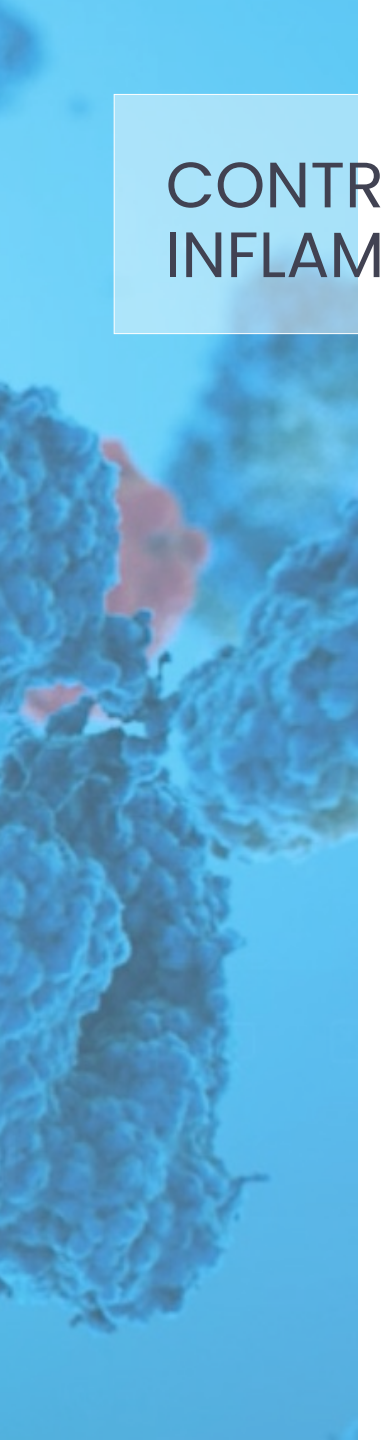
inflaRx

CORPORATE PRESENTATION

APRIL 2026



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inflaRx

Important Notice and Disclaimer

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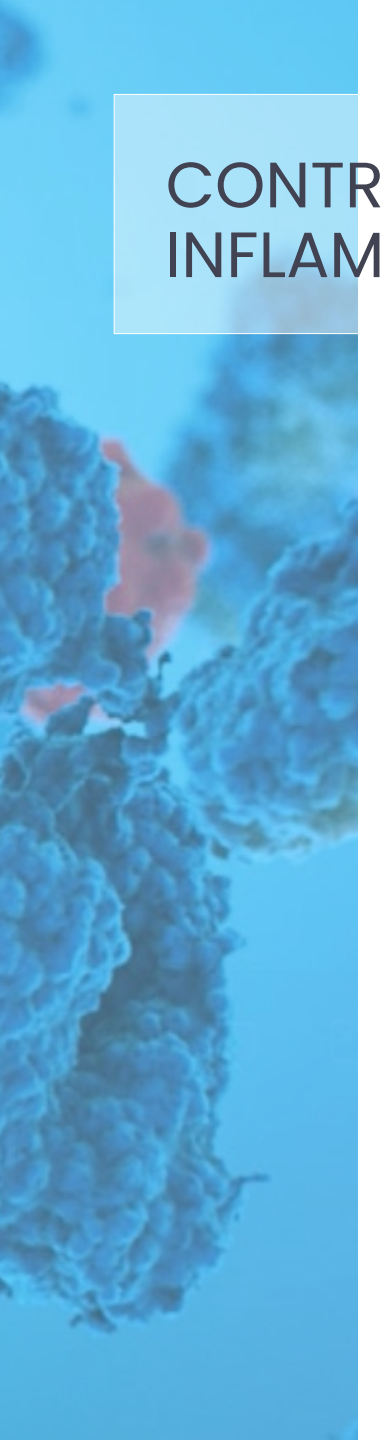
Forward-Looking Statements

This presentation contains forward-looking statements. All statements other than statements of historical fact are forward-looking statements, which are often indicated by terms such as "may," "will," "should," "expect," "plan," "anticipate," "could," "intend," "target," "project," "estimate," "believe," "predict," "potential" or "continue," among others. Forward-looking statements appear in a number of places throughout this presentation and may include statements regarding our intentions, beliefs, projections, outlook, analyses and current expectations concerning, among other things: the receptiveness of izicopan as a treatment for hidradenitis suppurativa (HS) and chronic spontaneous urticaria (CSU) patients and U.S. hospitals and related treatment recommendations by medical/healthcare institutes and other third-party organizations; our ability to successfully secure distribution channels and commercialize GOHIBIC (vilobelimab) as a treatment for COVID-19 patients and our ability to positively influence treatment recommendations by U.S. and European hospitals, guideline bodies and other third-party organizations; our expectations regarding the size of the patient populations for, market opportunity for, coverage and reimbursement for, estimated returns and return accruals for, and clinical utility of GOHIBIC (vilobelimab) in its approved or authorized indication or for vilobelimab and any other product candidates, under the Emergency Use Authorization (EUA) and in the future if approved for commercial use in the United States, Europe or elsewhere; our ability to successfully implement The InflaRx Commitment Program, the success of our future clinical trials for vilobelimab's treatment of debilitating or life-threatening inflammatory indications, including acute respiratory distress syndrome (ARDS) and other indications, and any other product candidates, including izicopan, and whether such clinical results will reflect results seen in previously conducted pre-clinical studies and clinical trials; the timing, progress and results of preclinical studies and clinical trials of vilobelimab, izicopan and any other product candidates, including for the development of vilobelimab in several indications, including to obtain full approval of GOHIBIC (vilobelimab) for COVID-19 and other virally induced ARDS, to treat HS and CSU, and statements regarding the timing of initiation and completion of studies or trials and related preparatory work, the period during which the results of the trials will become available, the costs of such trials and our research and development programs generally; our interactions with and the receptiveness and approval by regulators regarding the results of clinical trials and potential regulatory approval or authorization pathways, including our biologics license application submission for GOHIBIC (vilobelimab); the timing and outcome of any discussions or submission of filings for regulatory approval or authorization of vilobelimab, izicopan or any other product candidate, and the timing of and our ability to obtain and maintain full regulatory approval, the EUA and/or market authorization of vilobelimab or GOHIBIC (vilobelimab) for any indication; our ability to leverage our proprietary anti-C5a and anti-C5aR technologies to discover and develop therapies to treat complement-mediated autoimmune and inflammatory diseases; our ability to protect, maintain and enforce our intellectual property protection for vilobelimab, izicopan and any other product candidates, and the scope of such protection; whether the U.S. Food and Drug Administration (FDA) the European Medicines Agency (EMA) or any comparable foreign regulatory authority will accept or agree with the number, design, size, conduct or implementation of our clinical trials, including any proposed primary or secondary endpoints for such trials; the success of our future clinical trials for vilobelimab, izicopan and any other product candidates and whether such clinical results will reflect results seen in previously conducted preclinical studies and clinical trials; our expectations regarding the size of the patient populations for, the market opportunity for, the medical need for and clinical utility of vilobelimab, izicopan or any other product candidates, if approved or authorized for commercial use; our manufacturing capabilities and strategy, including the scalability and cost of our manufacturing methods and processes and the optimization of our manufacturing methods and processes, and our ability to continue to rely on our existing third-party manufacturers and our ability to engage additional third-party manufacturers for our planned future clinical trials and for commercial supply of vilobelimab and for the finished product GOHIBIC (vilobelimab) in the United States and Europe; our estimates of our expenses, ongoing losses, future revenue, capital requirements and our needs for or ability to obtain additional financing; our expectations regarding the scope of any approved indication for vilobelimab; our ability to defend against liability claims resulting from the testing of our product candidates in the clinic or, if, approved or authorized, any commercial sales; if any of our product candidates obtain regulatory approval or authorization, our ability to comply with and satisfy ongoing drug regulatory obligations and continued regulatory overview; our ability to comply with enacted and future legislation in seeking marketing approval or authorization and commercialization; our future growth and ability to compete, which depends on our retaining key personnel and recruiting additional qualified personnel; our competitive position and the development of and projections relating to our competitors in the development of C5a and C5aR inhibitors and other therapeutic products being developed in similar medical conditions in which vilobelimab, izicopan or any other of our product candidates is being developed or our industry; and the risks, uncertainties and other factors described under the heading "Risk Factors" in our periodic filings with the U.S. Securities and Exchange Commission. These statements speak only as of the date of this presentation and involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. Given these risks, uncertainties and other factors, you should not place undue reliance on these forward-looking statements, and we assume no obligation to update these forward-looking statements, even if new information becomes available in the future, except as required by law.



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InflaRx data presented

The data presented herein are topline results based on the number of patients indicated and are subject to final data review and quality checks. Data from patients still completing treatment are not included in the current analysis and may result in minor changes to the reported results.

For HS, two patients (one in the 60mg bid dosing group and one in the 90mg bid dosing group) are still completing treatment and are excluded from the data presented. For CSU, one patient in the 120mg bid dosing group is still under treatment and is excluded from the data presented. Data entry and verification for these patients is ongoing at the trial sites. While we do not expect the pending data from such patients to materially change the overall efficacy trends, particularly as the most pronounced efficacy in HS was observed in the 120mg bid dosing group, which is unaffected, minor changes may occur.

Final changes and corrections may occur upon full data review and quality checks, but we do not believe any such changes or corrections will have a material impact on the reported efficacy or safety trends. All data should be considered preliminary until the full dataset is available and final analyses are complete. We are committed to providing an update as soon as the remaining data are incorporated.

Information and sources

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

Third-Party Data

We have not conducted head-to-head clinical trial comparisons between izicopan and any third-party drug candidate or approved drug. Any third-party data displayed or referenced are intended solely for comparative orientation and are based on published data from various sources, including original publications, press releases, abstracts, posters, approval reviews and others. Except for a separate comparison to reported data from the use of avacopan in HS, all comparisons are focused on available data from drug candidates that are approved, have successfully completed Phase 3 development or have initiated Phase 3 development based on completed Phase 2 studies and have not been otherwise discontinued. These comparisons are not derived from head-to-head trials and the data displayed are from studies conducted under different protocols, with different inclusion and exclusion criteria, at different sites and at different times, among other differences. As such, the value of any such comparison may be limited, and we are unable to make comparative claims between izicopan and third-party drug candidates or approved drugs. We make no representation regarding the completeness of such comparative data and reference the sources of our comparisons where applicable.

About InflaRx

InflaRx (Nasdaq: IFRX) is a biopharmaceutical company pioneering anti-inflammatory therapeutics by applying its proprietary anti-C5a and anti-C5aR technologies to discover, develop and commercialize highly potent and specific inhibitors of the complement activation factor C5a and its receptor, C5aR. C5a is a powerful inflammatory mediator involved in the progression of a wide variety of inflammatory diseases. InflaRx's lead program is izicopan (INF904), an orally administered small molecule inhibitor of C5a-induced signaling via the C5a receptor, which has shown promising PK/PD characteristics as well as therapeutic potential in Phase 1 and Phase 2a clinical studies. The company is developing izicopan for the treatment of several inflammatory diseases, including hidradenitis suppurativa (HS). The Company has also developed vilobelimab, a novel, intravenously delivered, first-in-class, anti-C5a monoclonal antibody that selectively binds to free C5a and has demonstrated disease-modifying clinical activity and tolerability in multiple clinical studies.

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

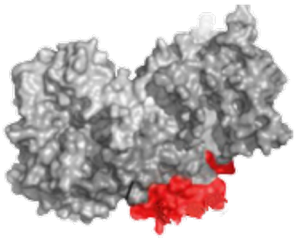
Harnessing C5a/C5aR with izicopan for controlling inflammation in I&I

- Lead asset **izicopan is an oral inhibitor of C5a/C5aR**, a validated target and critical part of the inflammatory cascade in disease areas in need of new mechanisms of action
- Phase 2a indicate izicopan has potential for **biologic-like efficacy in HS** and can improve clinical measures in CSU
- Izicopan also addresses large markets across immuno-dermatology and broader I&I, with the potential to be a **transformative anti-inflammatory pipeline-in-a-product**
- By improving upon the limitations of the marketed comparator, with clearly differentiated PK profile and inhibitory effects, **izicopan has best-in-class potential**
- A **newly streamlined corporate strategy** to focus on izicopan in HS and additional I&I areas, while continuing to **foster active dialog with potential collaborators** to expedite development across all I&I
- Strong balance sheet with **enough cash to fund operations to mid-2027** and advance toward next milestones
- **Strong IP position** and a team with proven track record of delivering clinical and regulatory successes

CSU [chronic spontaneous urticaria]; **HS** [hidradenitis suppurativa]; **PG** [pyoderma gangrenosum]; **I&I** [inflammation and immunology].

C5a/C5aR are validated targets promoting inflammation

C5



Cleavage of C5a

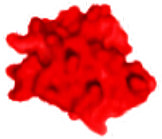
through complement pathway or enzymatic

Targeting strong pro-inflammatory mechanisms

vilobelimab
intravenous mAb

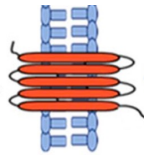
izicopan
oral small molecule

C5a



strong amplifier of inflammation

C5aR



expressed on many immune cells and upregulated in many tissues under disease conditions

Anaphylatoxin C5a is upstream of the cytokine network

- Induces histamine release – can lead to anaphylactic reactions
- Boosting effect on various pro-inflammatory cytokines from various immune cells (IL-17, IL-6, IL-8, IL-1 and others)
- Drives TH1 and TH17 T-cell differentiation

Strong activator of neutrophils and macrophages

- Chemotaxis of neutrophils / macrophages / monocytes into tissue
- O_2 radical generation + granular enzyme release from neutrophils
- NETosis induction (neutrophil extracellular traps)

Essential role in many inflammatory conditions

- Acute and chronic inflammation and other conditions
- Over 6,000 publications on role in numerous diseases

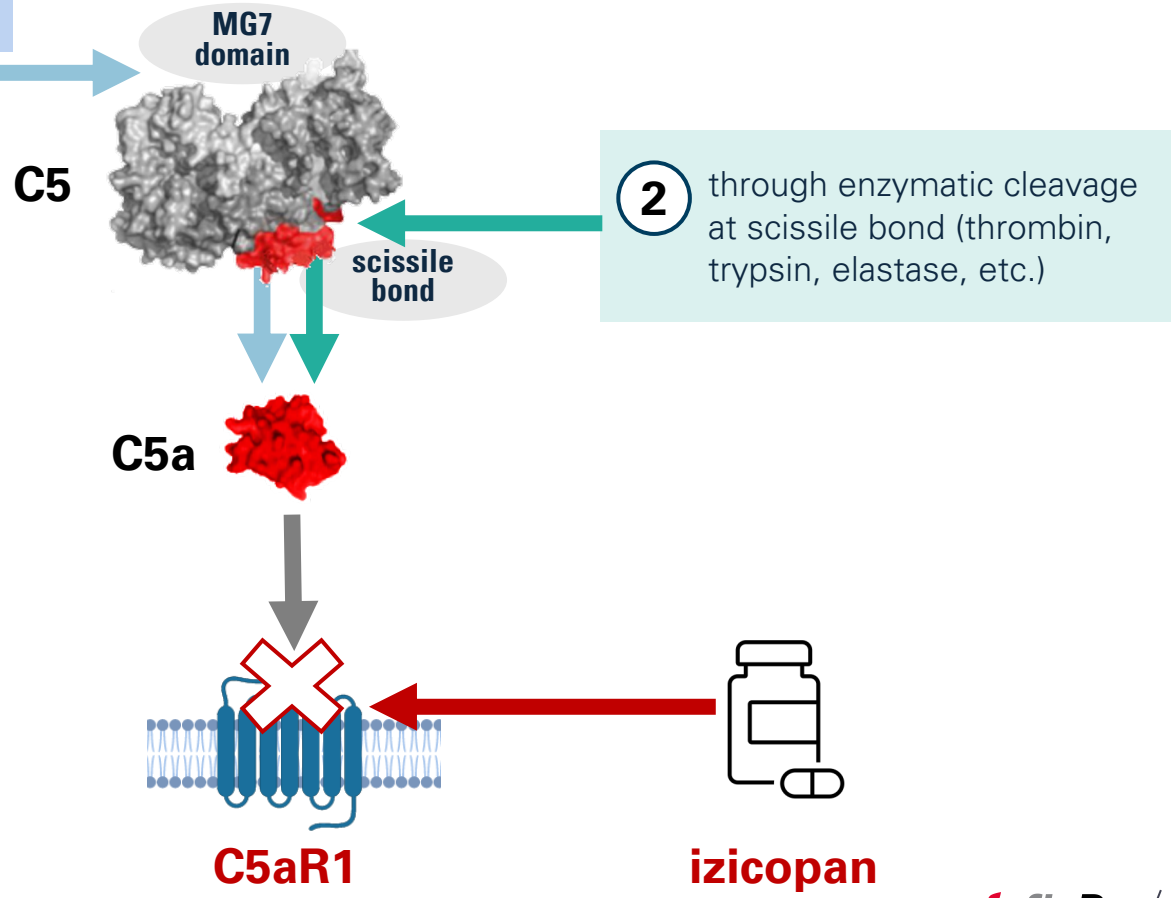
C5a/C5aR signaling inhibition: Izicopan prevents binding of C5a to C5aR

Complement Pathways		
Classical	Lectin	Alternative
Antibody binding	Mannose binding	Foreign surfaces

C3 activation

C5-convertases

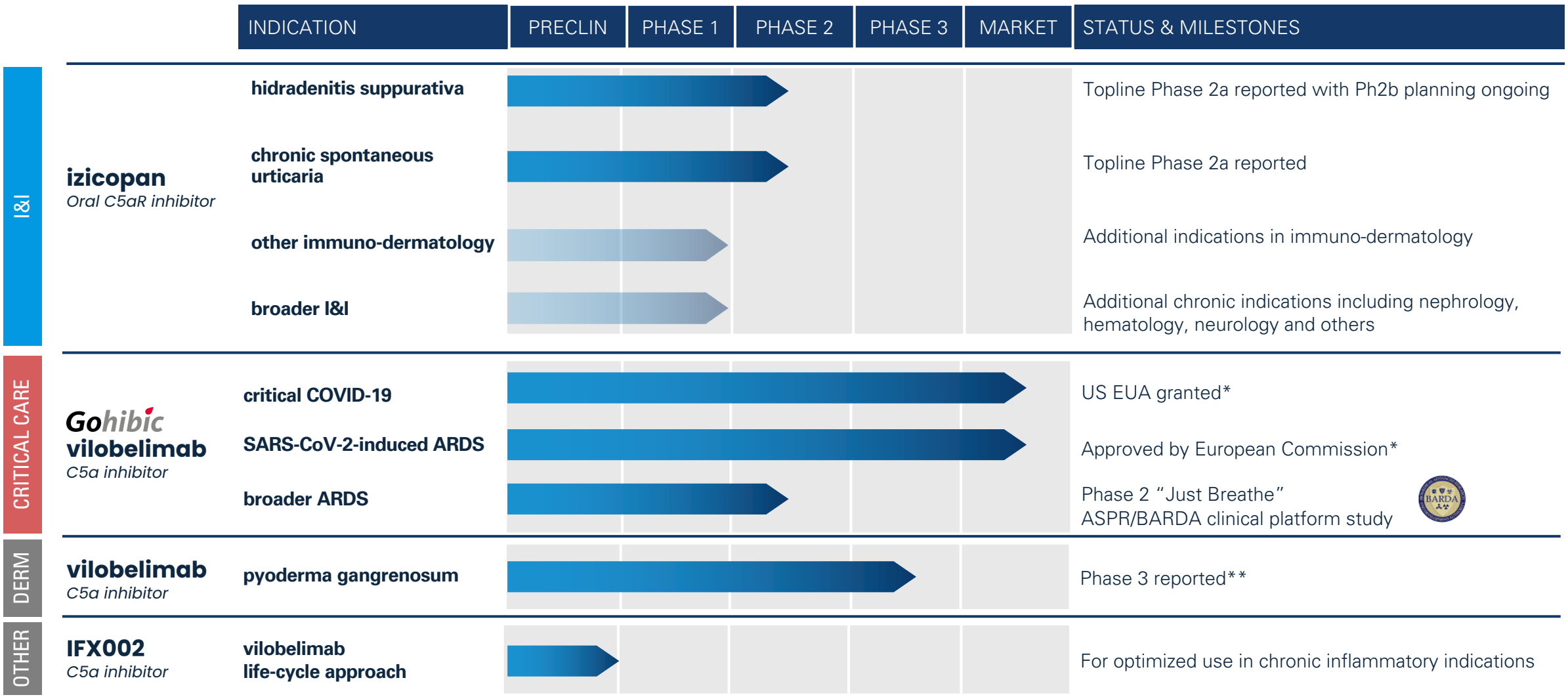
1 through C5 convertases (complement mediated)



Advantages of blocking C5aR with izicopan

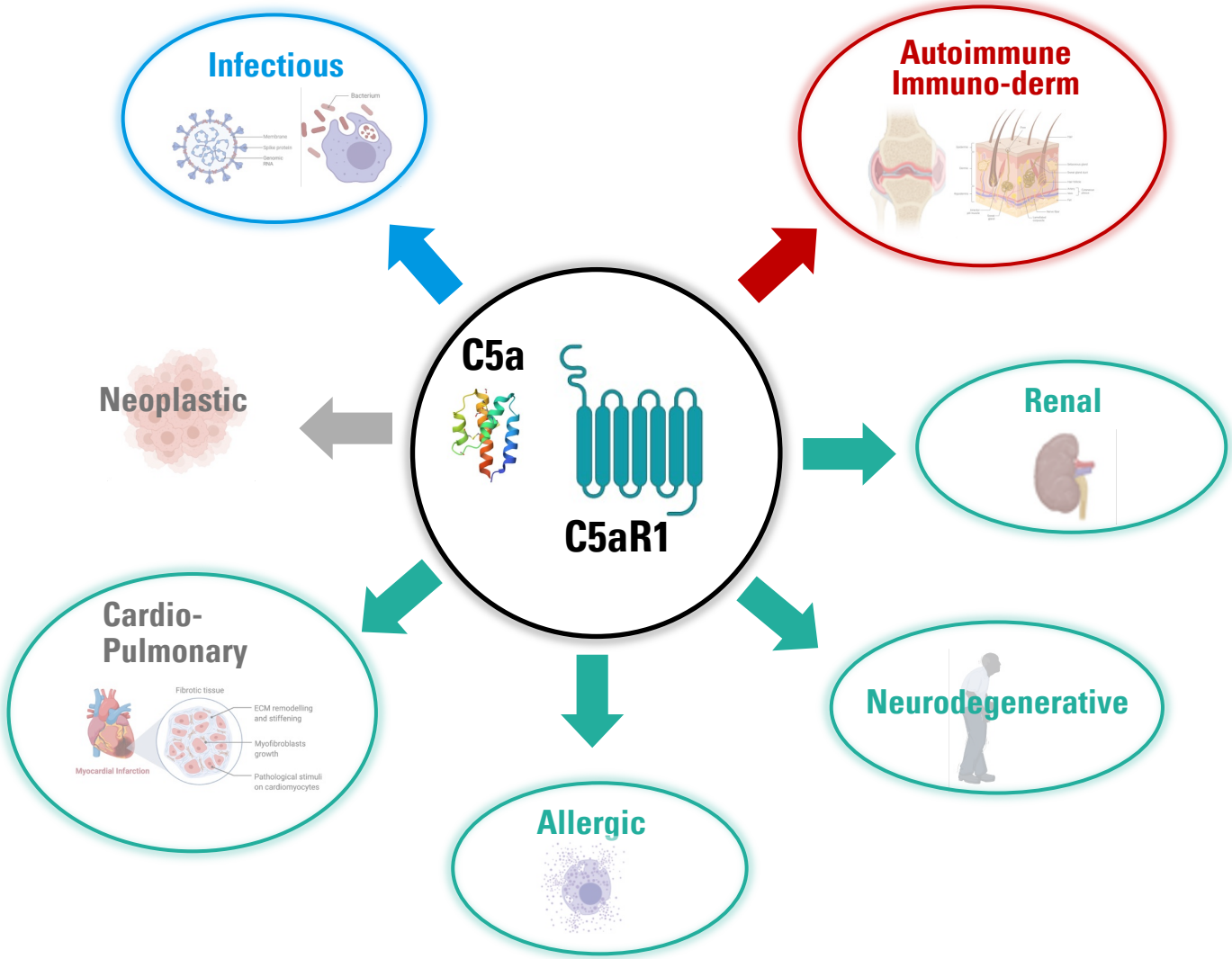
- Blocking C5 or further upstream (e.g., with eculizumab and related antibodies) does not adequately inhibit C5a/C5aR signaling
- A small molecule oral compound should have better tissue penetration and better ability to control C5a/C5aR signaling at the site of inflammation
- C5aR is selectively expressed on various immune and tissue cells, and blocking C5aR will not be impacted by increasing C5a generation

Izicopan-led pipeline targets multiple sizable markets



*Commercial partnering and distribution options in the US and EU being considered.
** Phase 3 stopped early for futility at interim analysis, FDA interaction considered.

C5a & C5aR inhibition has potential medical utility in broader I&I



Initial focus on HS and immuno-dermatology

Additional development expansion opportunity in various chronic inflammatory disease areas, including ANCA-associated vasculitis

Vilobelimab with proven life-saving anti-inflammatory effect in critically ill COVID-19 patients

Izicopan: An oral C5aR antagonist with best-in-class potential

Superior PK/PD profile in Phase 1 and Phase 2 studies compared to reported data from marketed comparator avacopan (graphs at right*/**)

- **~10-fold higher AUC_{last} and ~3-fold higher C_{max}** (Phase 1 SAD)
- **Significantly increased blocking activity** >90% blocking of C5a activity
- **Faster achievement** of therapeutic exposure

Safety profile

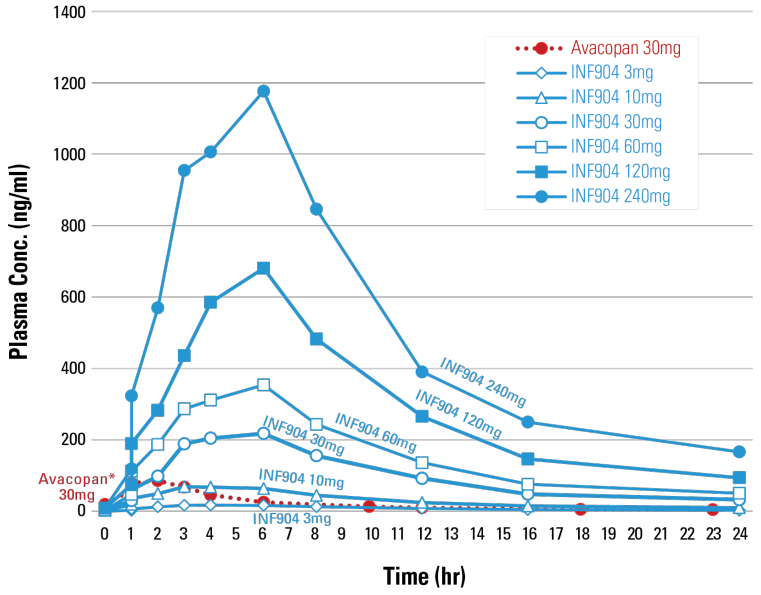
- **No signals of safety concern** (>180 humans exposed, completed GLP tox studies)
- **No time dependent CYP3A4 inhibition** (IC_{50} > 100 µM) as measured via Ki-based TDI study

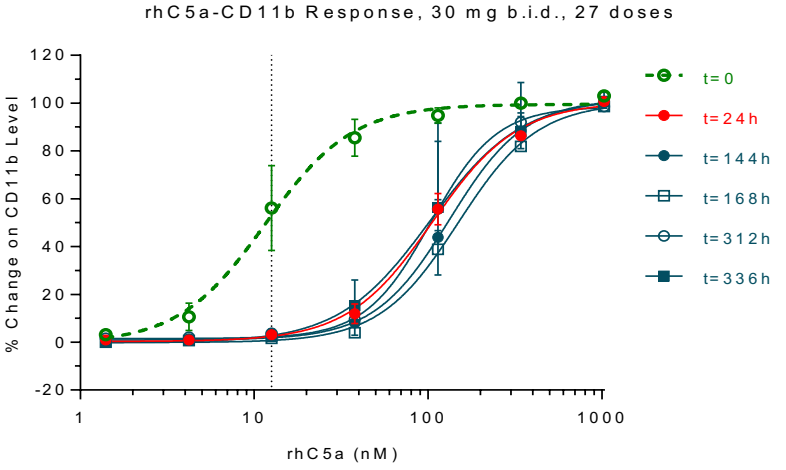
Dosing

- 30mg per capsule (vs 10mg for avacopan) **and potential for once-daily dosing**

Izicopan potential advantages

- Faster onset of action + higher target coverage + differentiated efficacy
- Cleaner safety profile
- More convenient dosing





*InflaRx data on file: PK Results From Single Ascending Dose (SAD) Phase 1 study – note: Avacopan data (Becker et al, 2016, PLoS One) are superimposed in graph for orientation. Avacopan was not included as a comparator in INF904 Phase I study.

**InflaRx data on file: PD Results from multiple ascending dose (MAD) Phase 1 study.

Izicopan's improved chemistry, metabolic & safety features

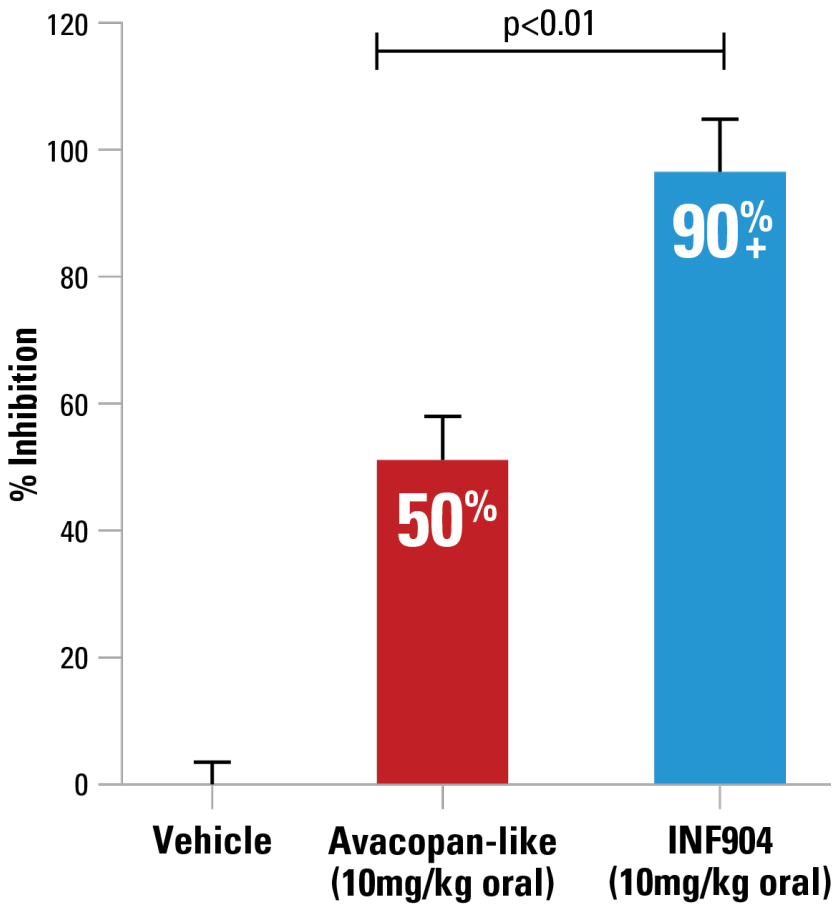
Feature	Avacopan	Izicopan	Key differentiation for izicopan
Medicinal chemistry	Used as a lead compound series for the development of izicopan	Further structure-activity optimization performed	Izicopan **has improved PK/PD profiling in humans and improved safety features** (e.g. metabolic stability and CYP inhibition), as listed below
Chirality	2 chiral centers	3 chiral centers	The extra stereocenter in izicopan **provides additional conformational control, significantly improving physiochemical properties and druggability**
Solid-state form	Crystalline	Amorphous	The amorphous form of izicopan supports **faster absorption and quicker attainment of therapeutic exposure** Izicopan therapeutic exposure reached in days to one week, vs 13 weeks for avacopan
CYP inhibition	Time dependent inhibitor of CYP3A4	Ki/Kinact assay demonstrates: No inhibition of CYP3A4 (IC50 >100 µM) – no time dependent inhibition (TDI)	**Avacopan CYP3A4 inhibition raises risk of drug–drug interactions** and may slow clearance of co-medications, **potentially contributing to liver toxicity**. This is **not a concern with izicopan**
Metabolic stability	Lower stability and high CYP-mediated turnover in HLM and HH: • **HLM:** short $t_{½}$ (≈30–70 min), high CL_{int} (~18–48 µL/min/mg) • **HH:** $t_{½}$ ≈ 140–276 min; CL_{int} ≈ 11–4.9 mL/min/kg Significant CYP-mediated bioactivation in HLM and in humans	Very high stability and very low CYP-mediated turnover in HLM and HH: • **HLM:** % remaining at 60 min >80%, long $t_{½}$ (≈240–370 min), very low CL_{int} (~0.6–3.7 µL/min/mg). • **HH:** $t_{½}$ ≈ 678–1675 min; CL_{int} ≈ 4.2–1.0 mL/min/kg. Significantly less CYP-mediated bioactivation	**Izicopan demonstrates high metabolic stability; 5–20× lower intrinsic clearance** vs avacopan Izicopan exhibits very low CYP-mediated metabolism, with very low microsomal clearance and long half-lives in both microsomes and hepatocytes, **suggesting a substantially lower risk in formation of reactive metabolites**

HLM [human liver microsomes]. **HH** [human hepatocytes]. **CL_{int}** = rate of intrinsic liver clearance. **$t_{1/2}$** = half-life.

Izicopan: Oral C5aR antagonist with best-in-class potential

Double the inhibitory effect in vivo in a pre-clinical model compared to avacopan

Inhibition of in vivo neutrophil activation by izicopan compared to avacopan-like molecule*



Plasma concentration sampled at **8 hours**:

Izicopan = **538 ng/mL**

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

APPROXIMATELY **2X** **izicopan doubled the in vivo inhibitory effect** at comparable dose when tested head-to-head with avacopan.

The **strongly improved PK features of izicopan** (plasma exposure) may drive the ability to increase efficacy in vivo.

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

Note: Izicopan dosing within this experiment exerts an approximately 4.5-fold higher plasma level 8 h after dosing when compared to the identical dosing with avacopan*.

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

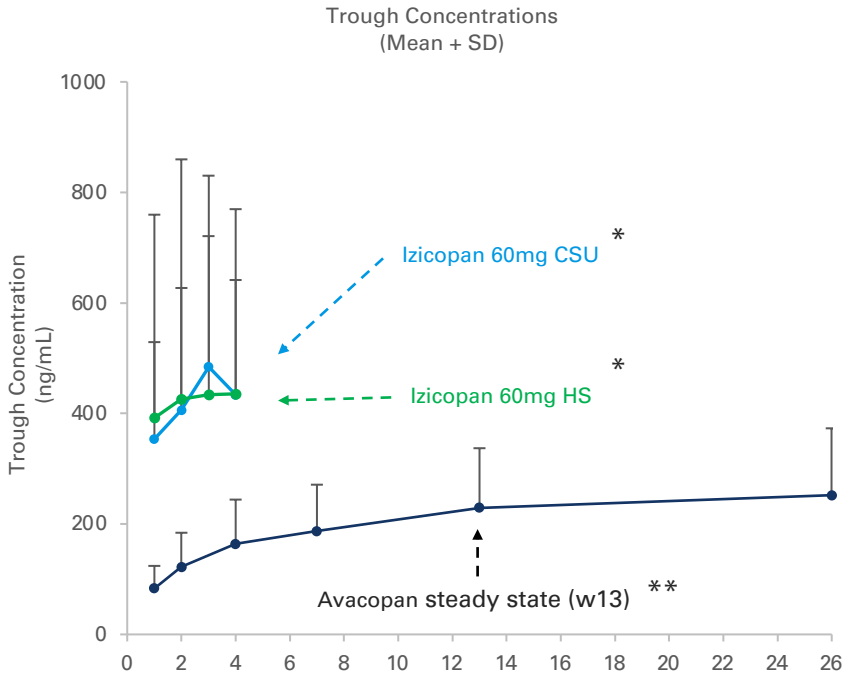
Izicopan therapeutic exposure reached within the first week in Phase 2a

Initial considerations

- Lower dose of 60 mg bid projected to result in **complete signal control based on Phase 1 data**
- **Explore efficacy at the high end** by adding 90 mg bid (HS) and 120 mg bid (HS and CSU)

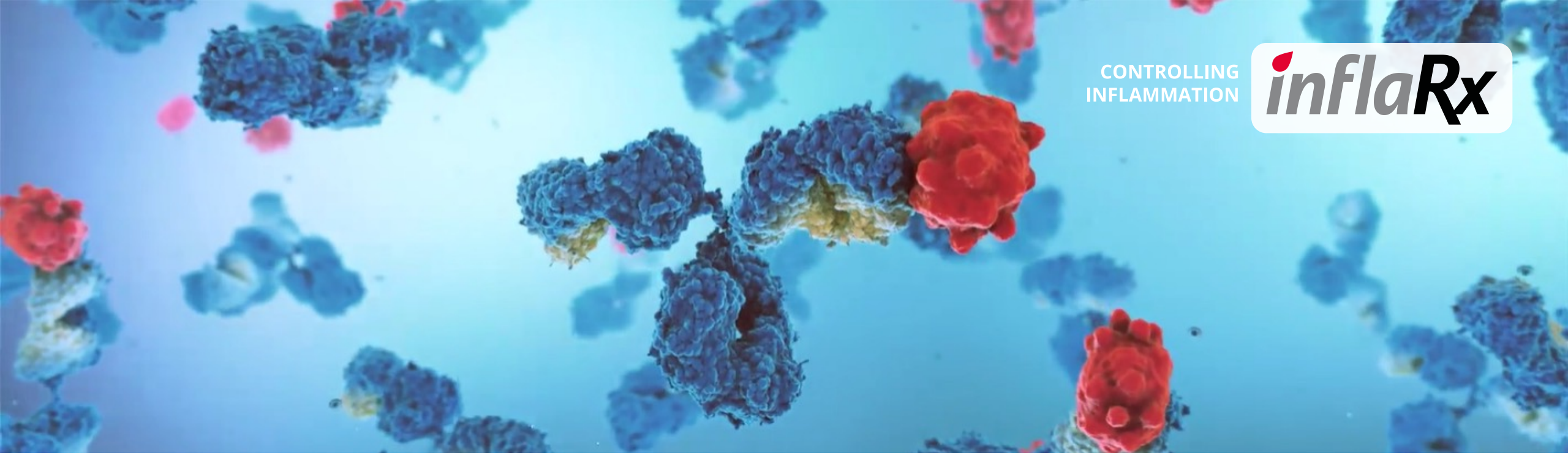
Preliminary PK results*

- **High plasma exposure can be reached approximately within the first week** (all doses tested), compared to 13 weeks for reported avacopan PK data



* Preliminary PK results of pooled patients: 10 - 11 HS patients (60mg bid) and 10 -11 CSU patients (60mg bid). InflaRx data on file final and full QC checked results will be reported after completion of the study.

** Steady state plasma levels of avacopan (NDA filing for ANCA-associated vasculitis, 75-100 patients) are reached by 13 weeks and the accumulation is approximately 4-fold.



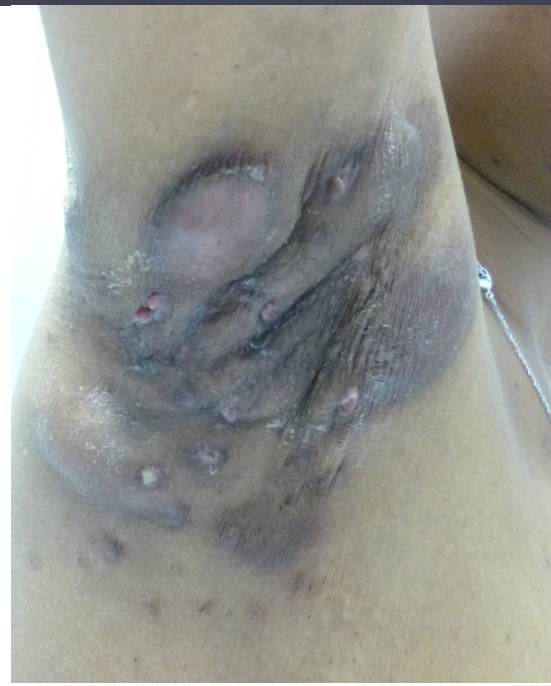
Izicopan for Hidradenitis Suppurativa

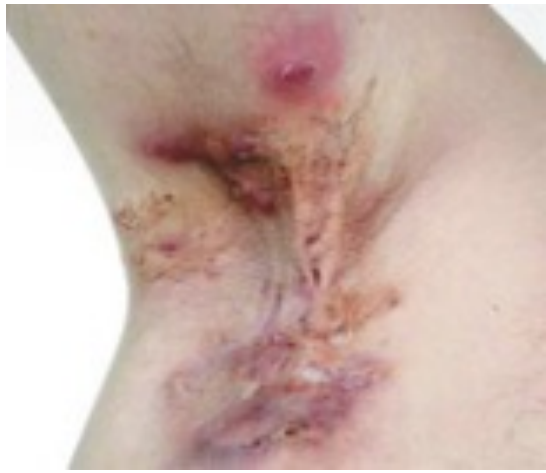
- All Ph2a data presented herein for end of treatment (EOT) at week 4 show results for n=29 evaluable patients
- All Ph2a data presented herein for end of study (EOS) at week 8 (after 4-week follow-up period) show results for n=25 evaluable patients
- Ph2a data from two additional patients (one in the 60mg bid dosing group and one in the 90mg bid dosing group) still completing treatment are excluded from the current analysis and data presented

** Ph2a data are topline and subject to final data review and quality checks. Results may change as additional patient data are incorporated.*

A strong rationale for developing izicopan in HS

- HS is a **chronic, recurring, debilitating neutrophil-driven inflammatory disease**
  - Prevalence in the US and EU is estimated to be 0.7% - 1.2%
  - We estimate there are clearly more than 200,000 moderate to severe HS patients in the US alone
- **New mechanisms are needed** to address the disease more completely
  - **Moderate to severe patients with active draining disease** have limited approved treatment options proven to be effective
  - Response to treatment with approved **anti-TNF-alpha or anti-IL17 agents is known to wane over time** in a significant number of cases
- HS patients have a preference for oral medications over injections (and surgical incisions)*
- Izicopan is an **oral C5aR inhibitor** with a **new mechanism**
  - Inhibits the known C5a/C5aR induced effects on neutrophil activation and tissue accumulation of immune cells including induction of NETosis – mechanisms implicated in HS progression and specifically in HS lesion formation (particularly dT´s and abscesses)
  - Clinical evidence existing that blocking the C5a/C5aR pathway reduces lesion counts in HS
- Market potential for izicopan in HS **could exceed US$ 1.5 Bn per year†**

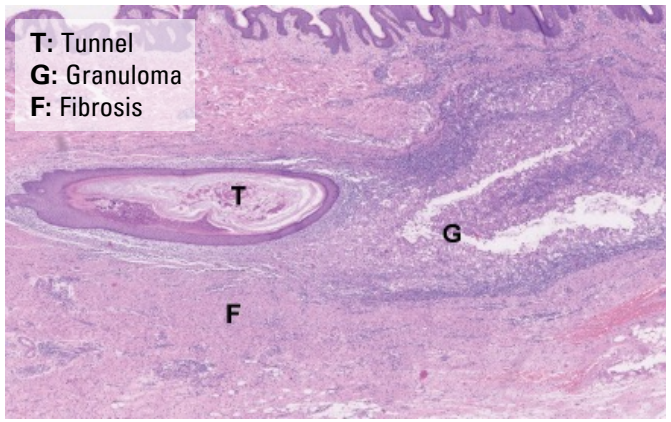




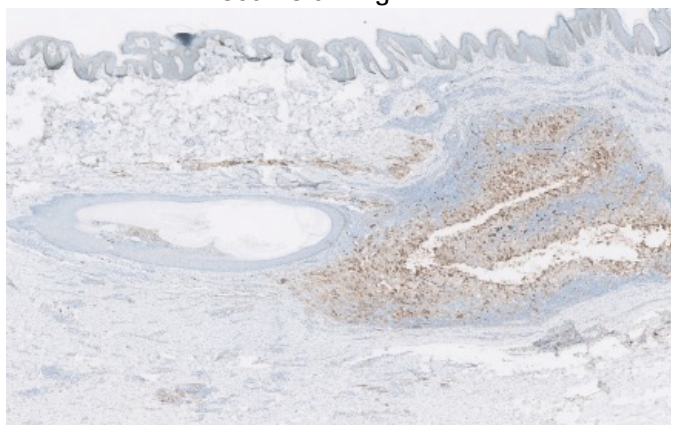
*Willems, D., Hinzpeter, EL., Van der Zee, H.H. et al. Patient 16, 153–164 (2023).
†IFRX proprietary market research, Clarivate.

C5aR and Neutrophils play a critical role in HS pathogenesis especially in draining tunnels (dTs) and abcesses

Standard H&E tissue staining



C5aR staining



Hurley stage III patient with tunnel formation and surrounding **granulomatous** inflammation with foreign body giant cells. **C5aR1 staining positive – neutrophils, histiocytes and giant cells**

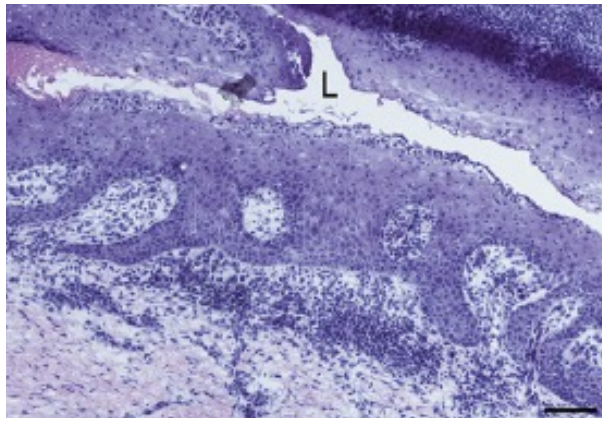
Of note: strong C5aR expression on these cells was found particularly around abscesses and draining tunnels at all stages of the disease

Van Straalen et al. 2022. *Front Immunol* 21.

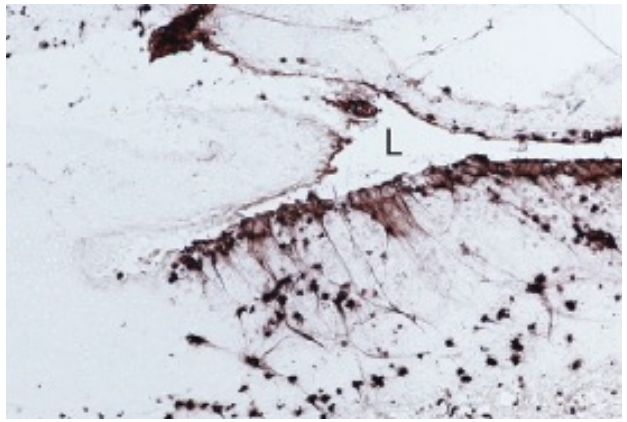
C5a is a key chemoattractant and a strong activator of neutrophils (which have high C5aR density) leading to neutrophil extracellular traps (NET) which are believed to be a disease driver in HS

HS Lesional Tunnel

Standard H&E tissue staining



NE: Neutrophil Elastase staining

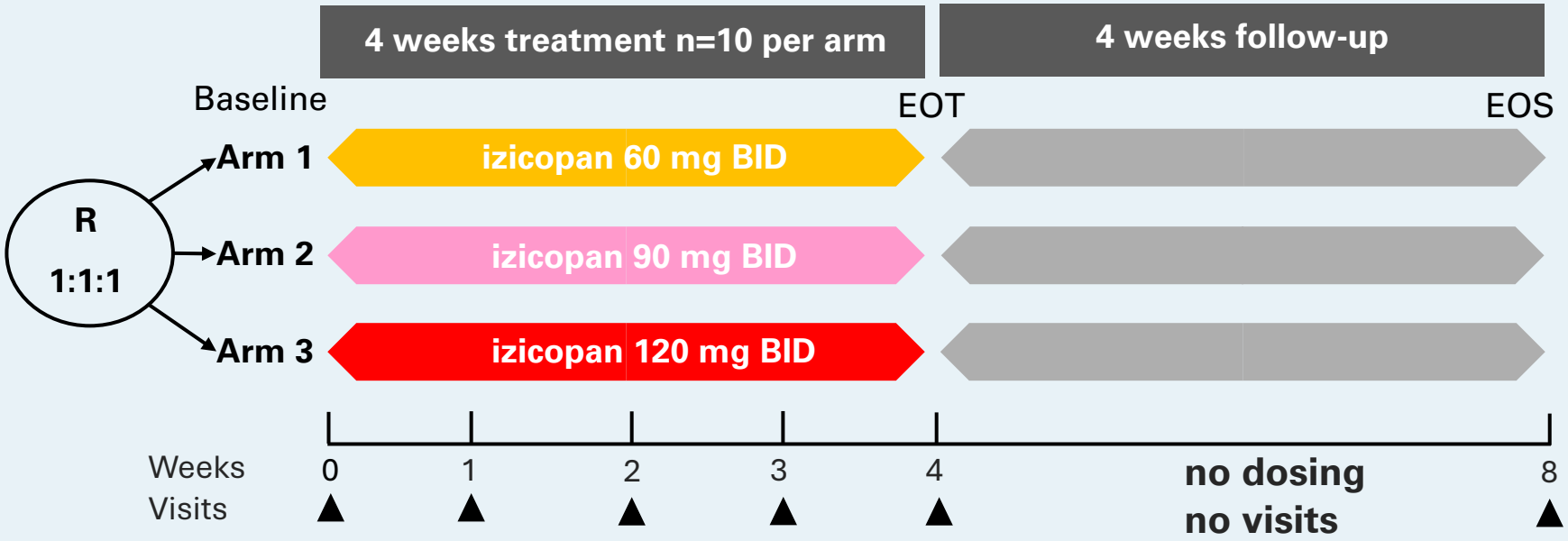


Neutrophils infiltrate inflammatory lesions in HS, including tunnels and the surrounding tissue

NETosis is believed to drive dT formation and C5a/C5aR activation is known to induce this mechanism

Navrazhina et.al, *J Allergy Clin Immunol*, 2021.

Izicopan Phase 2a in HS: Trial design



Izicopan capsules taken with food as described in the protocol.

EOT = End of treatment (week 4).
EOS = End of study (week 8).

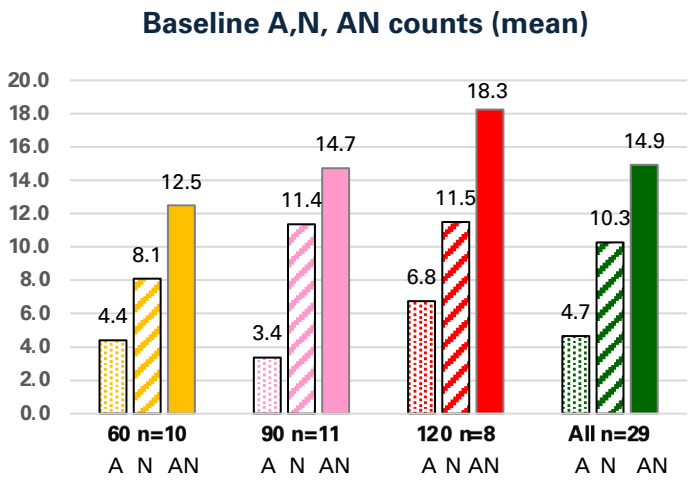
Primary & secondary objectives

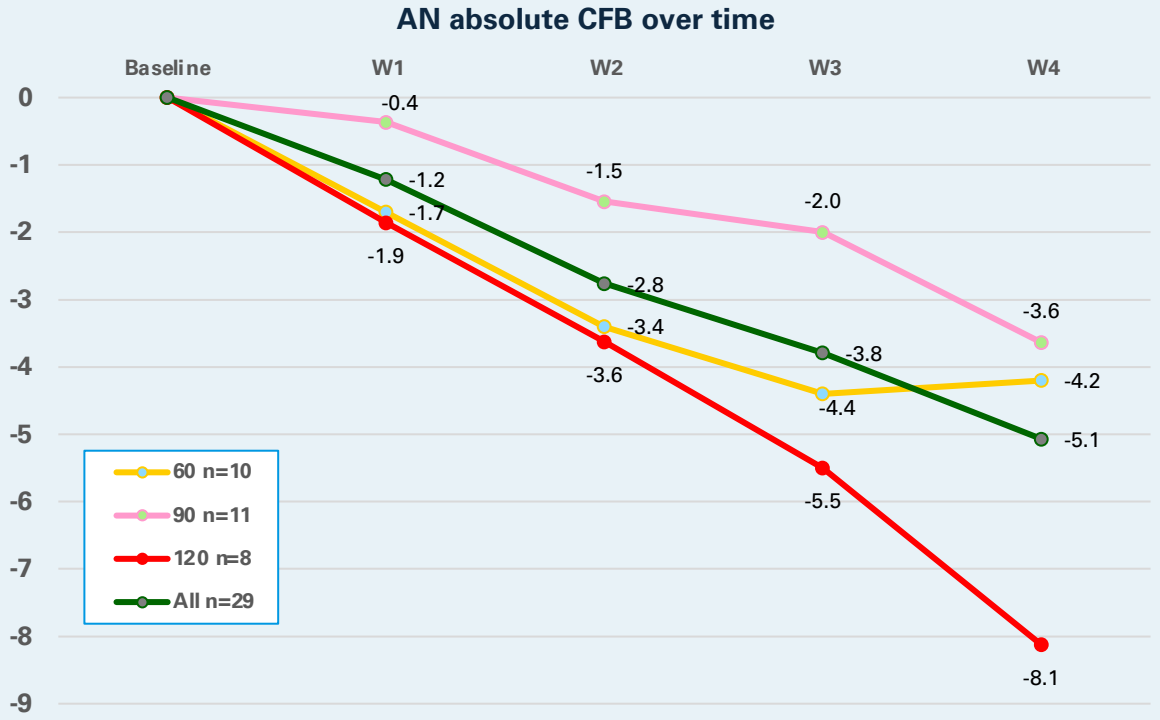
Safety & PK

Exploratory objectives

Clinical endpoints AN, dT, ANdT, HiSCR, NRS-Skin Pain, DLQI

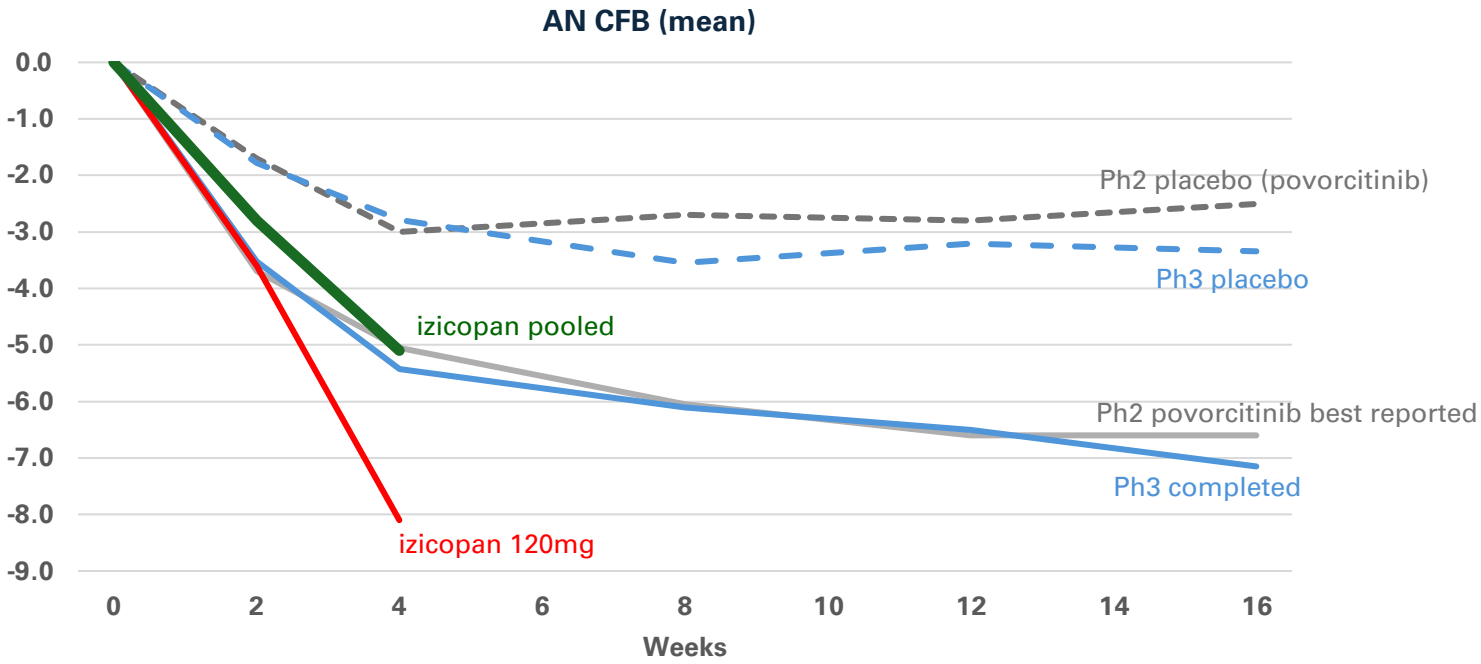
Izicopan Phase 2a in HS: Meaningful and consistent AN count reductions





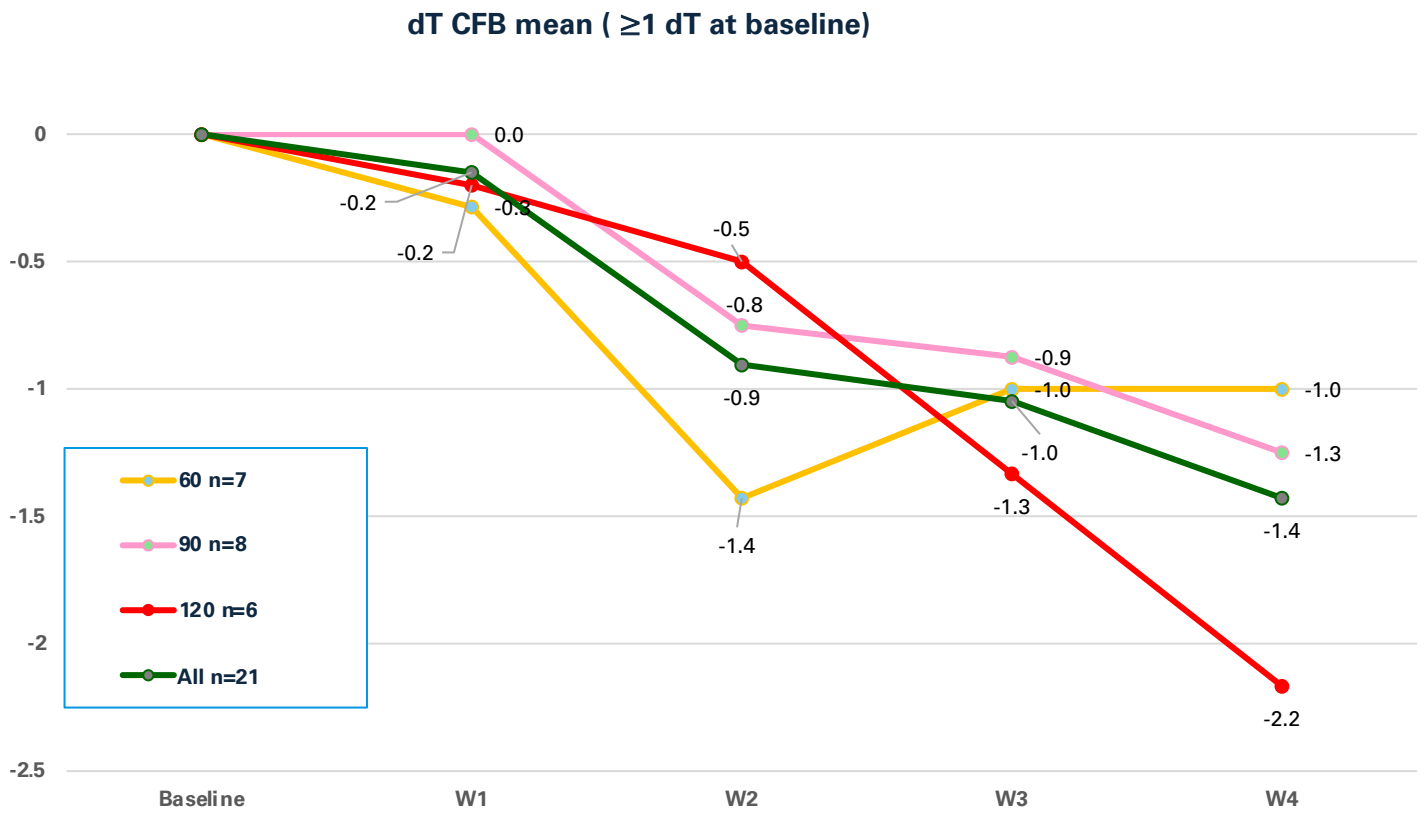
Izicopan Phase 2a in HS: AN reductions in line with successful studies

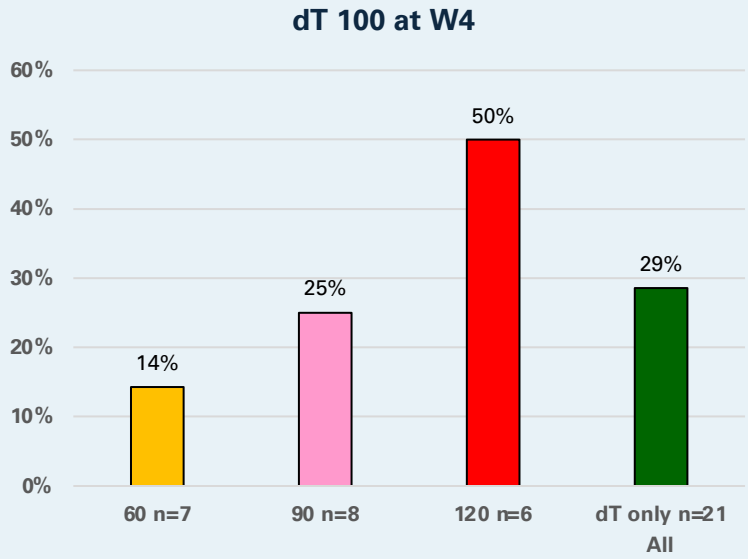
Comparison to average of reported successfully completed Phase 2 (moved into Phase 3) and all available completed Phase 3 drug data*



* Data are derived from different completed Phase 3 clinical trials and averages were created using the data available at each timepoint:
Adalimumab Phase 3 PIONEER I & II: week 12 data for all tested dose groups and placebo as reported by Kimball et al., N Engl J Med 2016; 375:422-34.
Secukinumab Phase 3 SUNSHINE & SUNRISE: week 2, 4, 8, 12, 16 data from all tested dosing regimens and placebo as reported by Kimball et al., Lancet 2023; 401(10378):747-761, absolute mean CFB were calculated from reported relative CFB means (assessed from graphs per timepoint).
Bimekizumab Phase 3 BE HEARD I & II: week 2, 4, 8, 12 and 16 data for all tested dose groups and placebo as reported from Kimball at al., Lancet 2024; 403(10443):2504-2519, absolute mean CFB data for week 2, 4, 8 and 12 are approx. numbers from graphs (data set with multiple imputations, HS-Antibiotics).
Povorcitinib Phase 2 : week 2, 4, 8, 12, and 16 data from the best reported doses (45mg QD & 75mg QD) and placebo as reported by Kirby et al. J Am Acad Dermatol; 90(3);521-529.

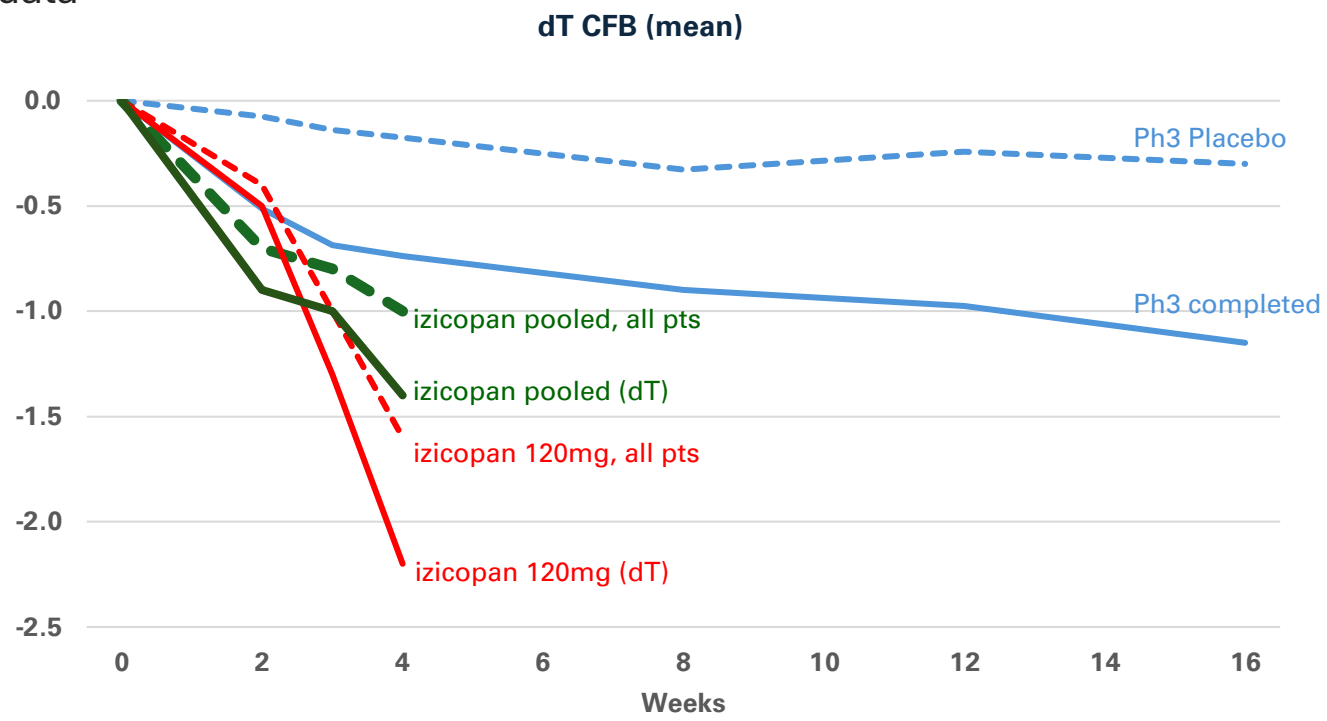
Izicopan Phase 2a in HS: Significant draining tunnel (dT) count reductions





Izicopan Phase 2a in HS: dT reductions vs reported successful Ph3 trials

Comparison to average of available reported completed Phase 3 drug data*



* Data are derived from different completed Phase 3 clinical trials and averages were created using the data available at each timepoint:

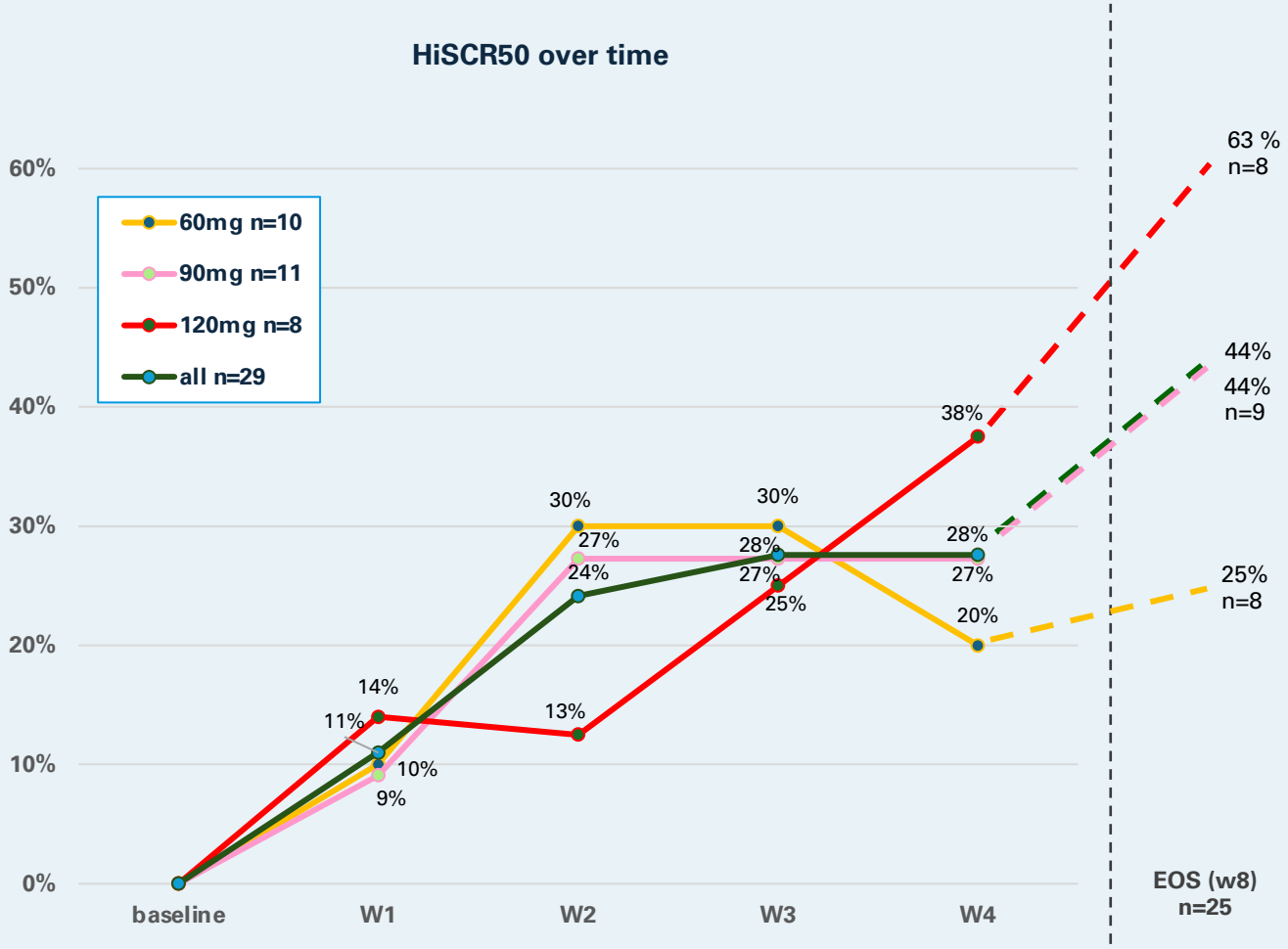
Adalimumab PIONEER I & II: week 12 data for tested dose and placebo as reported by Kimball et al., N Engl J Med 2016; 375:422-34.

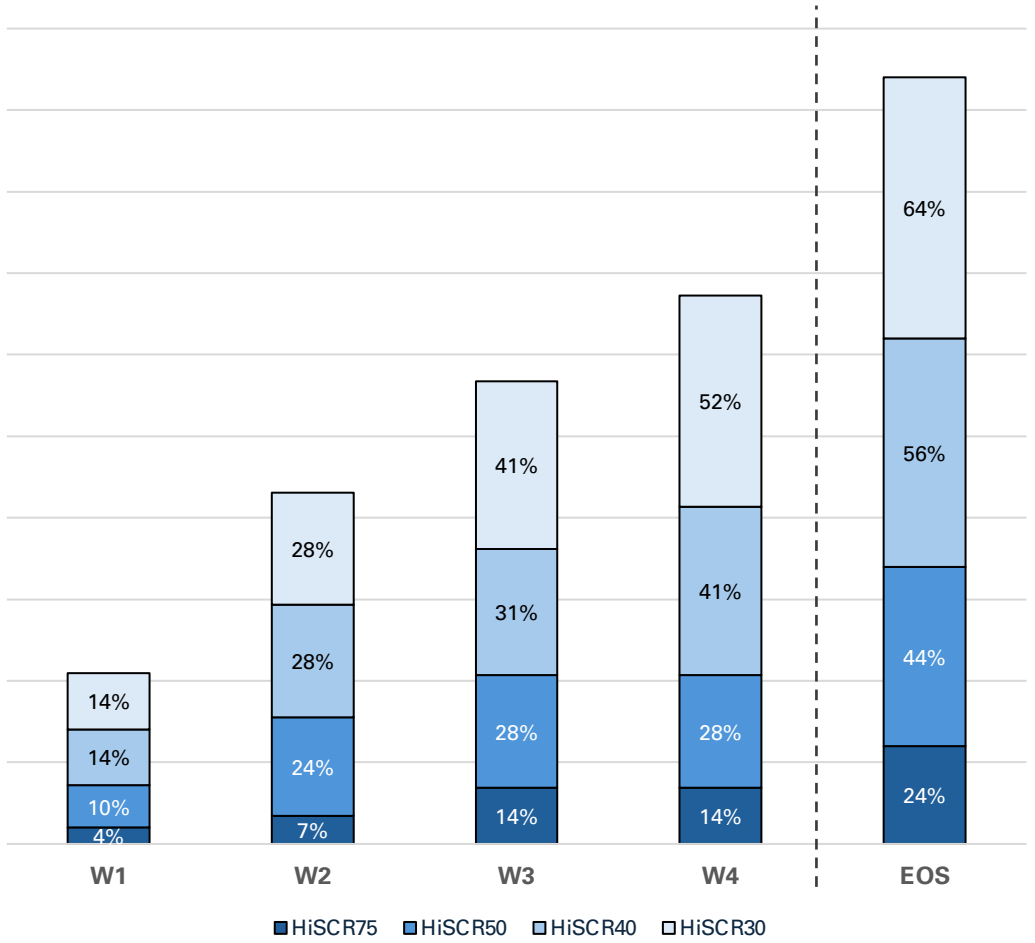
Secukinumab SUNSHINE & SUNRISE: week 2, 4, 8, 12, 16 data from all tested dose regimens and placebo as reported by Kimball et al., Lancet 2023; 401(10378):747-761, note: CFB was approximated by calculating differences in reported total means per week (estimated from published graphs) compared to baseline.

Bimekizumab BE HEARD I & II: week 2, 4, 8, 12 and 16 data for all tested dose groups and placebo as reported from Kimball at al., Lancet 2024; 403(10443):2504-2519, absolute mean CFB data for week 2, 4, 8 and 12 are approx. numbers from graphs (data set with multiple imputations, HS-Antibiotics).

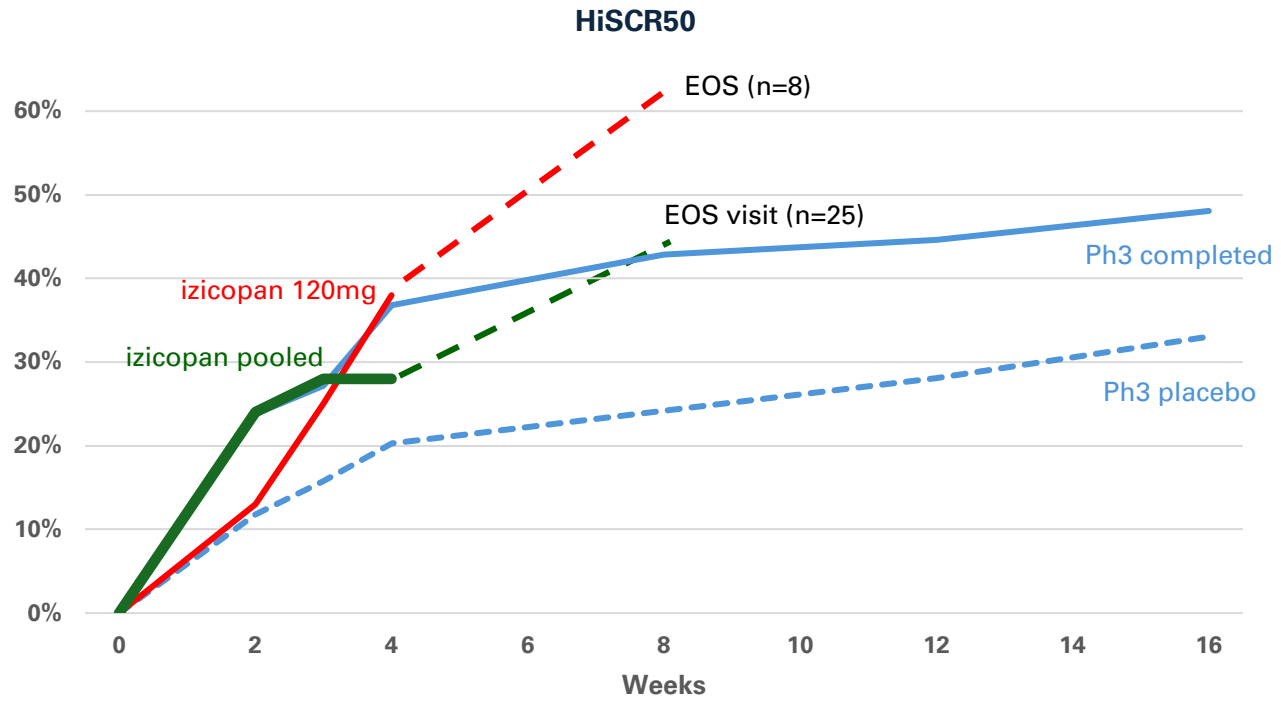
Povorcitinib STOP-HS1 & -HS2: week 3 and 12 data for all tested dosing groups and placebo (ITT population) as reported by Porter et al., EADV 2025 #D1T01.1C.

Izicopan Phase 2a in HS: Early improvements in HiSCR





Izicopan Phase 2a in HS: HiSCR vs successful Ph3 trials



* Data are derived from different completed Phase 3 clinical trials and averages were created using the data available at each timepoint:

Adalimumab PIONEER I & II : week 2, 4, 8, and 12 data for tested dose and placebo as reported by Kimball et al., N Engl J Med 2016; 375:422-34.

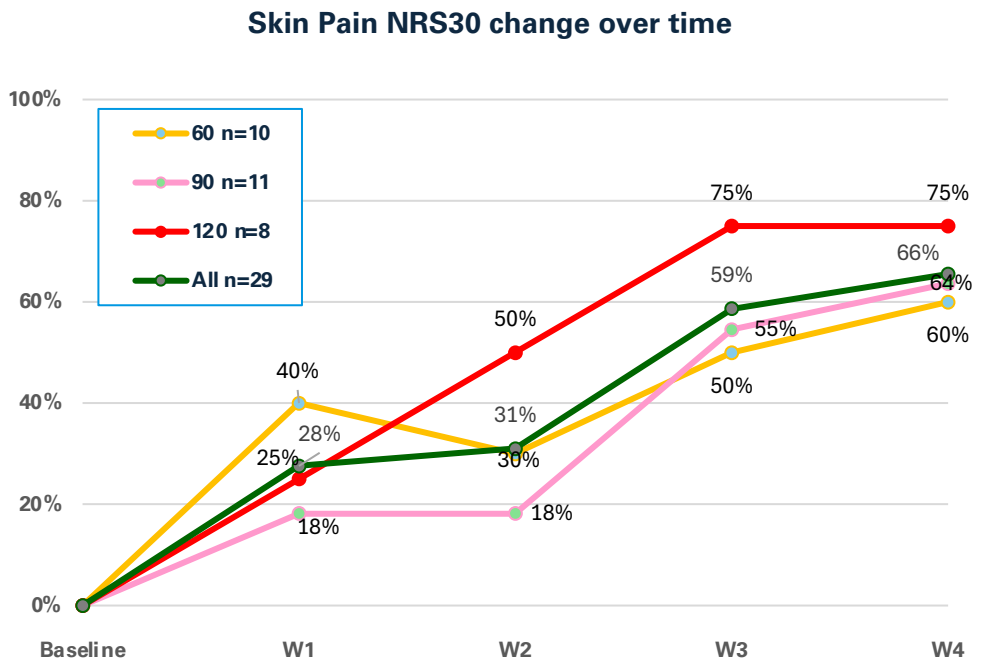
Secukinumab SUNSHINE & SUNRISE : week 2, 4, 8, 12, (approx. from published graph) and 16 (reported) data from all tested doses and from placebo as reported by Kimball et al., Lancet 2023; 401(10378):747-761 and on Clinicaltrials.gov (NCT03713619 , NCT03713632).

Bimekizumab BE HEARD I & II : week 2, 4, 8, 12 (approx. from published graph) and 16 (reported) data for all tested dose groups and placebo (ITT-mNRI [all antibiotics] data set) as reported by Kimball at al., Lancet 2024; 403(10443):2504-2519.

Povorcitinib STOP-HS1 & -HS2 :week 3 and 12 data for all tested doses and placebo (NRI data set) as reported by Porter et al., EADV 2025 #D1T01.1C.

Sonelokinumab VELA-1 & -2 : week 16 data for tested dose and placebo (composite strategy, ITT-mNRI), Moonlake R&D update, 29 September 2025.

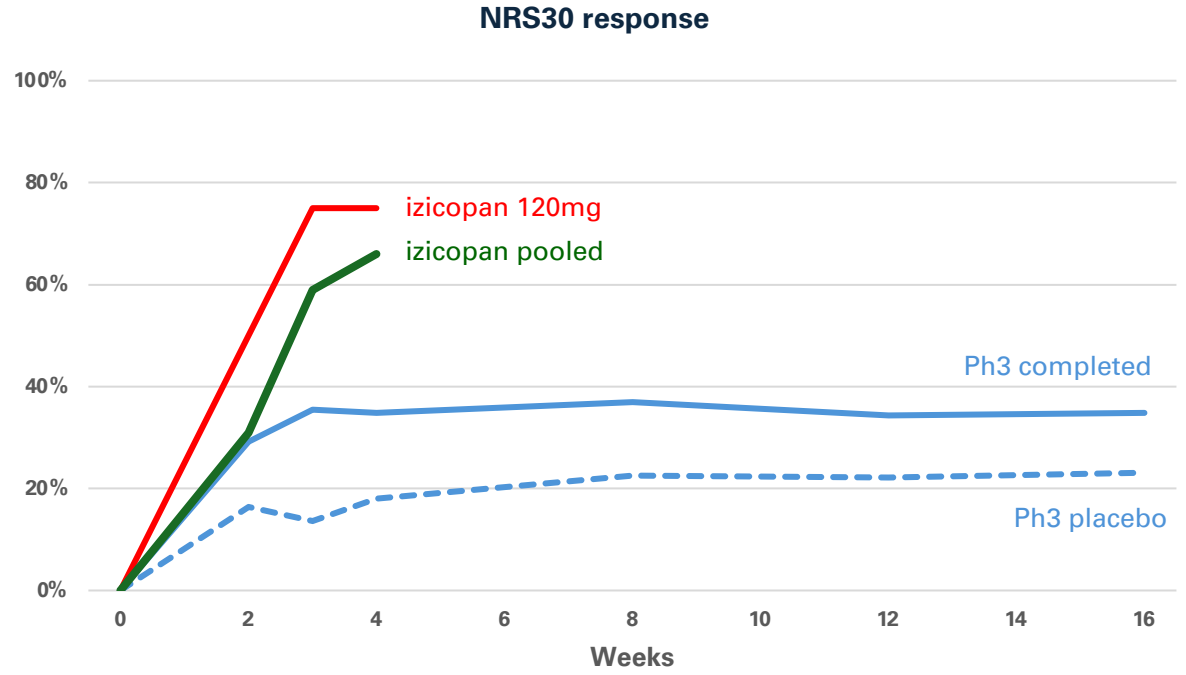
Izicopan Phase 2a in HS: High pain score reduction (NRS30)



NRS30 response is defined as ≥30% reduction from baseline **and at least 2 point reduction**

When applying the NRS 3 points reduction (in analogy to sonelokimab VELA data), the curve is identical

Comparison to average of available reported completed Phase 3 data*



* Data are derived from different completed Phase 3 clinical trials and averages were created using the data available at each timepoint:

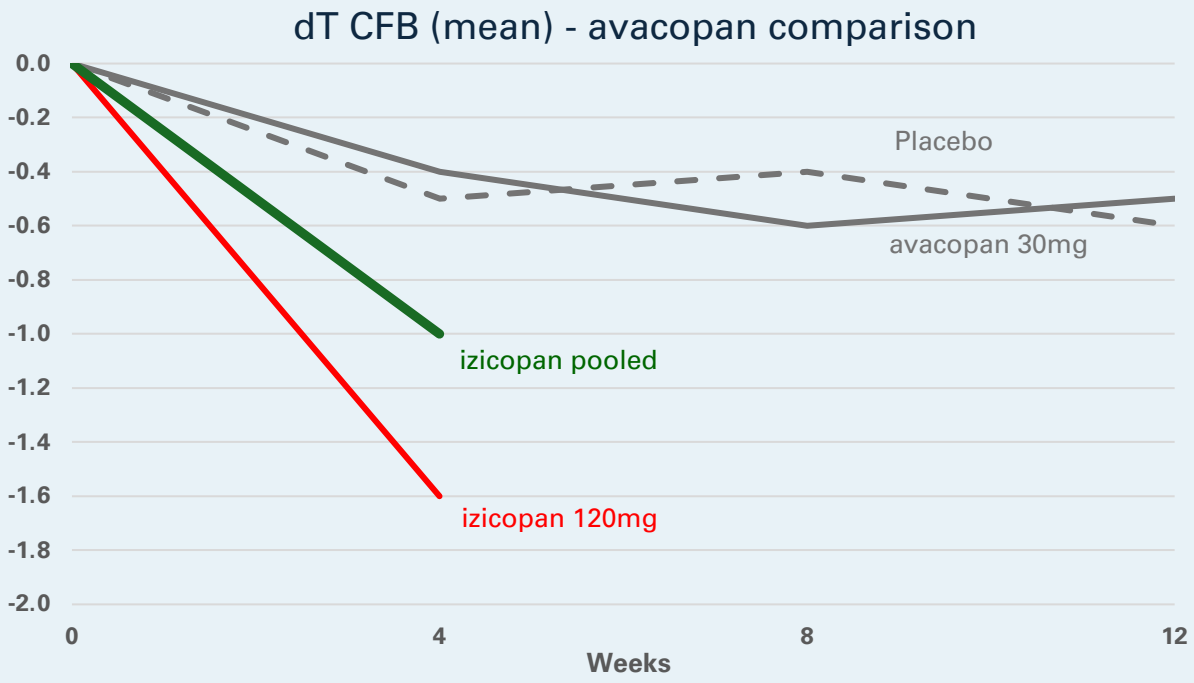
Adalimumab PIONEER I & II – week 2, 4, 8, and 12 data for tested dose and placebo as reported by Kimball et al., N Engl J Med 2016; 375:422-34.

Secukinumab SUNSHINE & SUNRISE – week 2, 4, 8, 12, (approx. from published graph) and 16 data from all tested doses and from placebo as reported by Kimball et al., Lancet 2023; 401(10378):747-761 and on Clinicaltrials.gov.

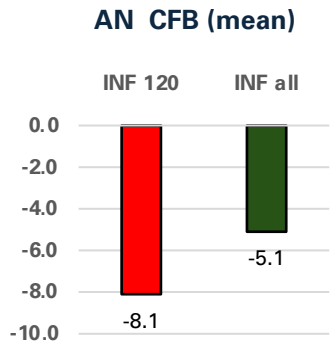
Povorcitinib STOP-HS1 & -HS2 – week 3 and 12 data for all tested doses and placebo, Incyte presentation, 17 March 2025.

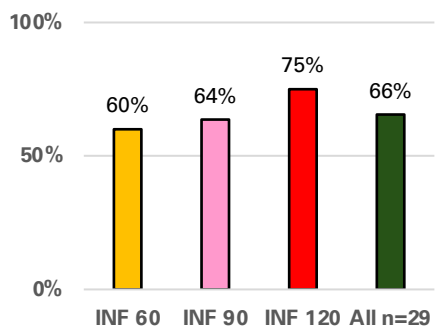
Note definitions: Adalimumab & Povorcitinib: NRS30 defined as ≥30% reduction and ≥1 point reduction from baseline for patients with baseline NRS ≥3 . Secukinumab: NRS30 defined as ≥30% reduction and ≥2 point reduction from baseline for patients with baseline NRS ≥3.

Izicopan Phase 2a in HS: Comparison to reported avacopan data*

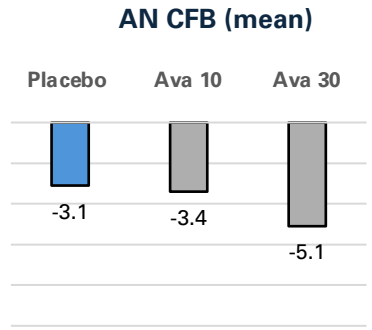


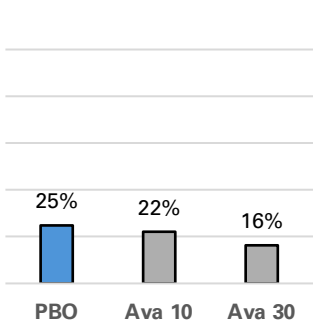
Izicopan Week 4





Avacopan Week 12



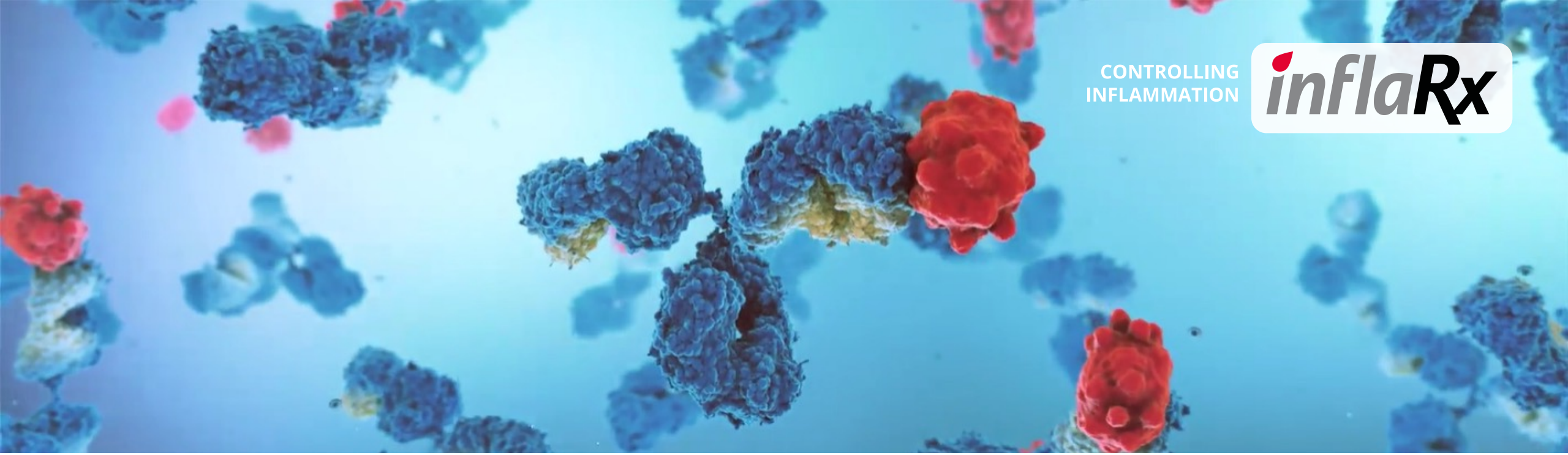


** Data from avacopan Phase 2 AURORA trial reported at clinicaltrials.gov (NCT03852472). NRS30 definition used in AURORA reported data: ≥30% reduction and ≥1 point reduction from baseline for patients with baseline NRS ≥3.*

Highlights of izicopan Phase 2a data in HS

- Izicopan showed **biologic-like efficacy in the first 4 weeks** with AN count reduction and HiSCR in line with successful Phase 3 comparators
  - Preliminary week 8 data indicated that **HiSCR response further deepens in the 4 weeks after end of treatment**
- Izicopan showed a **fast and deep reduction in dT** significantly differentiated from reported placebo responses and compares favorably to reported data of successfully completed Phase 3 drugs
- Izicopan treatment led to a **consistent pain reduction (NRS30)** at week 4 of **~65%** on average
- Improvement in both **NRS30 and DLQI are consistent with improvements of lesion reductions**
- Oral izicopan therapy **did not result in signals of safety concern**

Izicopan shows promise to be a safe oral agent with biologic-like activity and a differentiated new mechanism of action



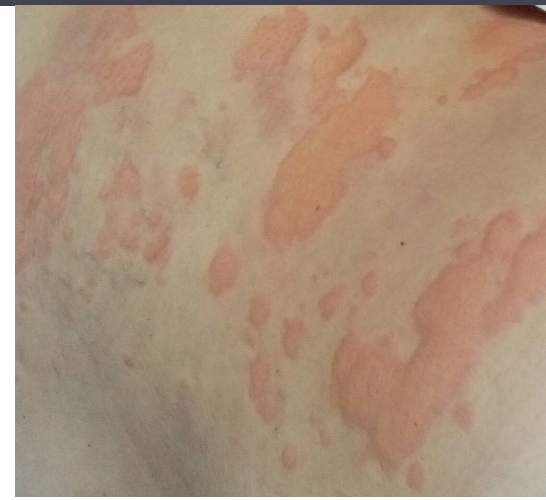
Izicopan for Chronic Spontaneous Urticaria

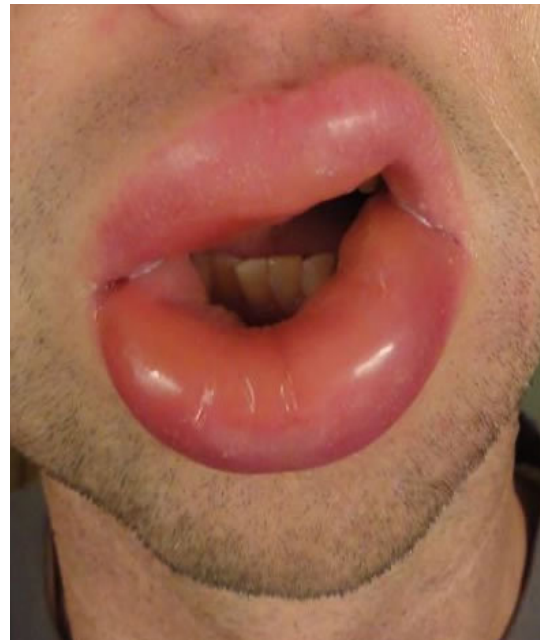
- All Ph2a data presented herein for end of treatment (EOT) at week 4 show results for n=30 evaluable patients
- All Ph2a data presented herein for end of study (EOS) at week 8 (after 4-week follow-up period) show results for n=23 evaluable patients
- Ph2 data from one additional patient still completing treatment are excluded from the current analysis and data presented

**Ph2a data are topline and subject to final data review and quality checks. Results may change as additional patient data are incorporated.*

A strong rationale for developing izicopan in CSU

- CSU is a **chronic inflammatory skin disorder**, with mast cell degranulation leading to various inflammatory cascades that leave patients predisposed to intensely debilitating itchy hives and wheals for ≥ 6 weeks and often associated with angioedema
- CSU has an **estimated prevalence of around 1% of the general population**, and 20% of this population experiences symptoms for more than 5 years
- **C5a is a potent mediator** that leads to mast cell degranulation and can further amplify IgE independent responses - known to attract neutrophils leading to release of mediators and NETosis potentially leading to chronic nature of the disease
- Despite availability of current treatment options such as anti-histamines and anti-IgE therapy, **approximately 30-60%* of these patients are estimated to remain non-responsive or symptomatic**
- Izicopan could be a **convenient oral therapeutic option** for those underserved with current therapies
- Overall maximum market potential for izicopan in CSU **could exceed US$1 Bn per year†**

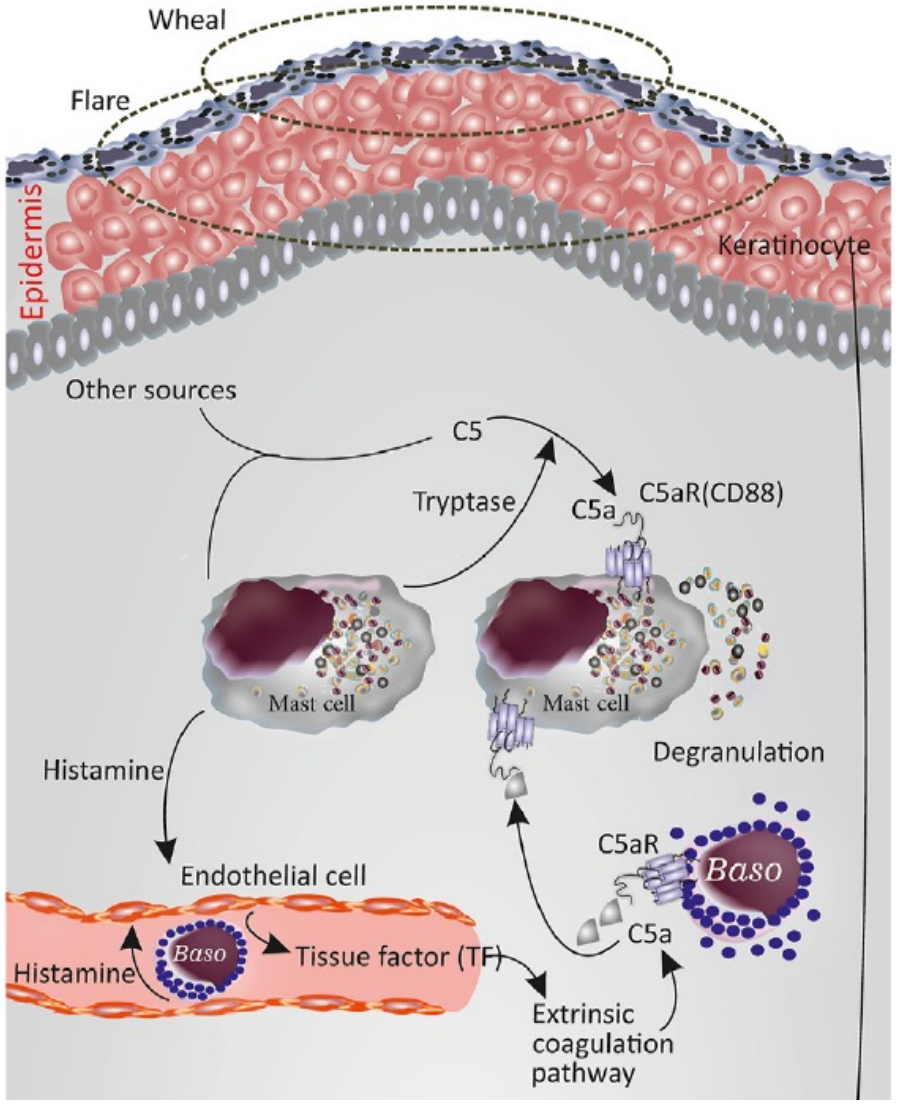




*Metz et al, Clin Rev Allergy Immunol. 2020; 59(1): 38–45.
†IFRX proprietary market research, Clarivate.

Role of complement C5a/C5aR1 and neutrophils in CSU

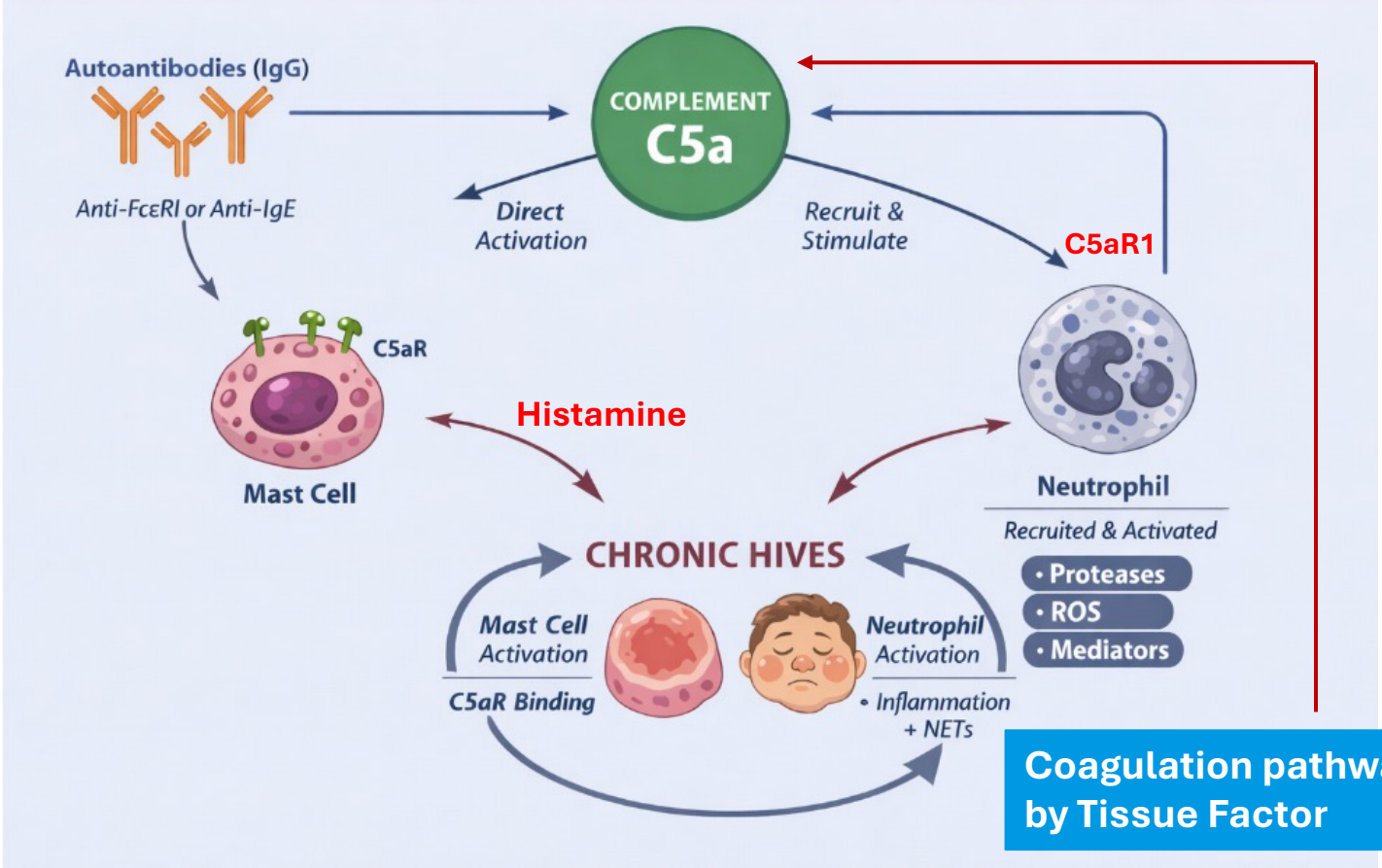
1. **C5a is a potent mast-cell and basophil activator** via C5aR1 which directly triggers degranulation and it can further amplify this response independent of IgE.
2. **C5a can be generated** via autoantibody-driven complement activation (**IgG**) on the FcRI receptor and from the coagulation cascade (**from tissue factor**).
3. **C5a is also a strong neutrophil chemoattractant and activator** leading to mediator release, increased vascular permeability and tissue injury.
   - Causes NET formation (NETosis) which in turn activates complement and mast cells
   - Cross-talk with mast cells
   - Neutrophils amplify mast-cell sensitivity
4. **C5a and neutrophils give rise to persistent mast-cell activation a perpetual loop which leads to chronicity of CSU**



Adapted from : Elieh-Ali-Komi D, Metz M, Kolkhir P, Kocatürk E, Scheffel J, Frischbutter S, Terhorst-Molawi D, Fox L, Maurer M. Chronic urticaria and the pathogenic role of mast cells. Allergol Int. 2023 Jul;72(3):359-368.

Pathogenic loop linking C5a, neutrophils & mast cells in CSU

Complement C5a & Neutrophils in CSU

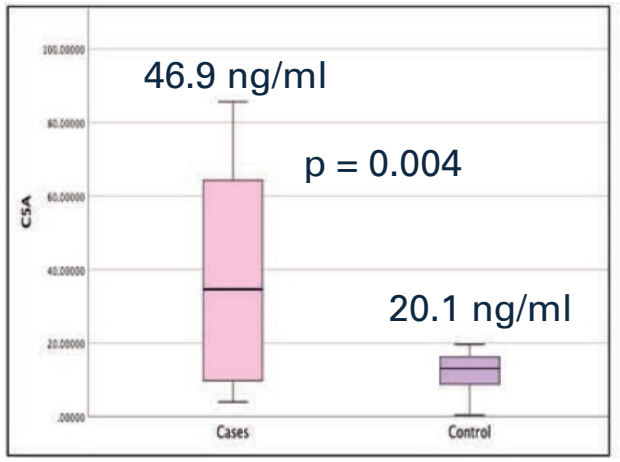


Coagulation pathway activated by Tissue Factor

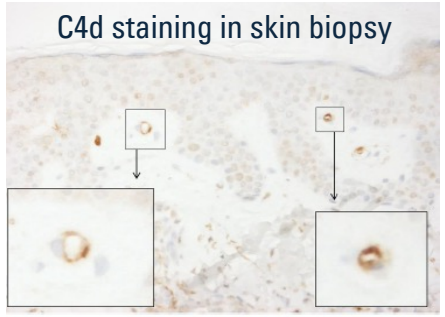
Kulthanan et al Asian Pac J Allergy Immunol 2023;41:179-185 DOI 10.12932/AP-180623-1638.

C5a in CSU and its role in IgE-independent histamine release

CSU patients have elevated C5a levels

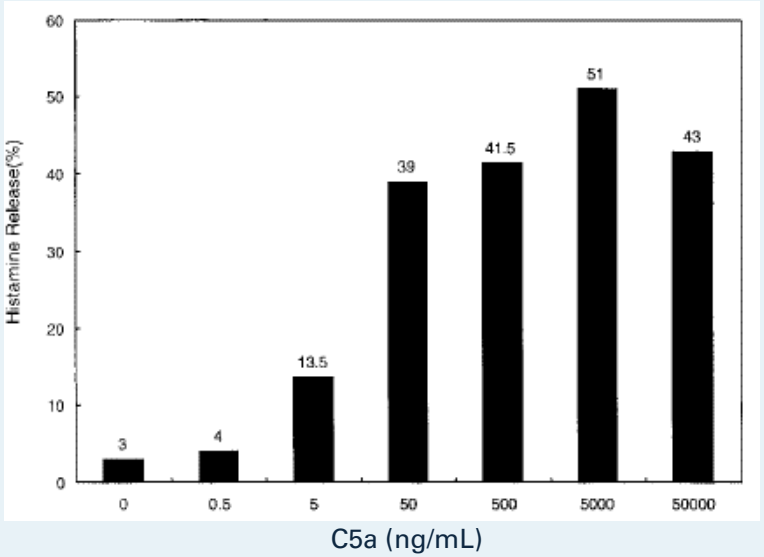


- CSU patients show evidence of complement activation in the skin



Bhatia et al. 2024 Asia Pacific Allergy 14; Aghdam et al. 2021 Clin Transl Allergy. 11

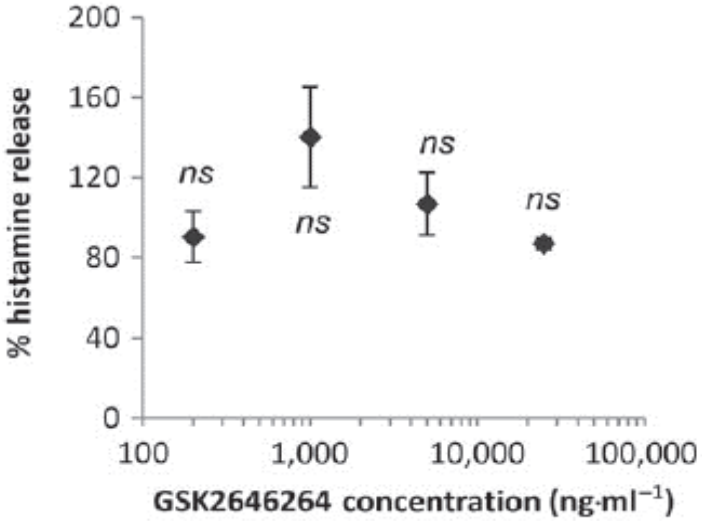
C5a induces histamine release from basophils in a dose-dependent manner



- Histamine release (percentage) from donor basophils stimulated with increasing levels of C5a

Kikuchi, 2002 J Allergy Clin Immunol:109

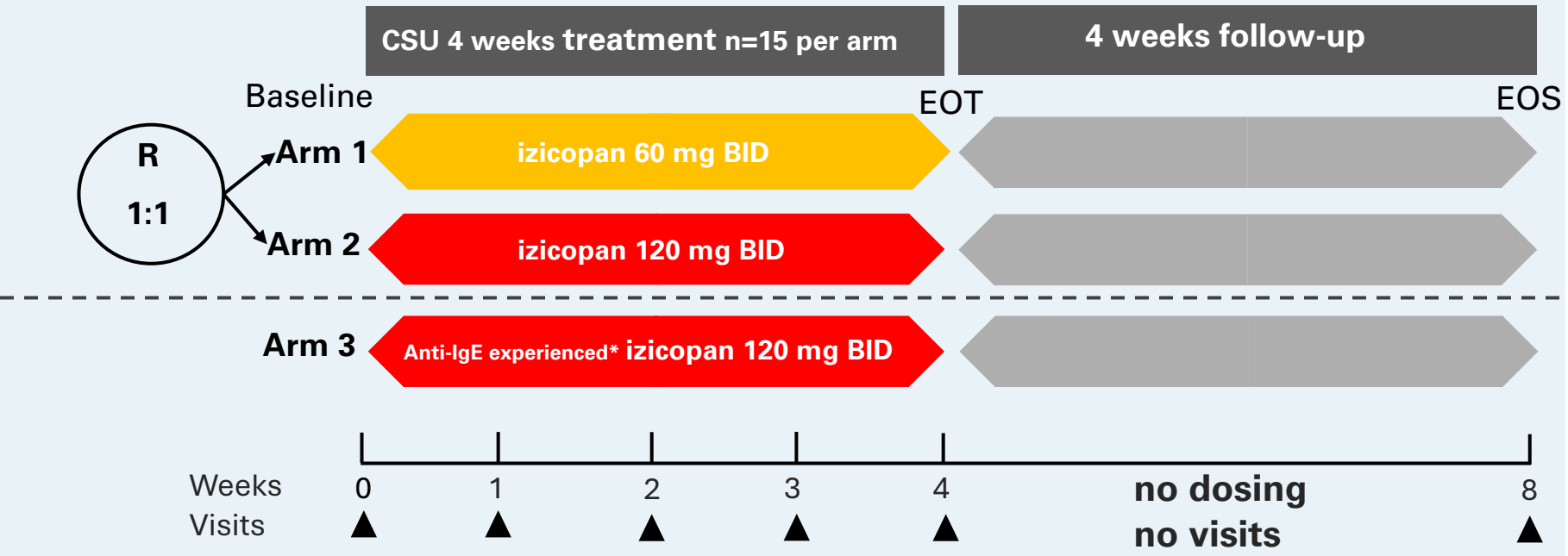
C5a mediated histamine release is independent of the IgE pathway



- Human Skin ex vivo Model: microdialysis tubing into the ex vivo human skin with 1nM C5a
- C5a stimulation of histamine releases is not affected by IgE pathway / SYK inhibitor GSK2646264

Molina et al; 2019 Br J Pharmacol: 176

Izicopan Phase 2a in CSU: Phase 2a trial design



Primary & secondary objectives

Safety & PK

Exploratory objectives

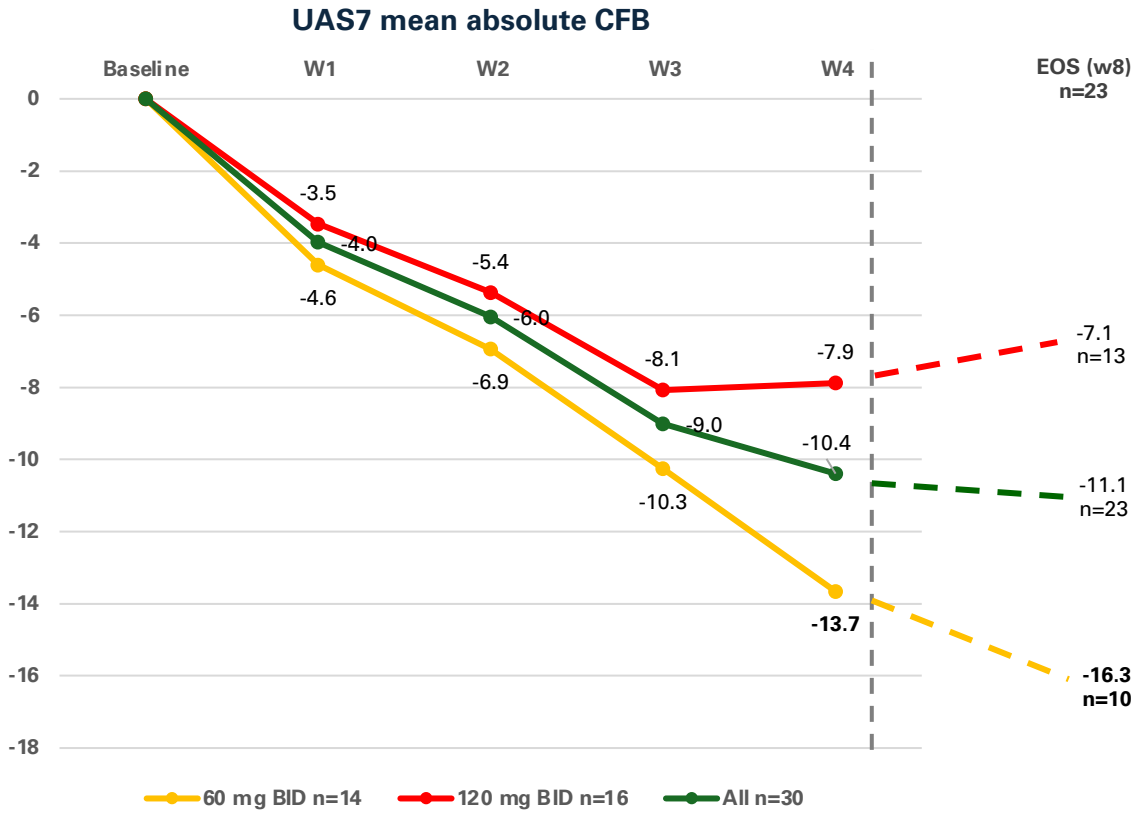
Clinical endpoints include UAS7,UCT7, Responders and QoL

* Incomplete/lack of response to anti-IgE treatment or patients with low baseline IgE < 40.

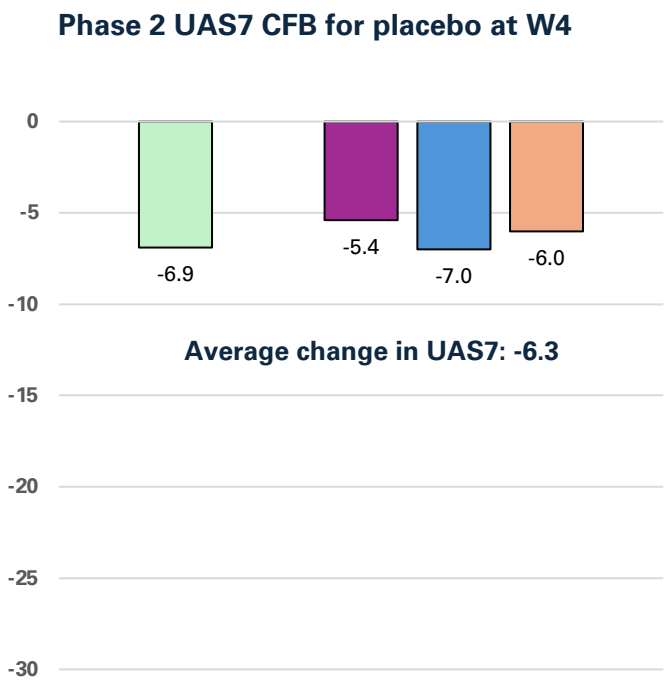
Izicopan capsules taken with food as described in the protocol.

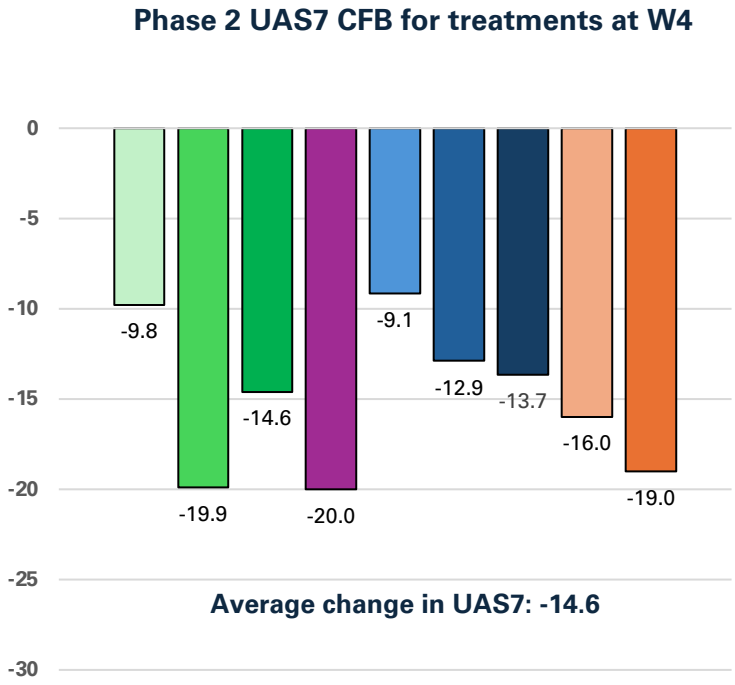
EOT = End of treatment (week 4).
EOS = End of study (week 8).

Izicopan Phase 2a in CSU: Effect on UAS7 at W4 continued to end of study (W8) after end of treatment



Izicopan Phase 2a in CSU: Phase 2 UAS7 data at W4 from competition (products progressed to Phase 3*)

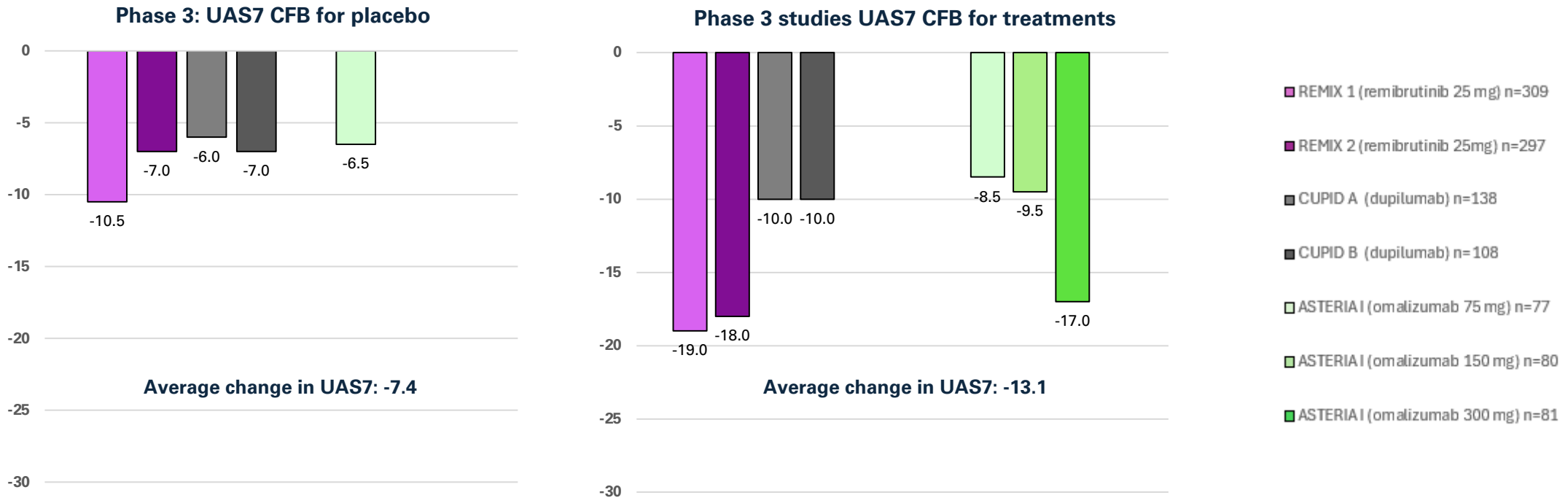




Izicopan UAS7 reductions are within the range of therapies successfully moved into Phase 3

Omalizumab Phase 2: Saini S et al J Allergy Clin Immunol. 2011 Sep;128(3):567-73.
Remibrutinib Phase 2: Maurer M, et al. J Allergy Clin Immunol. 2022 Dec;150(6):1498-1506.
Barzolvolima Phase 2: EAACI2025.
Rilzabrutinib Phase 2: Giménez-Arnau A, et al JAMA Dermatol. 2025 Apr 23.
Note: Week 4 data for rilza and barzo approx. numbers from graphs .
**Not all doses were progressed to Ph3.*

Izicopan Phase 2a in CSU: Comparative Phase 3 UAS7 data at W4

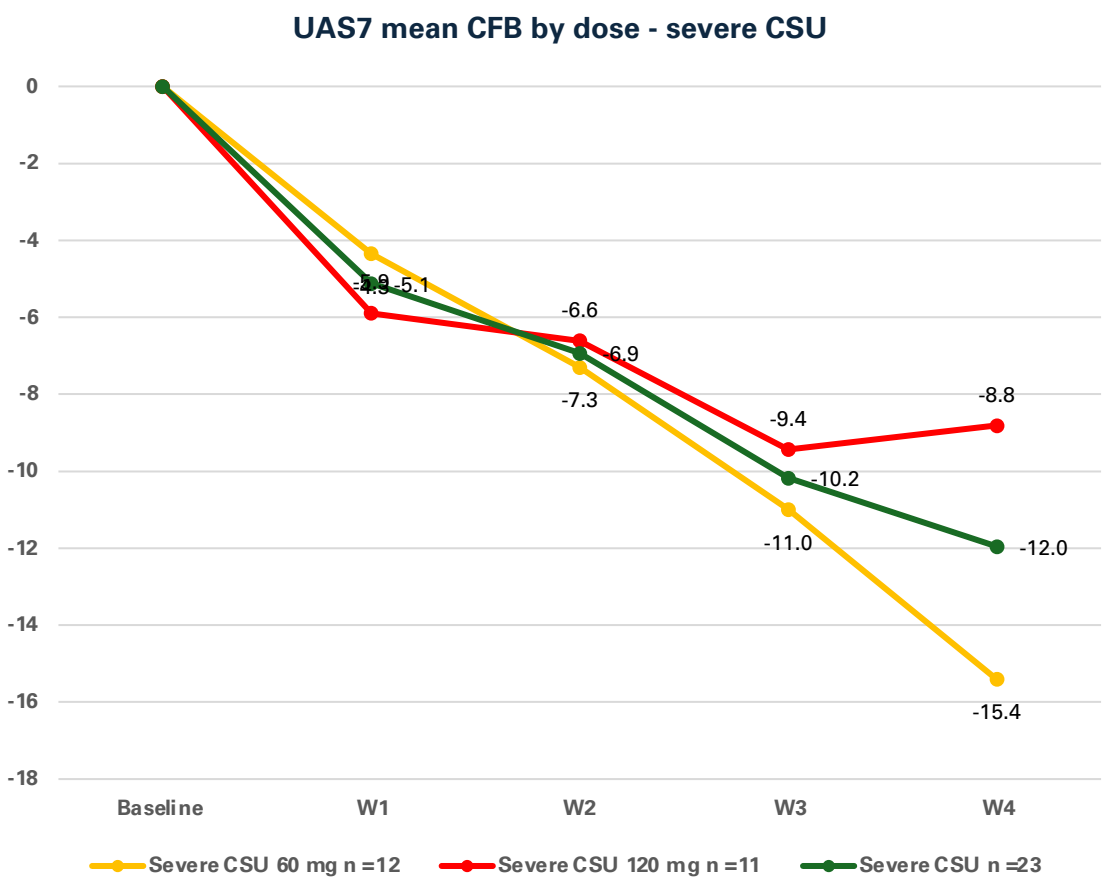


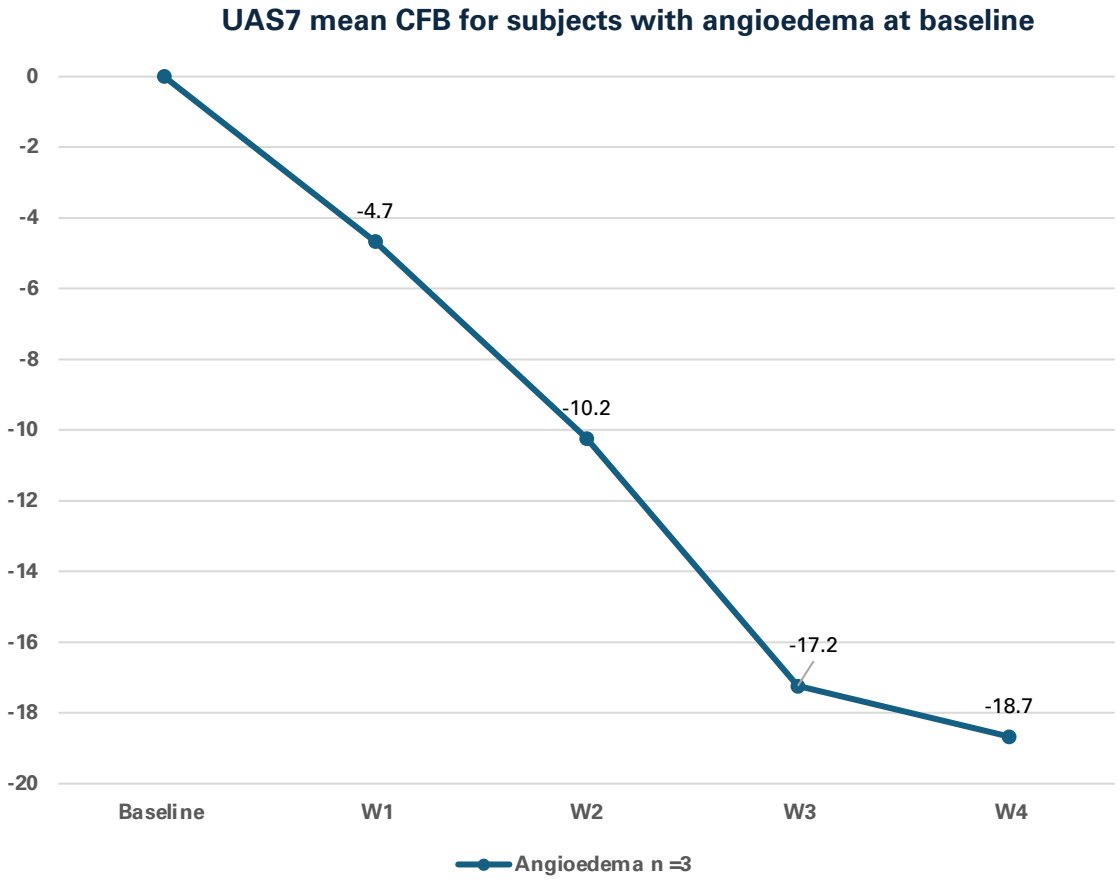
Izicopan UAS7 reductions are within the range of therapies successfully completing Phase 3

Omalizumab Phase 3 ASTERIA II: Maurer M N Engl J Med. 2013 .
Omalizumab Phase 3 ASTERIA I: Saini SS,. J Invest Dermatol. 2015.
Remibrutinib Phase 3: Metz M et al. N Engl J Med. 2025 Mar 6;392(10):984-994.
Dupilumab Phase 3: Maurer M et al J Allergy Clin Immunol. 2024 Jul;154(1):184-194 CUPID A and B, CUPID C clinical trial.gov.
Week 4 data for oma, dupi and remi are approx. numbers from graphs.

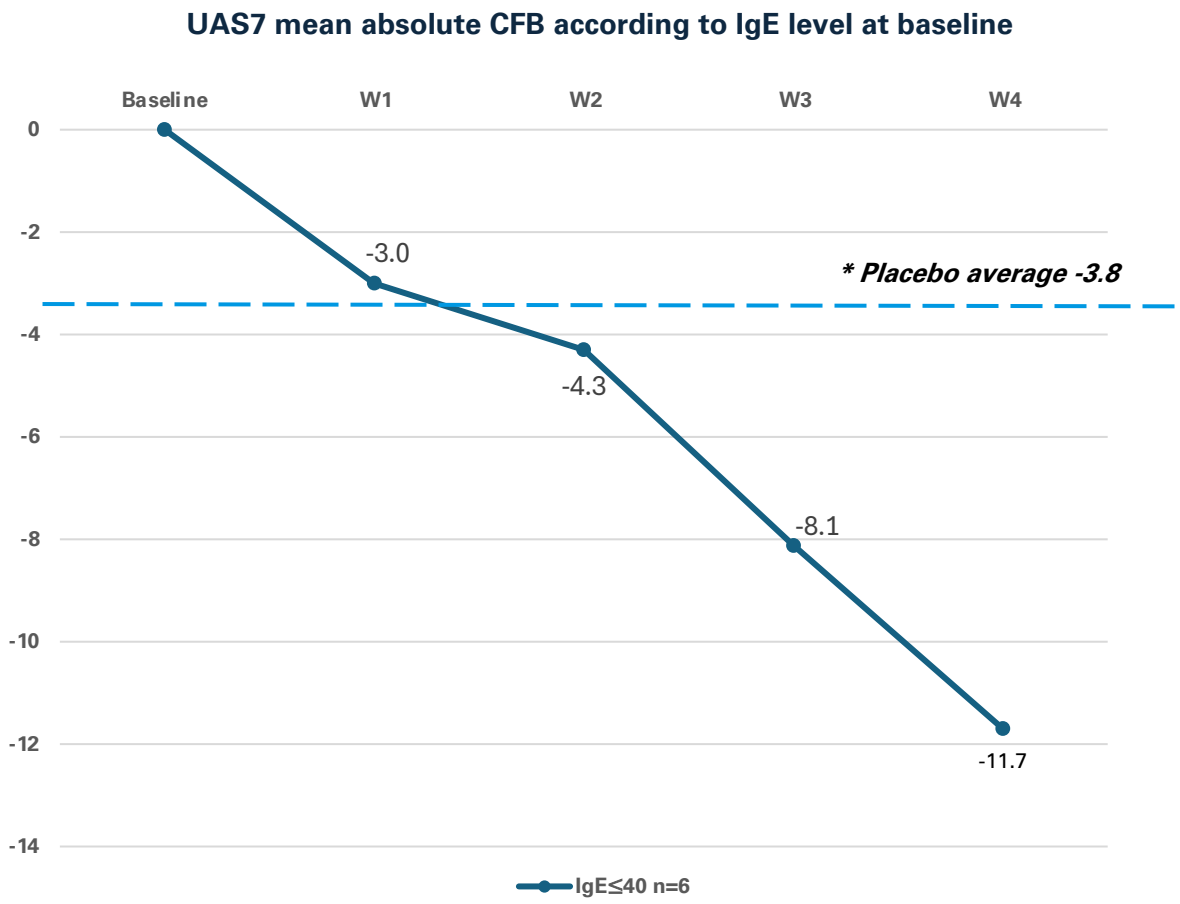
Izicopan Phase 2a in CSU: Substantial reduction in UAS7 in sub-populations

Patients with severe disease and those with angioedema





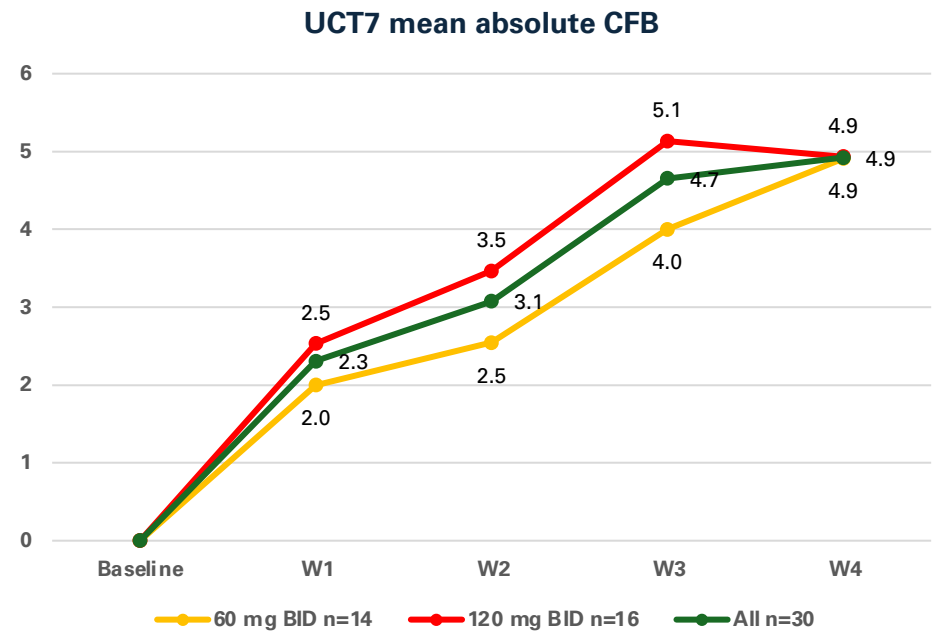
Izicopan Phase 2a in CSU: UAS7 reduced in patients with low IgE at baseline

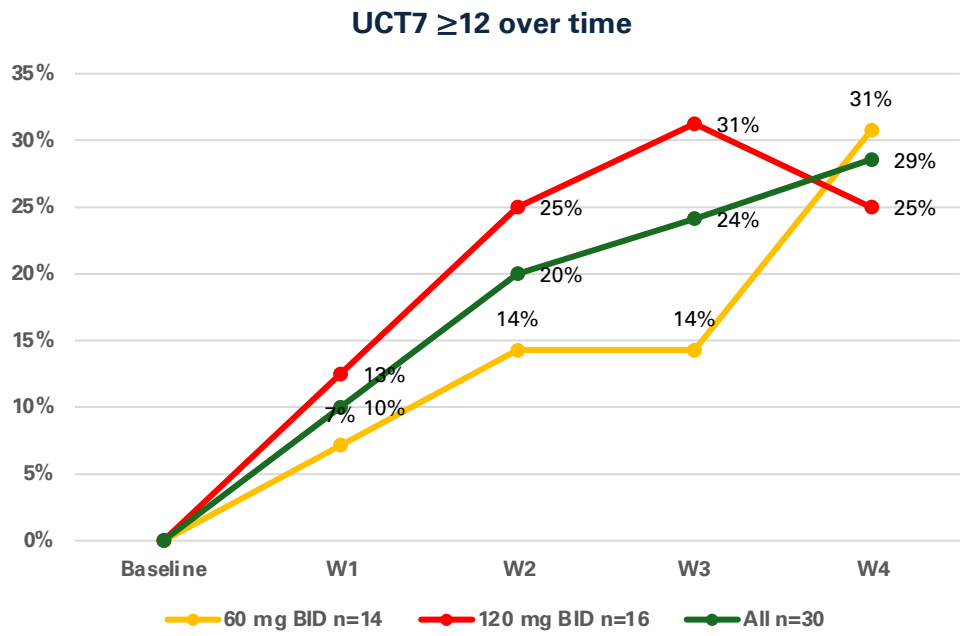


*Placebo rates tend to be lower in the low-IgE group from remibrutinib and barzovolimab Ph2 W12 data

** Dotted line represents the placebo average UAS7 CFB at Week 12 of remibrutinib and barzolvolimab Phase 2 data for IgE low patients (-1.6 and 6.1).*
Remibrutinib Research Abstract / Ann Allergy Asthma Immunol 129 (2022) S15—S81.
Barzovolimab EADV 2025 presentation.

Izicopan Phase 2a in CSU: Disease control with improvement in UCT7





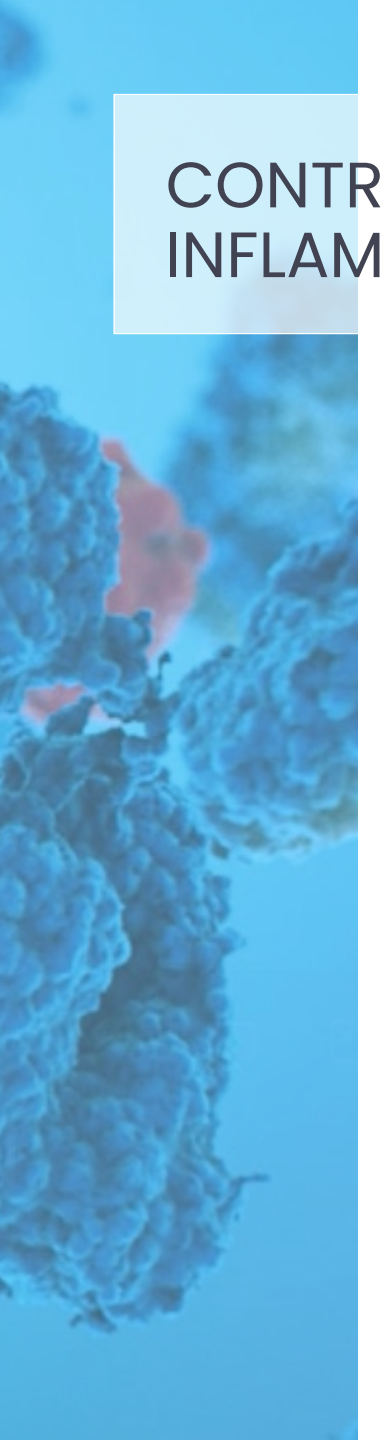
All doses achieved more than 4-point improvement in UCT7 score by W4

Highlights of izicopan Phase 2a in CSU

- Izicopan drives reduction in disease activity (UAS7 reduction) within a range indicative of clinical activity (as observed from Phase 2 & 3 comparators)
  - **60mg dose achieved the highest reduction of UAS7 –13.7**
  - In those with "**most severe"** CSU (defined as UAS7 of 28 -42), 60mg achieved a reduction of **UAS7 –15.4**
  - izicopan treatment in CSU patients with **angioedema** led to the highest reduction in **UAS7 –18.7**
  - izicopan achieves UAS7 reductions **in all comers**, **including those with low IgE levels** (indicative of Type IIb population) and those with high IgE levels at baseline
- Izicopan also shows **improvement in disease control** as measured by UCT7
- Oral izicopan therapy **did not result in signals of safety concern**

Izicopan has promise to be a safe oral agent in CSU, with multiple signals of efficacy on par with other agents

CONTROLLING
INFLAMMATION



InflaRx N.V.

Winzerlaer Str. 2
07745 Jena, Germany

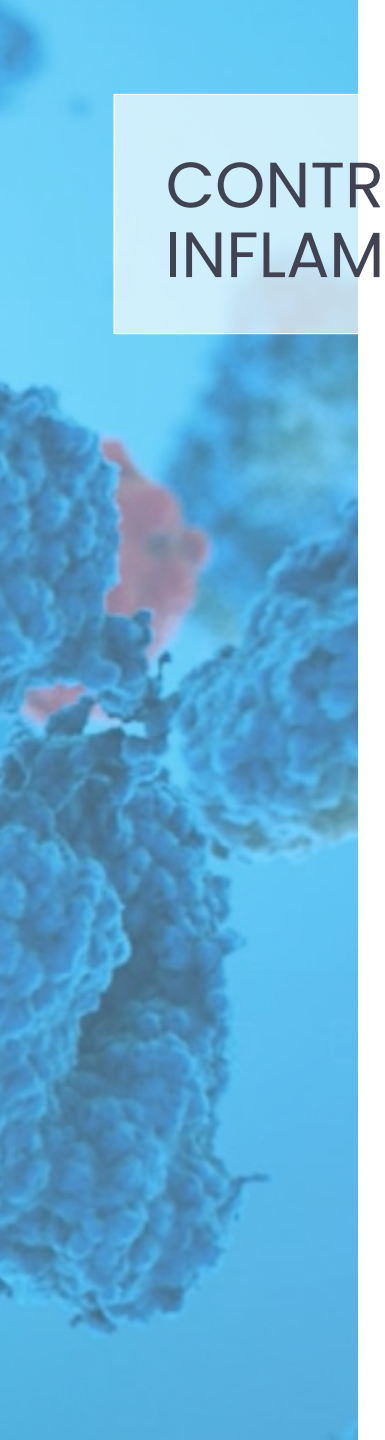
Email: IR@inflarx.com

Tel: +49-3641-508180

Fax: +49-3641-508181

www.inflarx.com

CONTROLLING
INFLAMMATION



Appendix

Izicopan Ph2a HS & CSU: Safety in patients taking at least 1 dose

HS (n = 33)

- **No signals of safety concern detected**
- No reported serious adverse events
- 3 adverse events (AEs) in 2 patients reported as possibly-to-likely related, all mild (Grade 1)

AE term	CTCAE grade	relationship to study drug
Food-induced diarrhea	Moderate = Grade II	Unrelated
Chemical burn R foot	Moderate = Grade II	Unrelated
Laceration (L anatomic lateral distal thumb)	Mild = Grade I	Unrelated
Post-nasal drip	Mild = Grade I	Unrelated
Food poisoning due to mushroom consumption	Mild = Grade I	Unrelated
Exacerbation of episodic headaches	Mild = Grade I	Unrelated
Bacterial Vaginosis	Moderate = Grade II	Unrelated
Motor Vehicle Accident	Mild = Grade I	Unrelated
Headache	Mild = Grade I	Unrelated
Acute viral infection	Moderate = Grade II	Unrelated
Headache	Mild = Grade I	Unrelated
Diarrhea	Moderate = Grade II	Unrelated
Vomiting	Mild = Grade I	Definitely related
Insomnia	Mild = Grade I	Probably related
Diarrhea	Mild = Grade I	Possibly related

CSU (n = 33)

- **No signals of safety concern detected**
- No reported serious adverse events
- 1 AE reported as possibly-related, mild (Grade 1)

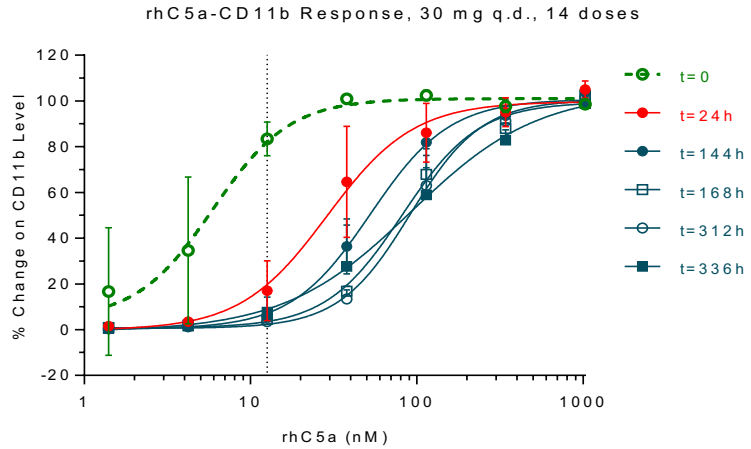
AE term	CTCAE grade	relationship to study drug
Headache	Mild = Grade I	Unrelated
Nausea	Mild = Grade I	Unrelated
Left Hand Swelling	Moderate = Grade II	Unrelated
Right Middle Finger Swelling	Mild = Grade I	Unrelated
Abdominal Cramping	Mild = Grade I	Unlikely related
Nausea	Mild = Grade I	Unlikely related
UTI	Mild = Grade I	Possibly related
Eczema behind right ear (due to scratching)	Mild = Grade I	Unrelated
Three insect bites right upper arm	Mild = Grade I	Unrelated
COVID-19 infection	Mild = Grade I	Unrelated

CTCAE: Common Terminology Criteria for Adverse Events

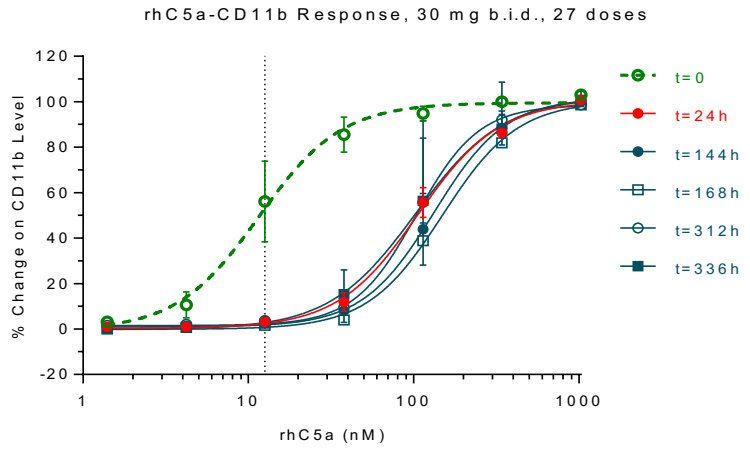
Izicopan: Oral C5aR antagonist with best-in-class potential

C5a-mediated CD11b upregulation on neutrophils ex vivo up to 14-day dosing

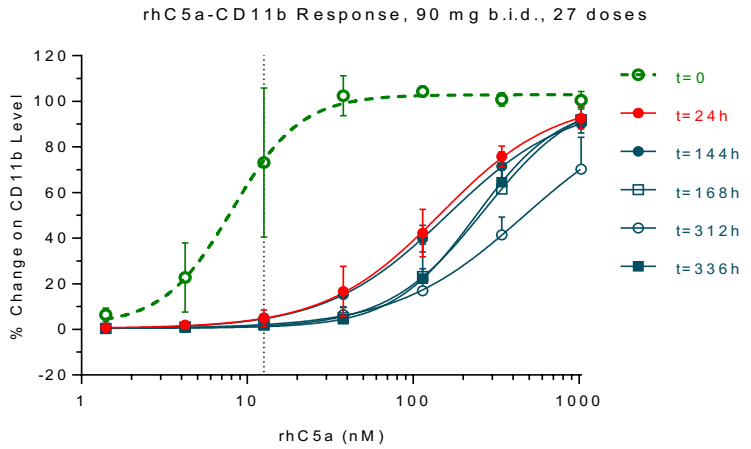
IZICOPAN: 30 MG QD



IZICOPAN: 30 MG BID



IZICOPAN: 90 MG BID



	Upon stimulation with 12.6 nM rhC5a (levels observed in disease state)														
	24 h			144 h (Day 6)			168 h (Day 7)			312 h (Day 13)			336 h (Day 14)		
	30QD	30BID	90BID	30QD	30BID	90BID	30QD	30BID	90BID	30QD	30BID	90BID	30QD	30BID	90BID
Blockade (%)	80	94	90	93	95	94	95	97	97	96	92	97	90	95	97
EC_{50} (nM)	35.6	106.2	145.6	52.4	134.7	160	74.2	149.0	268.2	92.4	126.3	465.7	94.6	110.9	238

PD MAD results confirm strong >90% C5a inhibition at C5a levels found in human diseases – this is clearly differentiated from reported avacopan results which have shown approximately 50% inhibition at a lower challenge of 10nM C5a (7-day dosing – trough)**

*EC_{50} (nM) is the half maximal effective C5a concentration ** Bekker et al. 2016, PLoSOne; 11(10): e0164646

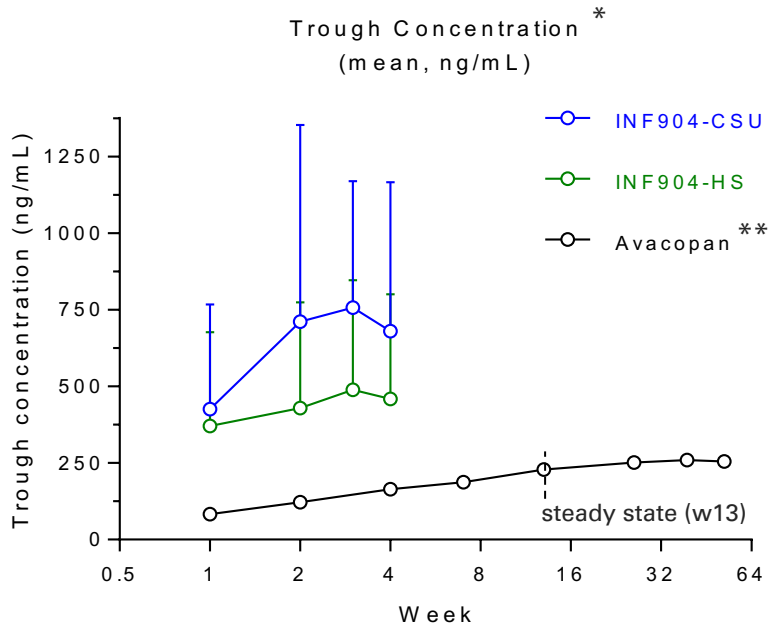
Izicopan therapeutic exposure reached within the first week in Phase 2a

Initial considerations

- Lower dose of 60 mg bid projected to result in **complete signal control based on Phase 1 data**
- **Explore efficacy at the high end** by adding 90 mg bid (HS) and 120 mg bid (HS and CSU)

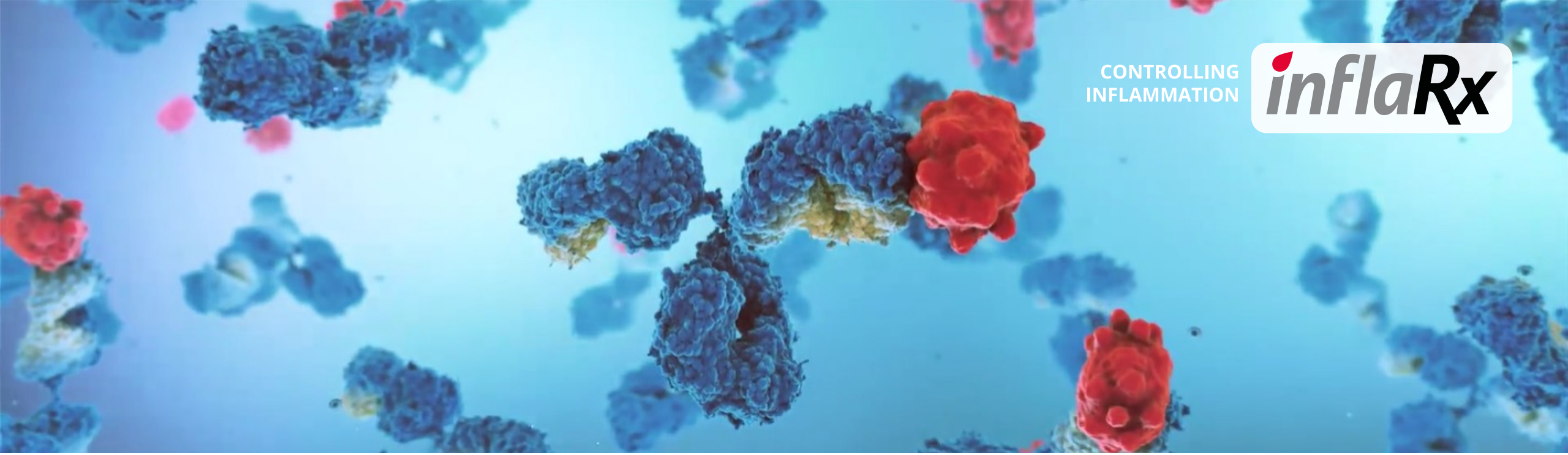
Preliminary PK results*

- **High plasma exposure can be reached approximately within the first week** (all doses tested), compared to 13 weeks for reported avacopan PK data



* Preliminary PK results of pooled 19-21 HS patients (60, 90, 120mg bid) and 14-17 CSU patients (60, 120mg bid) at topline data release. InflaRx data on file final and full QC checked results will be reported after completion of the study.

** Steady state plasma levels of avacopan (NDA filing for ANCA-associated vasculitis, 75-100 patients) are reached by 13 weeks and the accumulation is approximately 4-fold

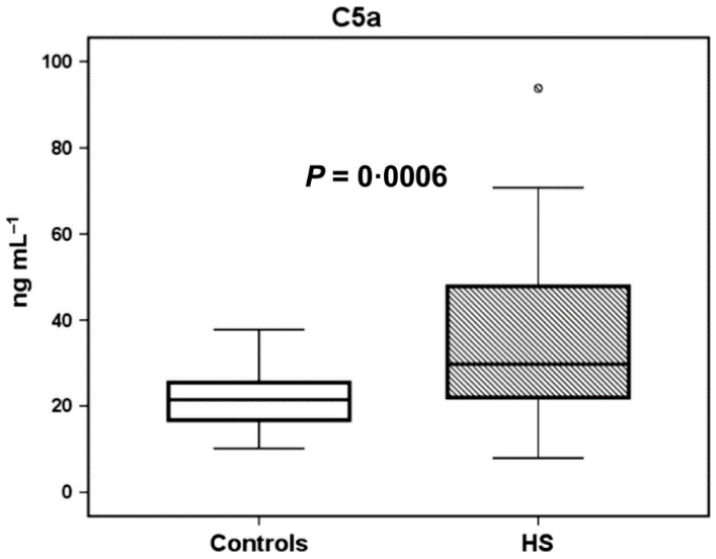


HS Appendix

Strong rationale for developing an anti-C5a/C5aR in HS

HS Patients Have Elevated C5a, a Major Neutrophil Activator That Can Be Blocked by an Anti-C5a/C5aR

HS patients have **significant complement activation** with elevated C5a levels



Concentrations of C5a in the plasma of 14 healthy controls and of 54 patients with HS. P-values symbolize significant differences between patients and controls.

Kanni et al, 2018

C5a/C5aR activation is a key neutrophil activator in HS patient plasma

- HS patient plasma strongly provokes neutrophil activation in healthy donor blood: this effect could be completely blocked by inhibiting C5a/C5aR.

Guo et al. 2019 Aug. US Patent No. 10,376,595
Source: InflaRx in house data on file

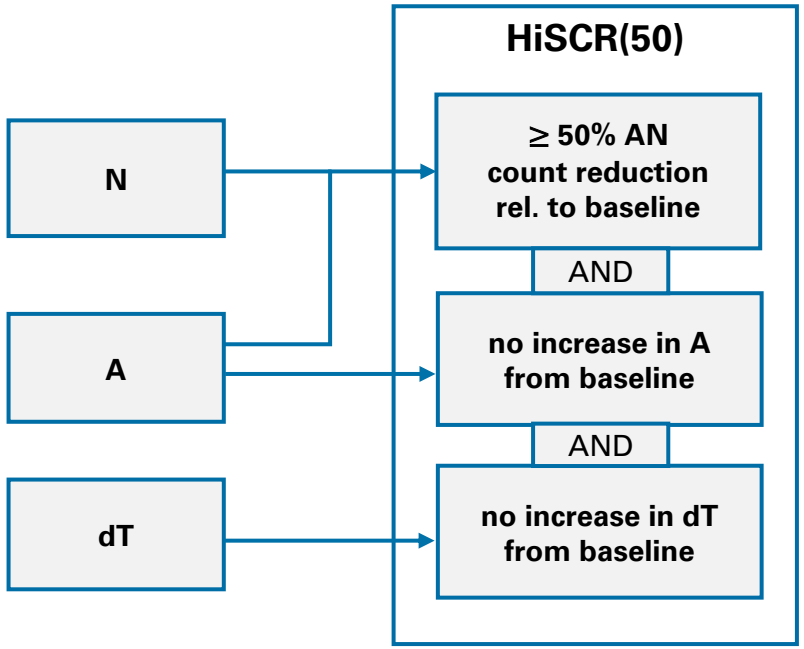
Izicopan Phase 2a in HS: Baseline characteristics

	n	AN-count mean	dT-count mean	ANdT-count mean	IHS4-score mean	Pain Score mean	DLQI mean	Hurley III (%)	mean HS diagnosis (years)	mean age (years)	% pts with ≥ 1 dT	prior biologic use	mean BMI	% female
60 mg bid	10	12.5	4.2	16.7	33.7	7.1	12.4	50%	14.6	42.2	70%	20%	36.7	60%
90 mg bid	11	14.7	4.5	19.3	36.3	6.6	14.2	36%	11.4	40.5	73%	18%	27.8	36%
120 mg bid	8	18.3	3.3	21.5	38.0	5.9	14.1	13%	8.1	35.9	75%	13%	28.9	38%
All	29	14.9	4.1	19.0	35.9	6.6	13.6	35%	11.5	39.8	72%	17%	31.2	45%

Baseline characteristics are **overall balanced and in line** with recent HS data sets

Hidradenitis suppurativa and HiSCR

- There are 3 inflammatory lesions: **inflammatory nodules (N), abscesses (A) and draining tunnels (dT)**
  - ANdT = total inflammatory burden (TIB)
- A and N are present in all disease stages, **can fluctuate strongly week to week which can drive placebo responses**
- dT are present in more progressed disease stages, **fluctuate less and show less placebo responses**



HiSCR = **Hi**dradenitis **S**uppurativa **C**linical **R**esponse

- has **high variability** due to AN fluctuation
- **dominated by the 50% AN count reduction criterion** and **does not account for reduction of dT**

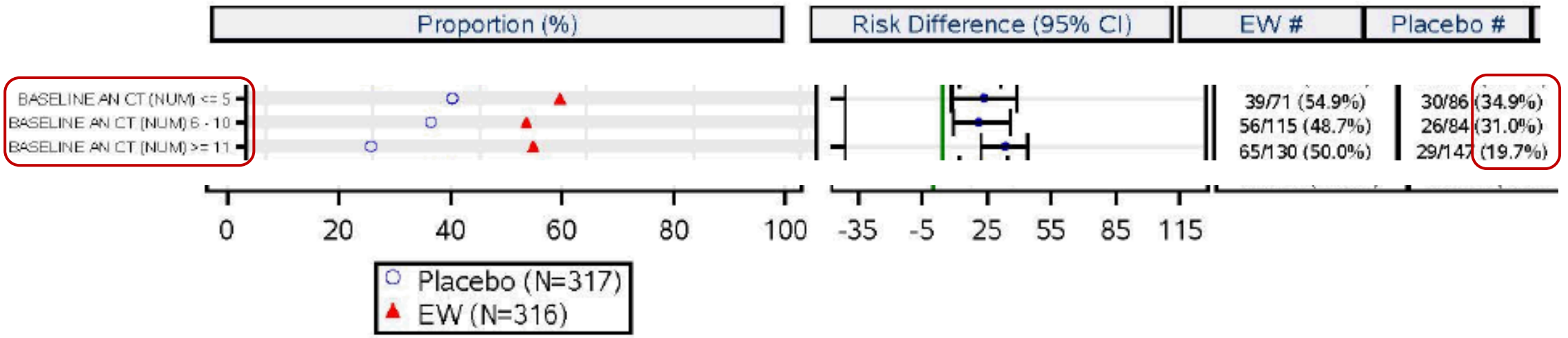
Note
response rates can depend on baseline AN count distribution: **placebo patients with low baseline AN have been reported to reach 50% reduction to a higher extent** than those with higher AN counts*

* EMA Assessment Report: Humira - No. EMEA/H/C/000481/II/0137 – page 82

Baseline AN count impact on HiSCR placebo response rates

Learnings from the PIONEER studies researching Humira in HS

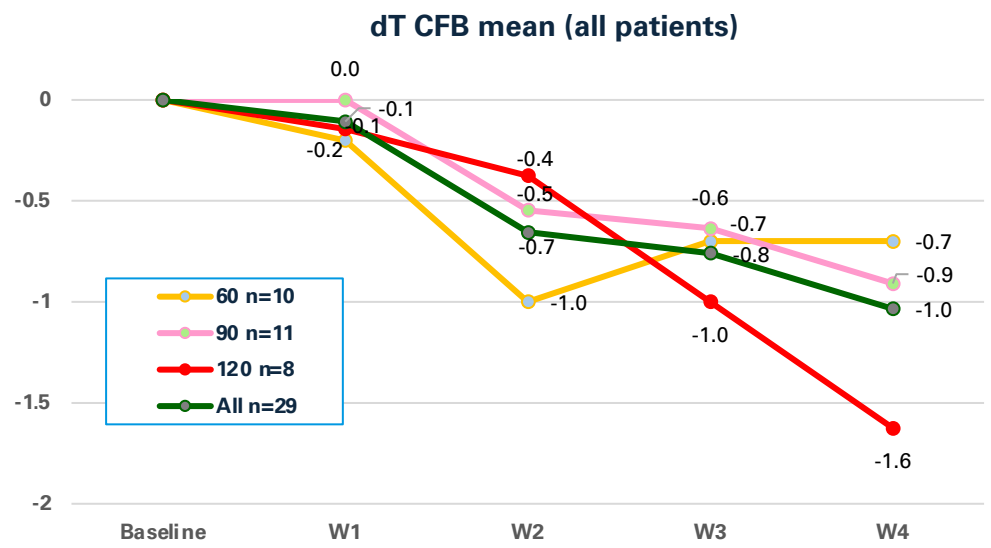
Proportion of subjects achieving HiSCR at week 12 (NRI) by subgroup (ITT_A population, integrated analyses)

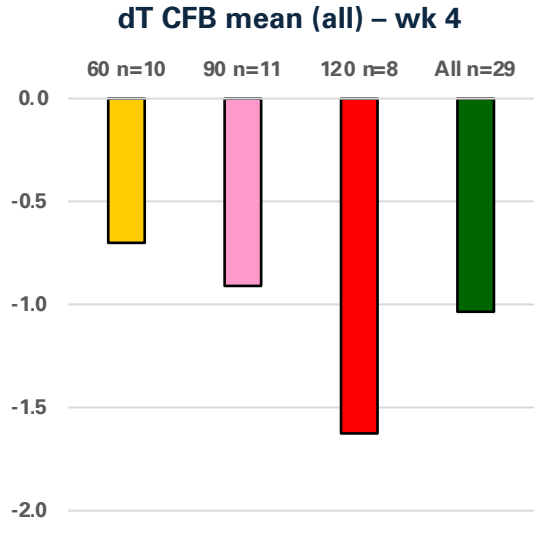


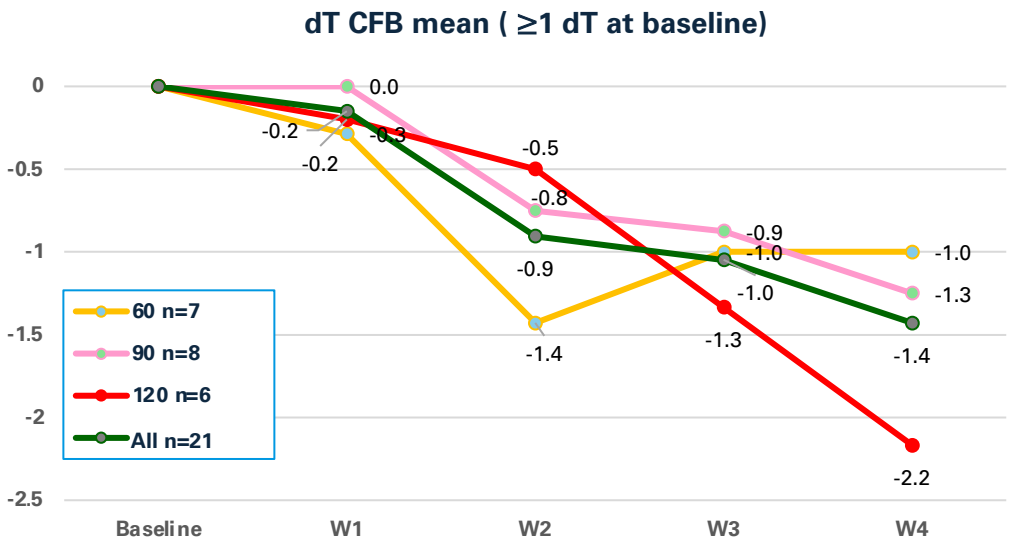
extracted from EMA Assessment Report: Humira - No. EMEA/H/C/000481/II/0137 – page 82

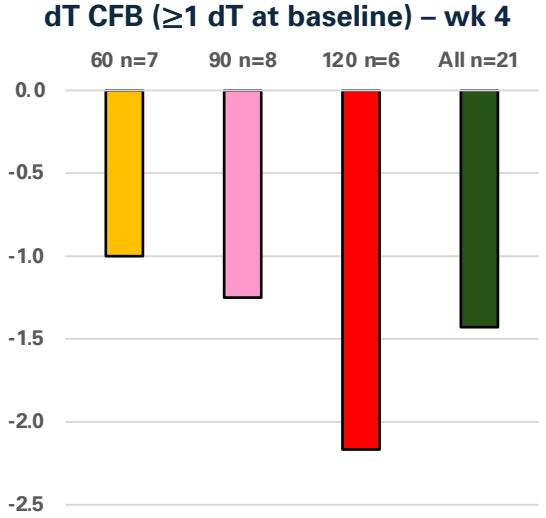
- Changing the cutoff of baseline AN count to ≥ 11 in the pooled PIONEER data set was reported to result in a reduction of the placebo response rate by absolute 15.2%
- Comparing baseline means between placebo and treatment groups does not inform about the baseline distribution of patients with low counts versus those with high counts.
- The currently used AN count cutoff for enrollment in clinical trials is AN ≥ 5

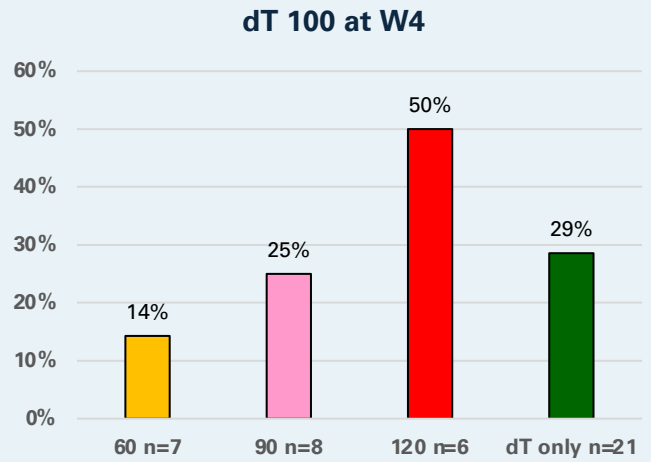
Izicopan drives draining tunnel (dT) count reductions





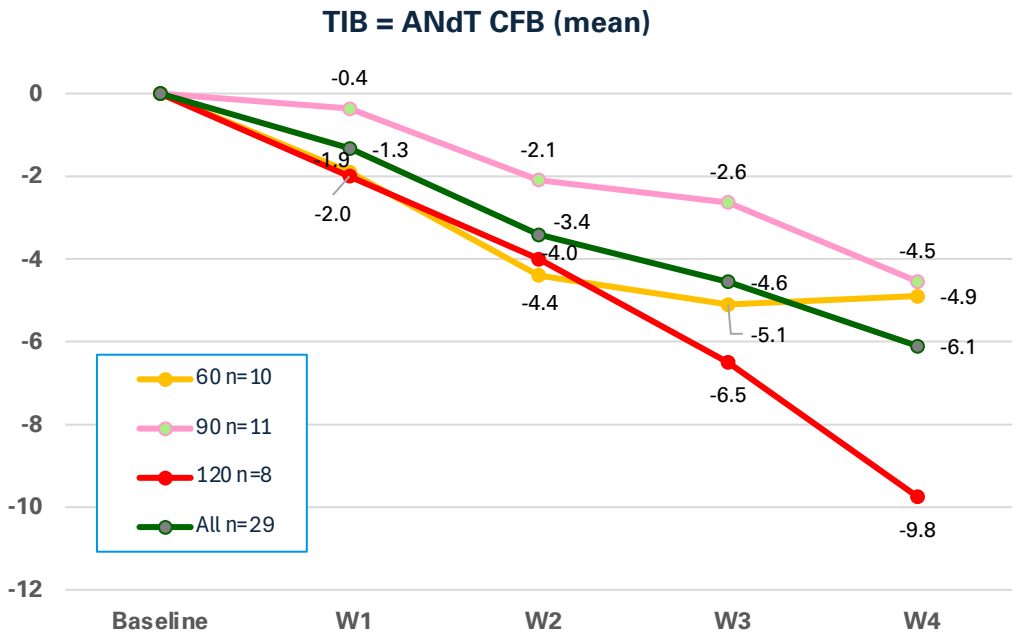




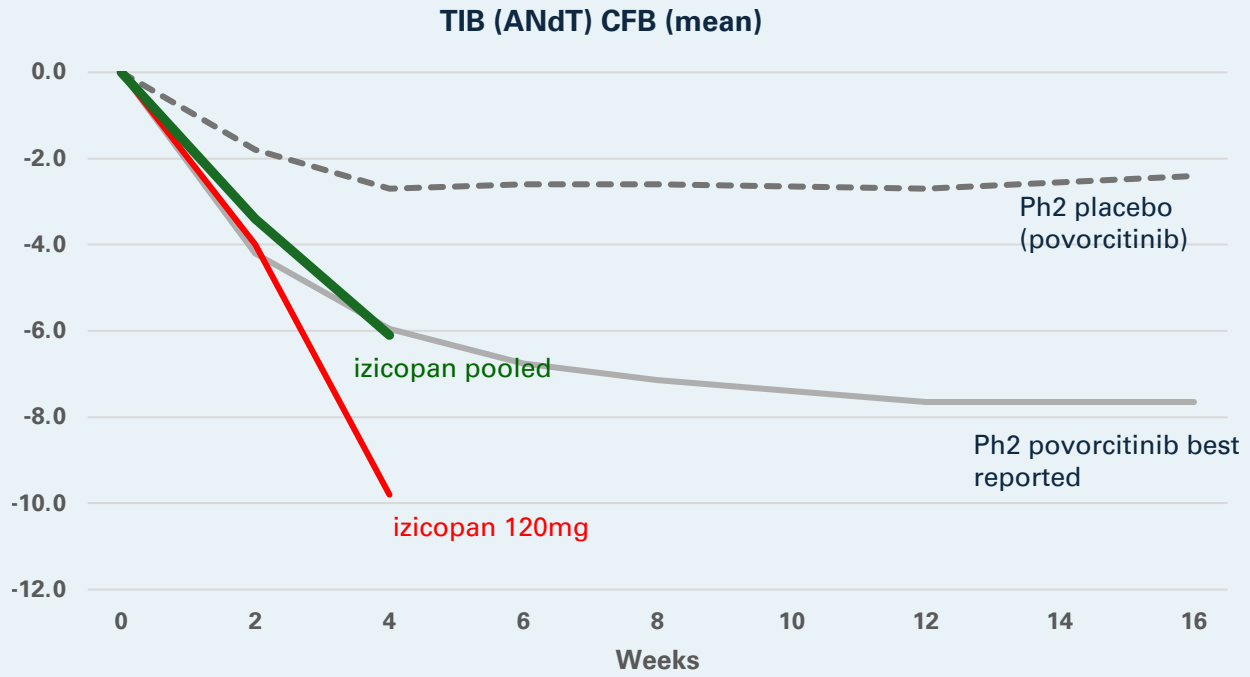


Izicopan drives reduction in total inflammatory burden (TIB)

TIB = ANdT count

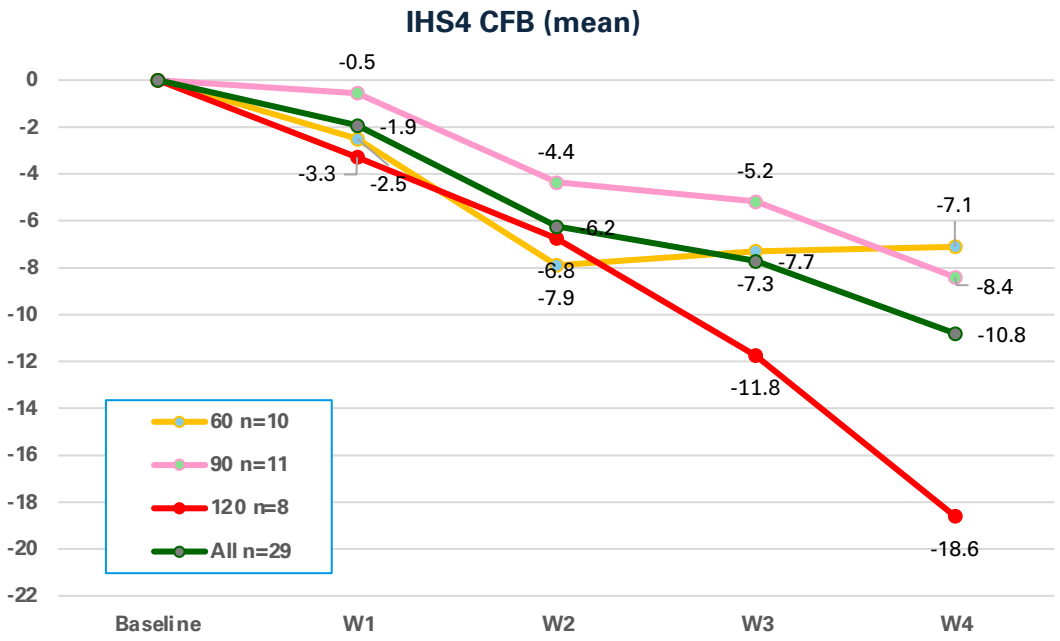


Comparison to average of reported successfully completed povorcitinib Phase 2 data*

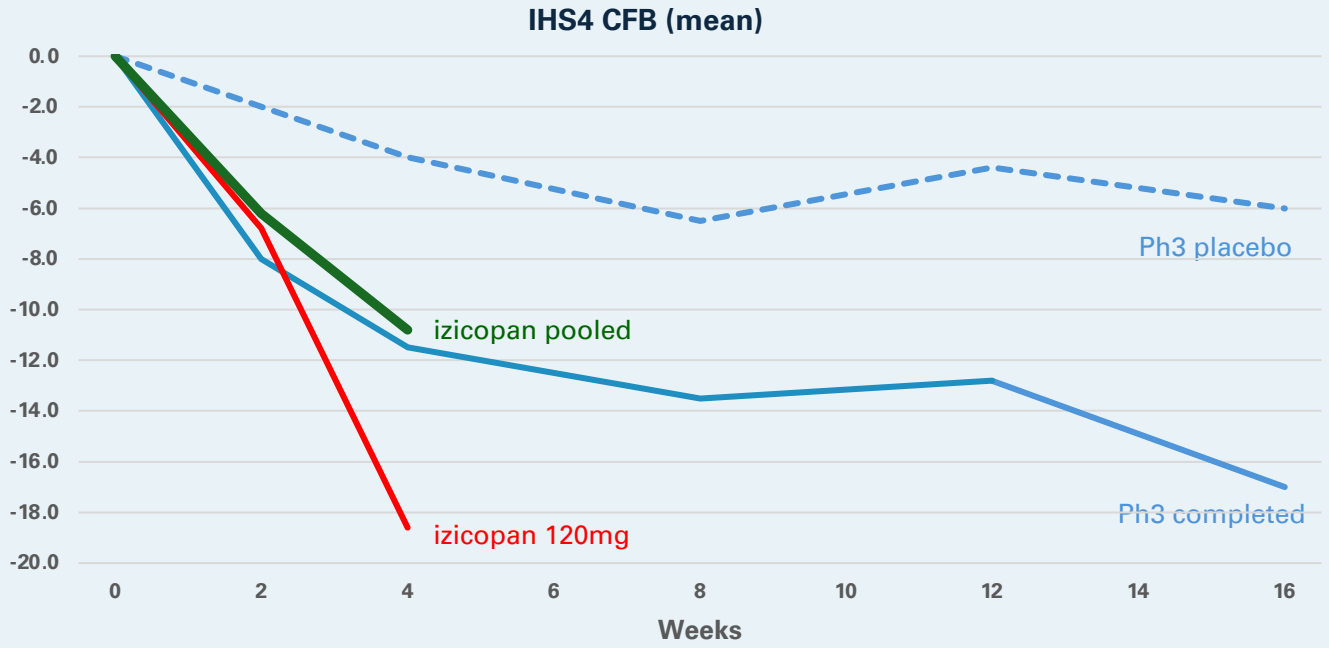


* Data from *Povorcitinib Phase 2* trial for week 2, 4, 6, 8, 12, and 16 as reported on Clinicaltrials.gov (NCT04476043). Average of data from the two best reported dosing regimens (45mg QD and 75 mg QD), also used in Phase 3, at each reported timepoint are reflected in line "Ph2 povo best reported". Respective placebo data at each timepoint are reflected in line "Ph2 placebo (povo)".

Izicopan drives reductions in IHS4 score



Comparison to average of available reported completed Phase 3 data*

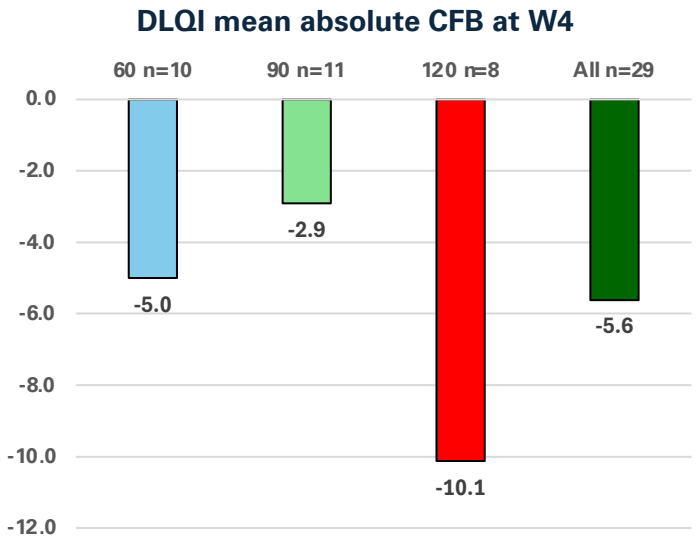


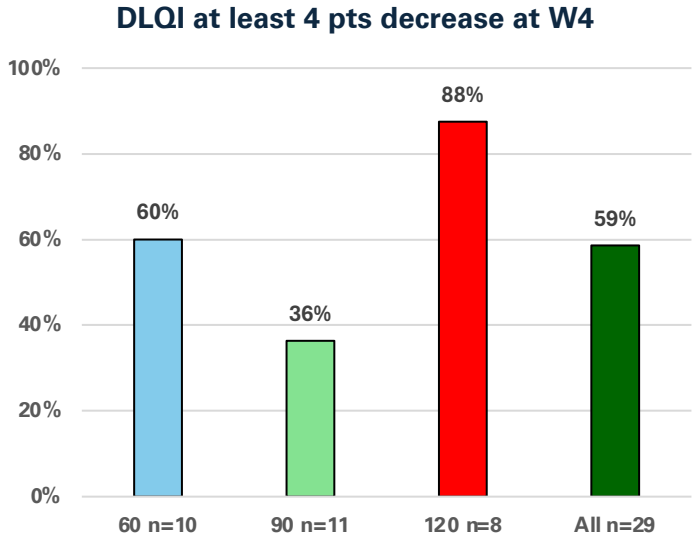
* Data are derived from different completed Phase 3 clinical trials and averages were created using the data available at each timepoint:

Adalimumab PIONEER I & II: week 12 data from tested dose groups and placebo as reported in post hoc analysis by Frew et al., J Am Acad Dermatol. 2020; 82(5):1150-1157;

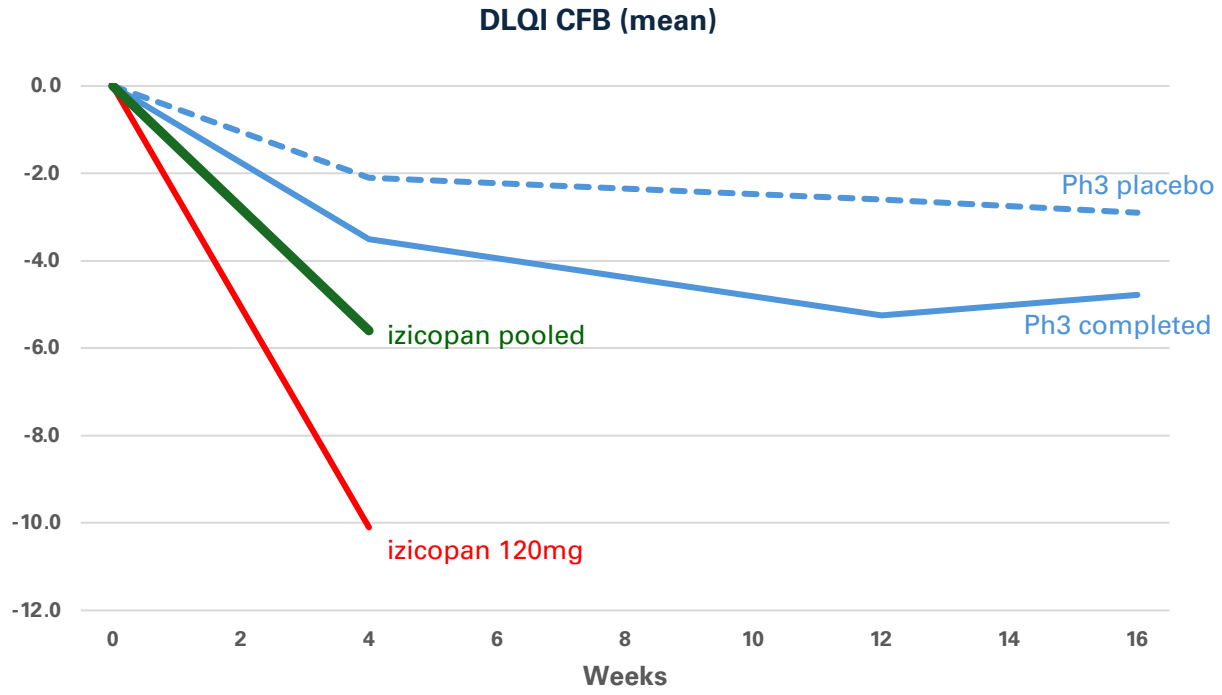
Bimekizumab BE HEARD I & II: week 2, 4, 8, 12, 16 data from all tested doses and placebo reported as pooled data from both trials by Zouboulis et al., 2025 EHSF poster T3-P-07; mean CFB data for week 2, 4, 8, 12 are approx. numbers from published graph.

Izicopan drives Dermatology Life Quality Index (DLQI) improvements





Comparison to average of available reported completed Phase 3 data*

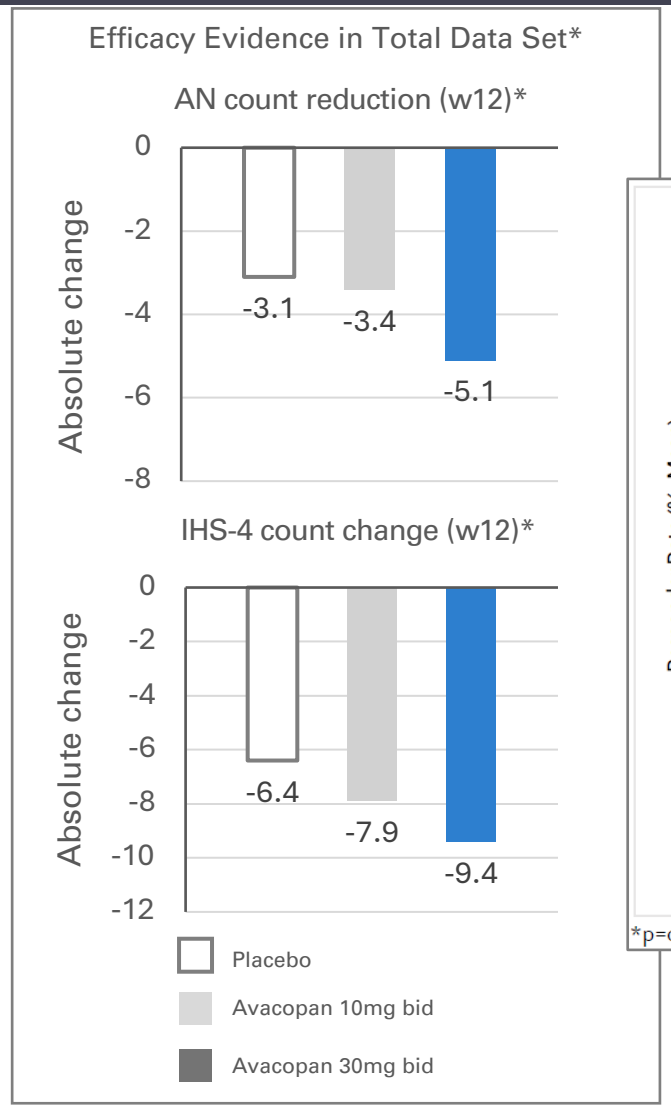


* Data are derived from different completed Phase 3 clinical trials and averages were created using the data available at each timepoint:

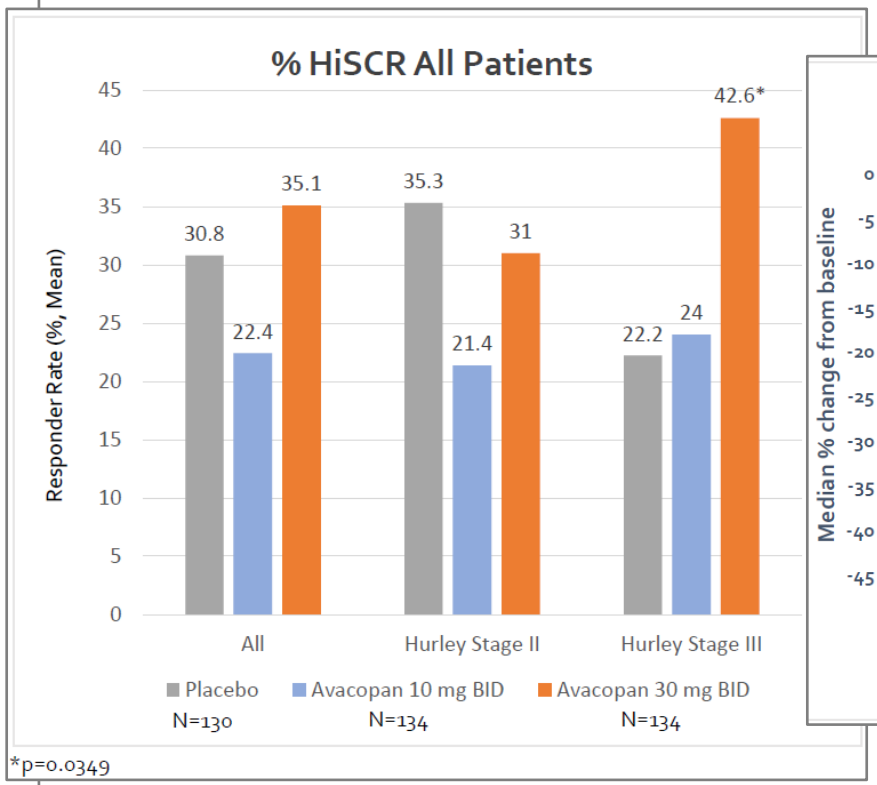
Adalimumab PIONEER I & II : week 12 data from tested dose groups and placebo as reported by Kimball et al., N Engl J Med 2016; 375:422-34.

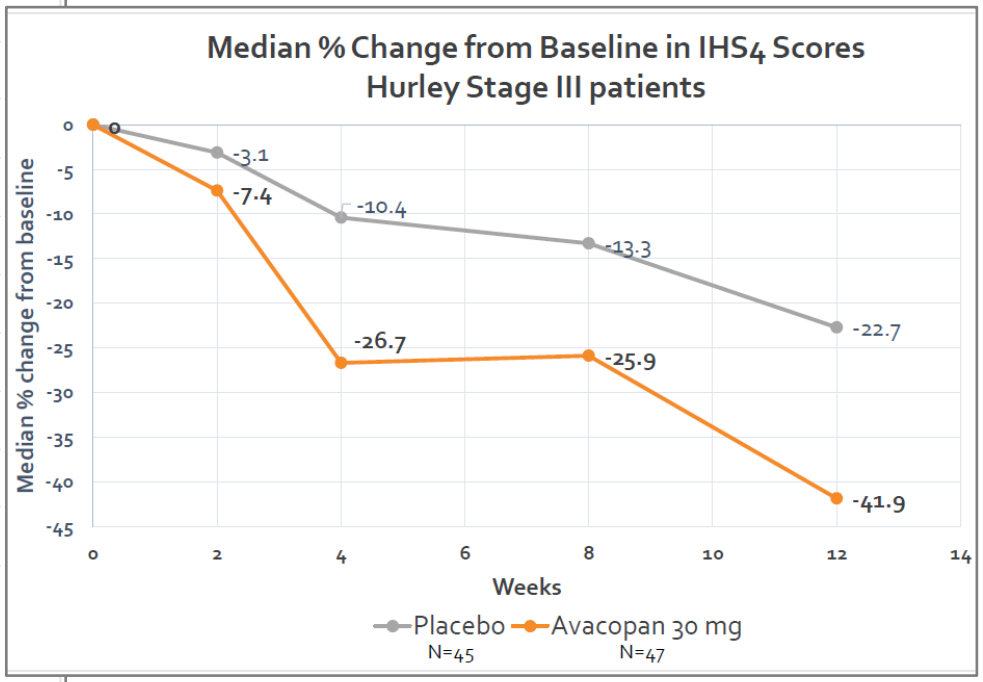
Bimekizumab BE HEARD I & II: week 12 data for all tested dose groups and placebo reported by Kimball at al., Lancet 2024; 403(10443):2504-2519. Week 4 CFB data are approximated using absolute differences in means reported for week 4 and baseline by Shi et al., 2025; 15(9):2553-2570.

Evidence for efficacy of C5aR inhibition in HS: Avacopan data



Primary outcome**

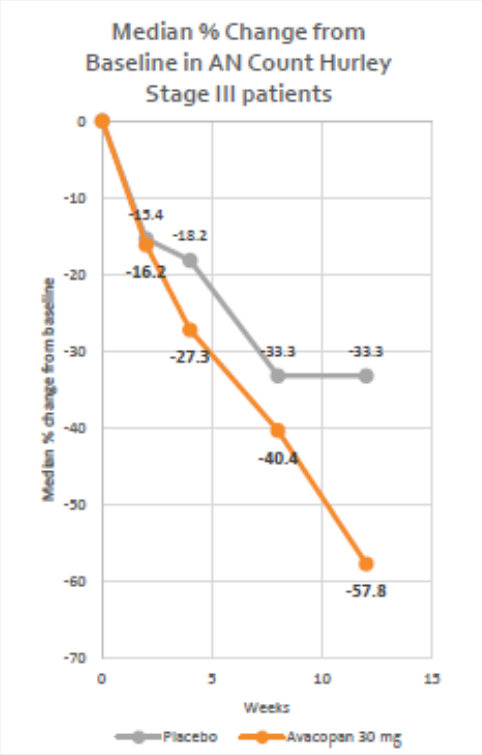


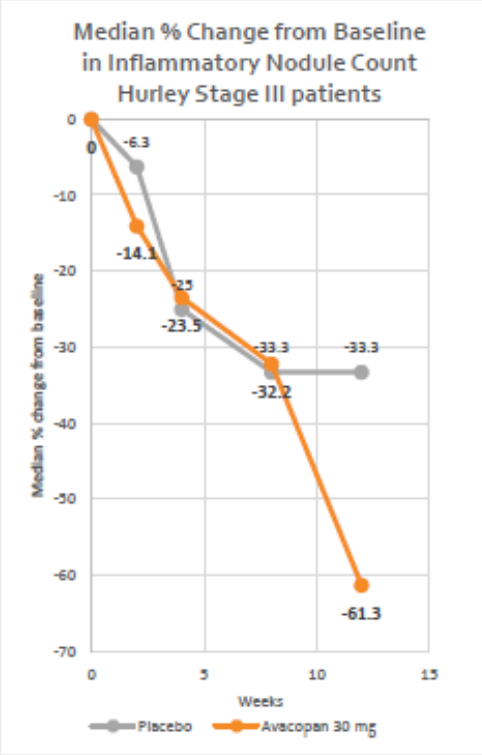


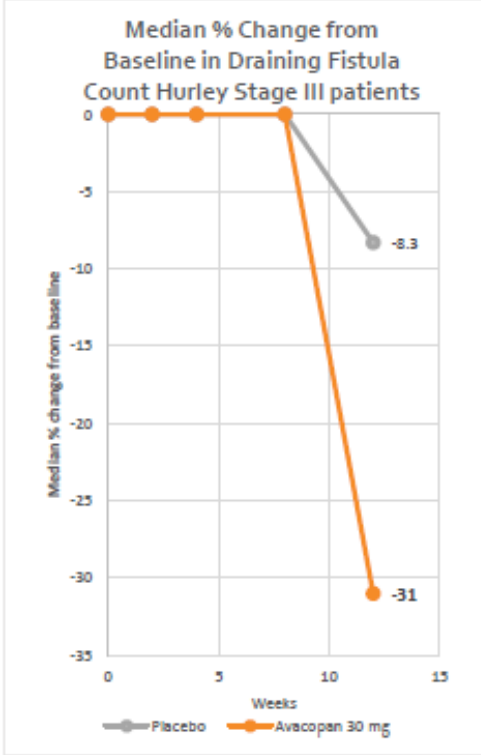
* Reported outcome results at clinicaltrials.gov, Chemocentryx, Avacopan HS Phase 2 trial (AURORA)
** Data from Chemocentryx presentation on Avacopan HS Phase 2 trial (AURORA) results, October 28, 2020: note: overall results were not stat. significant for HiSCR in all moderate to severe HS patients (primary endpoint)

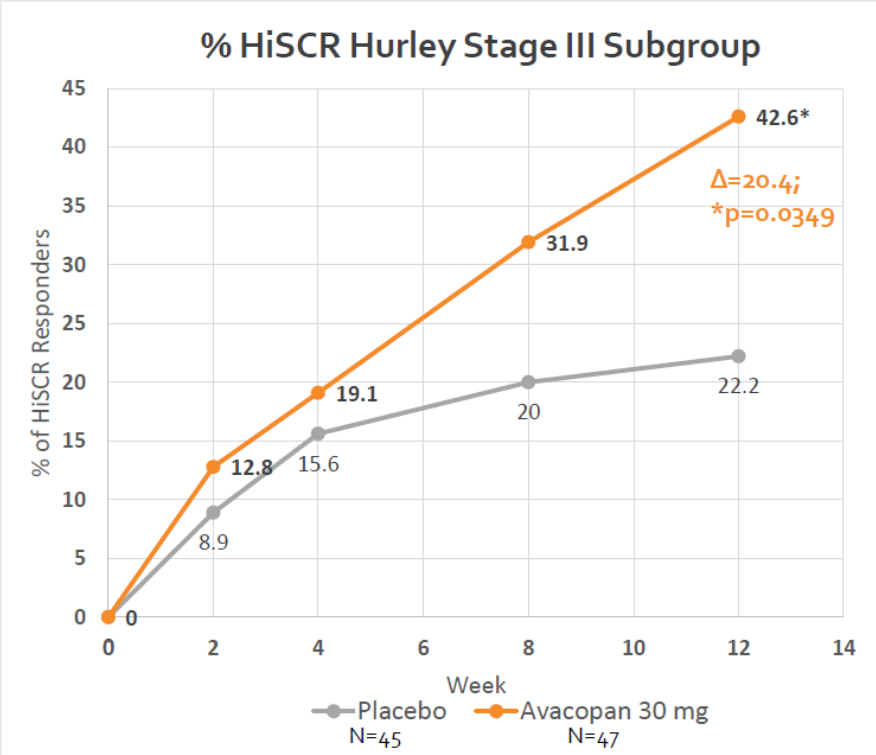
Avacopan data in Phase 2 showed efficacy emerging only at week 12

Results From Avacopan in HS Patients (Hurley III)*





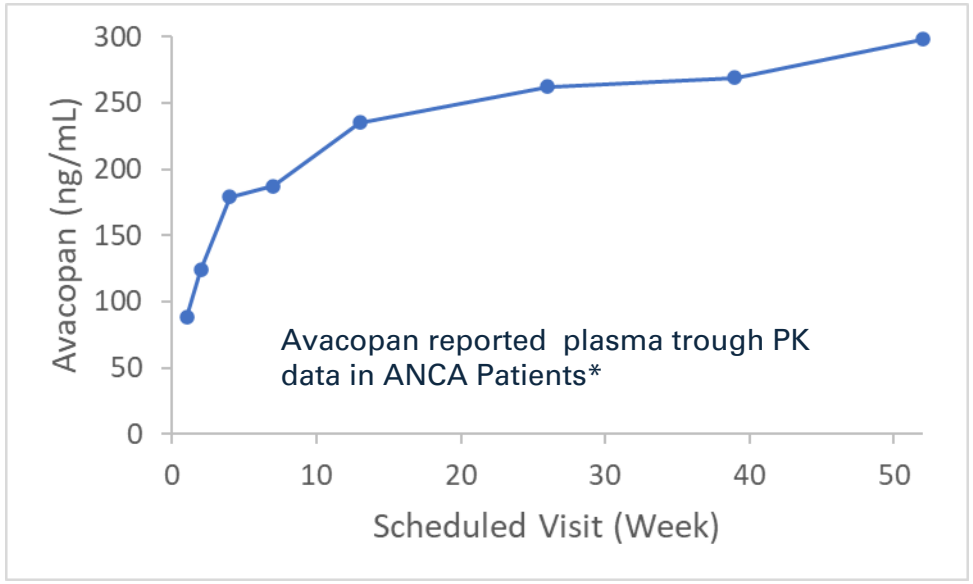




- Avacopan's efficacy (separation from placebo group) in HS only starts to emerge at W12 – please note: steady state reported from ANCA patients was only reached at approx. 3 months due to prolonged drug accumulation (x4)
- Avacopan's 30mg BID dosing regimen may be too low to show adequate clinical efficacy in HS

*Data from ChemoCentryx presentation on AURORA trial results, October 28, 2020: note: overall results were not stat. significant for HiSCR in all moderate to severe HS patients (primary endpoint)

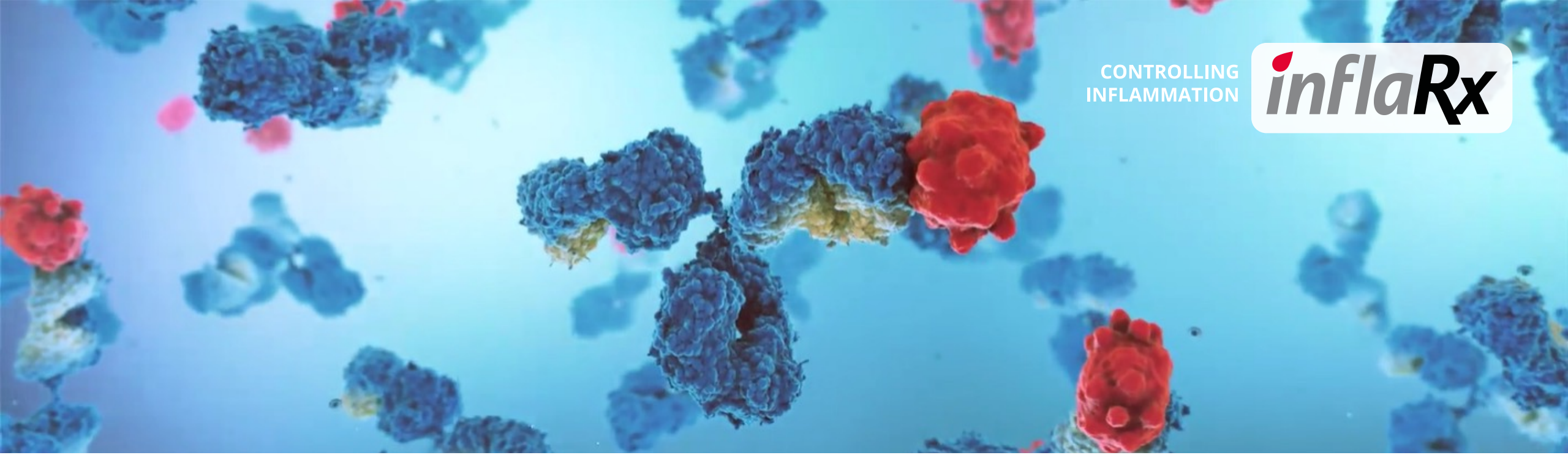
Avacopan data in ANCA patients show steady state reached by 13 weeks



*Data from avacopan NDA filing for ANCA-associated vasculitis: represented graphically.

- Steady state plasma levels of avacopan 30mg BID are reached by 13 weeks and the accumulation is approximately 4-fold
- Mean steady state plasma exposure estimates of avacopan are: 3466 h*ng/mL for the ($AUC_{0\text{-}12hr}$) in ANCA patients receiving 30 mg BID

Plasma accumulation may be a prerequisite for reaching blocking activity of C5aR1 on neutrophils, to sufficiently prevent activation and migration into tissue in order to show clinical efficacy



CSU Appendix

CSU endotypes: Type I auto-allergens and type IIb autoimmunity

C5aR signaling is suggested to be involved in both Type I and Type IIb endotypes

1 Type I autoimmune (IgE mediated)

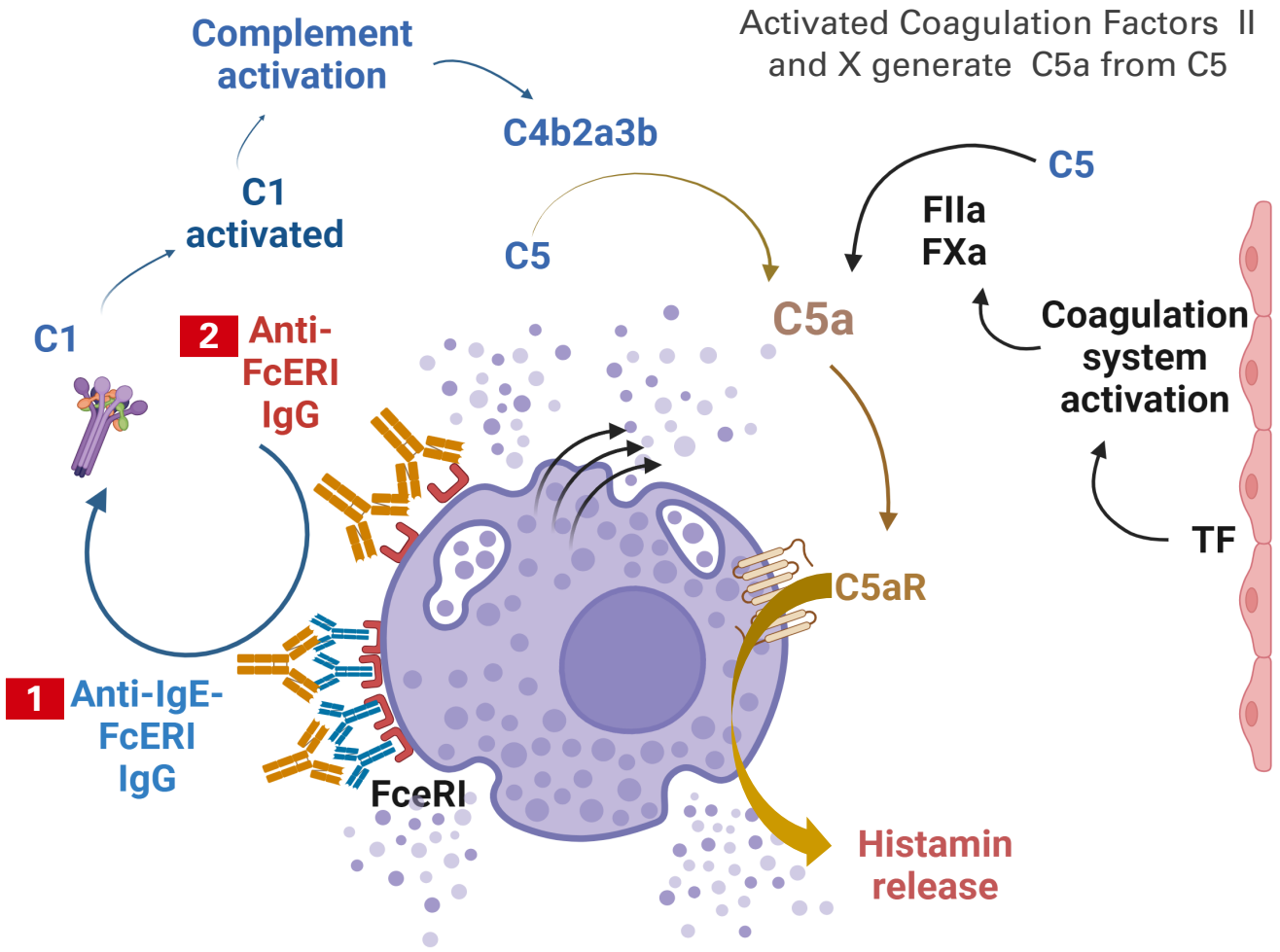
Also known as autoallergic CSU, is characterized by the presence of IgE antibodies against self-antigens.

The binding of the self-antigens to specific IgE leads to FcεRI cross-linking and mast cell degranulation via a type I hypersensitivity mechanism

2 Type IIb autoimmune (IgG mediated)

This is driven by IgG antibodies against either IgE or the high-affinity IgE receptor (FcεRI). Cross-linking of FcεRI results in activation of downstream kinases, which induces mast cell degranulation.

Activation of the coagulation and complement pathways can also trigger mast cell activation; with C5a being able to bind to C5aR on the surface of mast cells.

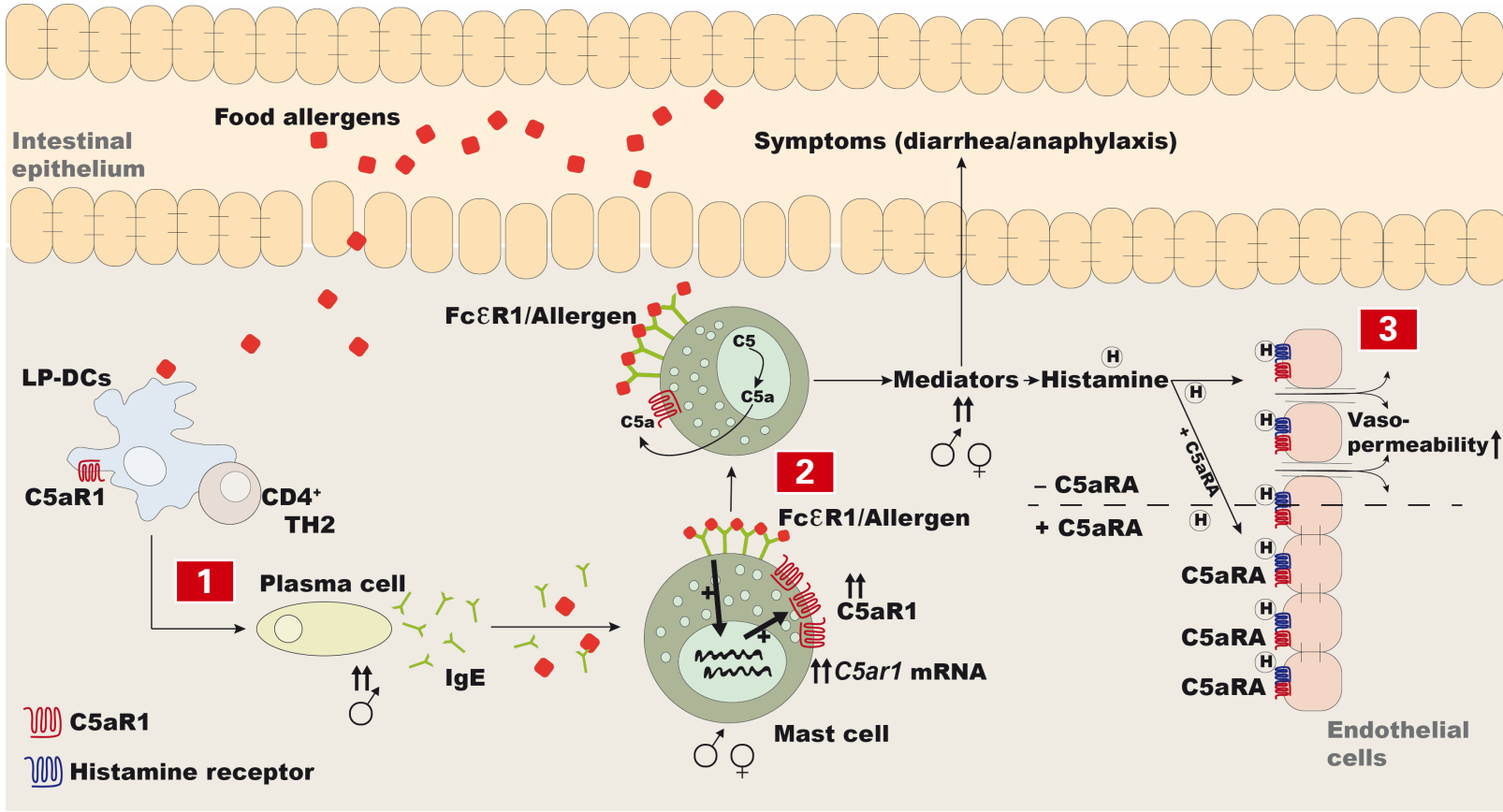


Modified from Kaplan AP et al Clin Exp Allergy 2009 and Yanase Y, J Allergy Clin Immunol 2021

C5aR1-mediated induction of anaphylaxis via mast cell activation

Activation of the C5a/C5aR1 axis drives the development of anaphylaxis at several levels

1. The regulation of the B cell response in male mice that leads to the production of antigen-specific IgE.
2. The enhancement of FcεR1-dependent degranulation of MCs.
3. The sensitization of the vascular system towards the MC mediator histamine.



Kordowski et al. Allergy 2019

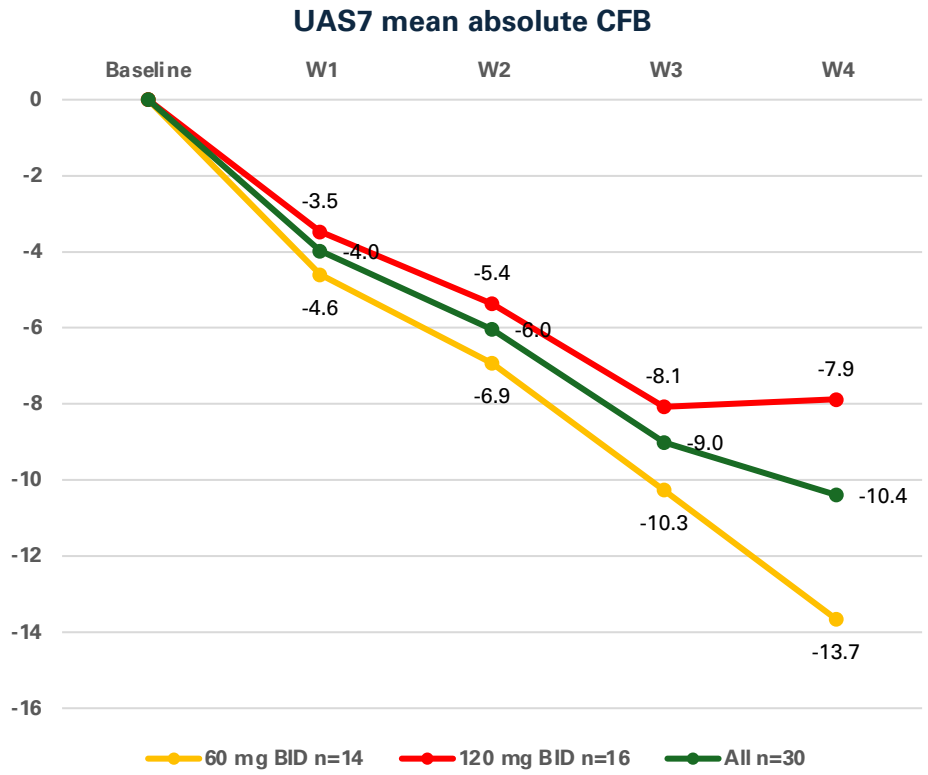
CSU: Baseline characteristics

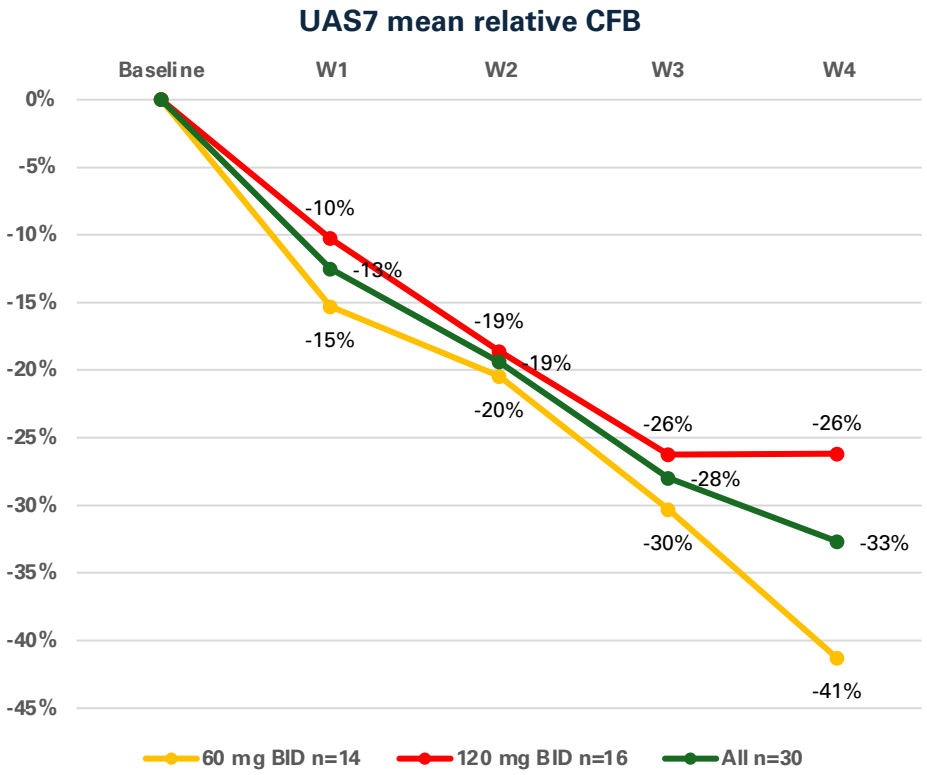
	mean age (years)	% female	mean CSU duration (years)	mean UAS7	mean UCT7	IgE (IU/L)	anti IgE experienced
60 mg BID n=14	41.3	86%	3.6	33.5	5.0	283.4	7%
120 mg BID n=16	47.8	81%	3.3	29.5	4.3	178.0	19%
All n=30	44.8	83%	3.5	31.4	4.6	230.7	13%
Anti IgE Naive n=25	43.7	80%	2.9	31.0	4.6	244.9	0%
Anti IgE Experienced n=5	50.2	100%	5.8	33.4	5.0	112.4	100%

Definitions

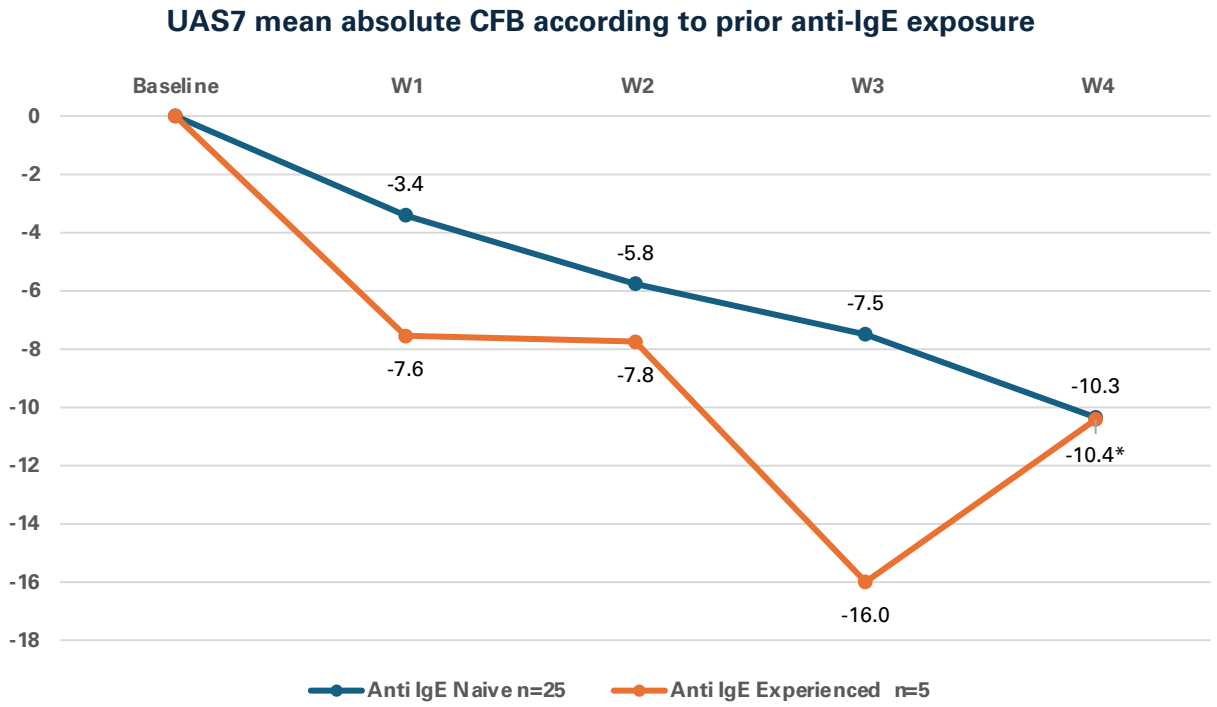
- UAS7: 28-42 (severe disease), 16-27 (moderate), 7-17 (mild)
- UCT7: 16 (complete disease control), 12-15 (well controlled), <12 (poorly controlled)

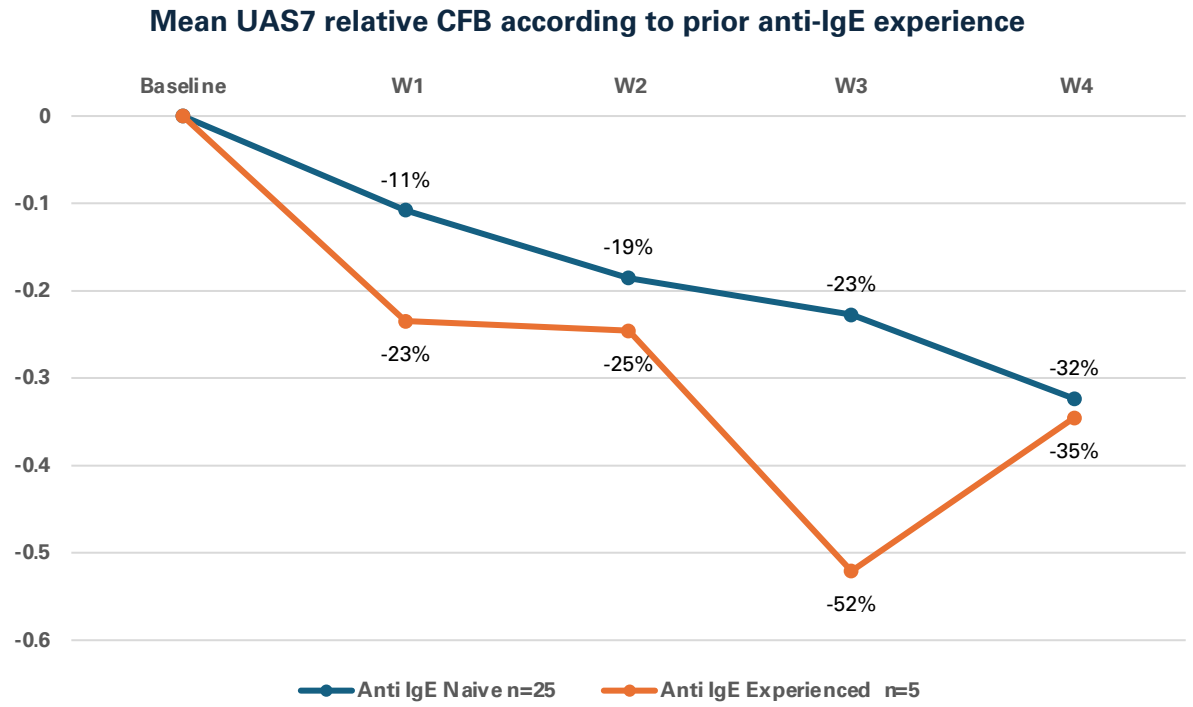
CSU: Izicopan achieved UAS7 reduction at W4





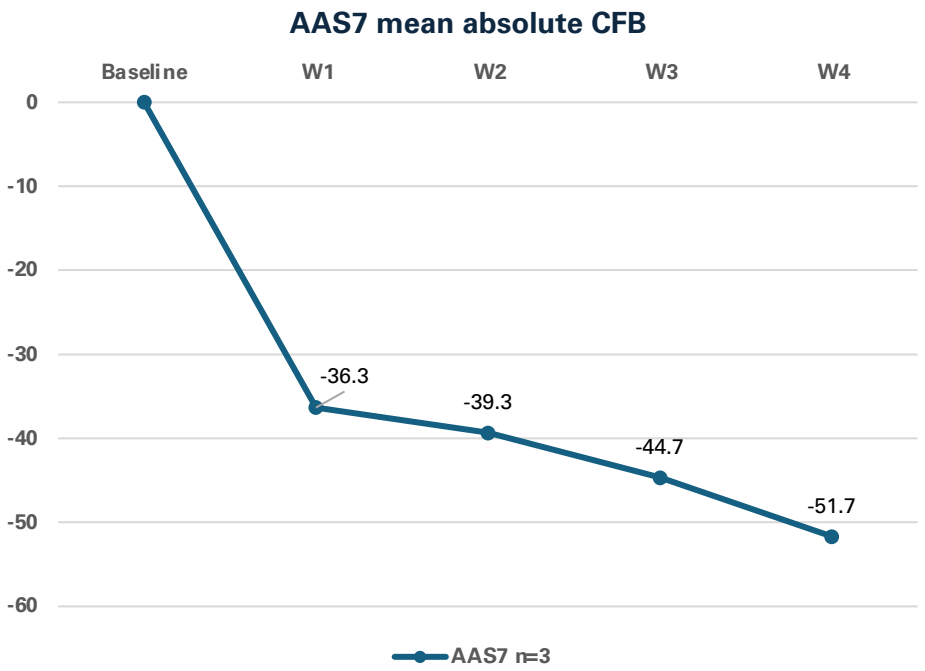
CSU: Izicopan reduced UAS7 in anti-IgE naïve and experienced patients

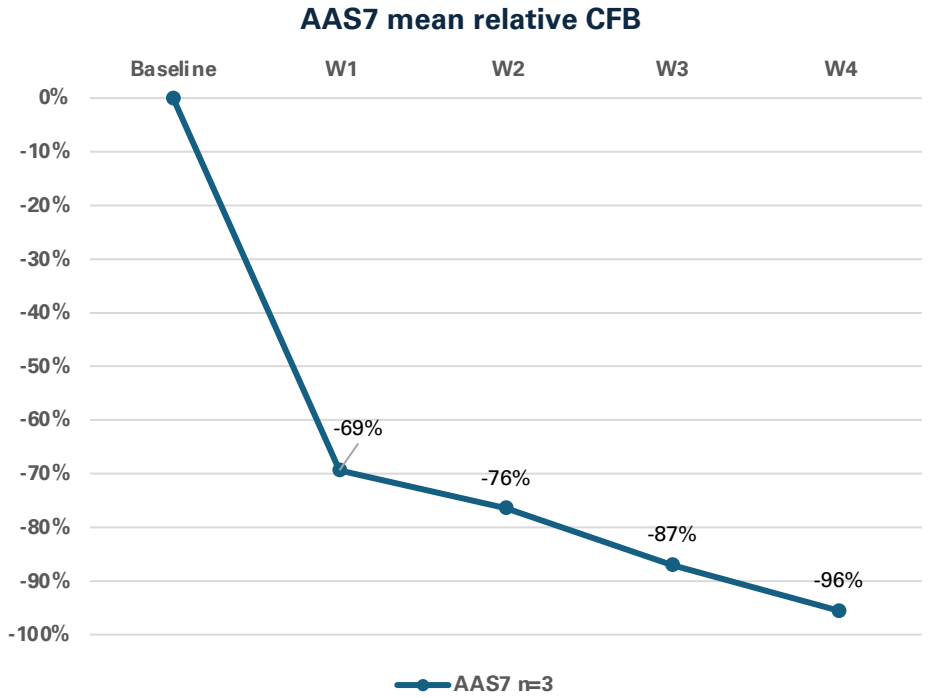




** 1 anti-IgE experienced patient had history of unexplained urticaria exacerbations from (stress and mental health shifts) which resulted in a dramatic increase in UAS7 at W4 but this was subsequently reduced after end of treatment with UAS7 reduction of -15.4 at EoS.*

CSU: Izicopan improves angioedema consistently up to W4



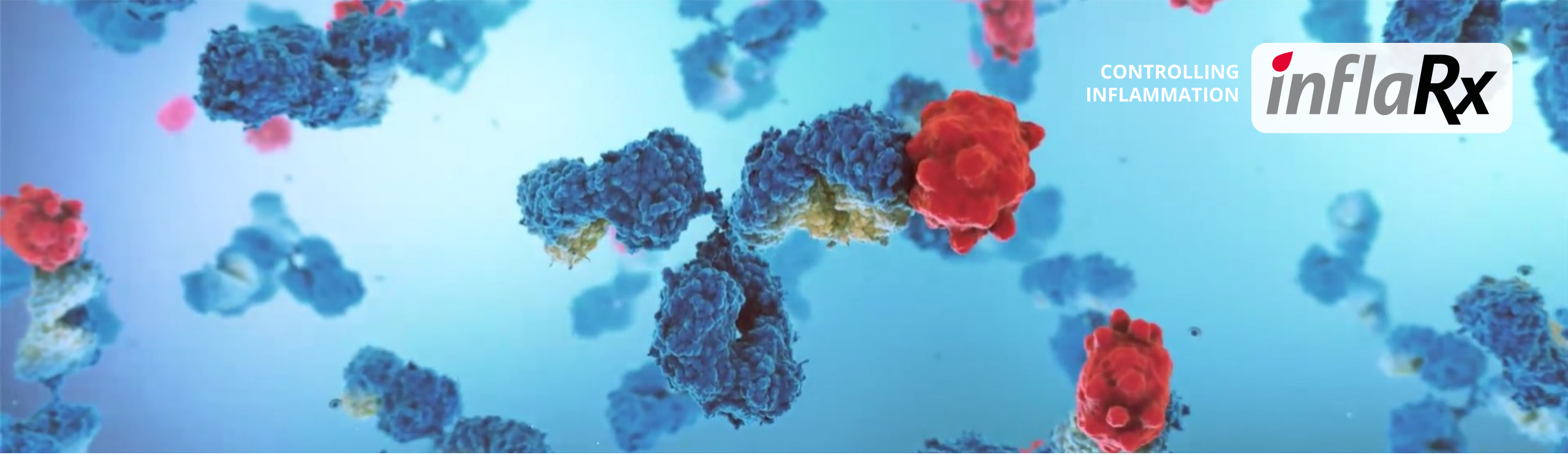


No episodes: 0
Low: 1 to 6
Moderate: 7 to 18
Severe: 19 to 105

CSU: Izicopan UAS7 and UCT7 at Week 4

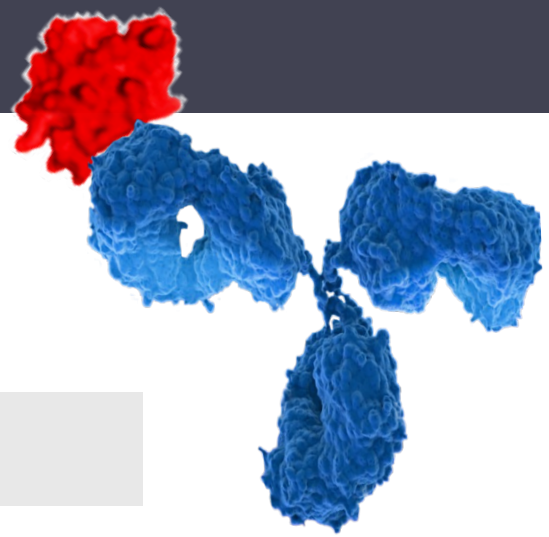
	UAS7 mean absolute CFB W4	responders UAS7 = 0 at W4	UAS7 ≤ 6 at W4	UCT7 ≥ 12 at W4
All n=30	-10.4	7%	11%	29%
60 mg BID n=14	-13.7	0%	8%	31%
120 mg BID n=16	-7.9	13%	13%	25%

UAS7 [responders of disease activity]. **UCT7** [responders of disease control].



Gohibic Appendix

Emergency Use Authorization (EUA) granted for Gohibic



Gohibic® (vilobelimab) Injection 200 mg/20 mL

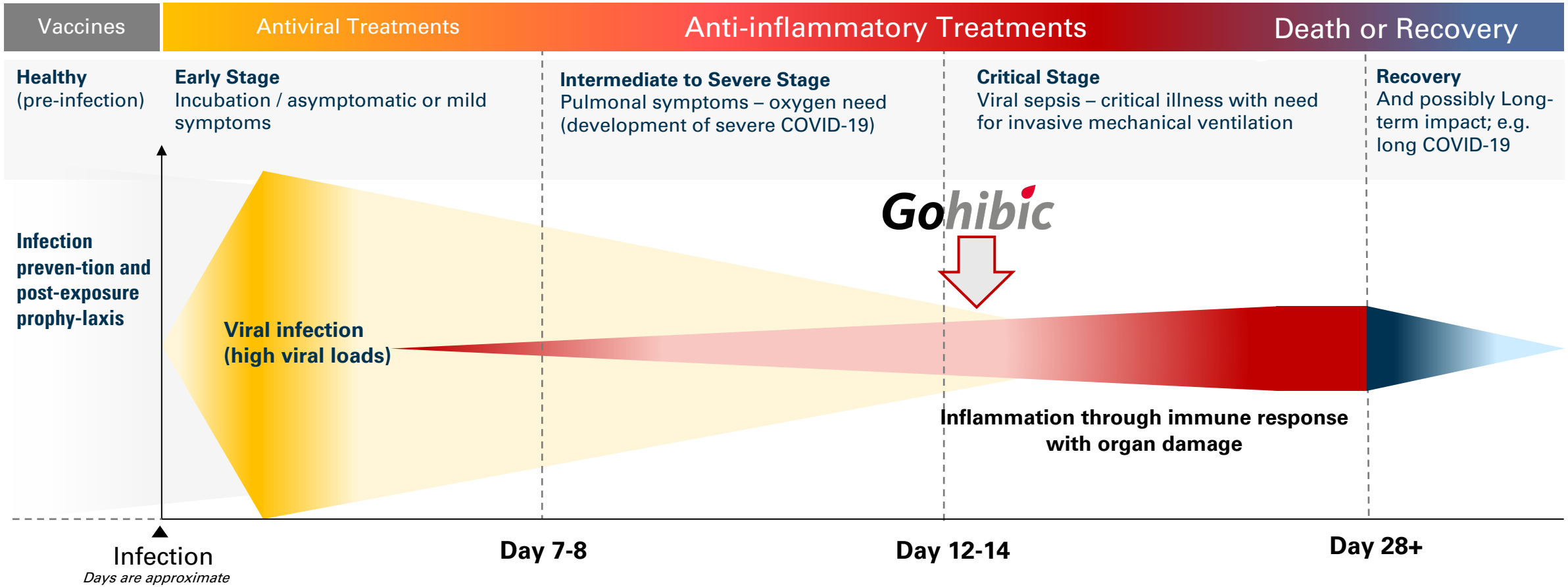
- Gohibic (vilobelimab) has not been approved, but has been authorized for emergency use by FDA under an EUA*, for the treatment of COVID-19 in hospitalized adults when initiated within 48 hours of receiving IMV†, or ECMO‡
- Authorization granted based on results from a Phase III clinical trial in critically ill, mechanically ventilated COVID-19 patients in which Gohibic treatment reduced mortality by 23.9% vs. placebo.
- Gohibic is the first authorized therapeutic targeting C5a as potential key player in the inflammatory host response
- Discussions with US FDA ongoing related to future BLA submission
- Phase 2 "Just Breathe" ASPR/BARDA clinical platform study for broader ARDS
- Approved by the European Commission for the treatment of adult patients with SARS-CoV-2-induced acute respiratory distress syndrome (ARDS) who are receiving systemic corticosteroids as part of standard of care and receiving IMV with or without ECMO
  - GOHIBIC is the first and only treatment approved in the European Union for the treatment of SARS-CoV-2-induced ARDS

*The emergency use of GOHIBIC is only authorized for the duration of the declaration that circumstances exist justifying the authorization of the emergency use of drugs and biological products during the COVID-19 pandemic under Section 564(b)(1) of the Act, 21 U.S.C. § 360bbb-3(b)(1), unless the declaration is terminated or authorization revoked sooner.

†IMV = invasive mechanical ventilation,
‡ECMO = extracorporeal membrane oxygenation

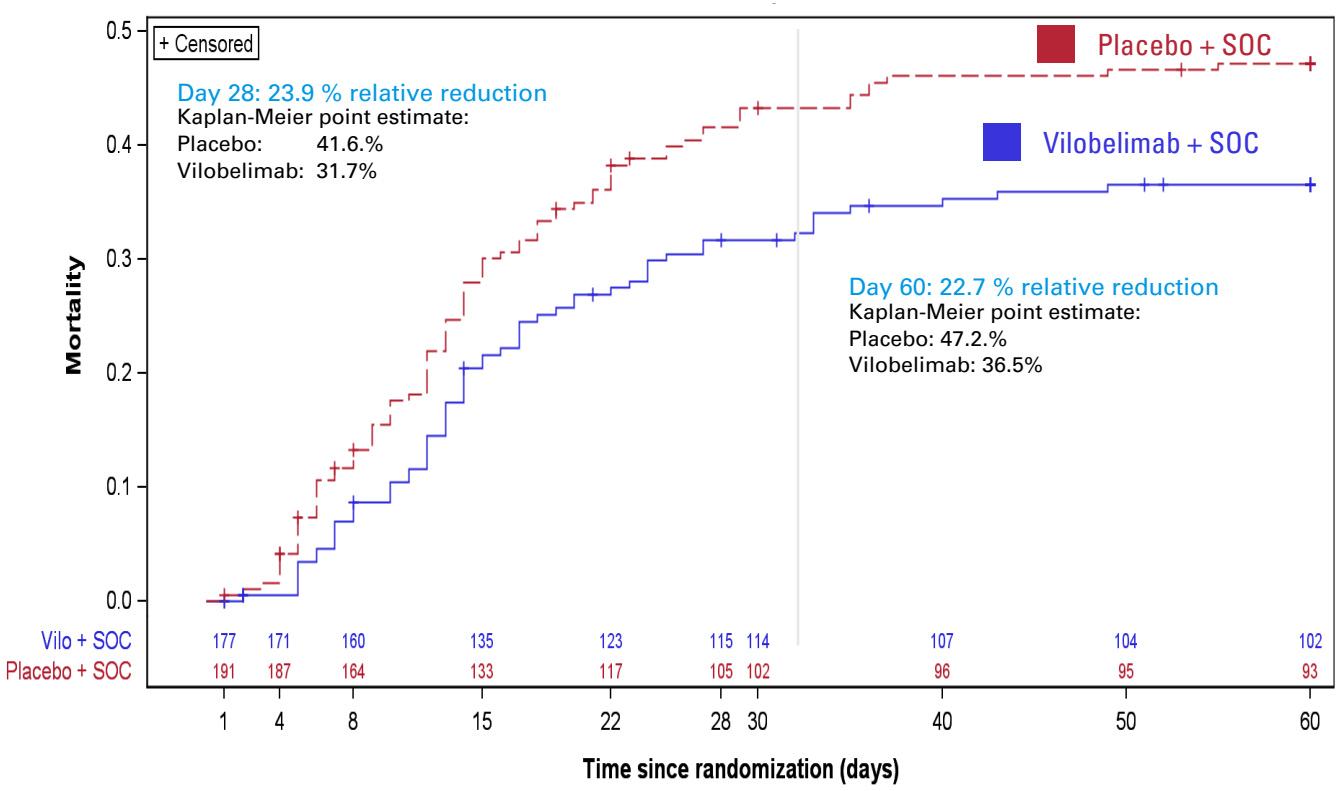
For additional and important information, please visit www.gohibic.com

COVID-19: Disease progression and therapeutic interventions



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

PANAMO Phase 3 primary endpoint: 28-day all-cause mortality



* Post-hoc analysis presented at ATS 2024

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

PANAMO Phase 3 trial

1:1 randomized, double-blind, placebo-controlled, multi-national trial in (n=369) invasive mechanically ventilated COVID-19 patients

SOC included concomitant use

- Corticosteroid use: 97%
- Anti-coagulant use: 98%
- Prior or concomitant use of other immunomodulators (**tocilizumab** > **baricitinib**): 20%

Tocilizumab / baricitinib subgroup analysis* (n = 71)

- 28-day all-cause mortality of **6.3%** for vilobelimab plus tocilizumab or baricitinib versus **40.9%** for placebo
  - Relative risk reduction of 84.6% (p=0.006)
- Co-administration of vilobelimab with baricitinib or tocilizumab not associated with safety concerns
- Demographics of subgroups were generally well-balanced and comparable to the overall study population

Number of patients needed to treat for saving one additional life = 9