



MoonLake Immunotherapeutics

Corporate Presentation

October 2023

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Certain information contained in this presentation relates to or is based on studies, publications, surveys and our own internal estimates and research. In this presentation, we rely on, and refer to, publicly available information and statistics regarding market participants in the sector in which we compete and other industry data. Any comparison of us to any other entity assumes the reliability of the information available to us. We obtained this information and statistics from third-party sources, including reports by market research firms and company filings. In addition, all of the market data included in this presentation involve a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Finally, while we believe our internal research is reliable, such research has not been verified by any independent source and we have not independently verified the information.

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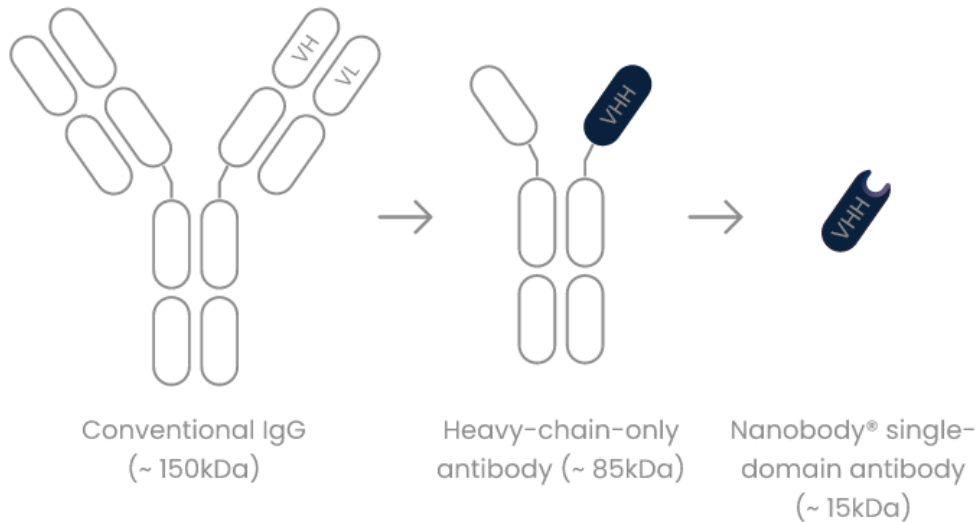


- **Founded in 2021** in Switzerland
- **Nanobody® technology** licensed in initial private round
- **Unique molecule with sonelokimab**, tri-specific IL-17A & IL-17F Nanobody® to elevate treatment in inflammation in a **\$40bn+ market**
- **Public on Nasdaq** in April 2022, gross proceeds of \$150m
- **Follow-on offering** in 2023, gross proceeds of \$460m
- **Nearly \$650m raised** to date
- **Clinical phase company** – successfully concluded phase 2b in psoriasis (n=313), primary end-point in phase 2b in HS (“MIRA”, n=234), and expecting imminent primary end-point in PsA (“ARGO”, n=207)
- Expecting readiness for **Ph 3 in at least 3 indications** by end of 2023
- Driven by a top-tier team, target is to unlock a **pipeline-in-a-product across large indications** from 2023 (>\$5bn in HS & PsA alone)

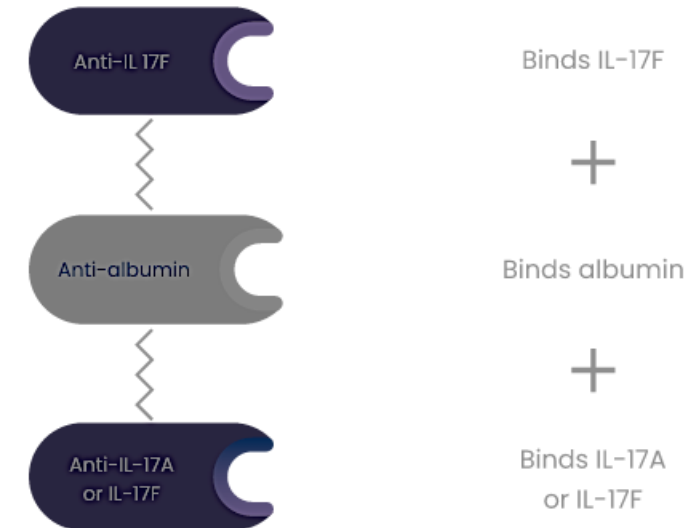


High interest in a differentiated molecule – Do you still Antibody?

Nanobodies® are much smaller than traditional antibodies



They can be designed to have multiple and different binding domains



IL-17A & IL-17F

Sonelokimab is a ~40kDa humanized Nanobody® consisting of three VHH domains covalently linked by flexible spacers - around a quarter of the size of traditional antibodies

With 2 domains, it binds with high affinity to IL-17A and IL-17F – a third domain binds human albumin

Subcutaneous administration, Q4W

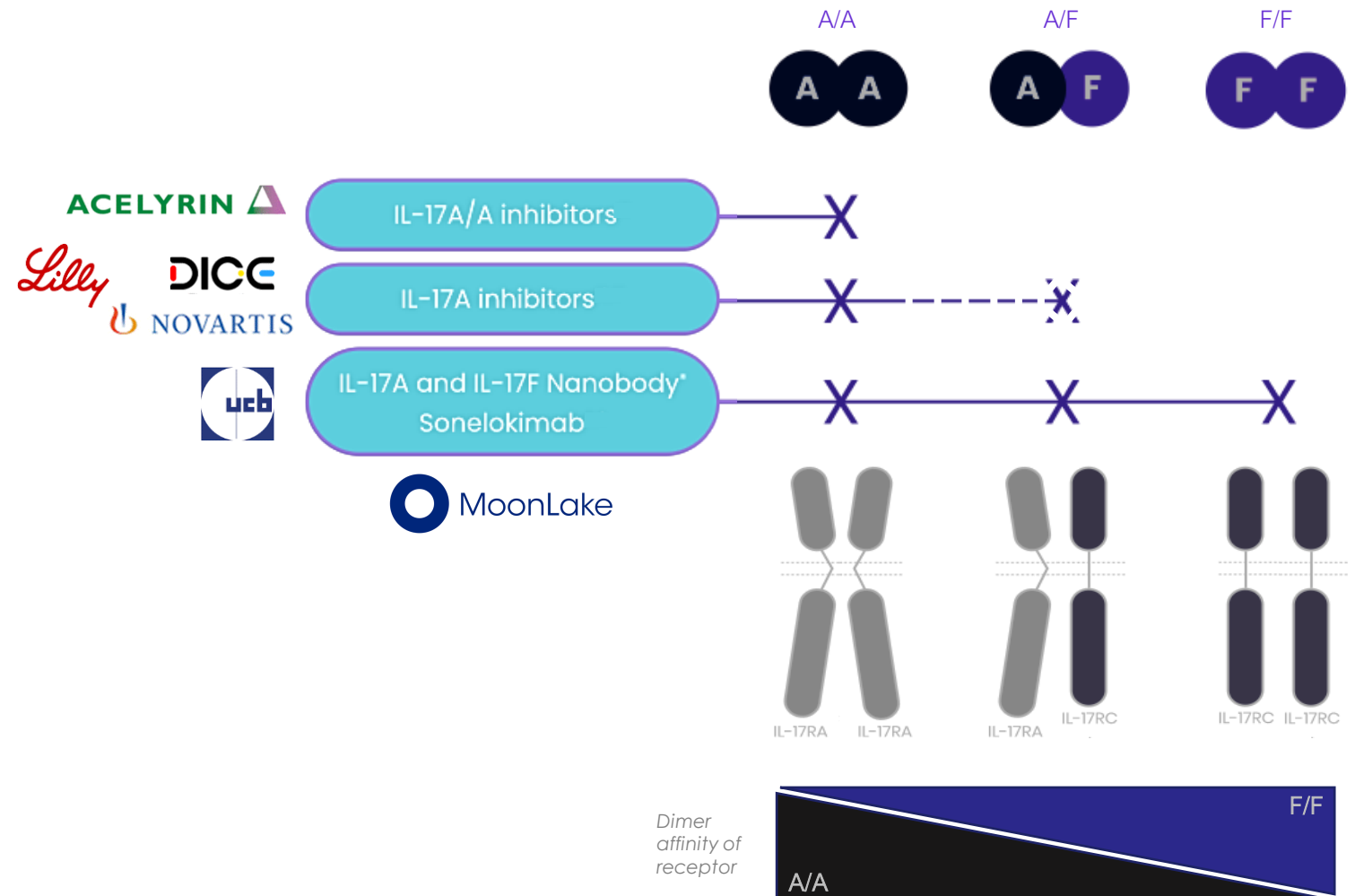
It's all about the dimers

Illustrative

IL-17A and IL-17F function as **dimers** to drive inflammation, through activation of IL-17RA & RC receptor complexes

Different IL-17RA & RC chains combine to form complexes, and those have **different affinity for different dimers**^{1,2}

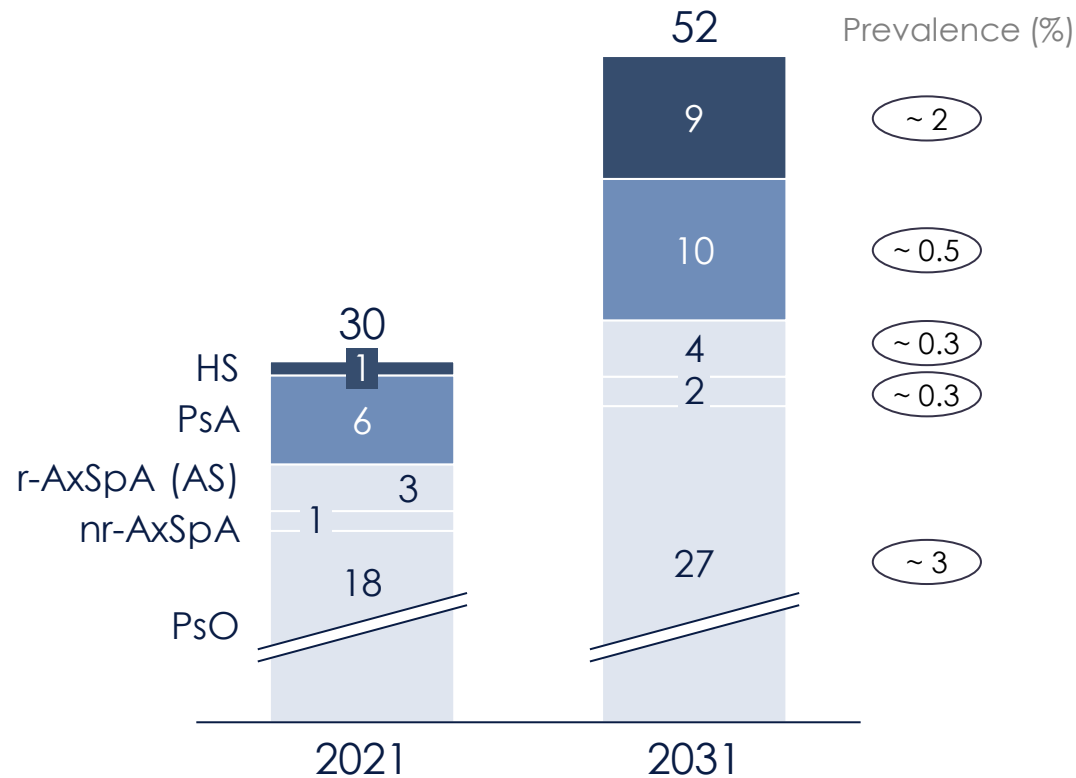
Not all IL-17-targeting therapeutics **can inhibit** IL-17A/A, IL-17A/F and IL-17F/F dimers



¹ Liu S, et al. Nat Commun. 2013;4:1888; ² Goepfert A, et al. Immunity. 2020 Mar 17;52(3):499-512

IL-17 inhibition is expected to lead in a growing \$50bn+ market

Global sales, USD Bn



IL-17 and other innovative biologics are expected to grow at CAGR 2-3x the rate of the market overall, to 2031

Hidradenitis Suppurativa (HS)

- **Driven by IL-17s** (60%) on base built by Humira™ as only therapy
- Several failures (e.g., IL-23, IL-36, TYK-2, IRAK, IL-1)

Psoriatic Arthritis (PsA)

- **Driven by IL-17s** with rates of 11%+ growth (1/3 market)
- Mostly IL-17 (incl. IZO) and some IL-23 but with latter not adding to joint treatment, (and JAKs)

Other: e.g., Axial Spondylarthritis (r-axSpA and nr-axSpA)

- **Driven by IL-17s** (20%+ growth) on base built by TNFs
- IL-23s failed

Other: e.g., Psoriasis (PsO)

- **Driven by newest IL-17** and IL-23 classes, eroding TNFs as the traditional class

IL-17A & F inhibition with further opportunity in many other disease areas across other Derm indications, Rheumatology, Ophthalmology, Hepatology, Nephrology, Oncology, and others



Why IL-17A & F is a highly attractive MoA

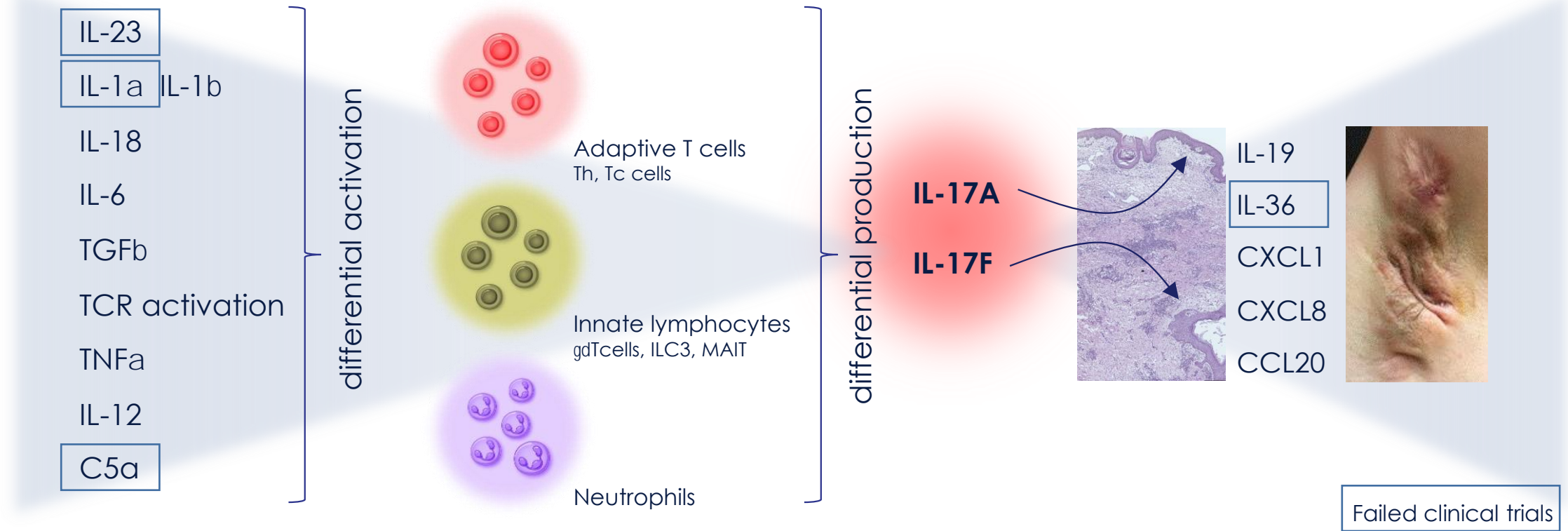
HS Example

Multiple stimuli induce subsets of immune cells to produce IL-17A and F

Different cell types preferentially produce IL-17A and/or F

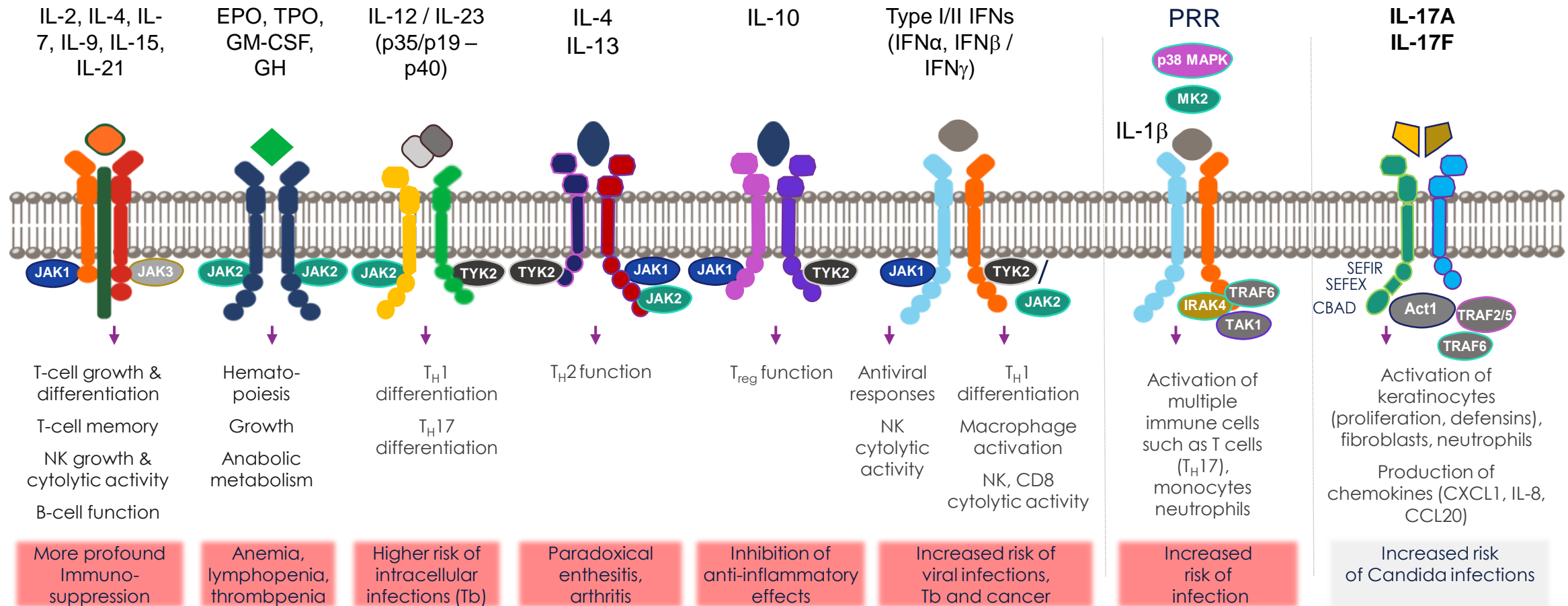
IL-17A and F as "bottleneck" in HS pathology

IL-17A and F induce multiple pro-inflammatory effectors, e.g. activation of keratinocytes







IL-17 represents a unique and potentially safer MoA vs. other options

- Jak/Tyk2 inhibitors affect multiple cytokine pathways explaining broad immunosuppressive and unwanted side effects
- MK2 and IRAK4 are involved in the epithelial reaction to danger signals



Act1, IL-17R adaptor protein; AP1, activator protein 1; Arid5a, AT-Rich interaction domain 5A; C/EBP β , CCAAT/enhancer-binding protein β ; CBAD, C/EBP β activation domain; CD, cluster of differentiation; DDX3X, DEAD-box helicase family member; EPO, erythropoietin; GH, growth hormone; GM-CSF, granulocyte-macrophage colony-stimulating factor; HuR, human antigen R; IL, interleukin; IFN, interferon; JAK, Janus kinase; MAPK, mitogen-activated protein kinase; NF- κ B, nuclear factor κ B; NK, natural killer; PRR, pattern recognition receptor; SEFIR, similar expression of fibroblast growth factor and IL-17Rs; SEFEX, SEFIR extension; TAK1, TGF β activated kinase 1; T_H, T-helper cell; TRAF, TNF-receptor associated factor; TPO, thrombopoietin; TYK, tyrosine kinase

SLK rapidly becoming a leader in large inflammatory diseases

	Trial	Patients (n)	Leading MoA	SLK leading asset
 HS	Phase 2b (MIRA)	234	IL-17A & F TNF & IL-17A	<input checked="" type="checkbox"/> Highest ever primary endpoint (HiSCR75), largest deltas to placebo, depth of responses
 PsO	Phase 2b	313	IL-17A & F IL-23 & IL-17A	<input checked="" type="checkbox"/> Largest delta vs market leader Cosentyx™ at PASI100, compared to BKZ, IL-23, etc.
 PsA	Phase 2b (ARGO)	200+	IL-17A & F TNF & IL-17A	<input type="checkbox"/> IL-17A & F inhibition shows best ACR/PASI data incl. TNF-IR pts
 <i>Other Rheum & Derm</i>	TBA	TBA	IL-17A & F Other	<input type="checkbox"/> IL-17A & F inhibition best data in AS, nr-AxSpA, enthesitis...

PsA primary endpoint data for SLK expected ahead of ACR 2023

PsO: SLK has a winning “next gen IL-17” profile in PsO

Phase 2 clinical data

THE LANCET

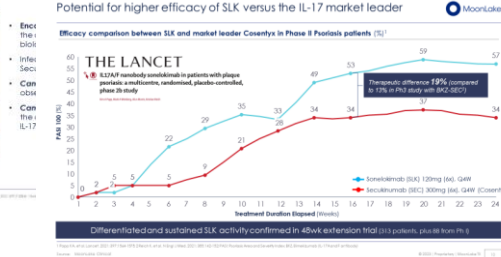
IL17A/F nanobody sonelokimab in patients with plaque psoriasis: a multicentre, randomised, placebo-controlled, phase 2b study

SLK has a differentiated safety profile to date

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Consult Table 3*

Study	Group	n	AEs	Serious AEs	Discontinuation	Death	Other
Phase 2b	SLK	100	10	0	2	0	0
	Placebo	100	15	0	3	0	0



- **Leading efficacy** in Inflammation (PASI 100 for most patients)
- **IL-17F adds to IL-17A inhibition** (vs. Cosentyx, 56% more patients to PASI100)
- **Clean profile** following historical IL-17 safety

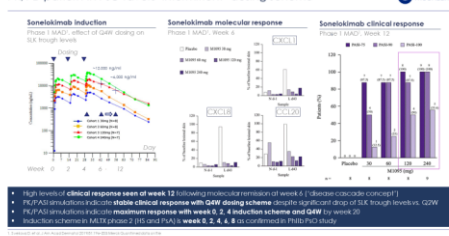
Phase 1 & Preclinical data

J AM ACAD DERMATOL
VOLUME 81, NUMBER 1

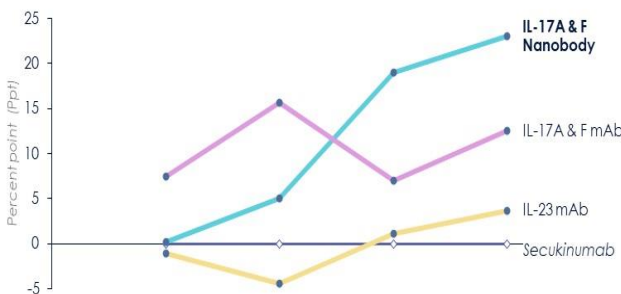
A randomized, double-blind, placebo-controlled phase 1 study of multiple ascending doses of subcutaneous M1095, an anti-interleukin 17A/F nanobody, in moderate-to-severe psoriasis

PK/PD pattern in PsO favors “intermittent” dosing scheme

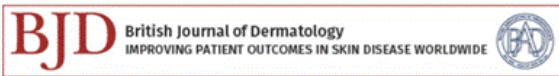
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PASI 100 delta to in-trial active comparator secukinumab



- **PK determined** for all testing doses (incl. 120 and 240mg)
- Stable clinical response with **Q4W dosing**
- **Molecular remission** & high **clinical response** over time
- **IL-17A & F inhibition** shows highest levels of skin clearance
- SLK shows **highest levels** of skin clearance (PASI100) **versus BKZ and IL-23s**



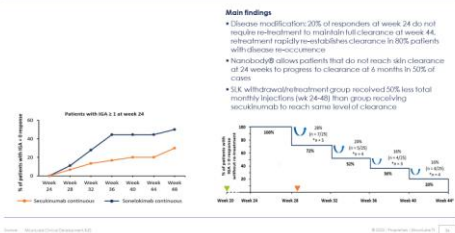
RESEARCH LETTER

Maintenance of response in moderate-to-severe psoriasis after withdrawal of the IL-17A and IL-17F nanobody sonelokimab – Is there a role for IL-17F in disease reoccurrence?

Kristian Reich, Eva Cullen, Mark Weinberg

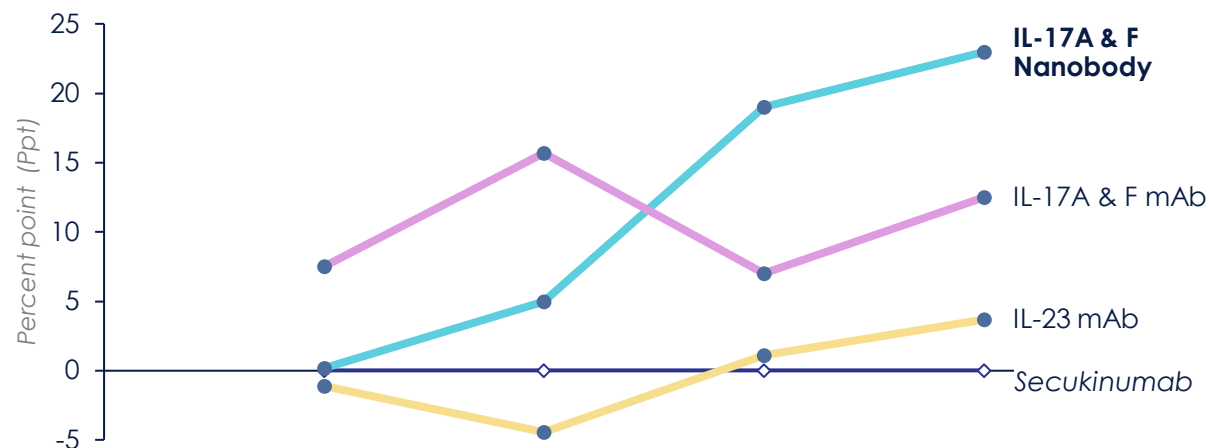
PD pattern supports disease control with intermittent inhibition

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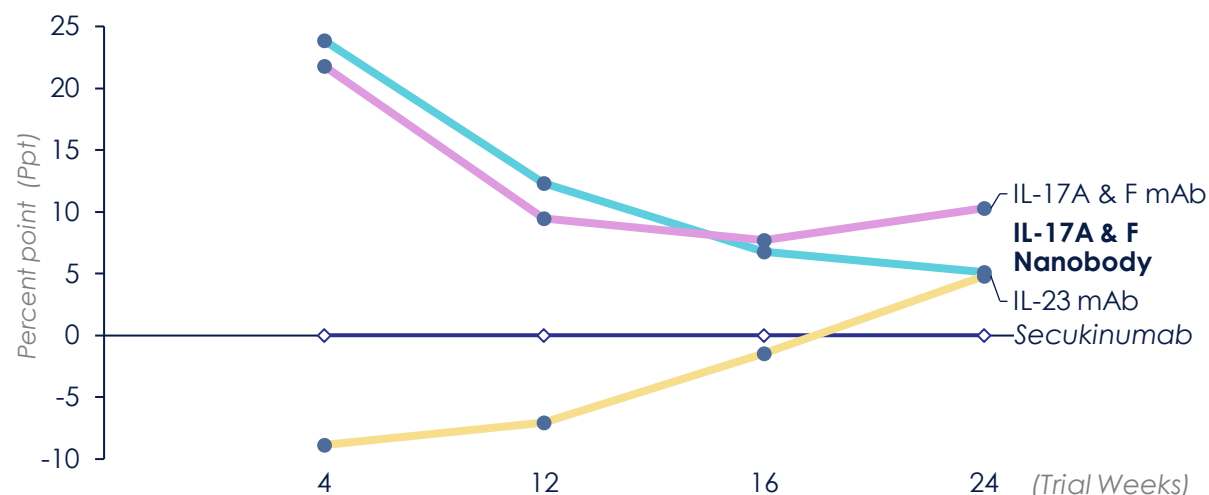


PsO: SLK has best relative performance in PsO for highest PASI

PASI 100 delta to in-trial active comparator secukinumab



PASI 90 delta to in-trial active comparator secukinumab



Key Notes

- All selected trials are double-blinded and use **secukinumab as active comparator**¹ – therefore permit **match-adjusted indirect comparisons** (MAIC) for same timepoints and same response scores
- SLK performs better at higher PASI – clear **leader on PASI100**
- SLK **never underperforms SEC** (at any time or PASI)
- SLK gap to BKZ at lower PASI always $\leq 5\%$, except **PASI100 where its $>10\%$ better**, over time to 24 wks
- **IL-23s** also lose advantage with high PASI, and **come under IL-17A and F MoA on PASI90 and 100**
- **SLK continues adding response** benefit and maintains response beyond 24 weeks²

¹ SLK (sonelokimab, IL-17 A & F Nanobody), Phase 2 trial (comparison is based on long-term data using the 120 mg load then Q4W (Figures of trial paper)); BKZ (bimekizumab, IL-17A & F mAb), BE RADIANT trial (comparison is based on long-term data using the 320mg Q4w arm (maintenance, data extrapolated from figures of trial paper)); GUS (guselkumab, IL-23 mAb), ECLIPSE trial (comparison is based on long-term data using the 100mg at wk 0 and wk 4 then Q8W (data extrapolated from figures of trial paper)); All trials are double blinded over the period and use same dosing regimen for secukinumab as approved. ² Reich et al., 2022, BJD, <https://doi.org/10.1111/bjd.21617>
Source: MoonLake, Peer reviewed publications

Approach to clinical design

- Trials started for **Hidradenitis Suppurativa (HS)** and **Psoriatic Arthritis (PsA)**, high unmet need diseases
- Trials illustrate our **pivotal design approach**:
 - **Larger size** than usual with **several arms**, incl. placebo and active reference **cross-overs**
 - Double-blinded, controlled trials, blinded post-cross over – **no open-labels, uncontrolled trials**
 - “Pivotal” designs to **accelerate** for well-planned superiority Phase 3s, including **dosing options**
 - Always **inclusive of Placebo AND active reference** (namely Humira) to plan Phase 3 and already mark differences to a “soon-to-be” global biosimilar
 - **Higher treatment goal as Primary Endpoint** vs standards (HiSCR75, ACR 50) to distinguish SLK, increase delta to placebo
- Anticipated read outs in 2023

Global Phase 2 program

Hidradenitis suppurativa



- Start date: **May 2022**
- End of screening: **Jan 2023**
- LP randomized: **Feb 2023**
- **234 patients** (vs. 210 target)
- **Fastest** recruitment in HS
- **57 activated sites** (US and Europe)
- **On-target baseline** comparable with main competitor pivotal trials
- PE read-out: **June 26 2023 (R&D Day)**
- **24-wk read-out** expected: Oct 2023

Psoriatic Arthritis



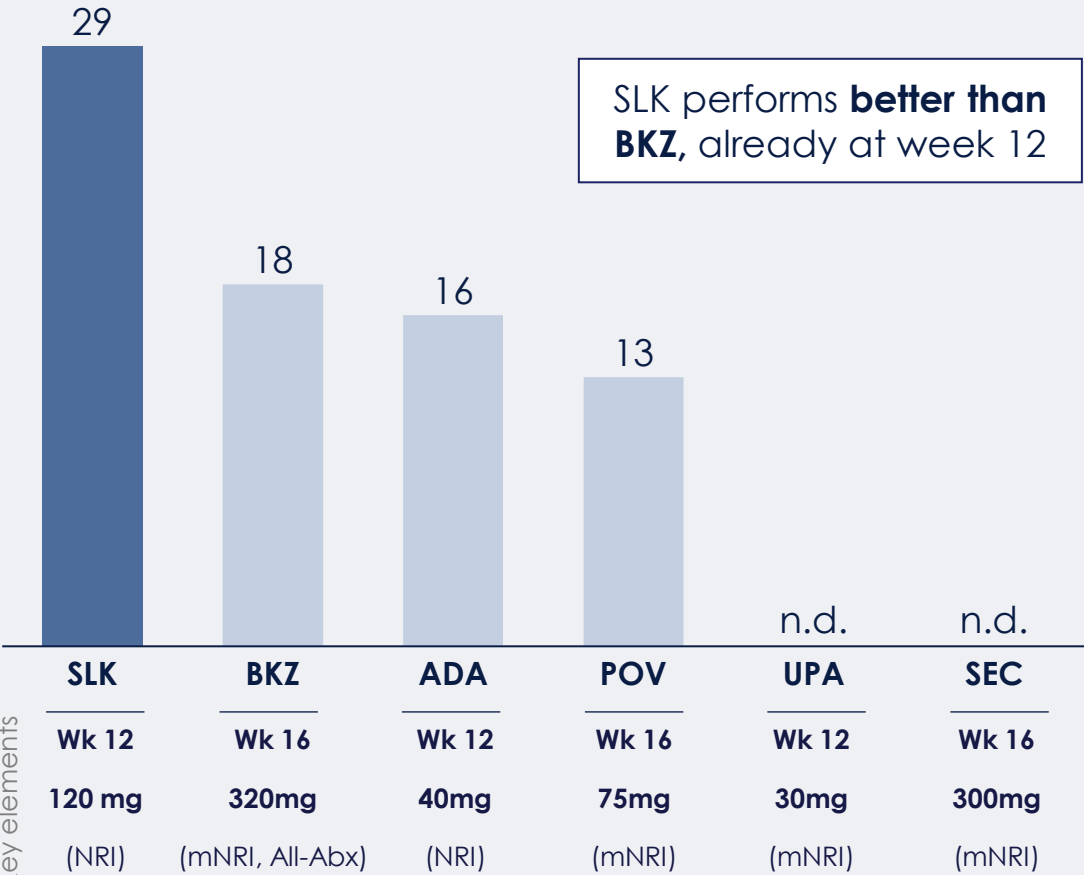
- Start date: **Dec 2022**
- Predicted LP randomized: **June 2023**
- Trial randomized **well ahead of plan**
- **5 arms**: 3 doses, placebo & Humira
- **207 patients**
- **~65 sites activated** (US and Europe)
- PE read-out expected: **Early Nov 2023**
- **24-wk read-out** expected: early 2024

HS: Setting a new bar in HS for primary endpoints

HiSCR75 delta to PLC (Primary endpoint only for SLK)

Percent delta for best doses, primary analysis

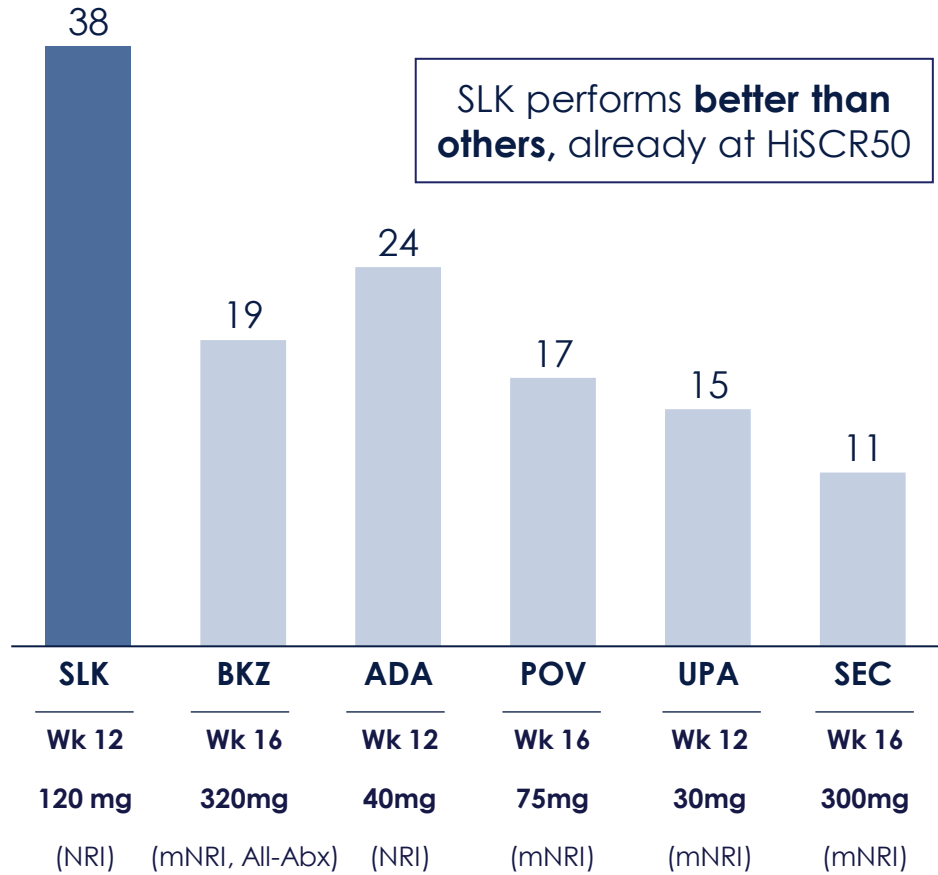
*p=0.0002



HiSCR50 delta to PLC (Primary endpoint for others¹)

Percent delta for best doses, primary analysis

*p<0.0001

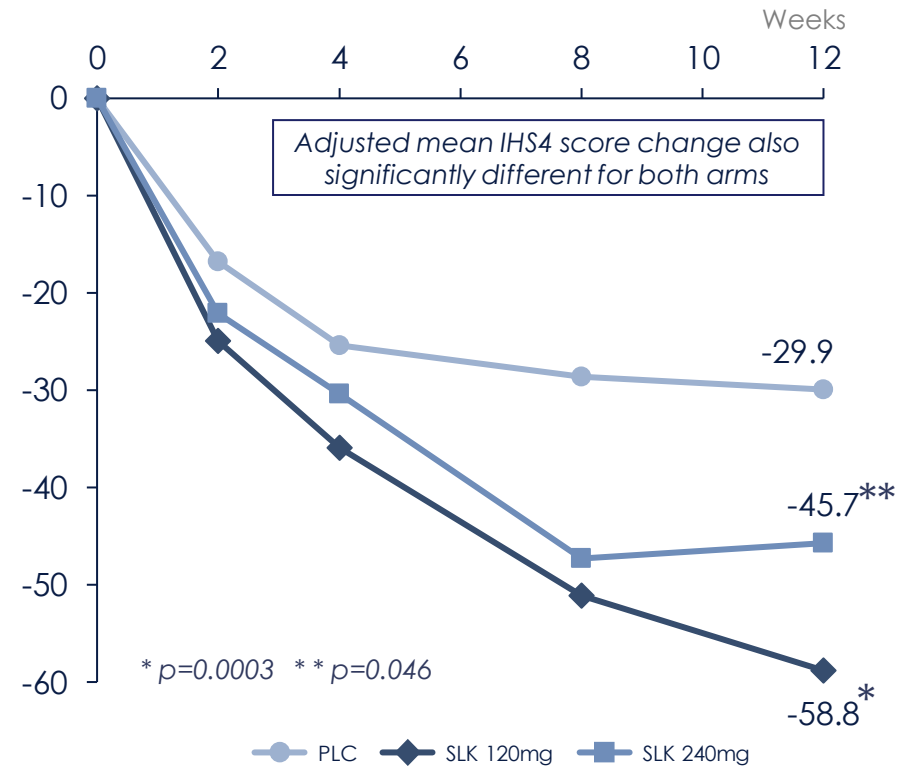


Note: This is a comparison across trials, with inherent limitations. No head-to-head trials. 1 POV used mean AN count reduction as primary endpoint 2 Estimation of HiSCR75 delta to PLC at wk 16 using the avg. response of the 2 crossed-over groups from PLC to SLK doses at wk 12 (PLC plateaus already from week 8) and the wk 16 response for the 120mg arm; PLC, Placebo; SLK, Sonelokimab (MIRA study); BKZ, Bimekizumab (pooled BE HEARD I/II); ADA, Adalimumab (pooled PIONEER I/II); POV, Povorcitinhib (NCT04476043); UPA, Upadacitinib (NCT04430855); SEC, Secukinumab (pooled SUNRISE/SUNSHINE)
Source: MoonLake Clinical

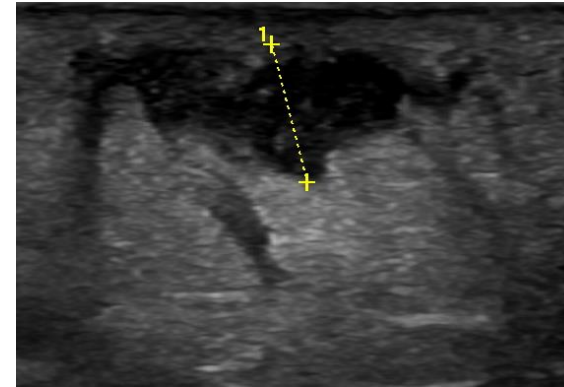
HS: SLK significantly improved deep complex lesions at Week 12

IHS4 adjusted mean change

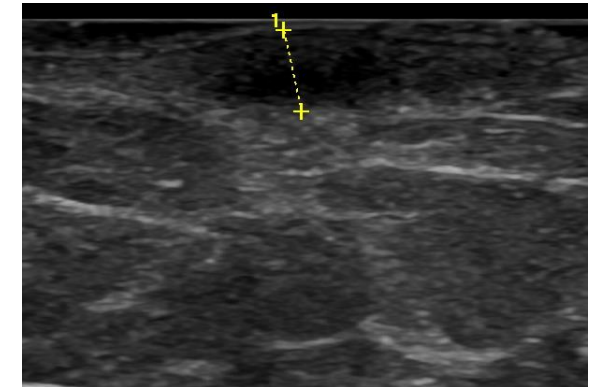
Percent (%) change from baseline over time, ITT



Direct evidence of DT changes



Deep dermal tunnel at baseline (before treatment)



Week 12 (120mg sonelokimab)

- Case from ultrasound sub-study
- Confirming reduction of tunnel activity and morphology in patients receiving sonelokimab
- Reduction of lumen observed, as well as disappearance of neutrophil influx ("pus")

SLK improves the IHS4, a weighted composite score that quantifies **changes in tunnels, nodules and abscesses** – indicates that SLK reduces draining tunnels in patients, the **most complex inflammatory lesion** in HS

1 IHS4 score is calculated as $\sum (n \text{ of nodules} \times 1, n \text{ of abscesses} \times 2, n \text{ of draining tunnels} \times 4)$

*, ** nominal p-values, from MMRM including co-variables: baseline IHS4; Hurley Stage; prior biologic use; visit; treatment and visit-by-treatment interaction

HS: SLK confirmed as only drug with deep responses across scores

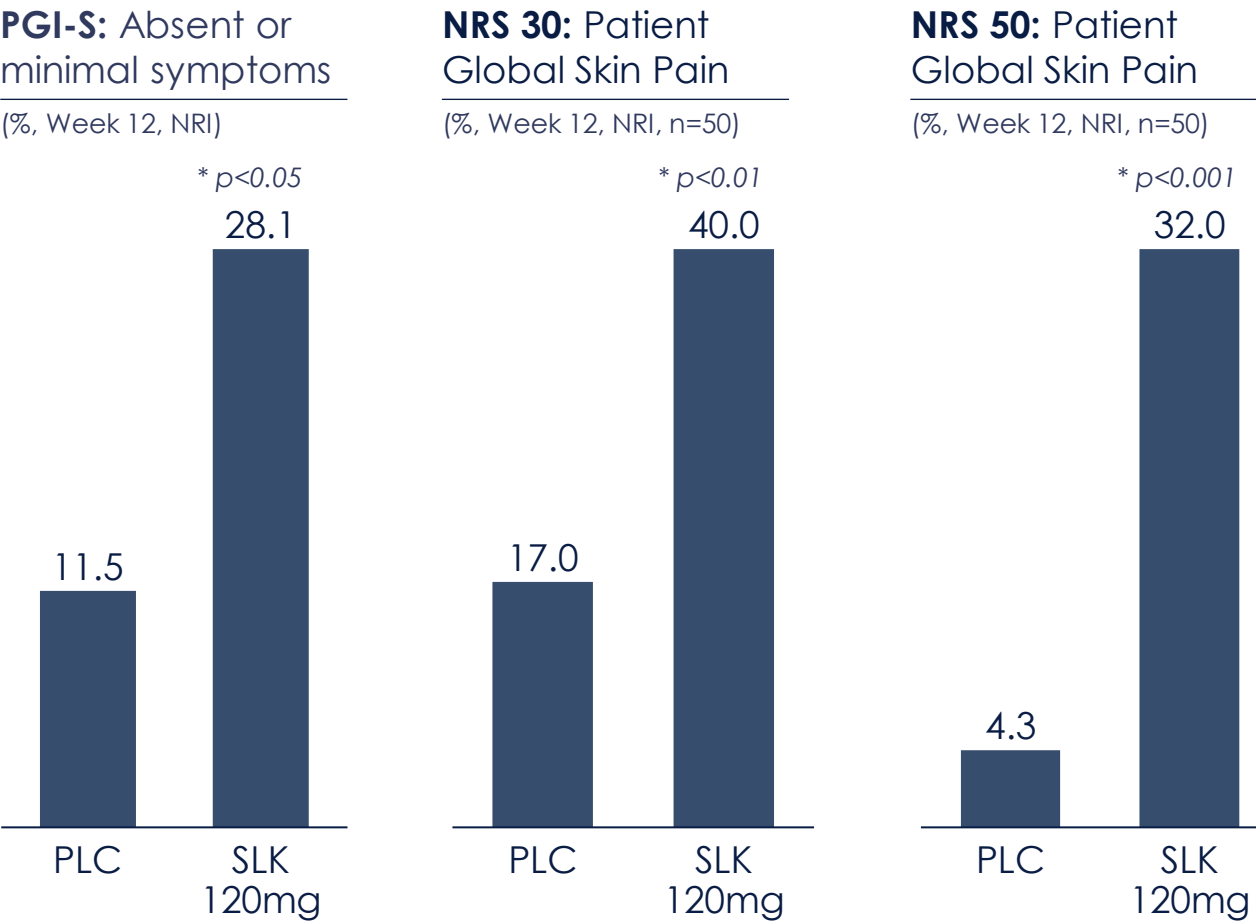


Late breaking session at EADV¹ (Oct 11th 2023)

- **Peer-reviewed** scientific conference presentation of **unique results for SLK** – first (and only time) **HiSCR75** as primary endpoint
- Confirmation of **differentiated depth of responses** across clinical scores, incl. HiSCR90, IHS4, DT100 & Patient Report Outcomes
- **Safety profile in line** with previous tested indications and favorable for patients
- **Matching** of leading **IL-17A & F inhibition** MoA **AND molecular characteristics** (not “either/or”) to meet KOL & patient hopes
- Recognition of **strength of pivotal-like design of MIRA trial**, in contrast with several recent HS trial failures

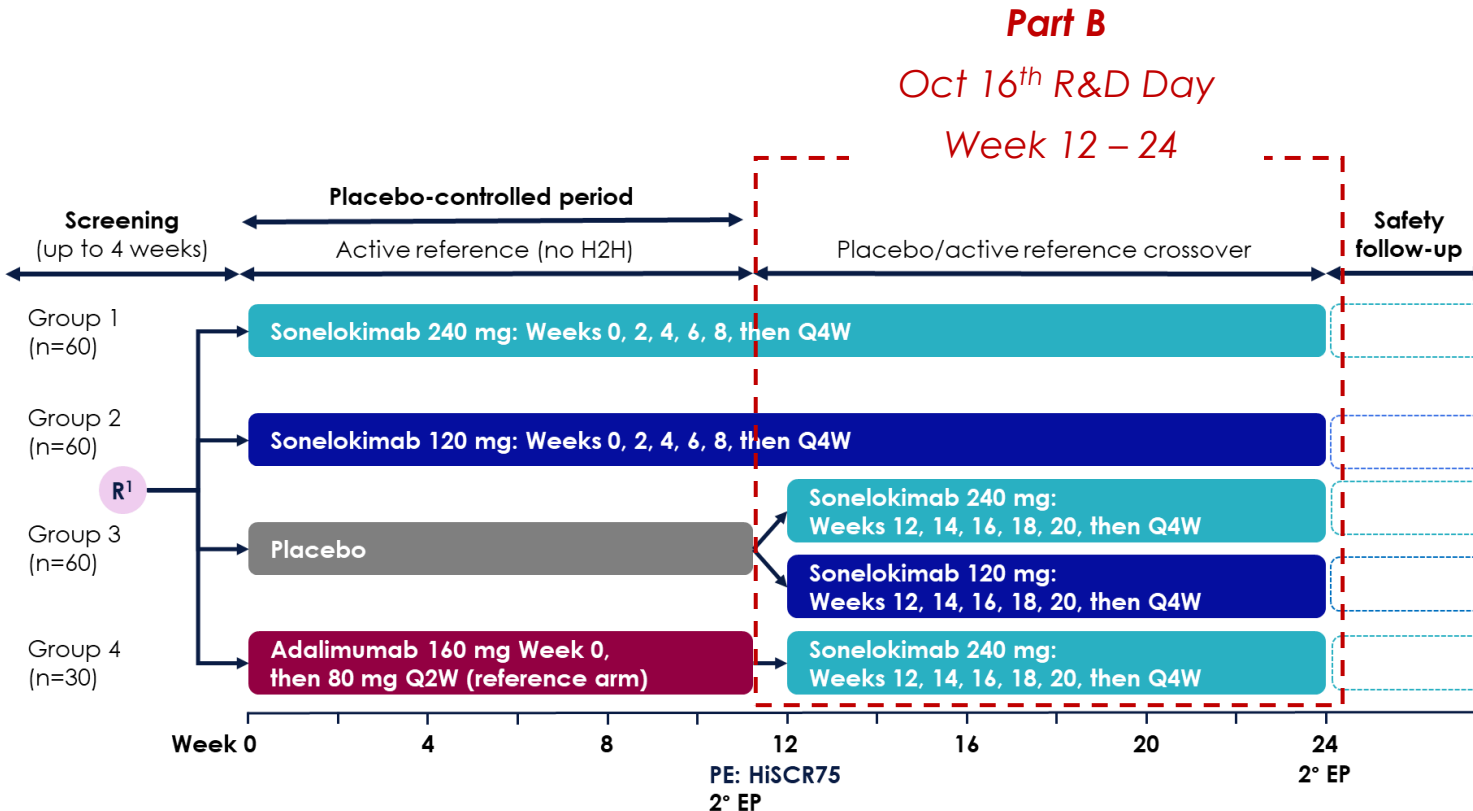


Additional insights on SLK depth of response at 12 weeks



“Totality of evidence” on SLK continues to accumulate and confirms promise of differentiated profile in HS







¹ <https://ir.moonlake.com/events/event-details/eadv-2023-moonlake-scientific-presentation> * Nominal p-value from a Cochran-Mantel-Haenszel test stratified by Hurley Stage and prior biologic use Notes: PGI-S absent or minimal symptoms is defined as a PGI-S score of ≤1 in participants with a PGI-S score of >1 at baseline; NRS is based on the Patient Global Assessment of Skin Pain. NRS 30 is defined as a ≥30% and a ≥1-point improvement from baseline in NRS score in participants with ≥3 NRS at baseline. NRS 50 (post-hoc analysis) is defined as a ≥50% from baseline in NRS score in participants with ≥3 NRS at baseline.



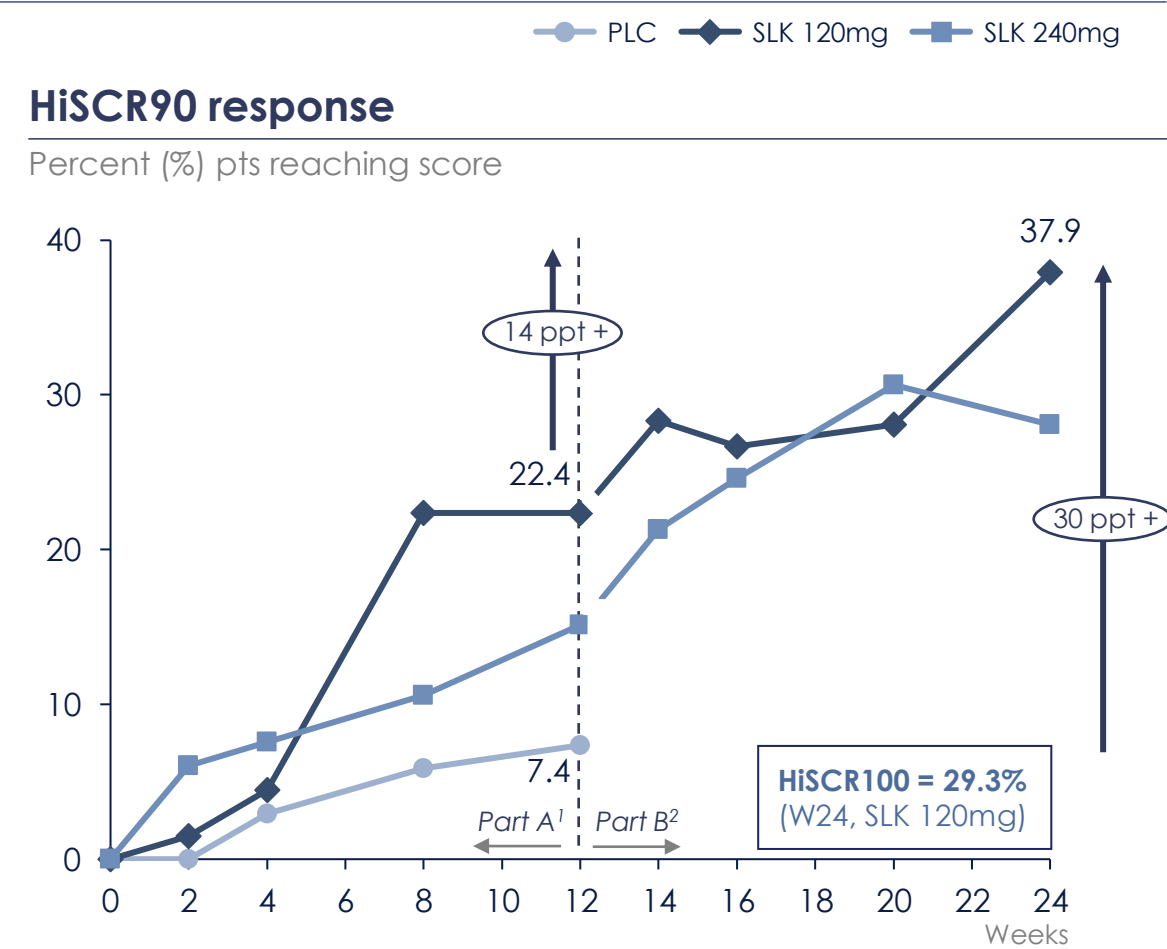
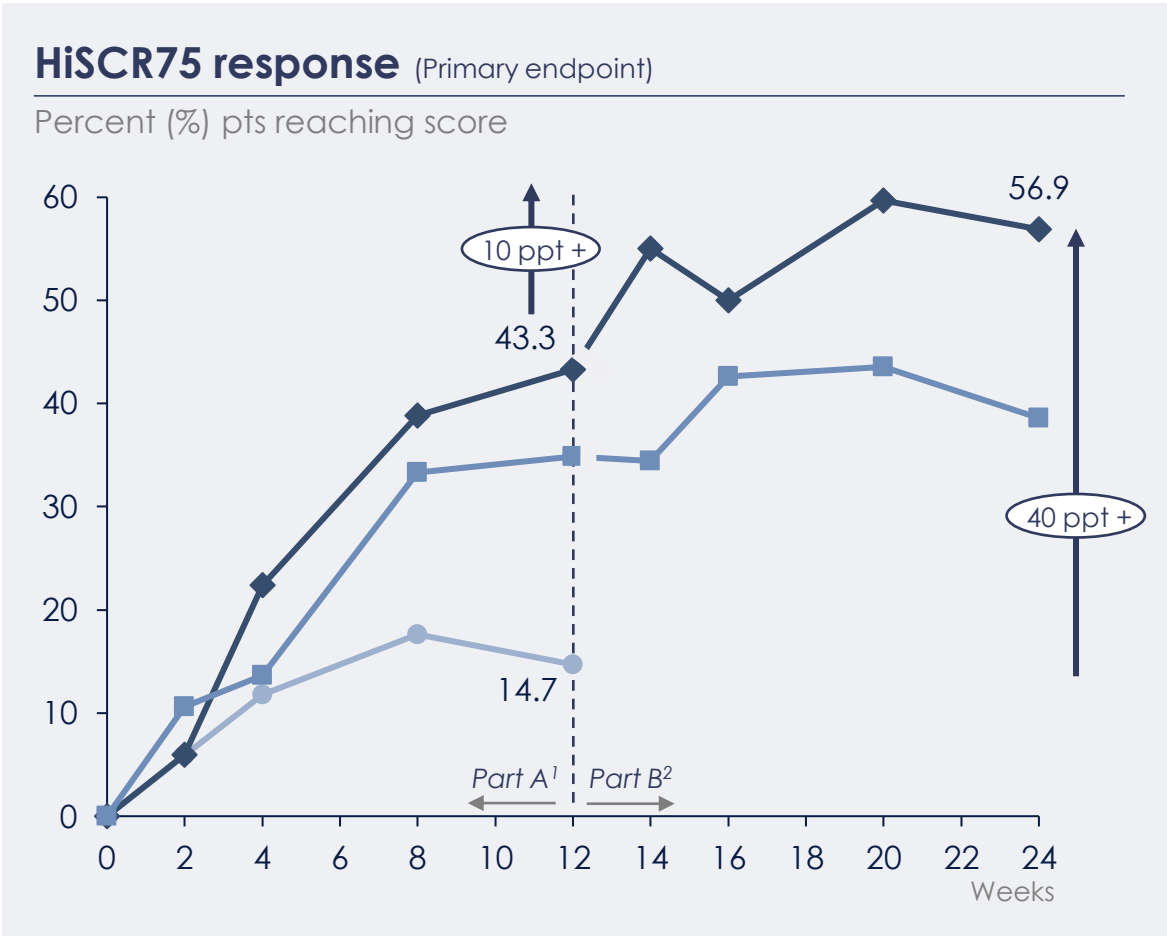
Objectives of Part B

- **Confirm & strengthen the differentiated profile** of SLK to ensure data package shows is unique
 - **Sustained or improved responses at deeper scores** across endpoints to permit **unique disease control** in HS
 - Confirm dosing for Ph3 & TNF switching
 - Verify **safety profile** & benefit-risk ratio
- **Address concerns** that might remain from Part A (e.g., dose behavior, trial quality, reproducibility) or from any **recent issues of competitors** (e.g., level of responses due to A&F as a better MoA, little discontinuations, no dramatic therapy issues)
- Create **transparency on the expected outcomes** from Phase 3 and the final anticipated **de-risking of SLK in HS**



-  **Higher HiSCR75 with Q4W dosing** 57% of patients reach **HiSCR75** at week 24 with 120mg (10ppt+)
-  **Greater depth of responses** 40% patients reach **HiSCR90 and IHS4-90** by week 24 (14ppt +)
-  **More disease control** 1 in every 4 patients in **inflammatory remission (IHS4-100)** & 40%+ report **absent or minimal** disease activity (PGI-S)
-  **Best dose confirmed** 120mg is **best performing dose** across the board and dose behavior **replicated** from wk 12
-  **Effect on TNF patients** At wk 24 **patients respond better with SLK** vs. ADA; **non-responders reach SLK-like responses** within 12 weeks
-  **Favorable safety profile** **No new** signals, **no IBD**, or **malignancy**, **mAb-like ISR** rate, *Candida* (if present) **transient** and with **no discontinuations**

HS: Response with SLK increases through week 24, with monthly dose



Significant increase in number of patients **reaching HiSCR75** with convenient **monthly injection**
Deepening of responses with monthly maintenance dosing, as close to **40% of patients reach HiSCR90** by week 24

1 ITT-NRI data up to Wk 12 (Part A)

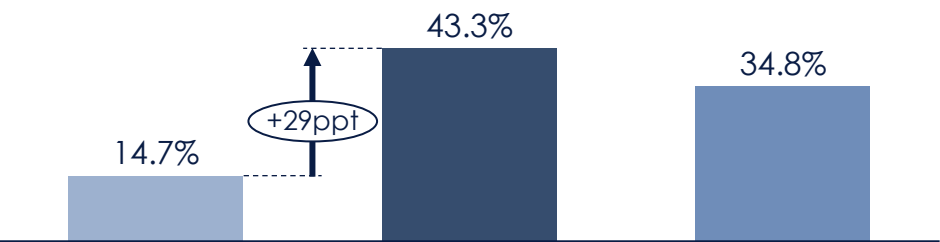
2 As observed data from Wk 14-24 (Part B)

HS: Placebo crossover confirms 120mg as the “winning dose” in HS

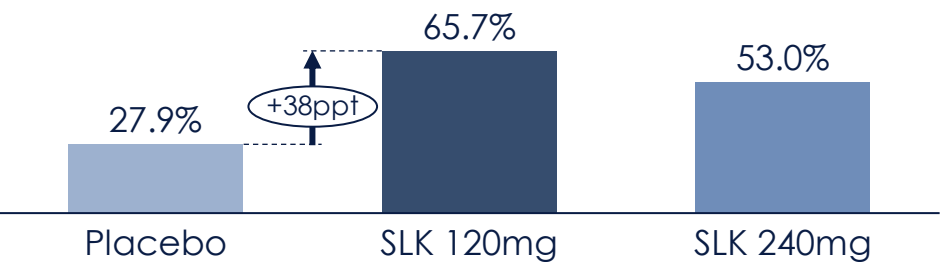
Part A Response Rates

Percent (%) pts reaching score at Week 12, NRI

HiSCR75

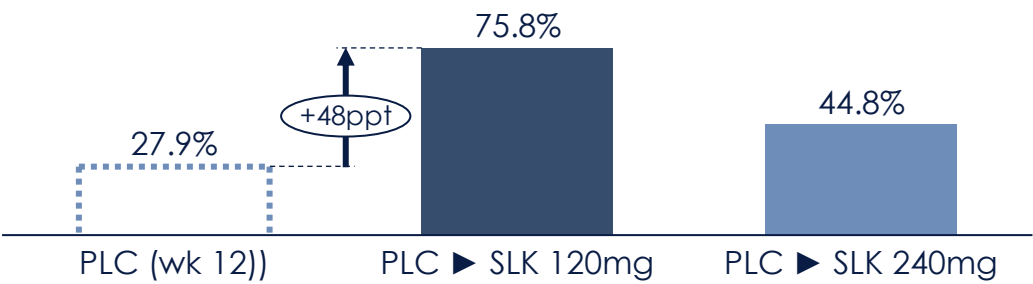
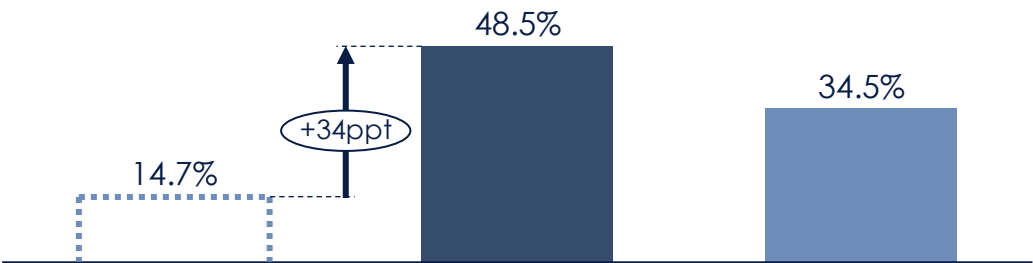


HiSCR50



Part B Response Rates (PLC crossover arms)

Percent (%) pts reaching score at Week 24, NRI



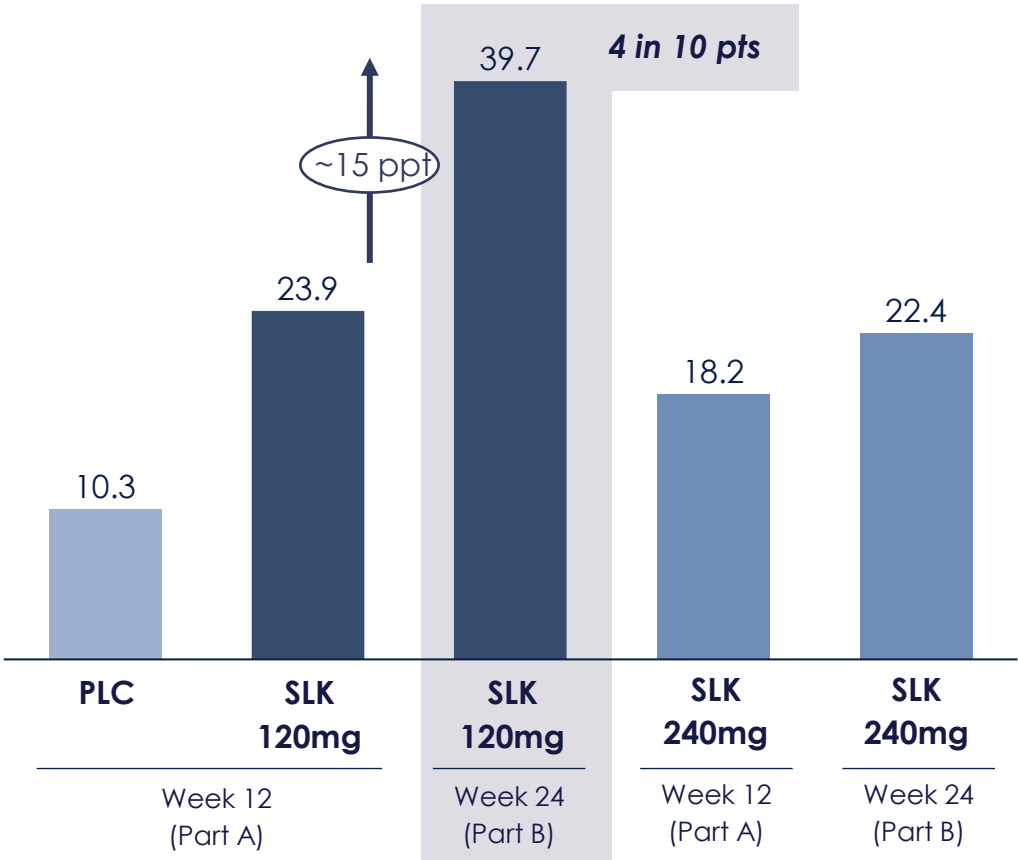
Placebo crossover arms **validate findings from the primary analysis**, including **substantial clinical improvement** for all patients crossed over from placebo, and **similar trends between the two dose arms** as observed in the first 12 weeks

HS: SLK allows patients & physicians to aim for inflammatory remission



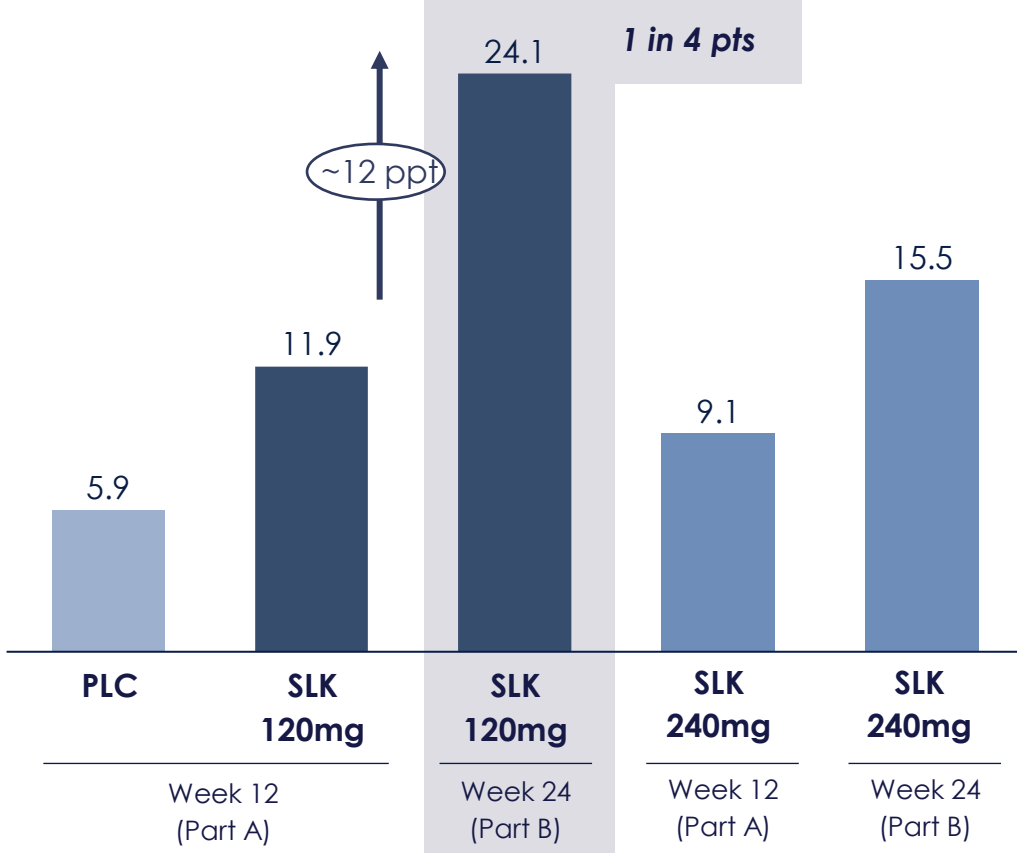
IHS4-90 response

Percent (%) pts reaching score¹



IHS4-100 response

Percent (%) pts reaching score¹



Monthly SLK deepens **IHS4 response** (e.g., IHS4-90 and IHS4-100)
About 1 in 4 four HS patients on monthly SLK 120mg achieve **inflammatory remission** (IHS4-100)

¹ ITT-NRI data up to Wk 12; as observed data after week 12

Research & Clinical Summary

A new bar, a new era

The scientific rationale for a unique molecule

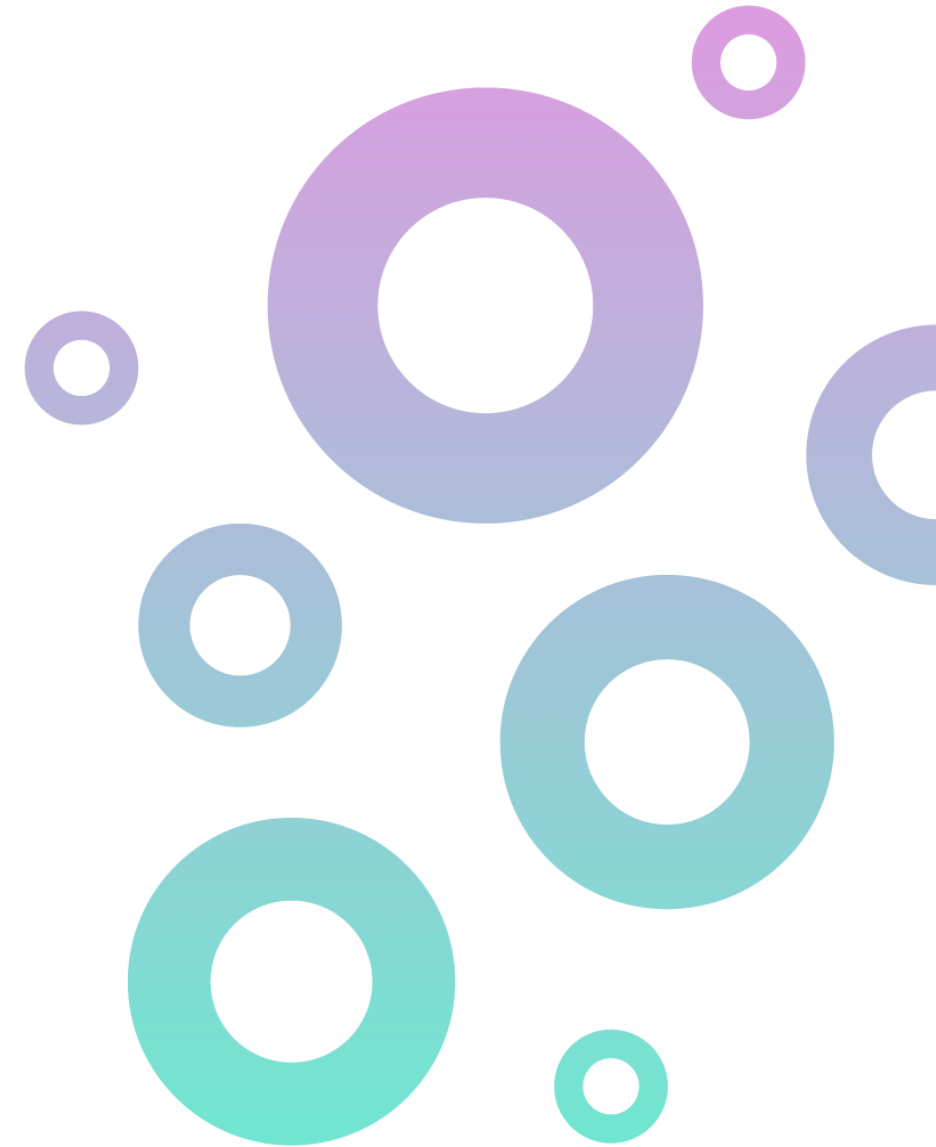
- SLK has unique IL-17F & A binding properties, two key inflammation targets
- SLK has enhanced tissue penetration, reaching where mAbs cannot

What MIRA shows – a full package for a leading drug

- HiSCR75 above all other trials & improving over time with monthly dosing
- Significant effect on tunnels (DT100, IHS4), the deepest inflammatory lesions
- Deepening of responses across different scores (e.g., HISC90)
- SLK allows for complete inflammatory remission over time (IHS4-100)
- Impact on what matters to patients: pain, quality of life, drainage
- Suggested beneficial switching of patients from TNF to SLK
- Favorable safety tolerability profile, as observed previously

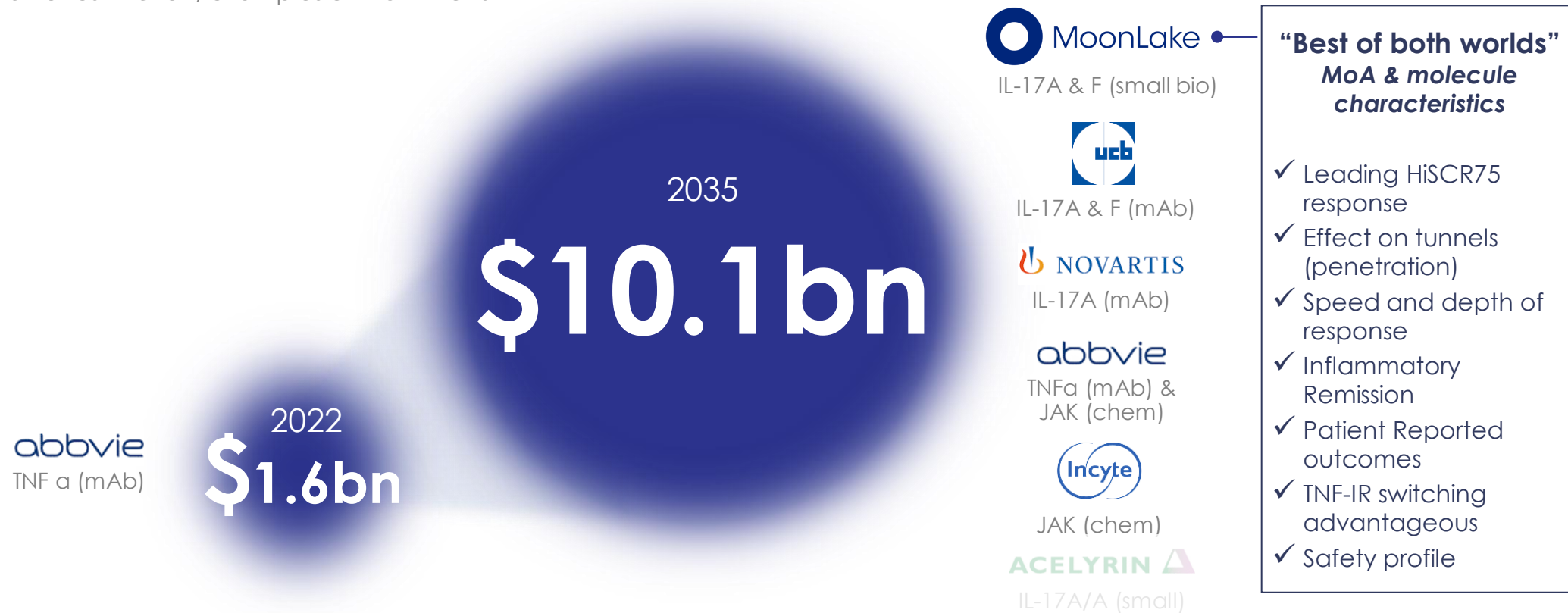
Optimal outcome for further clinical development – Winning dose regimen and endpoints known for phase III; clear PK and ADA further de-risks development

Note: any comparisons across trials that are not head-to-head trials with inherent limitations.



We expect the new data to re-affirm SLK's potential in a large market

US HS Biologics Market estimation, examples of main MoAs



Key drivers

Overall HS True Prevalence	2.1%	2.1%	(can be up to 4%, esp. in the US)
Proportion of Mod-to-Severe with HS Diagnosis	~7%	~19%	(growth as per current US claims)
Biologics Use	~7%	~13%	(as psoriasis over the last 12 years)

External estimations ranging now from 4-10bn, to our knowledge, with variation around prevalence and pricing

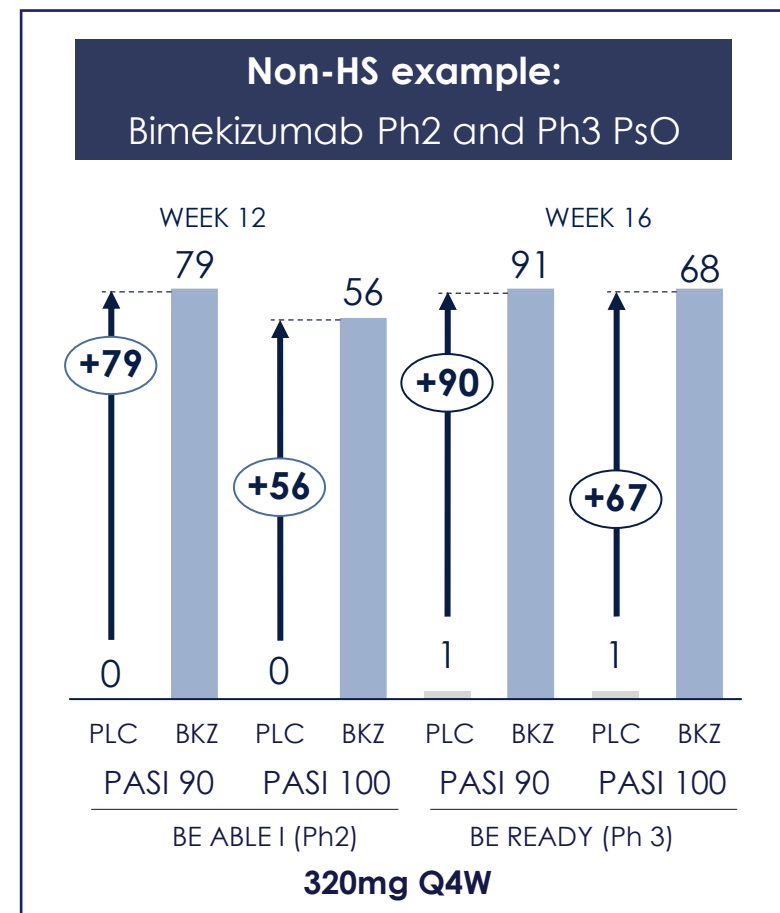
1 'Hurley III Hidradenitis Suppurativa Has an Aggressive Disease Course', Annika et al., Dermatology 2018, doi: 10.1159/000491547

HS examples

- ✗ Adalimumab Ph2 HS results (double-blind, placebo-controlled, two active dose arms, n = 154 patients, n = 26 sites; *milder patients, biologic-naive*) **were not fully replicated** in Ph3 results (approx. 12% drop in Δ HiSCR50)^{1,2}
- ✗ Secukinumab Ph2 HS results (open-label, uncontrolled, n = 20 patients, single-center) **were not predictive** of Ph3 (approx. 26% drop in HiSCR50 response)^{3,4}
- ✗ Bimekizumab Ph2 HS results (double-blind, placebo-controlled, active reference, one dose of BKZ, n = 88 patients, bayesian-augmented control design) **were not fully replicated** in Ph3 (approx. 11% drop in Δ HiSCR75, Be Heard 2)^{5,6}

Non-HS example

- ✓ Bimekizumab Ph2 PsO results (double-blind, placebo-controlled, 5 active dose arms, n = 250 patients, multiple sites in 6 countries) **were predictive** of Ph3 results^{7,8}



Pivotal-like MIRA design is replicated in Ph3, in contrast to other trials

BKZ Ph2^{1,2} 90 participants

BKZ Ph3 (BH II)^{3,4} 509 participants

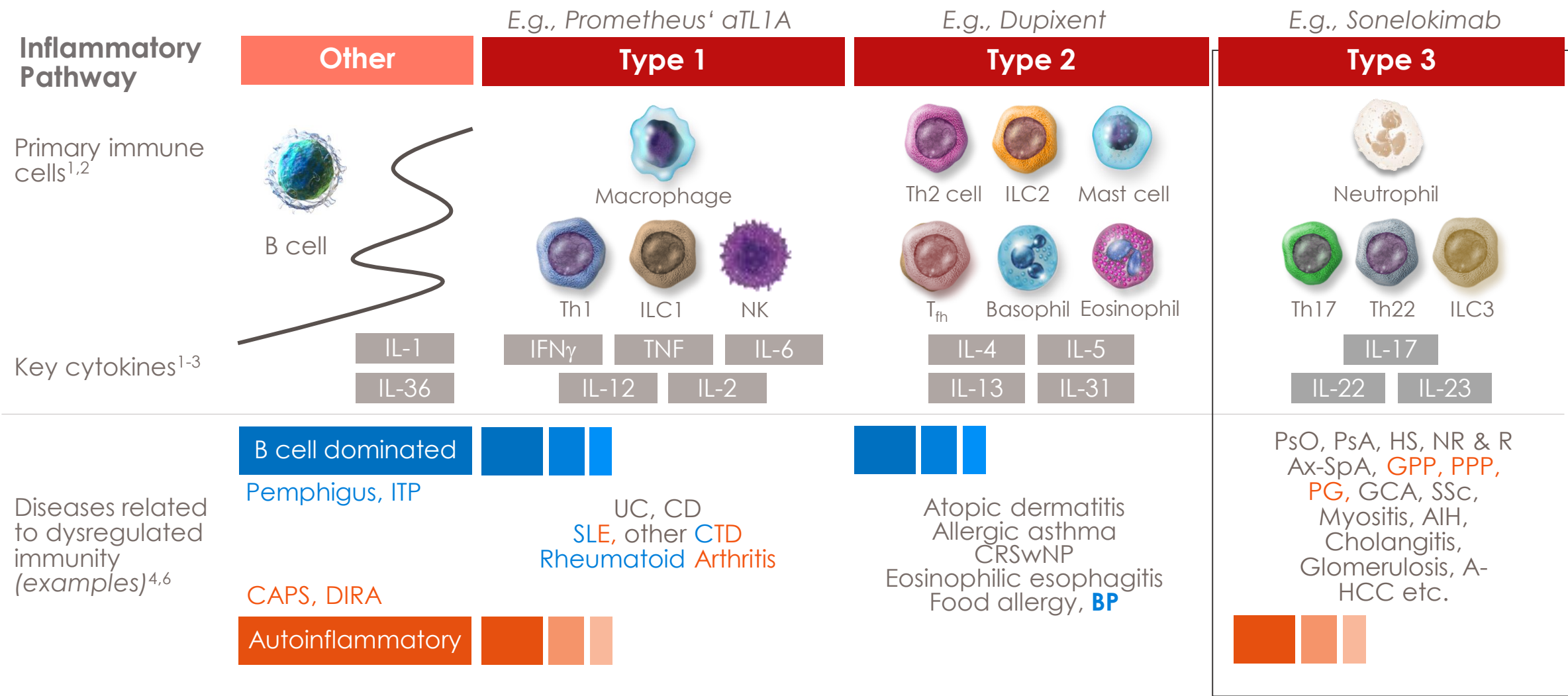
MLTX Pivotal-like (MIRA) 234 participants

Trial structure		
Only one dose tested	Two doses tested	Two doses tested
Loading dose	No loading dose	No loading dose
21 patients received placebo	74 patients received placebo	68 patients received placebo
Double Blinded/Placebo-controlled	Double Blinded/Placebo-controlled	Double Blinded/Placebo-controlled
Stats analyses		
PPS with no clear Abx Tx rules	ITT with Abx Tx rules	ITT with Abx Tx rules
NRI, as observed ⁵	mNRI	NRI
Bayesian augmentation +40 PBO, ADA	No Bayesian augmentation	No Bayesian augmentation
9% placebo HiSCR 75	16% placebo HiSCR 75	15% placebo HiSCR 75
12% discontinuations primary period	~8% discontinuations primary period	~5% discontinuations primary period
Cohort characteristics		
No prior TNFi or IL-17i	Not reported	Prior TNFi or IL-17Ai permitted ⁶
Mandatory topical antiseptic	No mandatory antiseptic	No mandatory antiseptic
≥3 AN lesions	≥5 AN lesions	≥5 AN lesions
Mean AN # 14.5 BKZ vs 22.1 PBO	Mean AN # 16.5 (not reported by arm)	Mean AN # 14.6 SLK 120 vs 14.5 PBO
No inflamm. comorbidities except inactive IBD	Active skin diseases, sarcoidosis, SLE, IBD excluded	Only skin/IBD excluded
49% Hurley II	61% Hurley II	64% Hurley II
No limit on concomitant Abx (% not reported)	Concomitant Abx limit not reported (9% at baseline in overall population)	30% limit on concomitant Abx (11% at baseline in overall population)
1 stratification factor (Hurley)	2 stratification factors (Hurley, ABX)	2 stratification factors (Hurley, prior Bx)

¹ Glatt et al. JAMA Dermatol 2021;157:1279–88; ² NCT03248531; ³ Kimball et al. AAD 2023;oral presentation; ⁴ NCT04242498; ⁵ Sensitivity analysis presented as key data in primary publication; ⁶ No primary failures or patients unsuitable for therapy; Note: comparisons across trials, with inherent limitations. Not head-to-head trials. Not all trial details might be captured in full.

Source: MoonLake Clinical

SLK is the potential leader in Type 3 diseases



A winning MoA...

- **Highest responses**

*IL-17A & F inhibition showed **highest & most durable responses** (BKZ & SLK)*

- **Safer inhibition**

Long history of consistent safety for IL-17, where Candida ("thrush") is main adverse event – vs. TB, ISRs, cancer, infections, CV events, death... (with TNFa or JAK1)

- **Only 2 molecules**

SLK & BKZ – top 2 typically get 2/3 of indication bio sales (avg. \$4bn+)¹

... and a differentiated molecule

- **Elevated Performance**

SLK shows highest performance at elevated treatment goals, HiSCR75, IHS4-100 (or PASI100), as well as additional key outcomes for patients

- **Higher goals**

*Highest primary clinical endpoint with **HiSCR75**, with comparisons to gold-standard Humira® (or Cosentyx® on PASI100)*

- **Improved convenience**

*Candida ("thrush") at **lower rates** vs BKZ and **monthly injections** vs. biweekly BKZ (in HS)*

¹ Based on analysis of 2023 sales of 11 indications (PsO, RA, Asthma, AD, AxSpA, CU, SLE, PsA, COPD, CD, UC) – 2030 ranges are even higher

On October 18, 2023, **UCB, Brussels, Belgium announced approval by the U.S. Food and Drug Administration** (the “FDA”) of its IL-17A and IL-17F inhibiting antibody for the treatment of adults with moderate to severe plaque psoriasis.

The label granted by the FDA includes **warnings and precautions relating to, among others, suicidal ideation and behavior (“SI/B”) and liver biochemical abnormalities.**

-----WARNINGS AND PRECAUTIONS-----

- Suicidal Ideation and Behavior (SI/B): May increase risk of SI/B. Advise patients, their caregivers, and families to monitor for the emergence or worsening of depression, suicidal ideation, or other mood changes. If such changes occur, advise them to promptly seek medical attention or call the National Suicide and Crisis Lifeline at 988. Carefully weigh risks and benefits of treatment with BIMZELX in patients with a history of severe depression and/or suicidal ideation or behavior. (5.1)
- Liver Biochemical Abnormalities: Elevated serum transaminases were reported in clinical trials. Test liver enzymes, alkaline phosphatase, and bilirubin at baseline and according to routine patient management. Permanently discontinue use of BIMZELX in patients with causally - associated combined elevations of transaminases and bilirubin. (5.4)

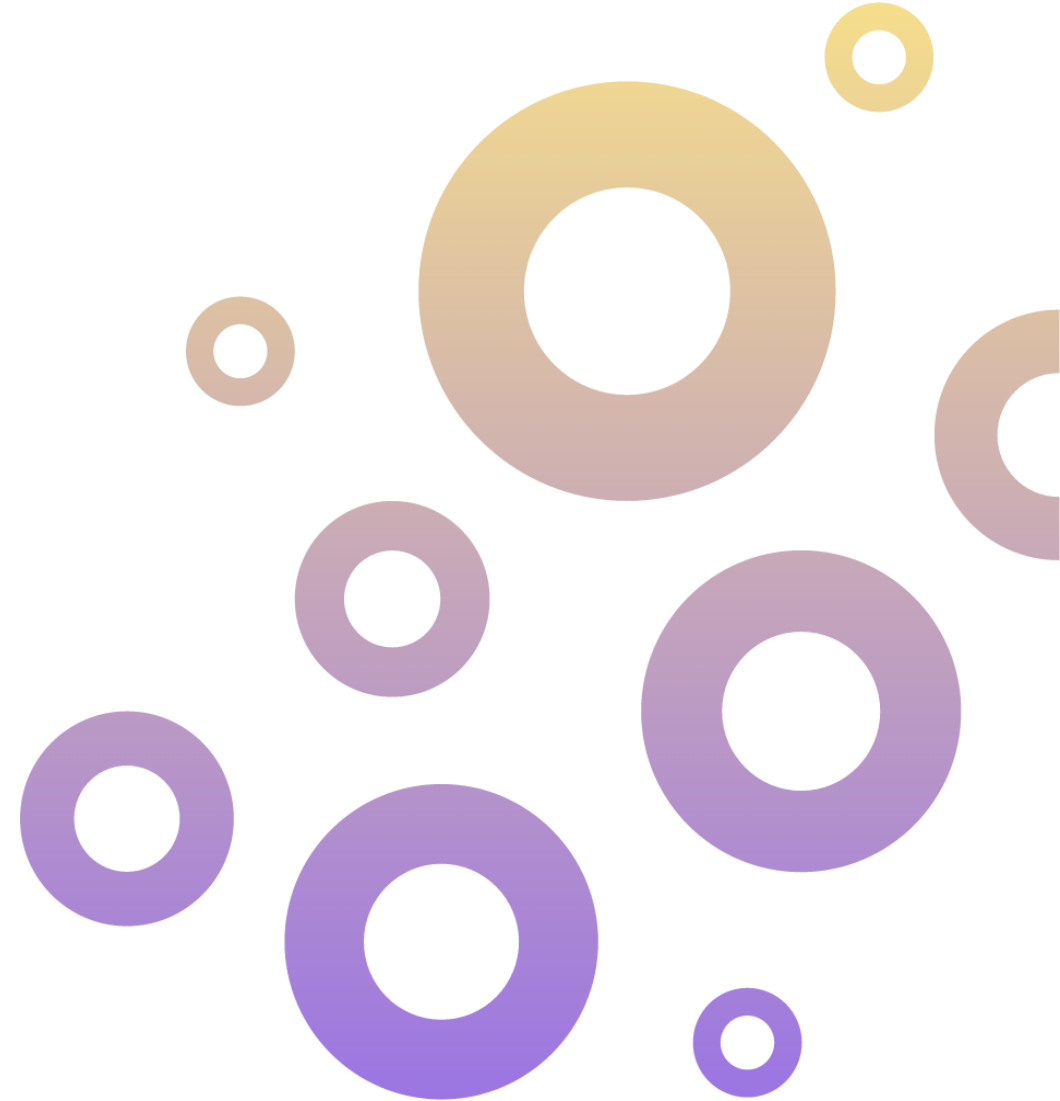
Statement from MoonLake (October 19, 2023)

MoonLake Immunotherapeutics has reviewed available safety data for its investigational tri-specific Nanobody® Sonelokimab (“SLK”).

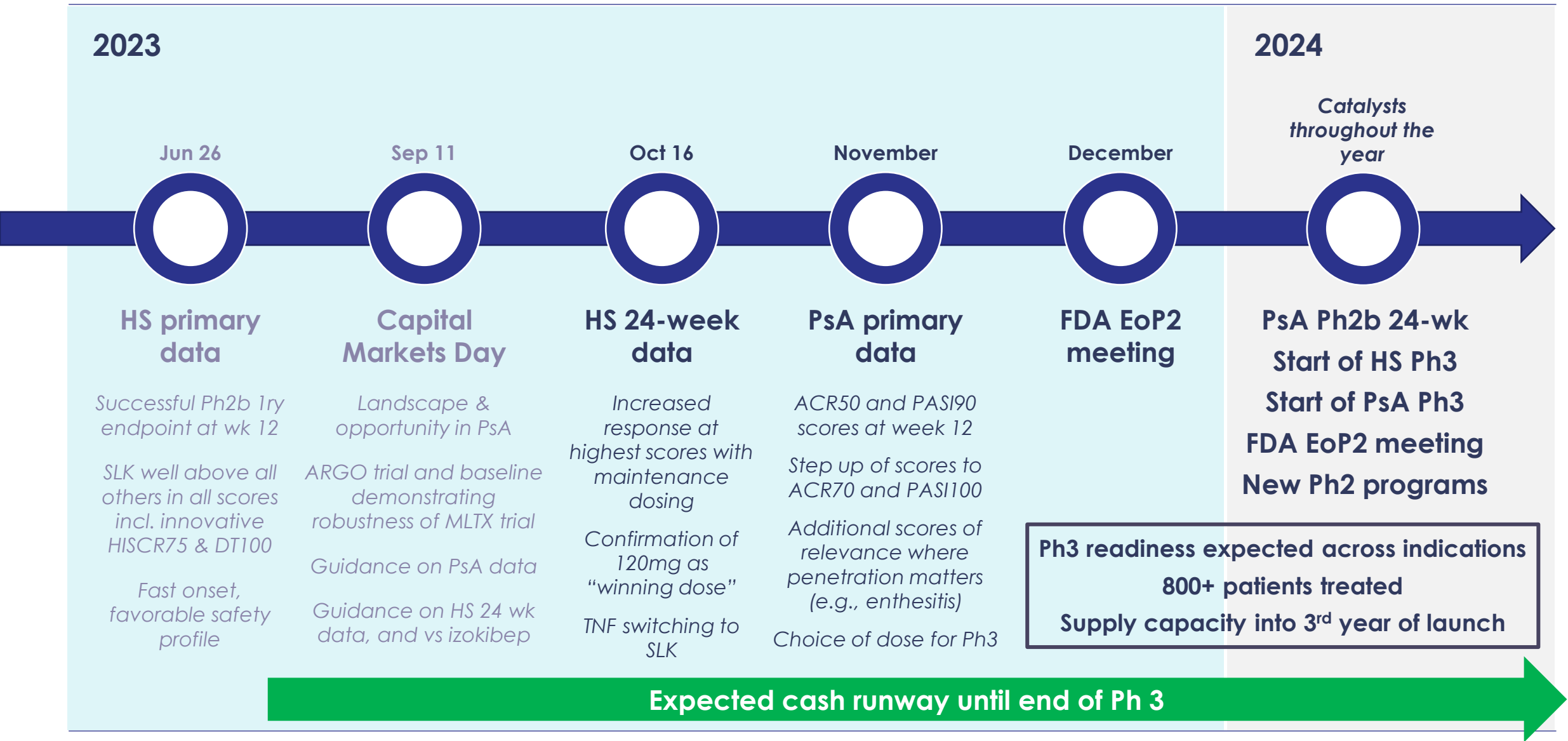
*In the overall SLK clinical development program of over 500 patients exposed to date across reported indications, MoonLake sees **no signal of SI/B related to SLK treatment.***

*Similarly, MoonLake **has not observed any signal related to liver enzyme elevations** in patients exposed to SLK.*

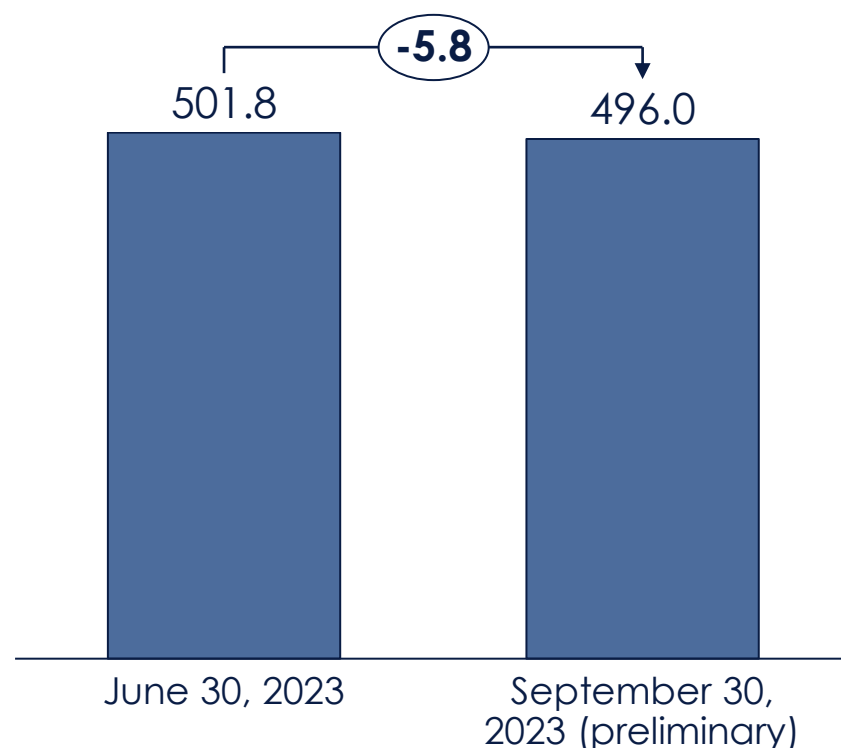
- **Best in class** – SLK is a unique molecule among “next gen IL-17s” and beyond, as now shown in HS and PsO
- **Highly differentiated** – Only two molecules can inhibit all IL-17 pro-inflammatory dimers, only SLK combines that MoA with unique molecular characteristics
- **MLTX = Robust trials** – Comparing apples-to-apples is critical, esp. in diseases like HS, PsA, PsO & others, and only pivotal-like designs derisked insight
- **Multi Bn drug** – SLK may impact very large markets that are growing fast now, with potential over \$70bn, as a leading asset in Type 3 inflammation
- **Our time** – MLTX expects all key readouts among “next gen IL-17s” to end of 2023, and operates from a position of financial stability and strength into 2024



MLTX has a full set of expected catalysts to year end, more in 2024



Cash, cash equivalents & short-term marketable securities in USD M



- **Strong balance sheet with USD 496M** in cash, cash equivalents and short-term marketable debt securities as per September 30, 2023
- **Low cash burn of USD 5.8M in Q3-2023** demonstrating cost-efficient set up and focus of MLTX
- **Expected cash runway until the end of 2026**, covering
 - Completion of ongoing Ph2 programs in HS & PsA
 - Ph3 program in HS
 - Ph3 program in PsA
 - Additional Ph2 program
 - Submission of BLA
 - All other base spend

¹ Differences may not add up due to rounding



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