



CORPORATE PRESENTATION

Oppenheimer 36th Annual Healthcare
Life Sciences Conference

February 25, 2026

CONTROLLING INFLAMMATION



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 izecopan 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 izecopan 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

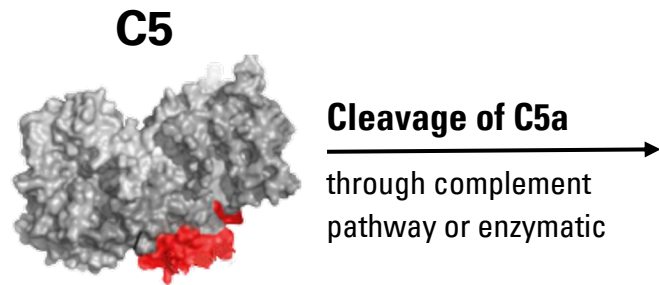
InflaRx (Nasdaq: IFRX) is a biopharmaceutical company pioneering anti-inflammatory therapeutics by applying its proprietary anti-C5a and anti-C5aR technologies to discover, develop and commercialize highly potent and specific inhibitors of the complement activation factor C5a and its receptor, C5aR. C5a is a powerful inflammatory mediator involved in the progression of a wide variety of inflammatory diseases. InflaRx's lead program is izecopan (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 izecopan 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

C5a/C5aR are validated targets promoting inflammation



Targeting strong pro-inflammatory mechanisms

vilobelimab
intravenous mAb

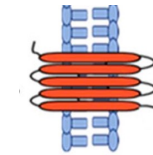
izicopan
oral small molecule

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

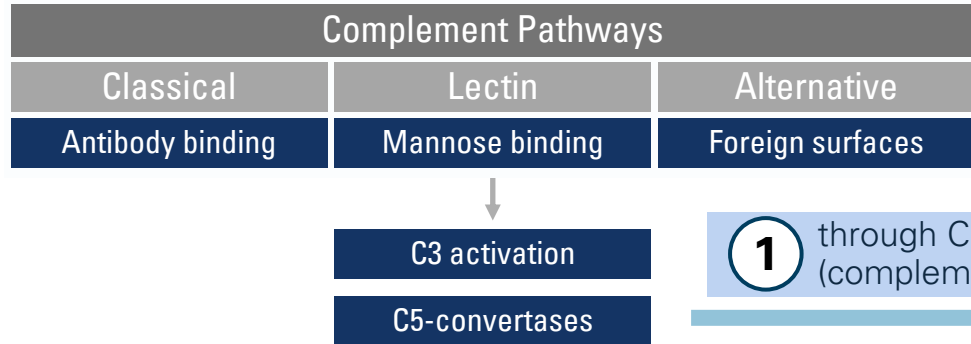
Strong activator of neutrophils and macrophages

- Chemotaxis of neutrophils / macrophages / monocytes into tissue
- O₂ radical generation + granular enzyme release from neutrophils
- NETosis induction (neutrophil extracellular traps)

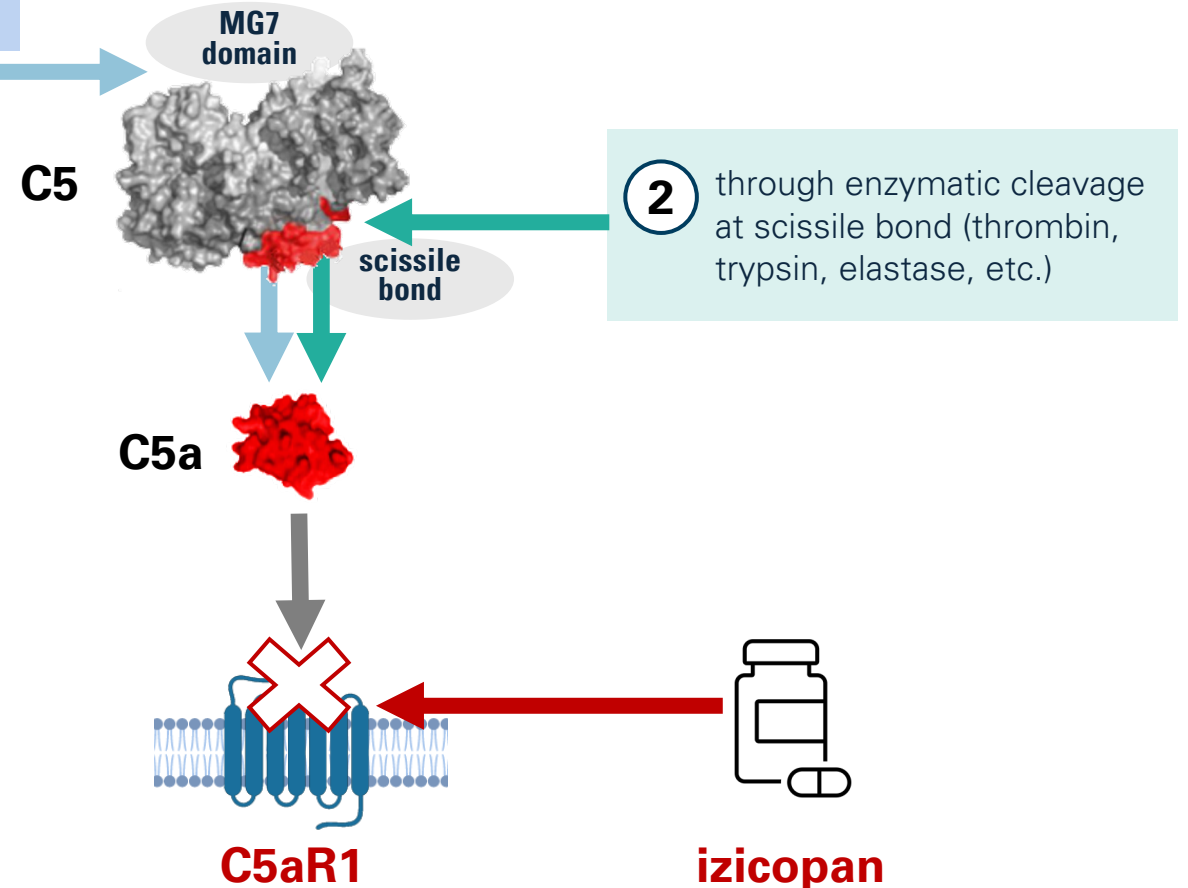
Essential role in many inflammatory conditions

- 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



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

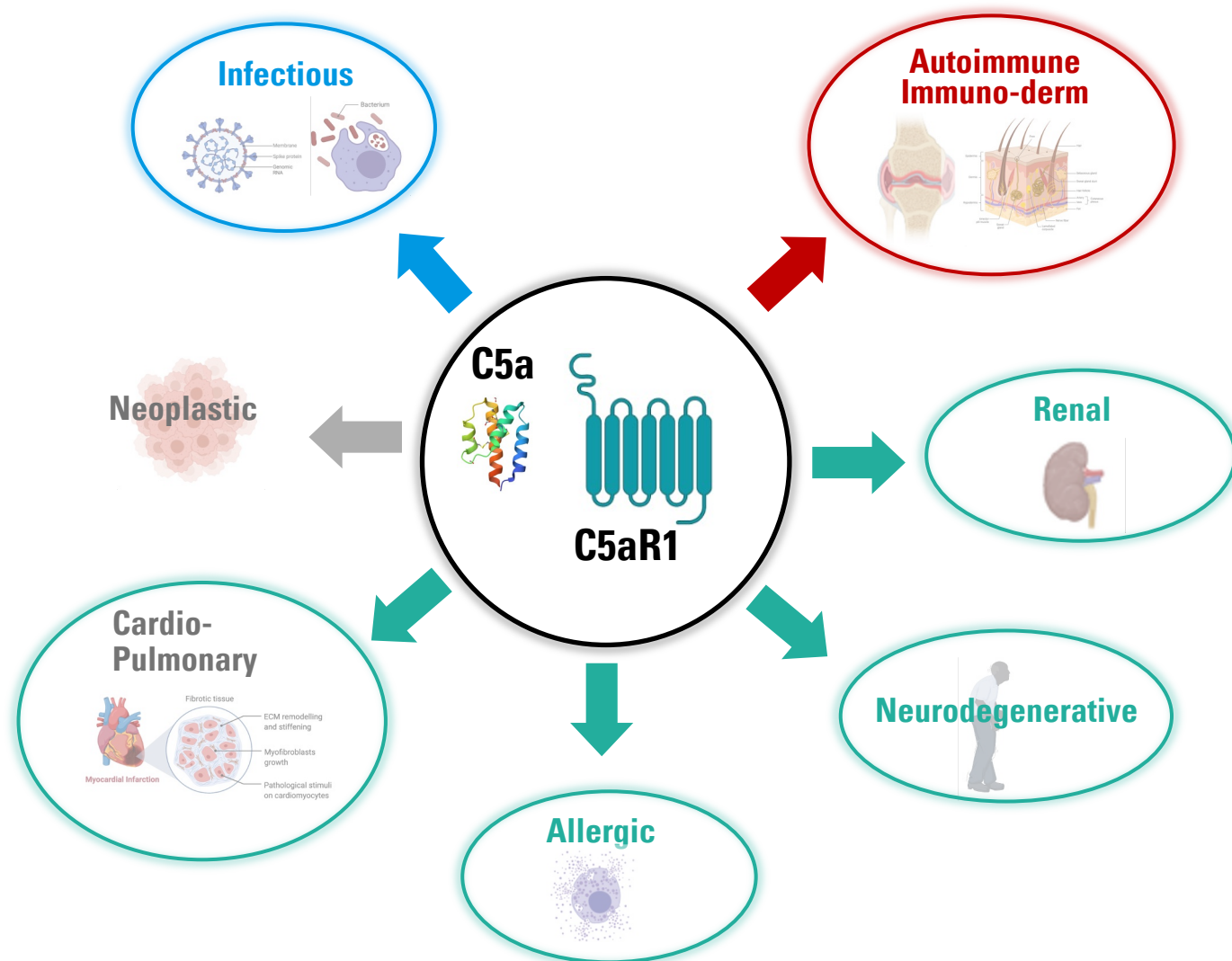
Izicopan-led pipeline targets **multiple sizable markets**

	INDICATION	PRECLIN	PHASE 1	PHASE 2	PHASE 3	MARKET	STATUS & MILESTONES
I&I	izicopan <i>Oral C5aR inhibitor</i>						Topline Phase 2a reported with Ph2b planning ongoing
	hidradenitis suppurativa						
	chronic spontaneous urticaria						Topline Phase 2a reported
	other immuno-dermatology						Additional indications in immuno-dermatology
	broader I&I						Additional chronic indications including nephrology, hematology, neurology and others
CRITICAL CARE	Gohibic vilobelimab <i>C5a inhibitor</i>						US EUA granted*
	critical COVID-19						
	SARS-CoV-2-induced ARDS						Approved by European Commission*
	broader ARDS						Phase 2 "Just Breathe" ASPR/BARDA clinical platform study 
DERM	vilobelimab <i>C5a inhibitor</i>						Phase 3 reported**
OTHER	IFX002 <i>C5a inhibitor</i>						
	vilobelimab life-cycle approach						For optimized use in chronic inflammatory indications

*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*/**)

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

Favorable safety profile with over 180 human subjects dosed

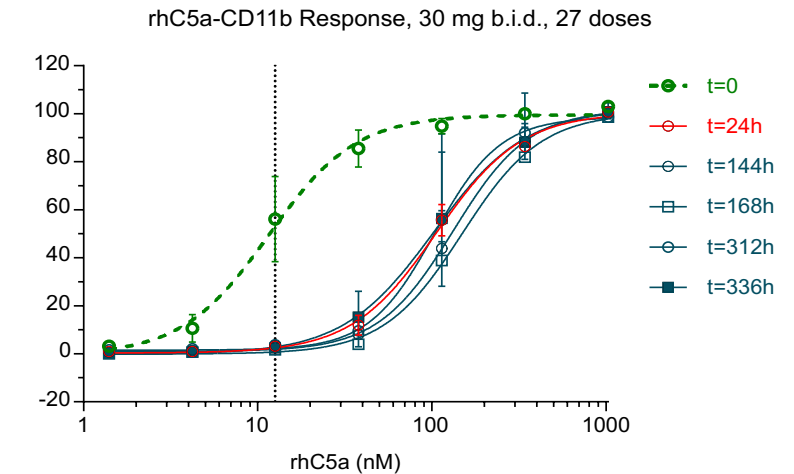
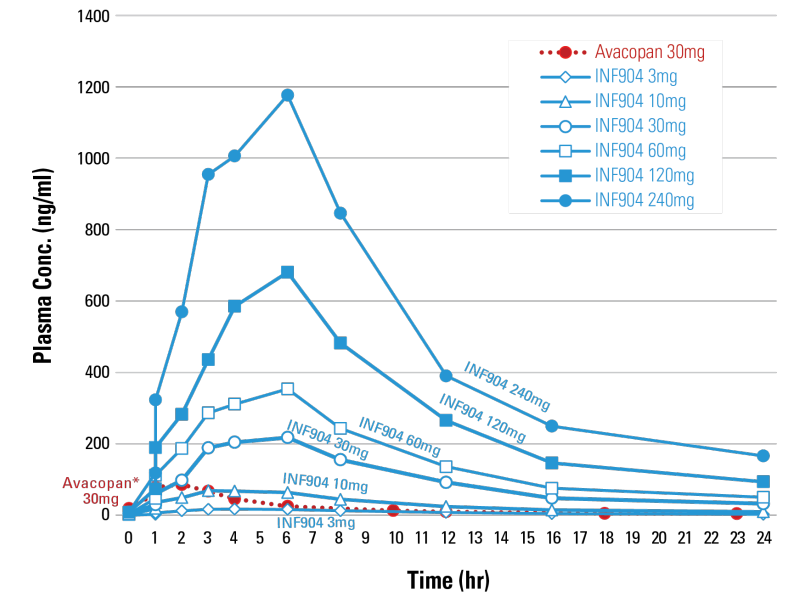
- Preclinical studies, Phase 1 (SAD and MAD) and Phase 2a in HS and CSU **show no signals of safety concern**

Other favorable features compared to avacopan

- **30mg per capsule** compared to 10mg for avacopan
- **Much weaker inhibitor of CYP3A4/5** in pre-clinical studies in vitro (36-fold higher in IC_{50})

These properties allow for exploring a significantly more potent C5aR inhibition with fast onset in patients which may lead to higher clinical efficacy for izicopan

This could open significant additional market opportunities

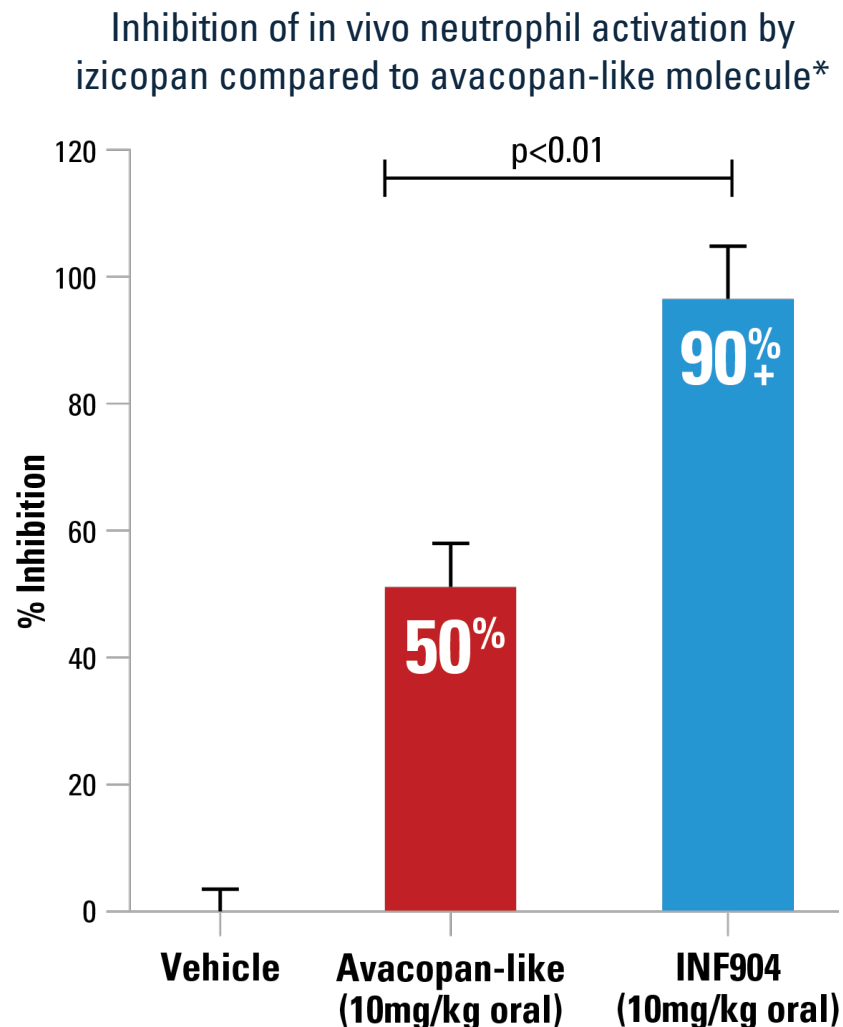


*InflaRx data on file: PK Results From Single Ascending Dose (SAD) Phase 1 study – note: Avacopan data (Becker et al, 2016, PLoS One) are superimposed in graph for orientation. Avacopan was not included as a comparator in izicopan Phase I study.

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

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

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



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*

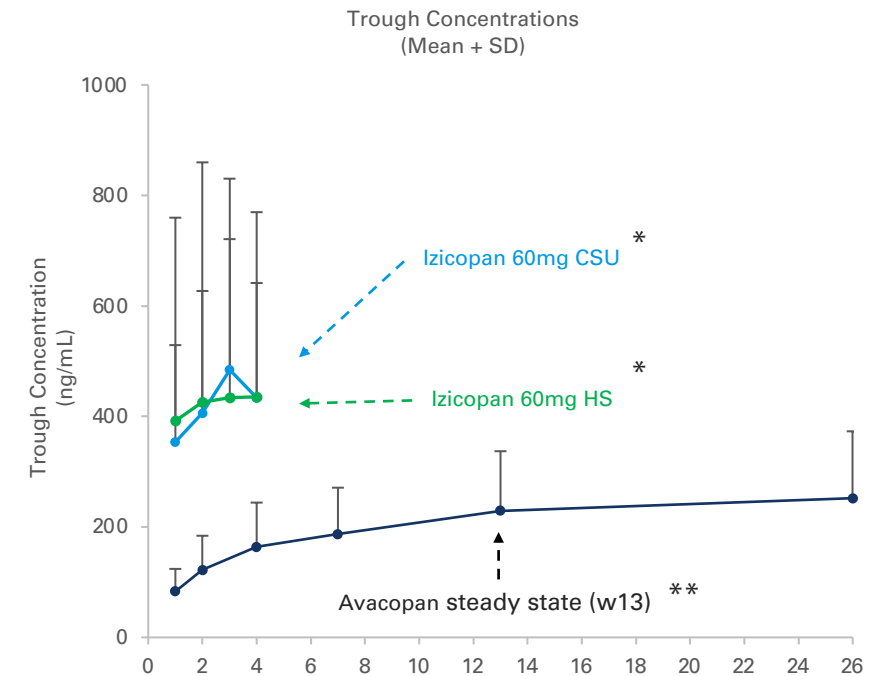
Izicopan therapeutic exposure reached within the first week in Phase 2a

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

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[†]**

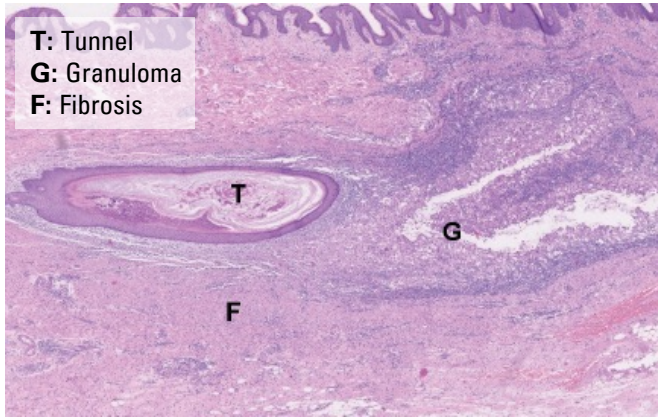


*Willems, D., Hinzpeter, EL., Van der Zee, H.H. et al. Patient 16, 153–164 (2023).

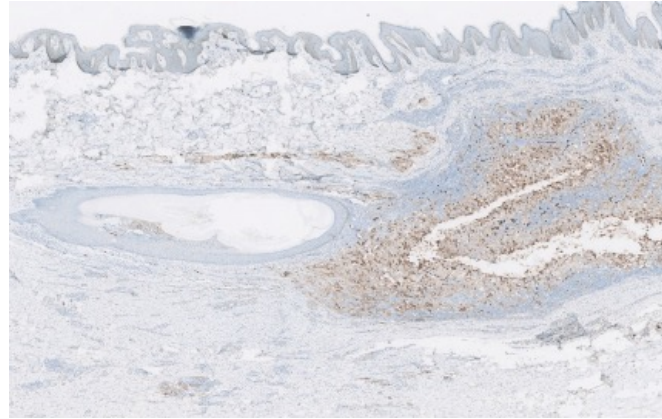
[†]IFRX proprietary market research, Clarivate.

C5aR and Neutrophils play a critical role in HS pathogenesis especially in draining tunnels (dT) and abscesses

Standard H&E tissue staining



C5aR staining



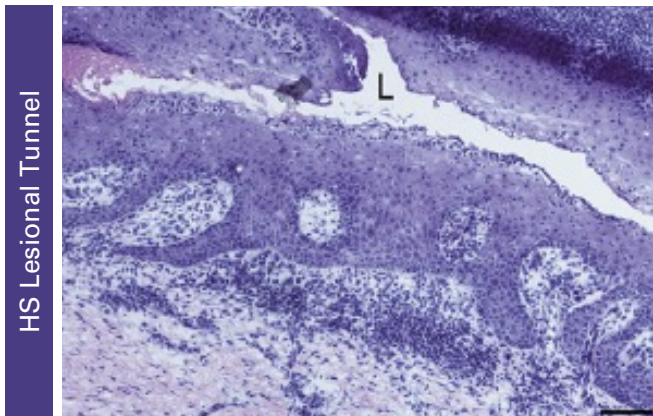
Hurley stage III patient with tunnel formation and surrounding **granulomatous** inflammation with foreign body giant cells. **C5aR1 staining positive – neutrophils, histiocytes and giant cells**

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

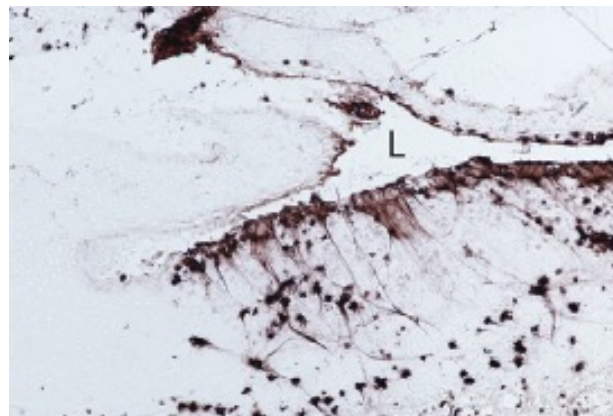
Van Straalen et al. 2022. *Front Immunol* 21.

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

Standard H&E tissue staining



NE: Neutrophil Elastase staining

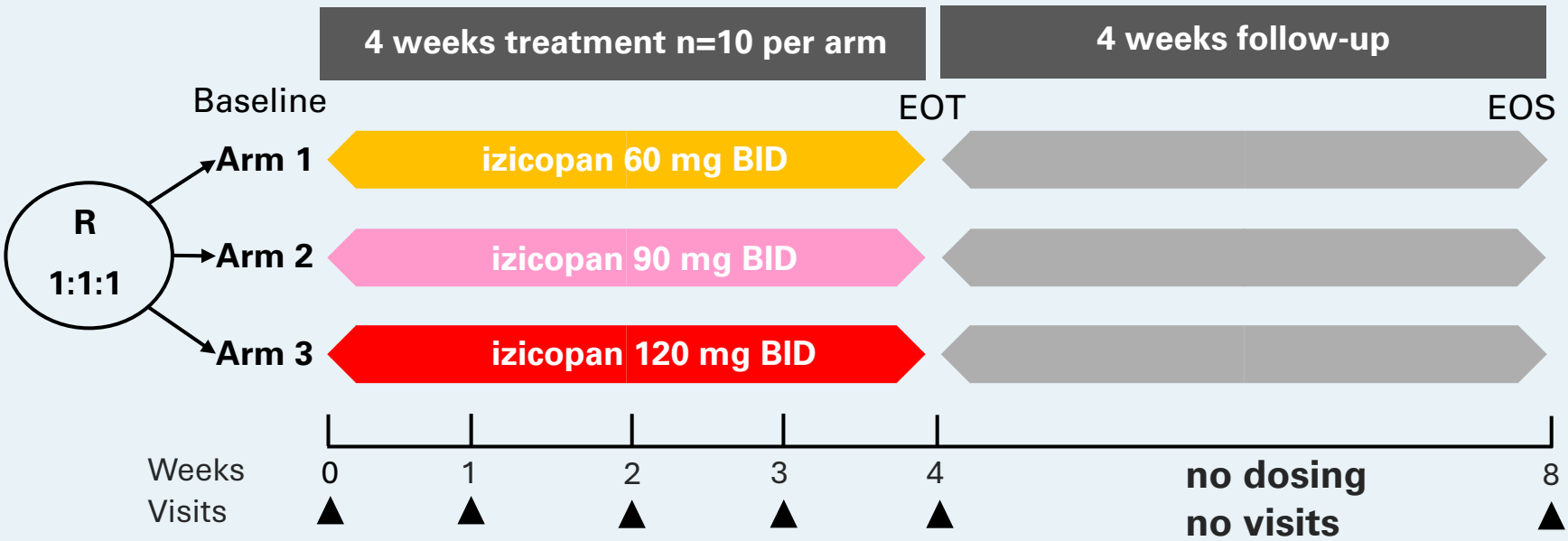


Neutrophils infiltrate inflammatory lesions in HS, including tunnels and the surrounding tissue

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



Primary & secondary objectives
Safety & PK

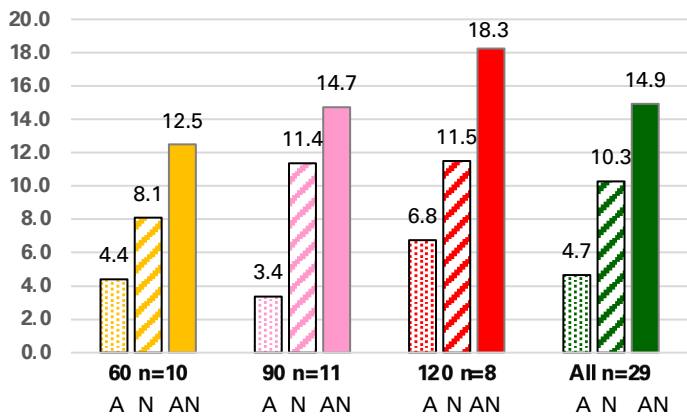
Exploratory objectives
Clinical endpoints AN, dT, ANdT, HiSCR, NRS-Skin Pain, DLQI

Izicopan capsules taken with food as described in the protocol

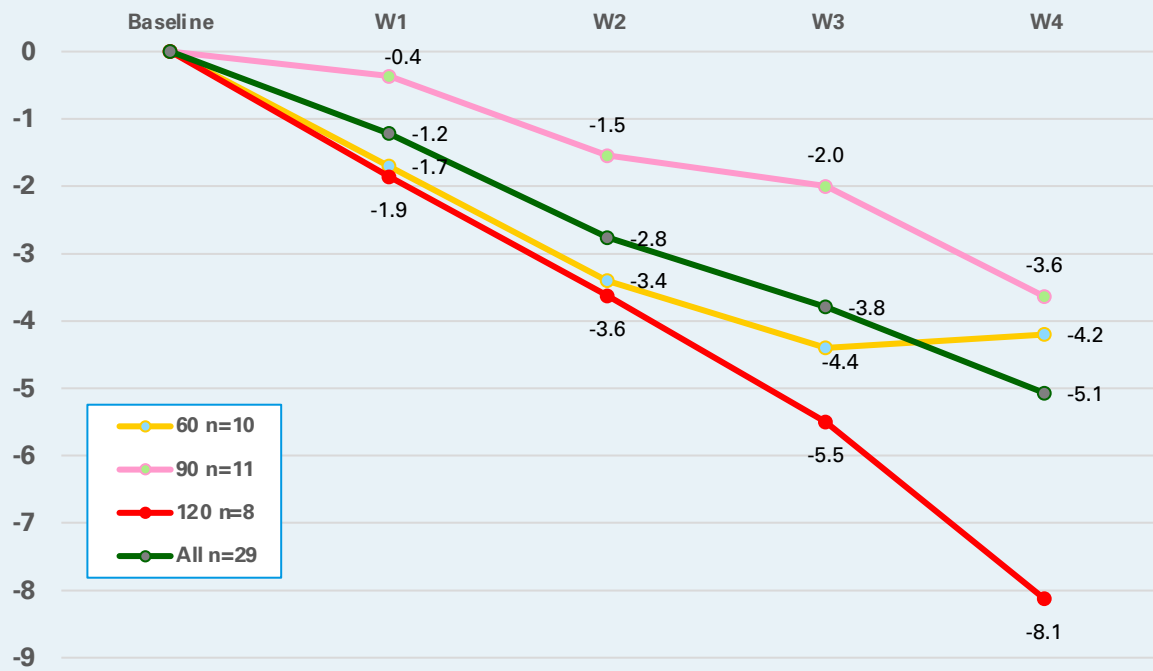
EOT = End of treatment (week 4)
EOS = End of study (week 8)

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

Baseline A,N, AN counts (mean)

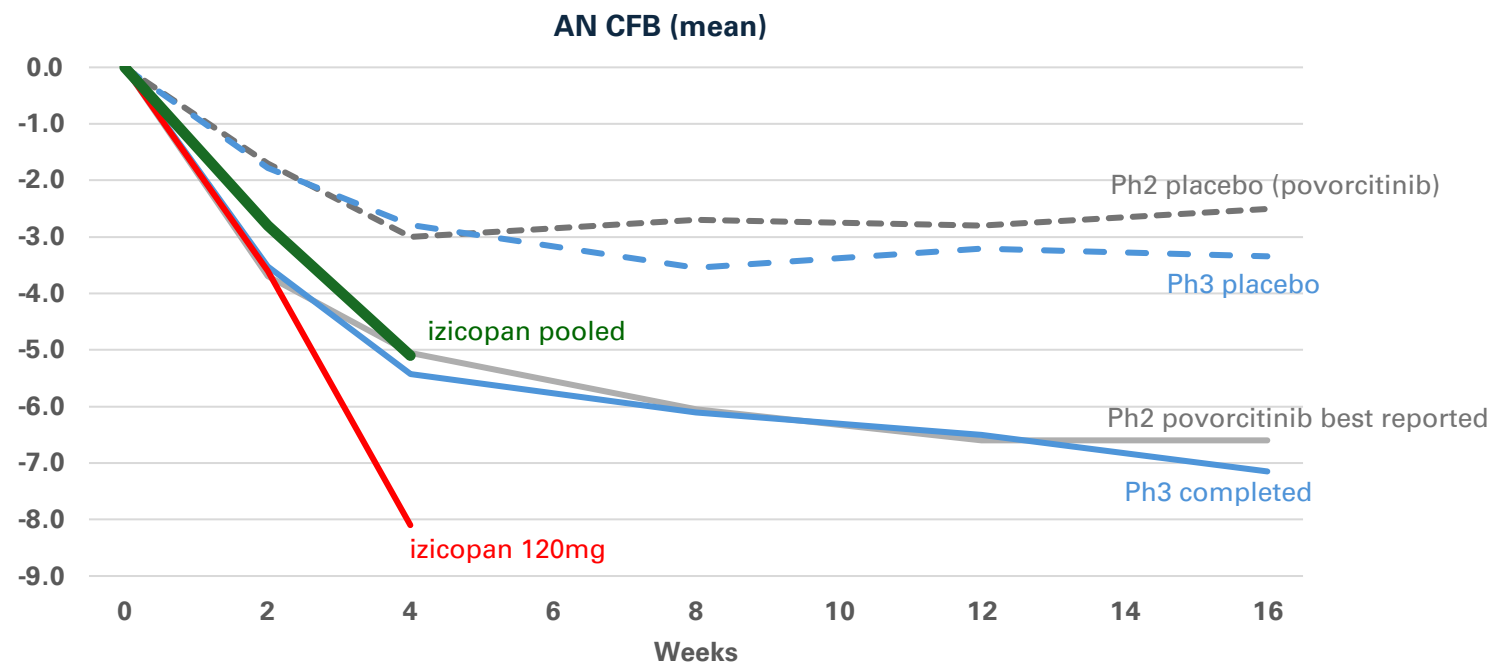


AN absolute CFB over time



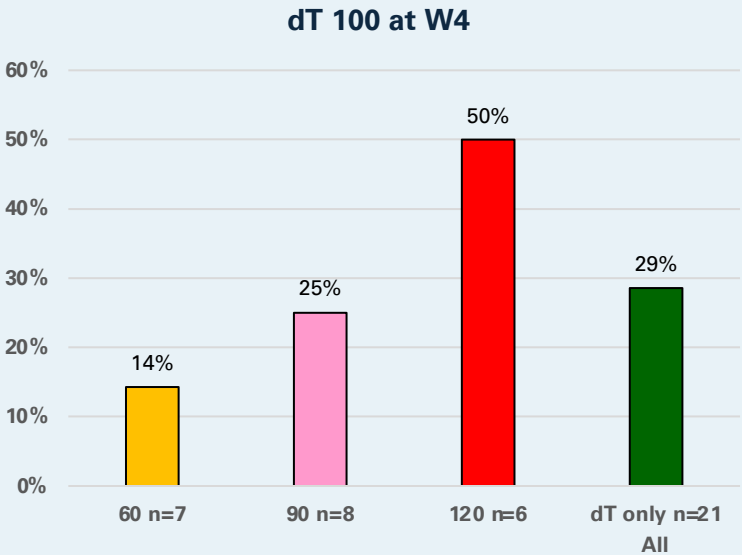
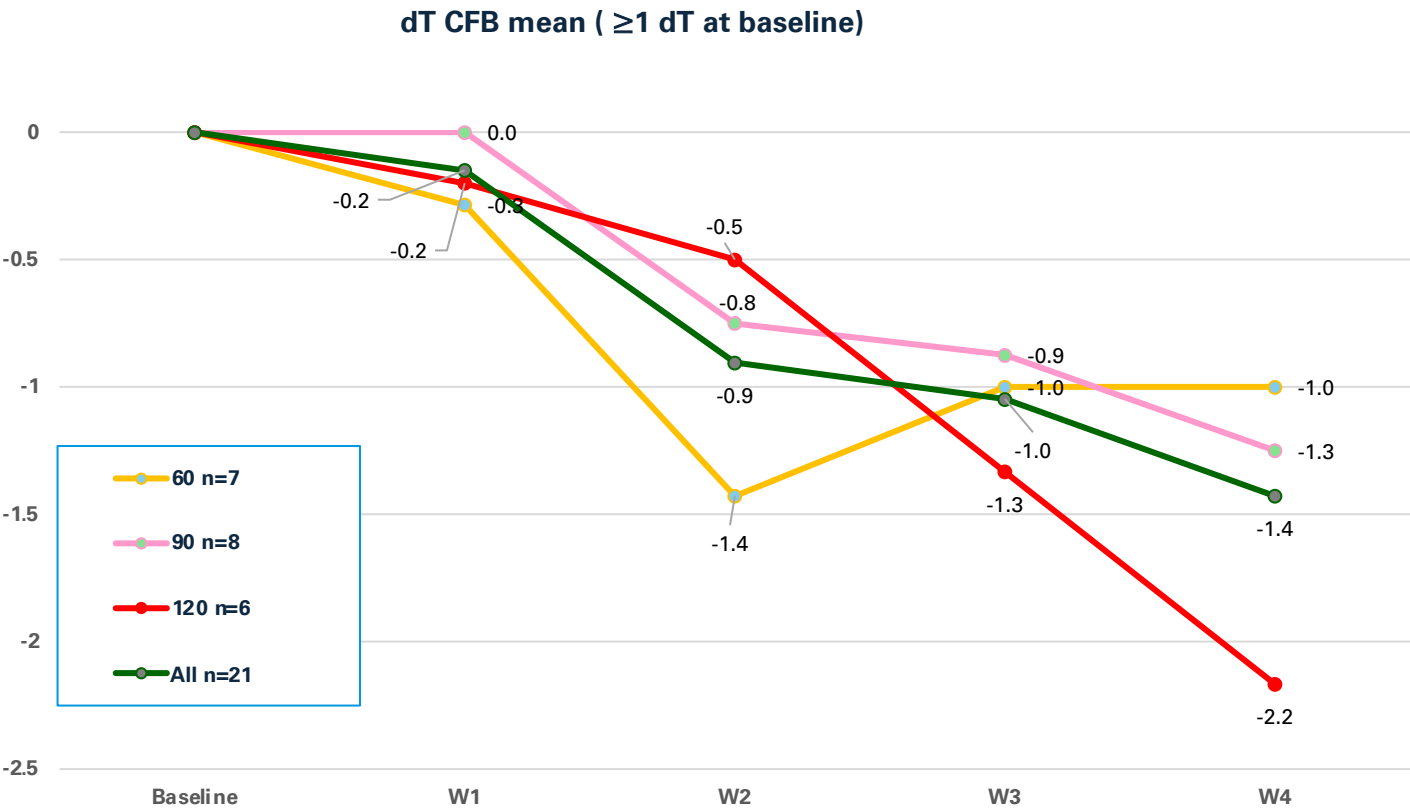
Izicopan Phase 2a in HS: AN reductions in line with successful studies

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

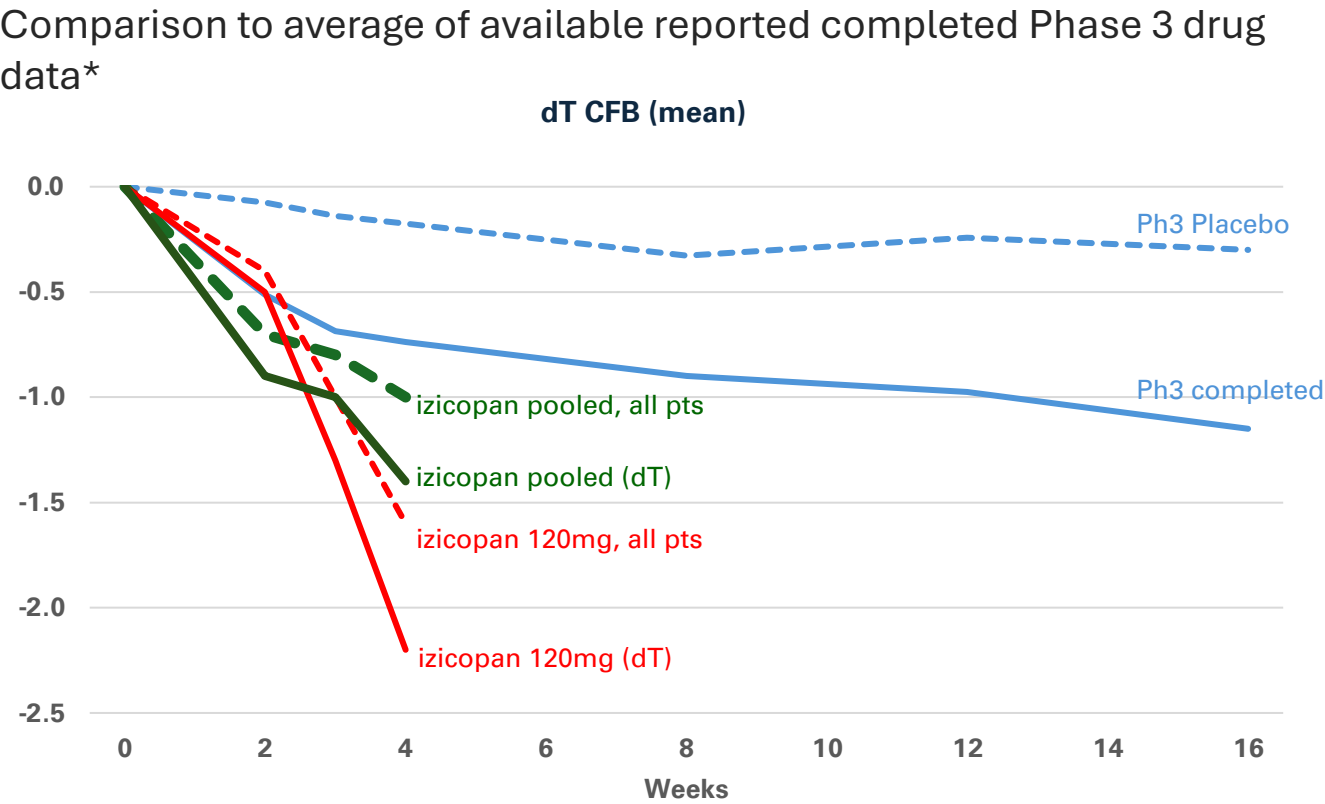


* Data are derived from different completed Phase 3 clinical trials and averages were created using the data available at each timepoint:
Adalimumab Phase 3 PIONEER I & II: week 12 data for all tested dose groups and placebo as reported by Kimball et al., N Engl J Med 2016; 375:422-34.
Secukinumab Phase 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 et 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).
For Phase 2 data from
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

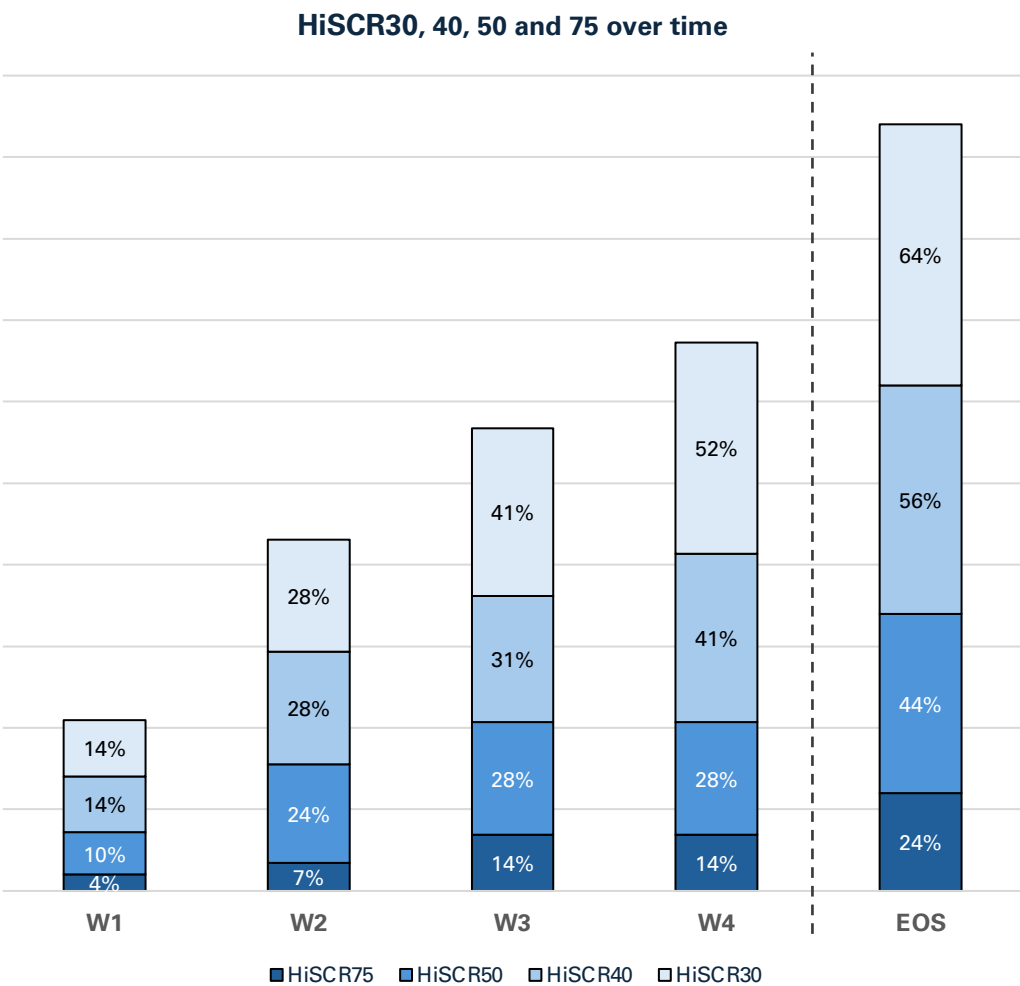
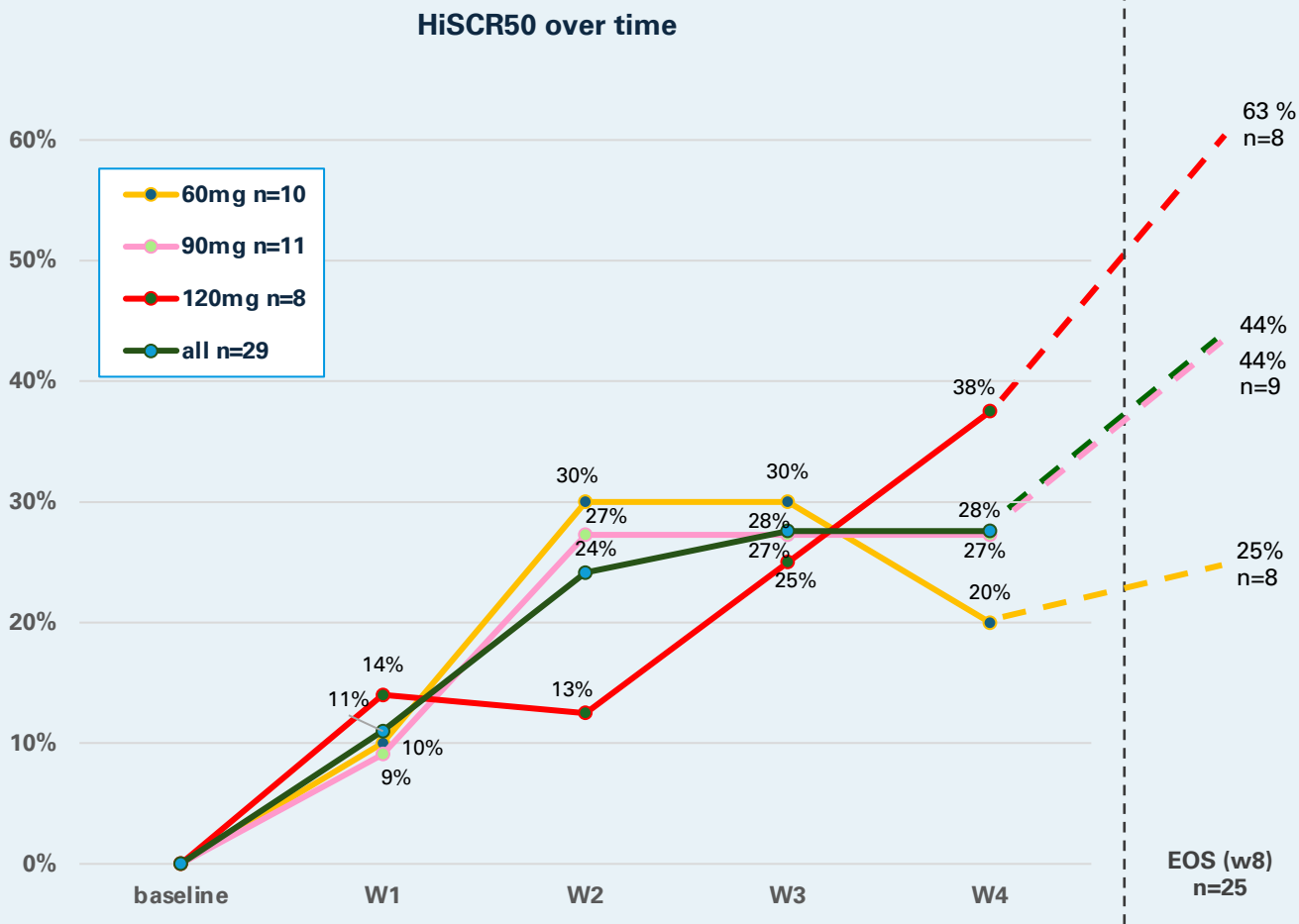


Izicopan Phase 2a in HS: dT reductions vs reported successful Ph3 trials

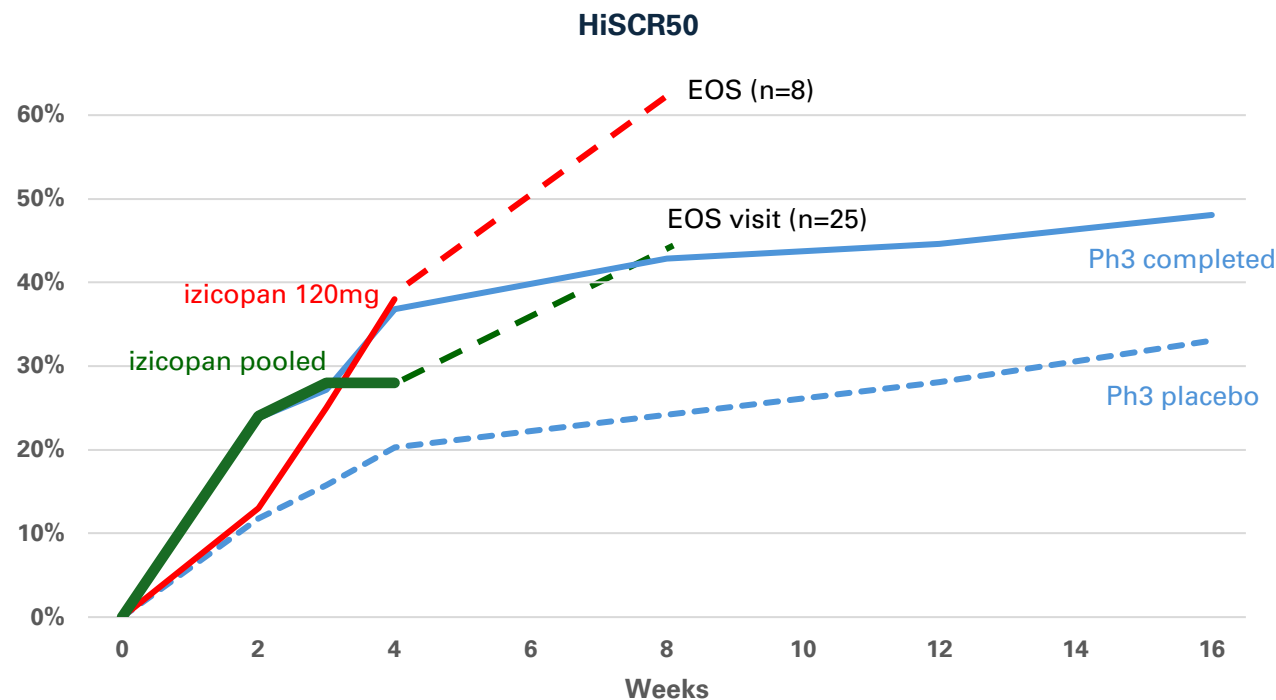


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Bimekizumab BE HEARD I & II: week 2, 4, 8, 12 and 16 data for all tested dose groups and placebo as reported from Kimball et al., Lancet 2024; 403(10443):2504-2519, absolute mean CFB data for week 2, 4, 8 and 12 are approx. numbers from graphs (data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

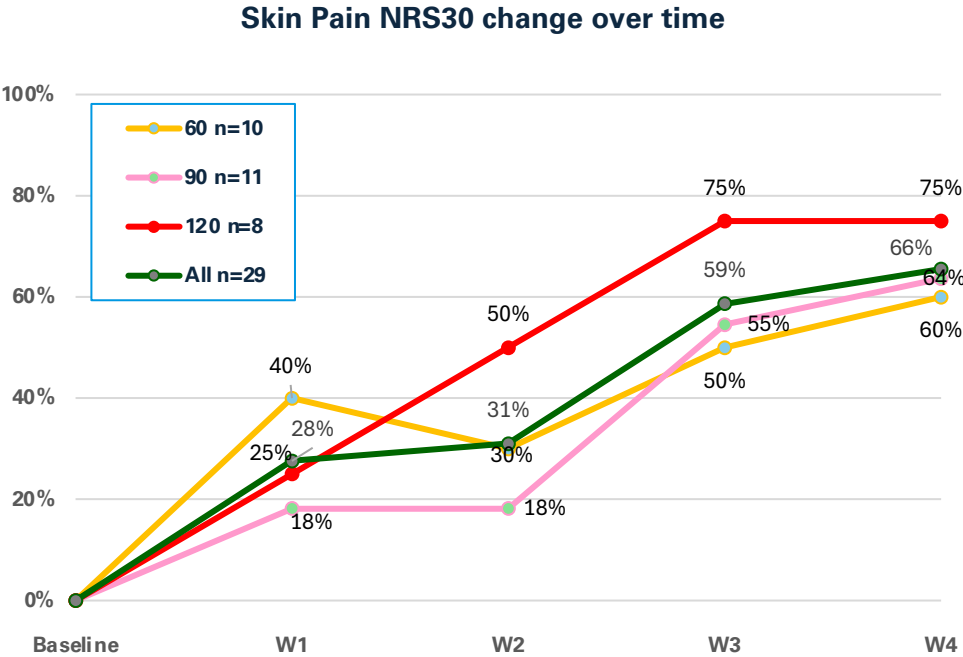
Secukinumab SUNSHINE & SUNRISE: week 2, 4, 8, 12, (approx. from published graph) and 16 (reported) data from all tested doses and from placebo as reported by Kimball et al., Lancet 2023; 401(10378):747-761 and on Clinicaltrials.gov (NCT03713619, NCT03713632)

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 et 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.

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

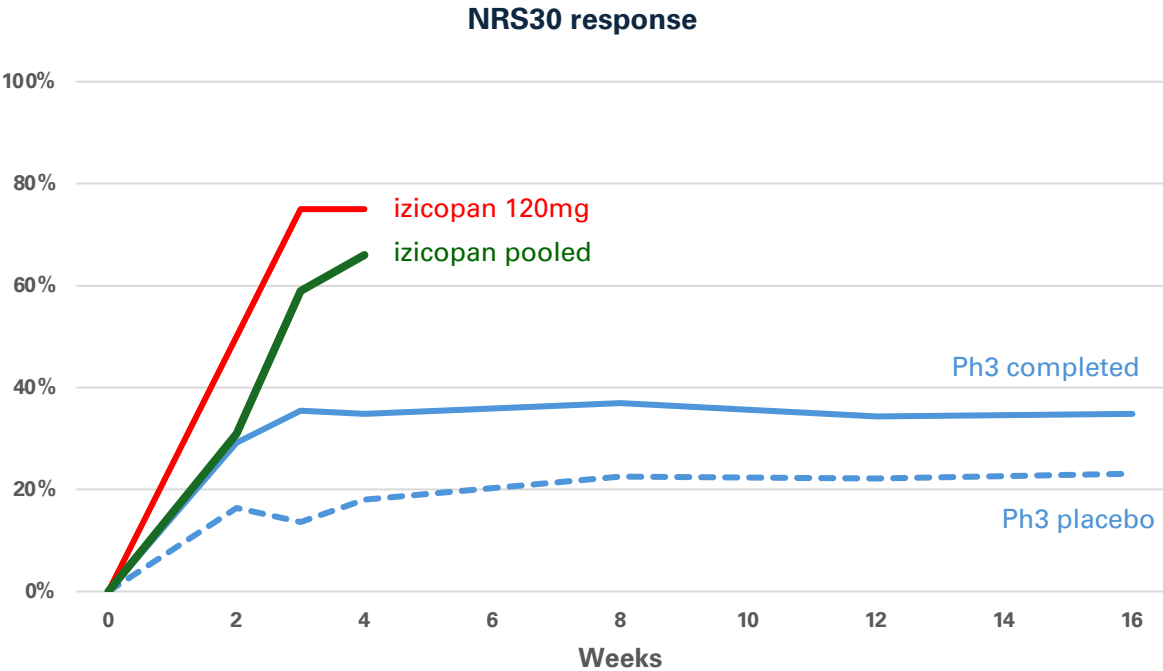
Izicopan Phase 2a in HS: High pain score reduction (NRS30)



NRS30 response is defined as $\geq 30\%$ 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

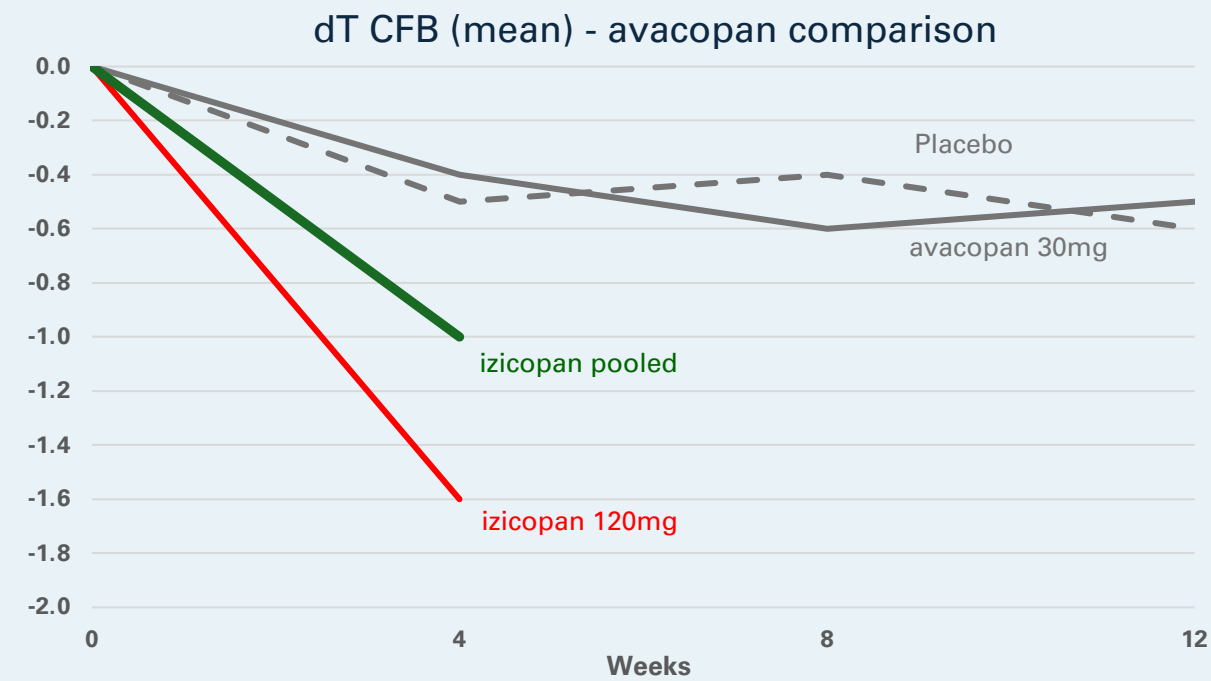
Comparison to average of available reported completed Phase 3 data*



* 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.

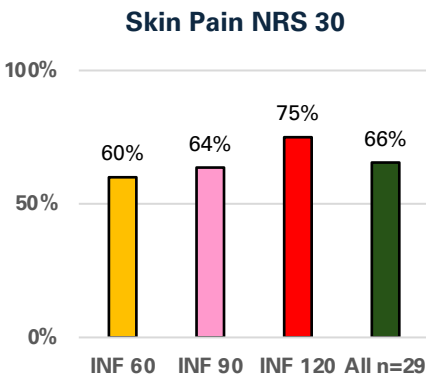
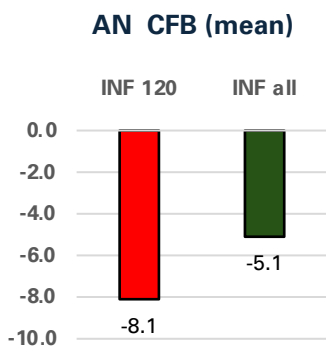
Note definitions: Adalimumab & Povorcitinib: NRS30 defined as $\geq 30\%$ reduction and ≥ 1 point reduction from baseline for patients with baseline NRS ≥ 3 .
Secukinumab: NRS30 defined as $\geq 30\%$ reduction and ≥ 2 point reduction from baseline for patients with baseline NRS ≥ 3

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

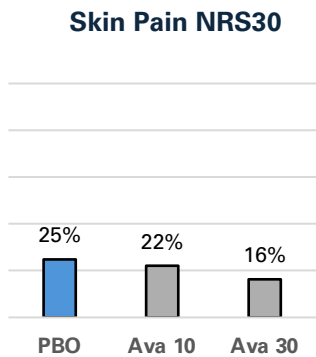
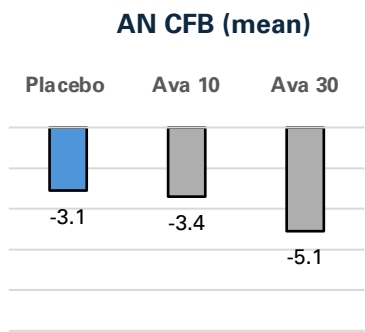


* Data from avacopan Phase 2 AURORA trial reported at [clinicaltrials.gov](https://clinicaltrials.gov/ct2/show/study/NCT03852472) (NCT03852472). NRS30 definition used in AURORA reported data: $\geq 30\%$ reduction and ≥ 1 point reduction from baseline for patients with baseline NRS ≥ 3

izicopan Week 4



Avacopan Week 12



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**

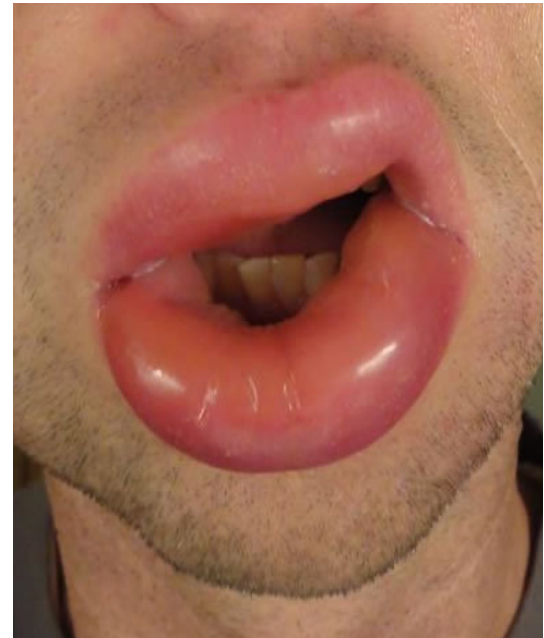
Izicopan shows promise to be a safe oral agent with biologic-like activity and a differentiated new mechanism of action

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

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[†]**

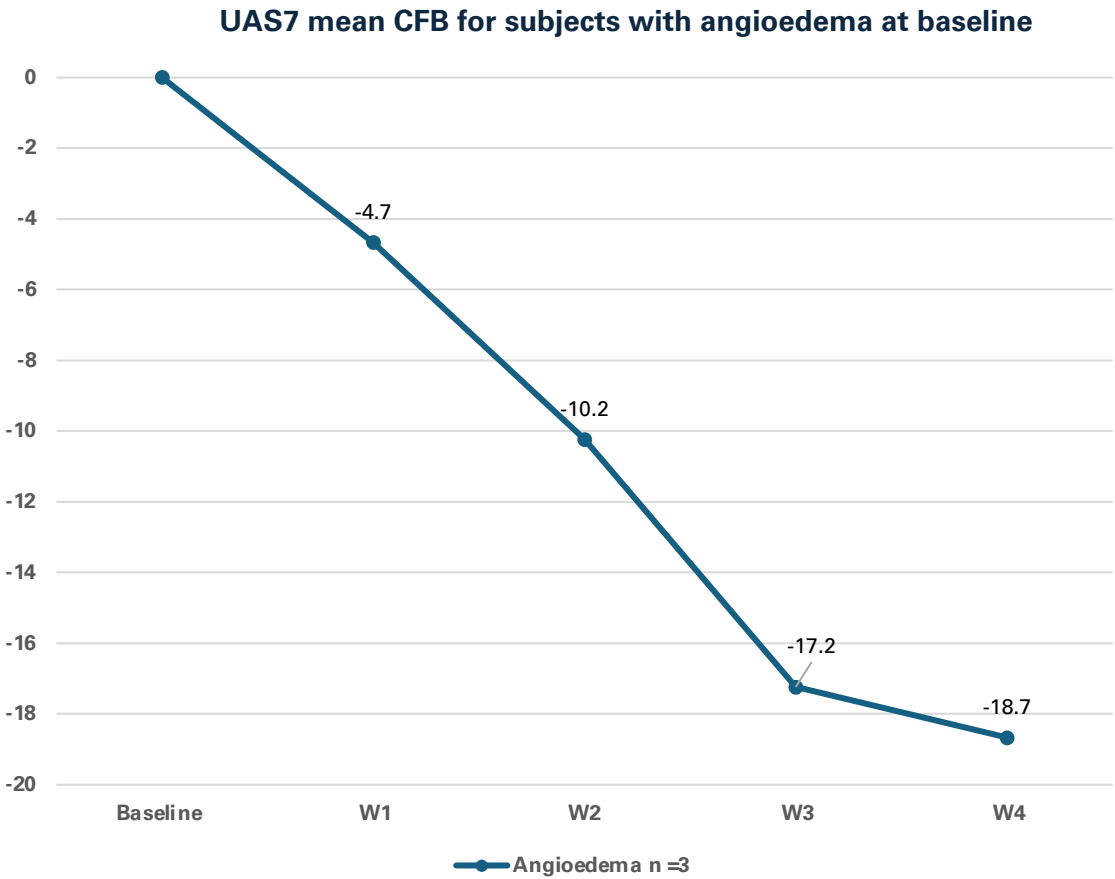
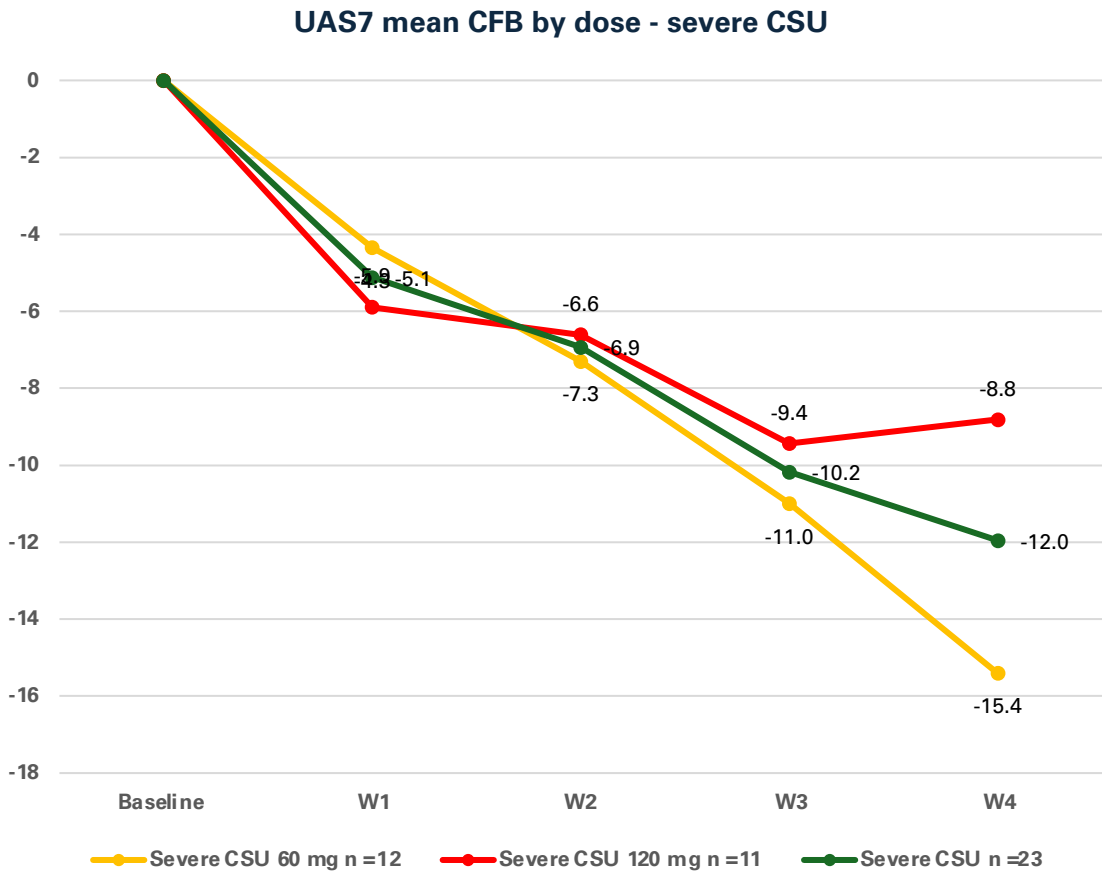


*Metz et al, Clin Rev Allergy Immunol. 2020; 59(1): 38–45.

[†]IFRX proprietary market research, Clarivate.

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

Patients with severe disease and those with angioedema



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

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