;• Undisclosed Indication</td>
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<td>• Development of TPP ongoing</td>
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<td><strong>IFX-2</strong>&lt;br&gt;<strong>C5a Inhibitor</strong></td>
<td><strong>Undisclosed Chronic Inflammatory and Autoimmune Diseases</strong>&lt;br&gt;• Not applicable</td>
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<td></td>
<td>• Developing as injectable with optimized use for other chronic inflammatory indications</td>
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</table>
The Terminal Complement Pathway

Membrane Attack Complex (MAC) triggers lysis of pathogens

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

C5 concentration in blood: 10 ~ 30 ng/ml (~1-2.5 nM)

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

Inflammation

C5aR

❖ cell activation
❖ cytokine generation

C5a

strong amplifier of inflammation

C5aR

❖ cell activation
❖ cytokine generation

PKC-signaling
HMGB-1 induction* (Inflammasome)

C5L2

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

other ligands: C3a, ASP, C4a etc

C5b-9 = MAC

C5b

Membrane Attack Complex (MAC) triggers lysis of pathogens

C5a 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.**


Colley et al. MABS. 2018,10 (1), 104
Songlin et. al. J. Biol. Chem. 2019; 294(21) 8384–839
Muenstermann et al.. Virulence, 2020; 10(1) 677-694

upregulated in many tissues during inflammation
IFX-1 FOR COVID-19 PNEUMONIA
DISEASE BACKGROUND

- Cause: severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) which likely uses ACE2 receptors for cell entry
- First identified in Wuhan, Hubei Province, China in December 2019

CLINICAL FEATURES

- Long latency: Incubation time prior to symptoms is estimated to be between 2 and 14 days (data analysis from CDC, US)
- Symptoms: flu-like symptoms including fever or signs of lower respiratory tract illness including throat ache, dry cough and shortness of breath and HYPOXEMIA - typically no runny nose
- Slow disease onset compared to SARS and MERS
- Death is typically caused by respiratory failure in presence of sepsis and multiple organ dysfunction, similar to other viral pneumonia-induced sepsis
- Risk factors: Age, smoking, hypertension, diabetes, COPD, immunodeficiency and others

Source: cdc.gov; Zhou et al. 2020, Lancet Infectious Disease
A VIRAL PNEUMONIA WITH A BROAD SPECTRUM OF IMMUNE-MEDIATED INJURY

LABORATORY FINDINGS
• Systemic inflammation: lymphopenia (>80%) + elevated CRP (>60%) at admission
• Neutrophil counts are positively correlated with disease severity and bad outcome
• Potential Prognostic Biomarker: NLR (neutrophil-to-lymphocyte ratio) – A patient with more neutrophils and less lymphocytes has the worst outcome.
• Moderately elevated levels of both Th1 cytokines (IL-6, TNF-α, IFN-Ƴ) and TH2 cytokines (IL-4 and IL-10)
• Other frequently increased markers: D-dimer, LDH, AST, ALT, troponin-I, ESR, serum ferritin et al.

DIAGNOSIS & PATHOLOGICAL EVENTS (HEALTH COMMISSION OF CHINA COVID-19 GUIDELINE, VERSION 7)
• Severe COVID-19 definition (at lease one): (1) Shortness of breath, RR≥30 times/min, (2) Oxygen saturation (Resting state) ≤93%, or (3) PaO2 / FiO2 ≤300mmHg
• CT findings: Bilateral or unilateral pneumonia, multiple mottling and ground-glass opacity
• Pathology in lung: Infiltration of monocytes and lymphocytes; hyaline thrombi in blood vessels; focal pulmonary hemorrhage and thrombosis of small vessels, necrosis; pulmonary interstitial fibrosis
• Pathology in heart: endothelial inflammation and thrombus formation; interstitial infiltration (monocytes, lymphocytes and/or neutrophils)
• Pathology in liver: Hepatocyte degeneration and focal necrosis are accompanied by neutrophil infiltration

COVID-19: White Blood Cell and Neutrophil Counts

White blood cell counts and neutrophil counts increase in non-survivors from day 9 on

Neutrophils are strongly activated by C5a – this can damage host tissue
Viral-induced Immune Injury in COVID-19 – Potential Role of C5a

Dual Play of Viral Inflammation and Immune-mediated Injury

**Tissue**
- IFN-γ
- IP-10
- IL-6
- IL-1β
- IL-8
- IL-14
- IL-10
- TNF-α

**Resolution:**
- Lymphocyte recovery (IgG, IgM)
- Inflammation under control
- Efferocytosis in lung

**Blood vessel**

**Initiation:**
- Type I IFN Signaling
- Local inflammation
- Lymphopenia

**Deterioration:**
- Lymphocyte suppression
- Neutrophil activation – tissue inflammation
- Organ damages (e.g., lung > heart, liver, vessel)
- Coagulopathy (e.g., DIC)

**Potential C5a effects:**
- Neutrophil activation with enzyme release and O2-radical production
- Tissue damage
- Release of tissue factor from endothelial cells – induction of coagulation

**Neutrophil facts:**
- 40-60% in WBCs
- Half-life: 6-8h
- 50-100 bn cells / day
- One neutrophil has approx. 200,000 binding sites for C5a

**Initiation:**
- Viral inflammation

**Progression:**
- Neutrophil-Mediated Injury
## Overview of IFX-1 Phase II/III Study in COVID-19 Pneumonia - Study Design

### STUDY OBJECTIVE

- Assessing safety and efficacy of IFX-1 in COVID-19 Pneumonia
- **Primary endpoint:** Relative change (%) from baseline in Oxygenation Index (PaO2 / FiO2) to day 5
- **Key secondary endpoints:**
  1. Number of patients (%) achieving an **Early Response** as defined by patients who experience relief of symptoms and laboratory parameter normalization on day 7
  2. Number of patients (%) achieving a **Late Response** as defined by resolution of clinical symptoms or discharge on day 28
  3. Frequency, severity, and relatedness to study drug of treatment-emergent adverse events and serious adverse events

### STUDY DESIGN

- Adaptive, open-label, randomized, multi-center in EU
- Target enrollment – **130 patients**
- First patient dosed – **March 2020**
- Phase II/III study consisting of two parts: In both study parts, patients will be randomized to two treatment arms (Arm A: best supportive care [BSC] + IFX-1; Arm B: BSC alone).
- After all patients are treated in Phase II (approx. n = 30), an interim analysis will be performed to assess the clinical benefit of the treatment using the assessed clinical parameters in order to potentially adapt and determine the confirmatory second part of the study.
IFX-1 FOR HIDRADENITIS SUPPURATIVA
Hidradenitis Suppurativa

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
• **InflaRx established** that HS patients have **significant complement activation** with elevated C5a levels

• **C5a is involved** in several **key pathophysiological mechanisms** in HS
  – Neutrophil activation is driven by C5a
  – Various C5a dependent players potentially involved (TNFa, IL-17, etc.)

• **C5a is key neutrophil activator in HS patient plasma**
  – HS patient plasma strongly provoked neutrophil activation in healthy donor blood; this effect could be completely blocked by the addition of IFX-1
**Main Period: n = 177 treated**

- Placebo
- IFX-1 minimal dose (400 mg q4w)
- IFX-1 low dose (800 mg q4w)
- IFX-1 medium dose (800 mg q2w)
- IFX-1 high dose (1200 mg q2w)

**Open Label Extension Period (OLE): n = 156**

- Week 16 HiSCR Responders: IFX-1 low dose (800 mg q4w)
- Week 16 HiSCR Non-Responders: IFX-1 medium dose (800 mg q2w)

**Screening**

16 weeks (double blind)

**16 weeks**

**28 weeks (24 weeks treatment + 4 weeks observation)**

**TOTAL TREATMENT TIME:** 9 months (week 40) + 1 month observation (week 44)

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**MAIN GOALS**

- Test a dose-dependent effect of IFX-1 on HiSCR* response at week 16 (primary endpoint)
- Assess long-term safety of IFX-1
- Test durability of response with lower maintenance therapy in open label extension period

**Important Note:** Patients entering the OLE were not unblinded to their initial therapy

*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
Primary endpoint: HiSCR dose response signal not met but signal towards improved AN count

* Full analysis set
SHINE Study: Outcome on Draining Fistula and IHS-4* Score Reduction – Week 16

Draining fistula change (mean %)**

IHS-4 score change (mean %)***

Statistically significant change in DF and in IHS-4 scores detected

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

** Full analysis set for patients with at least 1 DF at baseline, baseline adjusted

*** Full analysis set – baseline adjusted
Comparison of HiSCR for Week 16 Responder versus Non-responder Groups (OLE) Over Time

HiSCR response rate (%) per visit* (OLE) – with 95% CI

Responders: 71% maintain HiSCR response with low dose IFX-1
Non-responders: 42% become HiSCR responders with medium dose IFX-1

* Full analysis set
Inflammatory Lesion Reductions in All OLE Patients at End of Treatment (Week 40) Compared to Placebo Group Performance in Main Period (Week 16)

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 fistula
- Evidence for a high C5a turnover rate in HS, leading to increased dose requirements of IFX-1
- IFX-1 leads to a marked reduction of all inflammatory lesions in HS with a durable long-term effect detected even at non-optimal doses
- IFX-1 long-term treatment was well tolerated, no drug related SAEs* in the open label extension phase

**NEXT STEPS**

- Requested End-of-Phase II FDA Meeting in Q1 2020
- Discuss with FDA the path forward for regulatory approval towards a Phase III pivotal program
- Depending on meeting timing and feedback: define path forward by 2H 2020

* Serious adverse events
IFX-1 FOR ANCA-ASSOCIATED VASCULITIS
ANCA-Associated Vasculitis (AAV)

A life-threatening autoimmune condition

**CLINICAL FEATURES**
- Rare, life-threatening autoimmune disease, characterized by necrotizing vasculitis
- Life-threatening flare phases affect organs, leading to potentially fatal organ dysfunction and failure
- Predominantly affecting small vessels associated with anti-neutrophil cytoplasmic antibodies, or ANCA
- Disease activity is assessed using Birmingham Vasculitis Activity Score v3 (BVAS)

**PREVALENCE**
- Approx. 40,000 AAV patients in the US
- Approx. 75,000 AAV patients in Europe
- Orphan drug market

**CURRENT TREATMENT – MEDICAL NEED**
- Induction of remission critical during flare phases – induction treatment differs from maintenance therapy and consists of high dose corticosteroids plus either cyclophosphamide or rituximab
- Induction of remission therapy has significant side effects

Source: Chen, Jayne and Zhao. Complement in ANCA-associated vasculitis: mechanism and implication for management
IFX-1 in AAV
Clinical PoC established for Role of C5a / C5aR Pathway in AAV

RATIONALE

• 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 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
** Hao & Wang et al 2013, PLoS ONE, 8(6)
IFX-1 – P2.6 Phase II Study in AAV in the US (IXPLORE)
Study Design

<table>
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<th>GROUPS:</th>
<th>TREATMENT: 16 WEEKS</th>
<th>FOLLOW UP: 8 WEEKS</th>
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<tr>
<td>A</td>
<td>IFX-1 low dose + SOC (n=12)</td>
<td>Remission Induction Phase</td>
</tr>
<tr>
<td>B</td>
<td>IFX-1 high dose + SOC (n=12)</td>
<td>Maintenance Phase</td>
</tr>
<tr>
<td>C</td>
<td>Placebo + SOC (n=12)</td>
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</tbody>
</table>

Randomization 1:1:1

TREATMENT: 16 WEEKS
SOC = Rituximab or Cyclophosphamide + glucocorticoids

**Study objective (target: n=36)**

- Assessing safety and efficacy of IFX-1 in AAV – **First Patient Dosed in October 2018**
- **Primary endpoint:** Safety
- **Secondary endpoint:** Response rate based on the Birmingham Vasculitis Score (BVAS), various other secondary endpoints

* Guideline maintenance allows for: azathioprin / methotrexate / mycophenolate mofetil / low dose corticosteroids
IFX-1 – P2.5 Phase II study in AAV in Europe (IXCHANGE)

Study Design

Study objective (target: n=81)

• First patient dosed: May 2019
• Primary objective: Efficacy of IFX-1 treatment as replacement for glucocorticoid (GC) therapy in GPA and MPA
• Secondary objectives: To assess safety and tolerability of IFX-1 & compare toxicity of standard-dose GC with IFX-1

* Guideline maintenance allows for: azathioprin / methotrexate / mycophenolate mofetil / low dose corticosteroids
IFX-1 FOR PYODERMA GANGRAENOSUM
Pyoderma Gangraenosum (PG)

Source: Demis.net

• PG is a rare ulcerative skin disorder that can lead to chronic painful and difficult-to-treat wounds / ulcers occurring predominantly in people in their 40s and 50s
• Many PG patients also suffer from other autoimmune disorders, including inflammatory bowel diseases like ulcerative colitis, rheumatoid arthritis, and hematological diseases
• Patients suffer from severe pain, long healing times, and frequent relapses
• Diagnosis is based on the exclusion of other conditions and typical ulcers

CLINICAL FEATURES

INCIDENCE
• Rare - Estimated that up to 50,000 patients in the US and Europe are affected
• Orphan drug market

CURRENT TREATMENT – MEDICAL NEED
• No drugs currently approved in the US or EU
• Current treatment options include the use of systemic immunosuppression in rapidly progressing cases or, for less severe cases, topical or intralesional treatments can be used, including topical steroids
## Overview of IFX-1 Phase IIa Study in PG - Study Design

### STUDY OBJECTIVE

- Assessing safety and efficacy of IFX-1 in PG
- **Rationale:**
  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
- Multi-center in US and Canada
- Target enrollment – **18 patients**
- First patient dosed – June 2019

### TREATMENTS

- Trial started with 1 dose group: amendment approved to introduce a dose escalation to test 3 dose groups
- Subjects receive IFX-1 dosing every other week
- Dose: 800 mg biweekly first 5 patients
• 5 patients have been treated

• 2 out of the first 5 patients showed complete closure of the target ulcer with both patients in full disease remission. Both remained healed even after finishing the study

• In one additional patient, initial wound healing activity was detected in the first 2-3 weeks of treatment – but no wound size decrease or closure detected

• 2 additional patients with extensive disease (very large ulceration / ulcer reaching the entire circumference of the leg) did not heal the target ulcer and are still under treatment

• The “responders” have higher baseline C5a levels

• PD analysis (C5a levels) warrants higher dosing

• Dose escalation recently approved by relevant authorities
Female patient with extensive genital PG disease and target ulcer on lower extremity (no concomitant IBD)

- Various failed treatment attempts including high dose corticosteroids, etc.
- Significantly elevated baseline C5a levels
- Patient completely healed of all PG lesions at the end of treatment and remained healed after the end of the study

PATIENT EXAMPLE 2

- Male patient with treatment resistant PG disease and Addison’s disease from high-dose GC (no concomitant IBD)
- Significantly elevated baseline C5a levels
- Patient completely healed of all PG lesions at the end of treatment and remained healed after the end of the study
Our Strategy

GOALS AND STRATEGY

• **Advance** our lead program IFX-1 for HS towards Phase III / approval based on regulatory guidance

• Explore application of IFX-1 for AAV, COVID-19, PG and oncology in clinical development

• Extend pipeline with initiation of clinical development of IFX-1 in other complement-mediated autoimmune / inflammatory diseases

• Pursue the **development of IFX-2** 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 (End of Q3 2019: ~US$151 million)