PHASE IIB SHINE STUDY Q&A DOCUMENT – FEBRUARY 2020

As a result of the phase IIb SHINE study with IFX-1 in Hidradenitis Suppurativa (HS), the Company has engaged in an in-depth analysis of the full data set, both for internal purposes and to respond to inquiries from investors, analysts, health care professionals and others directly or indirectly involved in the industry and development of treatments for HS. This analysis revealed a substantial amount of information which will be utilized in the Company's efforts moving forward. In order to provide potentially valuable information and answer questions that the Company has been receiving, the Company has created this document to make some of the information and answers readily accessible.

Table of Content

1) The efficacy signals in the phase IIb SHINE study are generated by post-hoc analysis. Tell us how biases and caveats are mitigated in your post-hoc analysis. 2

2) Some publications show that c5a levels are not correlated with HS disease severity. What's your explanation? 2

3) How do we know IFX-1 blocks the C5a signaling pathways with the current dosing regimen? 3

4) The phase IIb SHINE study failed to meet its primary endpoint, why do you think there is a positive signal from this study? 3

5) Is it possible that the positive signal is generated from concomitant treatment such as surgery or antibiotic treatment? 6

6) What factors could have contributed to high placebo response rate? 7

7) What strategies can be deployed to minimize placebo response rates in pivotal trials? 7

8) What are the plans for the further development of IFX-1 in HS? 8

Important Notice and Disclaimer 8

1) The efficacy signals in the phase IIb SHINE study are generated by post-hoc analysis. Tell us how biases and caveats are mitigated in your post-hoc analysis.

The originally chosen primary endpoint, the HiSCR, is the assessment of the three inflammatory lesions (abscesses "A", inflammatory nodules "N", and draining fistulas "F"), which are then computed into the HiSCR by accounting for a reduction of the AN count (not including the draining fistulas), with no increase in A or F over baseline.

The efficacy analysis presented "post-hoc" is based on the same three assessed inflammatory lesions (ANF). Thus, the actual data remain untouched, as no changes were made in the underlying signal detection method (assessment of inflammatory lesions) and no new or other signals were generated for this analysis and the conclusions derived from this analysis. The analysis shows that high dose IFX-1 treatment statistically significantly reduced the three inflammatory lesions together (without any imputation) or when using the IHS4 score, which reflects reduction of all three inflammatory lesions in a weighted fashion (1 point for N, 2 points for A, 4 points for F). The analysis also resulted in a tatistically significant dose response signal when applying the originally chosen MCP-mod analysis at week 16 (as originally chosen as primary endpoint using the HiSCR in the SHINE study).

For lesion reduction and IHS4 scores, the Company used additional sensitivity analyses and imputation methods to verify that point estimates at different time points would also reflect an efficacy signal for the IFX-1 high dose group.

For the open label extension part (OLE) of the SHINE study, patients remained blinded to their original treatment. Placebo and minimal IFX-1 dose patients who were non-responders utilizing HiSCR, showed additional reduction of lesions (ANF and IHS4) when transitioned to the IFX-1 medium dose (800mg q2w). Note that this analysis was done using the last observation carried forward method (LOCF) to avoid detected long term benefits being caused by enhancement effects through the "non-benefitting" patients transitioning out of the trial over time.

2) Some publications show that c5a levels are not correlated with HS disease severity. What's your explanation?

This question typically relates to the publication of Kanni et al. (Br J Dermatol. 2018 Aug;179(2):413-419). Some confusion may exist due to the presentation seen in Fig 2 and Fig 3, in which Hurley stage III patients and severe patients do not appear to have higher levels of C5a.

Firstly, data from this publication show that HS patients in general have statistically significantly higher C5a levels when compared to controls. The data do not contradict the concept that C5a-driven inflammation drives HS disease. These data only display real life C5a levels of patients with a varying extent of inflammation and were not gathered in a prospective manner.

Secondly, C5a is a marker of complement-driven inflammation. However, it is not a marker of disease stage (i.e., Hurley III vs II). In each stage of HS, according to Hurley, one may detect patients with higher (e.g., flares) or lower inflammatory status. For example, acutely inflamed abscesses during a flare that may need surgical intervention will exhibit strong tissue inflammation and are likely induce higher C5a

levels. In contrast, abscesses in a healing process with less tissue inflammation will tend to exhibit lower C5a levels. Both types of patients may be categorized in the same Hurley stage while having very different C5a levels.

Finally, there is a large inter-individual and intra-individual variability in C5a levels in humans. It is therefore difficult to make conclusions on populations with small sample sizes (here n=8 to n=31). Given this variability in group differences, the small sample size of the above-mentioned retrospective study may not render a conclusion.

3) How do we know IFX-1 blocks the C5a signaling pathways with the current dosing regimen?

Sustained control over C5a levels was established with the high dose in the SHINE Phase IIb study (1200mg q2w) and the phase IIa study (800mg qw). The phase IIa C5a data have been published in the Company's F1 document. While it is impossible to prove an absolute C5a blockade in tissue with currently available measures, InflaRx has conducted extensive PK/PD analyses as well as PB/PK analyses on the basis of the available PK/PD data from the SHINE study, as well as on data from the phase IIa study. On the basis of these analyses, it appears that the high doses of IFX-1, used in these trials, offered sustained C5a control and tissue presence of IFX-1 in HS. In addition, InflaRx employed a secondary PD assay in several other studies, demonstrating that detected IFX-1 levels in patient plasma, when taken ex vivo, are able to block C5a-induced activation in neutrophils in fresh human blood up to 100% in a 1:1 concentration (antibody added recombinant C5a molecules).

4) The phase IIb SHINE study failed to meet its primary endpoint, why do you think there is a positive signal from this study?

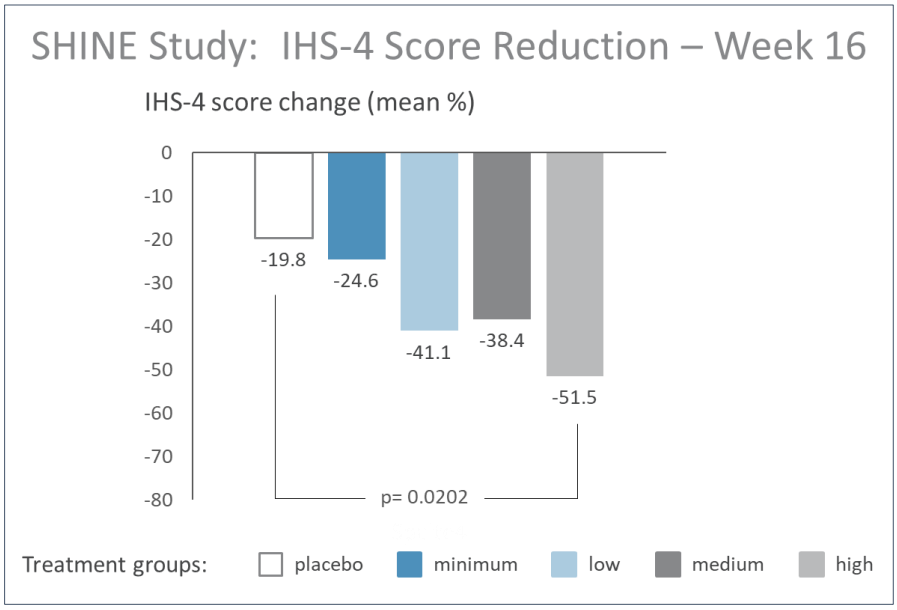
The Company's phase IIb SHINE study failed to meet its primary endpoint on HiSCR, which is defined as a ≥ 50% reduction of total body abscesses + inflammatory nodules count ("AN count"), with no increase in abscesses or draining fistulas when compared with baseline. The data show a large variability / natural fluctuation in the AN count, which implies a large variability of the HiSCR. This high variability was previously unknown (never published) to the field and has major implications on the power calculation of a trial.

The 95% confidence intervals at approximately 35 patients per group (group size in the SHINE study) covered ranges of up to > 40%. Thus, it is possible that the responder rates in the HiSCR by chance also display levels over 40% in placebo groups.

However, a number of positive signals were generated in the trial and are based on the inflammatory lesions which underly the HiSCR. For example, IFX-1 was shown to reduce the number of lesion as well as improve scores derived from the lesions other than the HiSCR.

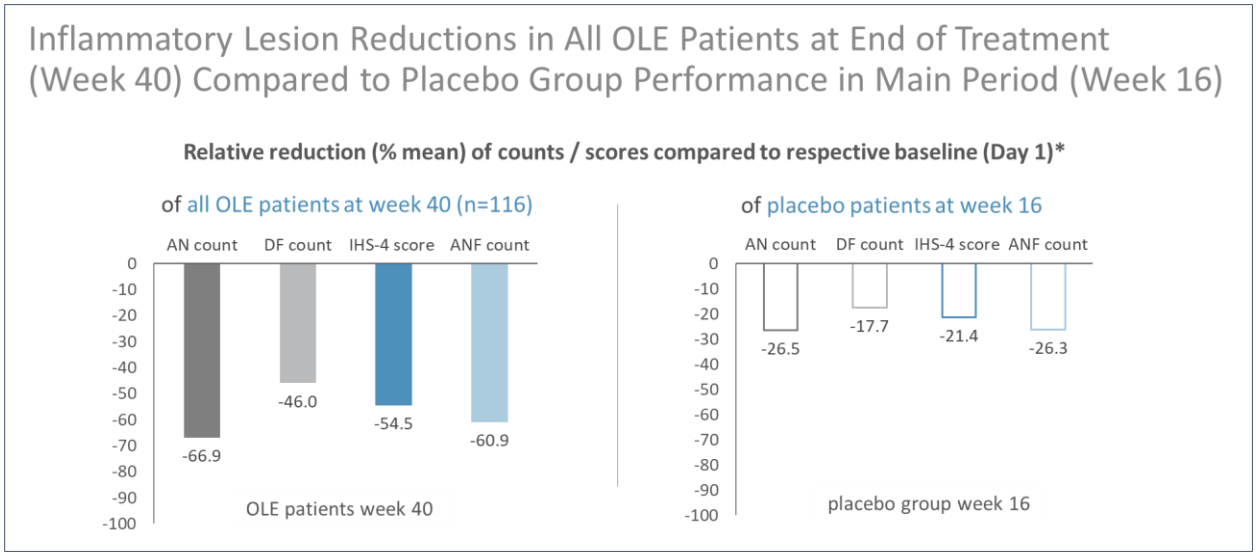
Additionally, there is a statistically significant signal on the reduction of all three inflammatory lesions combined (inflammatory nodules, abscesses and draining fistulas) and on draining fistula reduction at week 16, as well as other timepoints throughout the trial, and measured with various scores. These scores included 1) ANF count (counts abscesses, inflammatory nodules and draining fistulas all together) and 2)

IHS4 count (1 point per inflammatory nodule, 2 points per abscess, and 4 points per draining fistula). These effects were also tested with different sensitivity analyses (like LOCF, MMRM, etc.). IFX-1 high dose treatment statistically significantly reduced the number of inflammatory lesions compared to placebo at week 16, as well as improved IHS4 scores (published figure example from IFRX press release). When applying the MCP-mod statistical analysis (utilized in the SHINE study to analyze the HiSCR) on the IHS4, the SHINE study data demonstrate a statistically significant dose response at week 16.

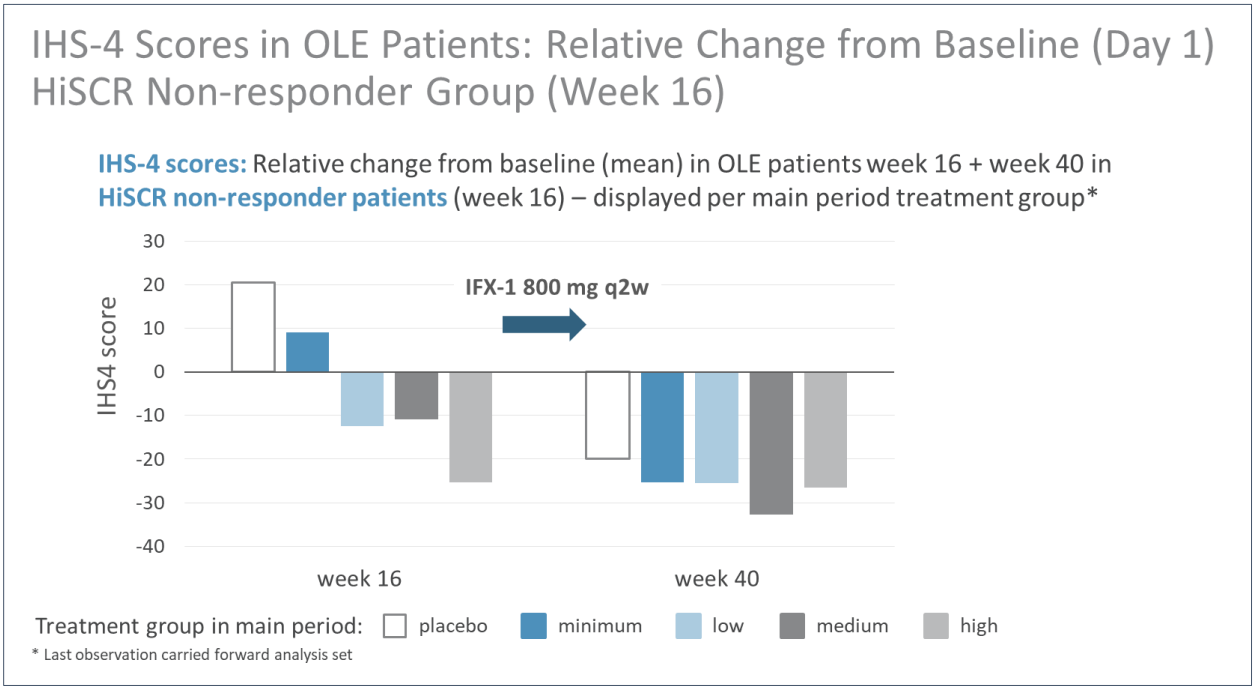


Efficacy analysis from Open Label Extension (OLE) phase of the SHINE study until end of treatment at week 40:

Overall, close to 70% of patients starting the SHINE study completed the OLE phase (9 months of treatment plus 1 month of observation). The lesion reduction of the group of patients completing the OLE period was compared to a) the patient's own OLE group baseline lesion counts on day 1 of the SHINE study, and b) to the "well" performing placebo group lesion reduction at week 16 of the SHINE study. In both analyses, the patients completing the OLD period showed a significant long-term lesion reduction after IFX-1 therapy, even if treated with what InflaRx believes to be a non-optimal dose of IFX-1 for HS treatment during the OLE phase (the high-dose was not carried forward in the OLE). Both relative lesion reduction and absolute lesion reduction showed potentially promising results (example in figure below from IFRX November press release and conference call for relative lesion reductions). This same efficacy signal holds when sensitivity analyses, such as LOCF, are applied.

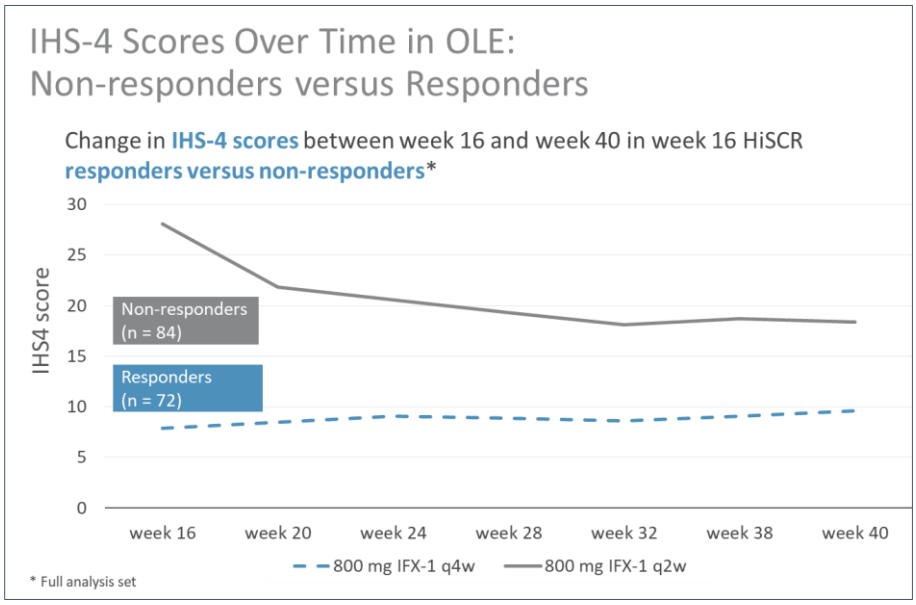


During the OLE part of the study, in which all patients remained blinded to their original treatment, former placebo patients deemed non-responders using HiSCR exhibited signs of response and lesion reduction (refer to figure below from the November IFRX conference call document). To a lesser extent, this effect was also seen with the low dose group patients (800mg q4w) and medium dose patients from the initial treatment period, who were transitioned to or maintained on the medium dose in the OLE. The only patients who did not show an additional benefit were high dose patient who were HiSCR non-responders, who transitioned to a lower dose in the OLE.



In addition, in the OLE, a lower dose of IFX-1 appears to maintain some lesion reduction in those patients who had a strong initial lesion reduction.

IFX-1 showed a durable and consistent benefit as treatment duration increased (refer to figure below from November conference call document).



5) Is it possible that the positive signal is generated from concomitant treatment such as surgery or antibiotic treatment?

Any impacting larger surgery was not allowed in the phase IIb SHINE study or the phase IIa study.

There are two different reasons for use of antibiotics in the trial:

1. Concomitant oral antibiotics (minocycline, doxycycline) - These drugs are used in HS because of their stabilizing anti-inflammatory properties. All patients had to fail a treatment attempt for HS with these antibiotics before enrolling in the phase IIb SHINE study and had to be stable on these drugs before enrollment.

2. Systemic antibiotics for any reason other than for treatment of HS (e.g., AEs, flu, etc.)

ad 1) Concomitant antibiotic use was overall well distributed over all groups during the entire course of the SHINE study. The low number of patients in the dose groups who were on stable allowed antibiotics (refer to 1) above) (n= 4 - 8) limits any potential impact on the overall study results. The high-dose treatment group had the lowest patient number in this category (n=4). Overall, there was no signal that concomitant allowed antibiotic use for HS impacted the significant reduction of lesions detected in the high-dose group, when compared to the placebo group.

ad 2) Systemic antibiotic use other than for HS: After detailed analysis of antibiotic use other than for HS, we found:

- No indication of more antibiotic use in the high dose group with a balanced antibiotic use in all treatment groups
- No indication of more antibiotic use during the Extension Period (compared to Main Period)
- Similar frequency of antibiotic use in the Main Period Responders and Non-Responders throughout the Extension Period

- No signal for an impact on the overall performance in HiSCR because of antibiotic use
- No signal for an impact on the overall performance of the IHS4 score related to antibiotic use

6) What factors could have contributed to high placebo response rate?

It is relevant to distinguish that a high placebo response rate, when measured utilizing HiSCR, does not necessarily reflect patient improvement. Thus, HiSCR response rate may not translate to a performance of the placebo group resulting in better patient outcomes. Indeed, statistically significant differences in the overall inflammatory lesion reduction (as well as other endpoints) were detected for the high dose IFX-1 group when compared to the placebo group.

One may posit that this is a result of the fact that it is now known that HiSCR has an extremely high variability. Combining this extremely high variability with the fact that the SHINE study had approximately 35 patients per group and given large 95% confidence intervals at week 16 of the trial, observed HiSCR rates could have been high by chance.

Nevertheless, there is no dispute that the placebo group performed better than expected when looking at overall lesion reduction and other scores. This may potentially be explained by the following:

1) 4:1 randomization IFX-1 to placebo, which implied a high chance for patients to be dosed with IFX-1. This assumption is reasonable because, like in other dermatological diseases, HS is known to be impacted by psychological factors

2) Intensified care within the SHINE study: Through biweekly i.v. dosing, patients were subjected to frequent “touch points” with HS specialists at which patients got access to wound cleaning, HS evaluation, i.v. treatment etc. This “intensified” HS care versus “normal” standard of care aspect has been suggested as a possible explanation by HS experts.

Importantly, InflaRx has done a thorough and in-depth analysis on the quality of the SHINE study and concluded that the trial was conducted according to high GCP quality standards and that patients were recruited by HS specialists with the sites comparable to the ones used in other large studies. InflaRx further analyzed whether larger biweekly detected fluctuations in AN counts for single patients could be attributed to a change in the examiner counting these lesions for each respective patient, but no such signal could be detected. InflaRx therefore determined that the detected AN count fluctuation is likely present in many HS patients and, as such, a natural course of the disease.

7) What strategies can be deployed to minimize placebo response rates in pivotal trials?

InflaRx believes that the following measures are useful to achieve this:

- Simple trial design, with if possible, 1:1 randomization to minimize the psychological expectation effect to be on drug;
- Increase of the trial group size to account for high lesion count variability;
- Have a thorough understanding along with PK/PD modeling of the dose to be explored; and
- Ideally, utilize a primary endpoint which is less dependent on a fixed relative reduction of the highly fluctuating AN count.

8) What are the plans for the further development of IFX-1 in HS?

InflaRx plans to discuss with the FDA in an end-of phase II meeting a potential path forward towards phase III development of IFX-1 in HS. Discussions are anticipated to cover the choice of an alternative approvable primary endpoint, as well as various other aspects related to potential further development. The FDA meeting request is planned to be submitted in Q1 2020. Depending on the timing and the outcome / feedback from the FDA meeting, InflaRx currently plans to define the potential path forward for the development of IFX-1 in HS by 2H 2020.

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