UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 OR 15(d) of The Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): September 11, 2023

MOONLAKE IMMUNOTHERAPEUTICS
(Exact name of registrant as specified in its charter)

Cayman Islands	**001-39630**	**98-1711963**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

Dorfstrasse 29 Zug, Switzerland	**6300**
(Address of principal executive offices)	(Zip Code)

41 415108022
(Registrant's telephone number, including area code)

N/A
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Securities Exchange Act of 1934:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Class A ordinary share, par value $0.0001 per share	MLTX	The Nasdaq Capital Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

On September 11, 2023, MoonLake Immunotherapeutics (the "Company") will be posting to its website an investor presentation to be used in the Company's September 11, 2023 Capital Markets Day event, including information regarding the Company's financial position, near-term catalysts and publication roadmap. A copy of the presentation is included with this Form 8-K for convenience and attached hereto as Exhibit 99.1. The investor presentation and replays of the webcast will be available on the Company's website at https://ir.moonlaketx.com.

The information in this current report on Form 8-K and the exhibit attached hereto shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act") or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, regardless of any general incorporation language in such filing, unless specifically so incorporated.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	MoonLake Immunotherapeutics Capital Markets Day Presentation dated September 11, 2023
104	Cover Page Interactive Data File-the cover page XBRL tags are embedded within the Inline XBRL document

Signature

Pursuant to the requirements of the Securities Exchange Act of 1934, as amended, the Registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

MoonLake Immunotherapeutics

Date: September 11, 2023

By: /s/ Matthias Bodenstedt
Name: Matthias Bodenstedt
Title: Chief Financial Officer


MoonLake
MoonLake Immunotherapeutics
Capital Markets Day
New York, NASDAQ
September 11th 2023
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W: moonlaketx.com | E: info@moonlaketx.com

Disclaimer

Forward Looking Statements

Certain statements in this presentation may constitute "forward-looking statements" within the meaning of the U.S. Private Securities Litigation Reform Act of 1995. Forward-looking statements include, but are not limited to, statements regarding our expectations, hopes, beliefs, intentions or strategies regarding the future including, without limitation, statements regarding: plans for preclinical studies, clinical trials and research and development programs; the anticipated timing of the results from those studies and trials; potential market opportunities, estimates of market size, and estimates of market growth; potential indications; the timing of regulatory meetings; the occurrence and timing of market engagement; expectations regarding the time period over which our capital resources will be sufficient to fund our anticipated operations; the timing and likelihood of success, plans and objectives of management for future operations and future results of anticipated product development efforts; and effects on liquidity and capital resources, including cash position. In addition, any statements that refer to projections, forecasts, or other characterizations of future events or circumstances, including any underlying assumptions, are forward-looking statements. The words "anticipate", "believe", continue", "could", "estimate", "expect", "intend", "may", "might", "plan", "possible", "potential", "predict", "project", "should", " strive", "would" and similar expressions may identify forward-looking statements, but the absence of these words does not mean that such statement is not forward looking. Forward-looking statements are based on current expectations and assumptions that, while we and our management consider reasonable, as the case may be, are inherently uncertain. New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. Factors that may cause actual results to differ materially from current expectations include, but are not limited to, various factors beyond management's control including general economic conditions and other risks, uncertainties and factors set forth in the section entitled "Risk Factors" and "Cautionary Statement Regarding Forward-Looking Statements" in our Annual Report on Form 10-K that was filed with the U.S. Securities and Exchange Commission (the "SEC") on March 20, 2023, as well as factors associated with companies, such as MoonLake Immunotherapeutics, that operate in the biopharma industry. Nothing in this presentation should be regarded as a representation by any person that the forward-looking statements set forth herein will be achieved or that any of the contemplated results of such forward-looking statements will be achieved. You should not place undue reliance on forward-looking statements in this presentation, which speak only as of the date they are made and are qualified in their entirety by reference to the cautionary statements herein. We neither undertake nor accept any duty to release publicly any updates or revisions to any forward-looking statements to reflect any change in our expectations or in the events, conditions or circumstances on which any such statement is based. This presentation does not purport to summarize all of the conditions, risks and other attributes of MoonLake Immunotherapeutics.

Industry and Market Data

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.

Trademarks

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Source: MoonLake Corporate

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Instructions for this session

Please **take note of the disclaimer** on the previous page

You can **submit your questions** through the Q&A function – questions are only visible to the moderators – we will address **as many questions as possible** at the end of this session

The presentation and a **replay** will be made available on our IR website

For any **technical issues** during the webcast, please also use the Q&A function to request support

Other requests should be directed to ir@moonlaketx.com or media@moonlaketx.com

Source: MoonLake Corporate

Welcome to our Capital Markets Day

MoonLake

Logistics

Date: September 11th, 2023
Time: 11:30-13:00 EDT
Location: Nasdaq Marketsite 10FL, 4 Times Square, New York *(Webcast also available)*

Agenda

Topic	Sub-topics	Speaker	Timing
Introduction	- Welcome & session details - Where MLTX stands and next catalysts	Jorge Santos da Silva	15 mins
Psoriatic Arthritis (PsA) – Unmet need & evolving landscape	- A multi-domain disease with high unmet needs - IL17s in PsA - IL-17F and size as key factors for new therapies - SLK in PsA	Prof. Joseph Merola	20 mins
MLTX PsA ARGO trial update	- Why IL-17 and SLK matter in PsA - What to expect from the ARGO trial - ARGO – Status, baseline, disposition	Prof. Kristian Reich	20 mins
Guidance on upcoming data	- Expectations for ARGO PsA 12wk data - Opportunity in PsA - Focus areas for MIRA HS 24wk data	Jorge Santos da Silva	15 mins
Financial Update	- Q2 Financials and path forward	Matthias Bodenstedt	5 mins
Q&A session			To end

Source: MoonLake Corporate

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Introduction

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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)

Source: MoonLake Corporate

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

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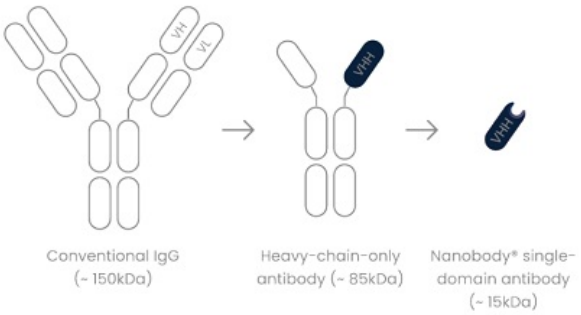
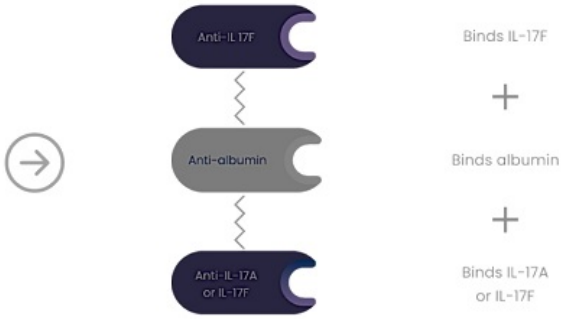
Nanobodies® are much smaller than traditional antibodies



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

Source: MoonLake Research

It's all about the dimers

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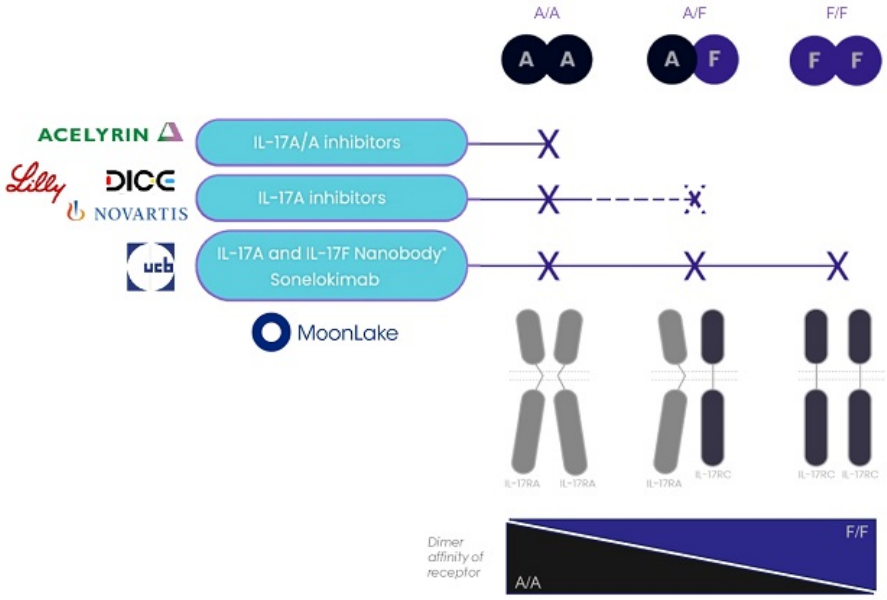
Illustrative

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



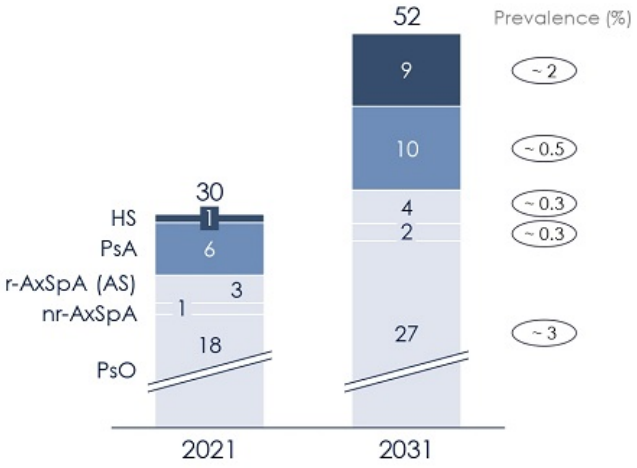
1 Liu S, et al. Nat Commun. 2013;4:1888; 2 Goepfert A, et al. Immunity. 2020 Mar 17;52(3):499-512

Source: MoonLake Research

The broad IL-17 class is expected to lead in a growing market

MoonLake

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

Source: Clarivate's Disease Landscape & Forecast 2021-2031 (last update: PsO (moderate to severe), PsA in December 2022; SpA in September 2022). HS 2031 projections based on LifeSci MLTX initiation report (assuming SUK value share identical to volume share)

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

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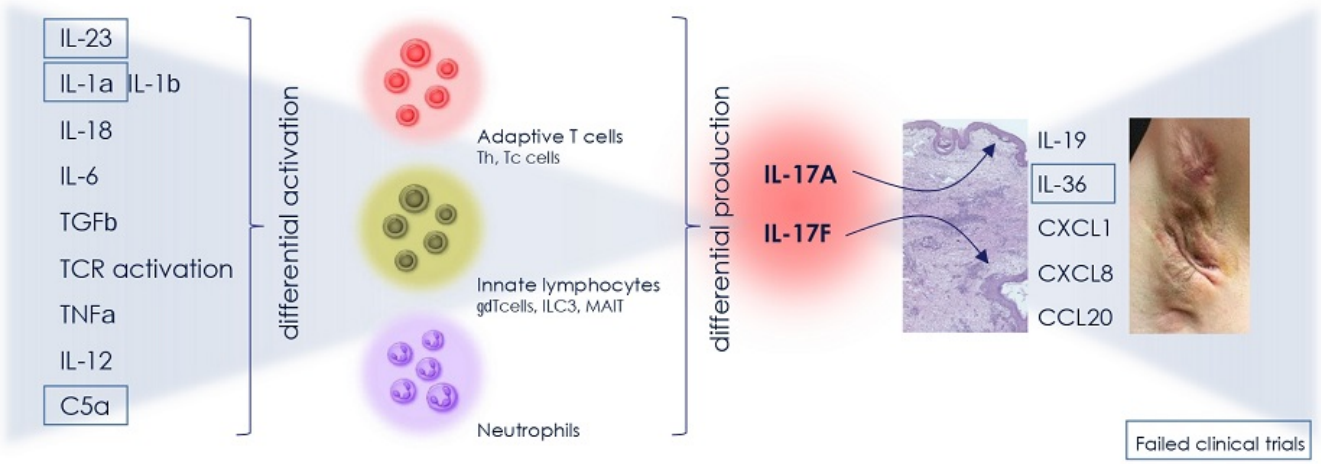
HS Example

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

Different cell types preferentially produce IL-17 A 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



Source: MoonLake Clinical

SLK rapidly becoming a leader in large inflammatory diseases

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	Trial	Patients (n)	Leading MoA	SLK leading asset
 **HS**	Phase 2b (MIRA)	234	IL-17A & F TNF & IL-17A	**Highest** ever primary endpoint (HiSCR75), **largest** delta to placebo at HiSCR75 and 50
 **PsO**	Phase 2b	313	IL-17A & F IL-23 & IL-17A	**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	IL-17A & F inhibition shows **best** ACR/PASI data incl. TNF-IR pts
 *Other Rheum & Derm*	TBA	TBA	IL-17A & F Other	IL-17A & F inhibition **best** data in AS, nr-AxSpA, enthesitis…

PsA primary endpoint data for SLK expected ahead of ACR 2023

Source: MoonLake Corporate

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

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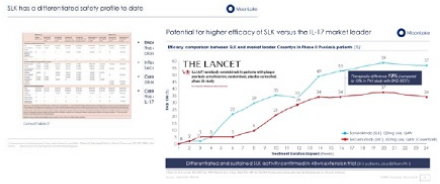
Phase 2 clinical data

THE LANCET

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



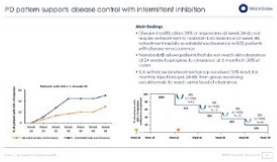
- **Leading efficacy** in Inflammation (PASI 100 for most patients)
- **IL-17F adds to IL-17A** inhibition (vs. Cosentyx, 56% more patients to PASI100)
- **Clean profile** following historical IL-17 safety

BJD British Journal of Dermatology IMPROVING PATIENT OUTCOMES IN SKIN DISEASE WORLDWIDE

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

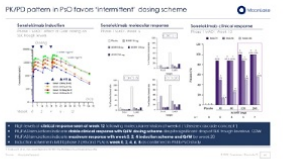


- **Duration of IL-17A & F** response over time
- **Long-term anti-inflammatory** effect of SLK even after withdrawal
- **Continued dosing benefit** in non-/slow responders

Phase 1 & Preclinical data

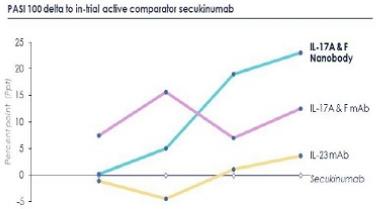
J Am Acad Dermatol
Volume 81, Number 1

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 determined** for all testing doses (incl. 120 and 240mg)
- Stable clinical response with **Q4W dosing**
- **Molecular remission** & high **clinical response** over time

PASI 100 delta to in-trial active comparator secukinumab



- **IL-17A & F inhibition** shows highest levels of skin clearance
- SLK shows **highest levels** of skin clearance (PASI100) **versus BKZ and IL-23s**

Source: MoonLake

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

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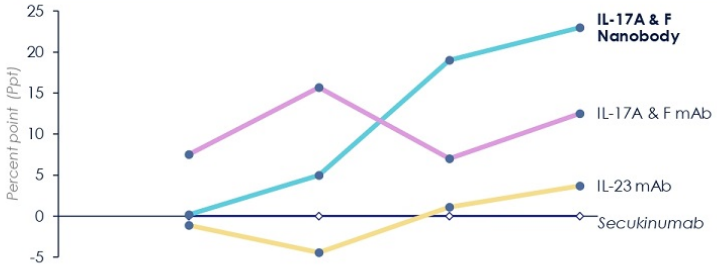
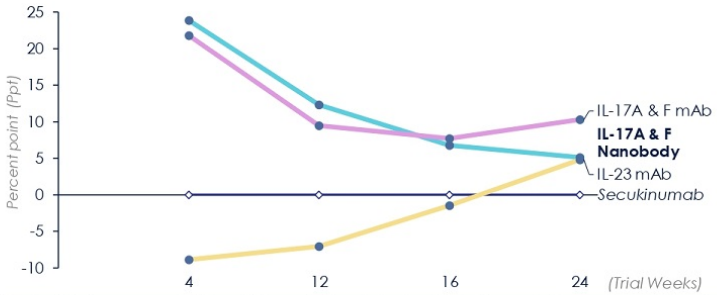
PASI 100 delta to in-trial active comparator secukinumab



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 ≤ 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 weeks[2]

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

We drive large global Ph2 trials to open the SLK pipeline-of-indications

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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

MIRA
NCT05322473

- 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

ARGO
NCT05640245

- 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

Source: MoonLake Clinical Development

HS: SLK reaches the **highest** scores vs others, including **HiSCR75**

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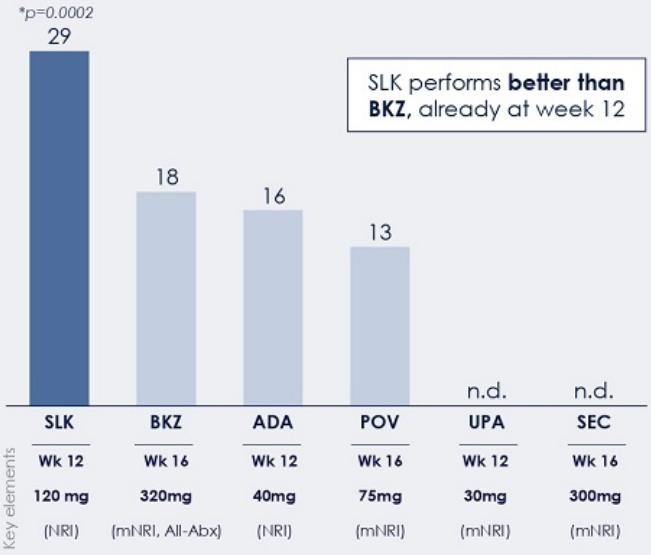
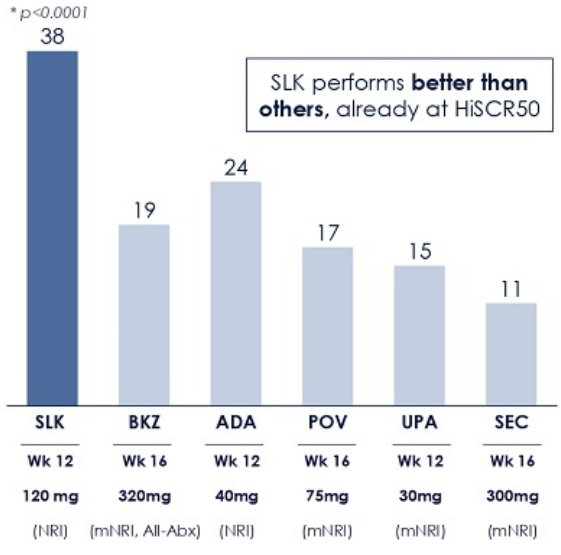
HiSCR75 delta to PLC (Primary endpoint for SLK)

Percent delta for best doses, primary analysis



HiSCR50 delta to PLC (Primary endpoint for others[1])

Percent delta for best doses, primary analysis



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 **improves IHS4** and **changes morphology** of tunnels

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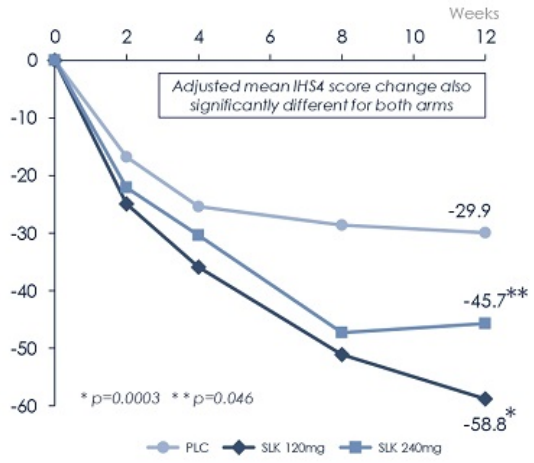
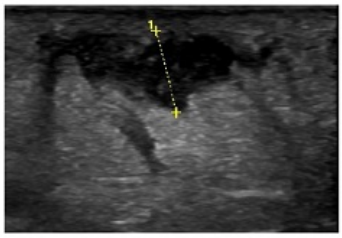
IHS4 adjusted mean change

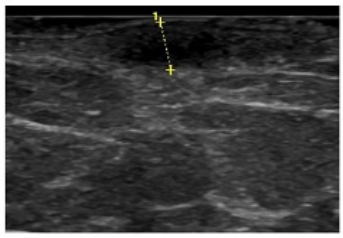
Percent (%) change from baseline over time, ITT



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

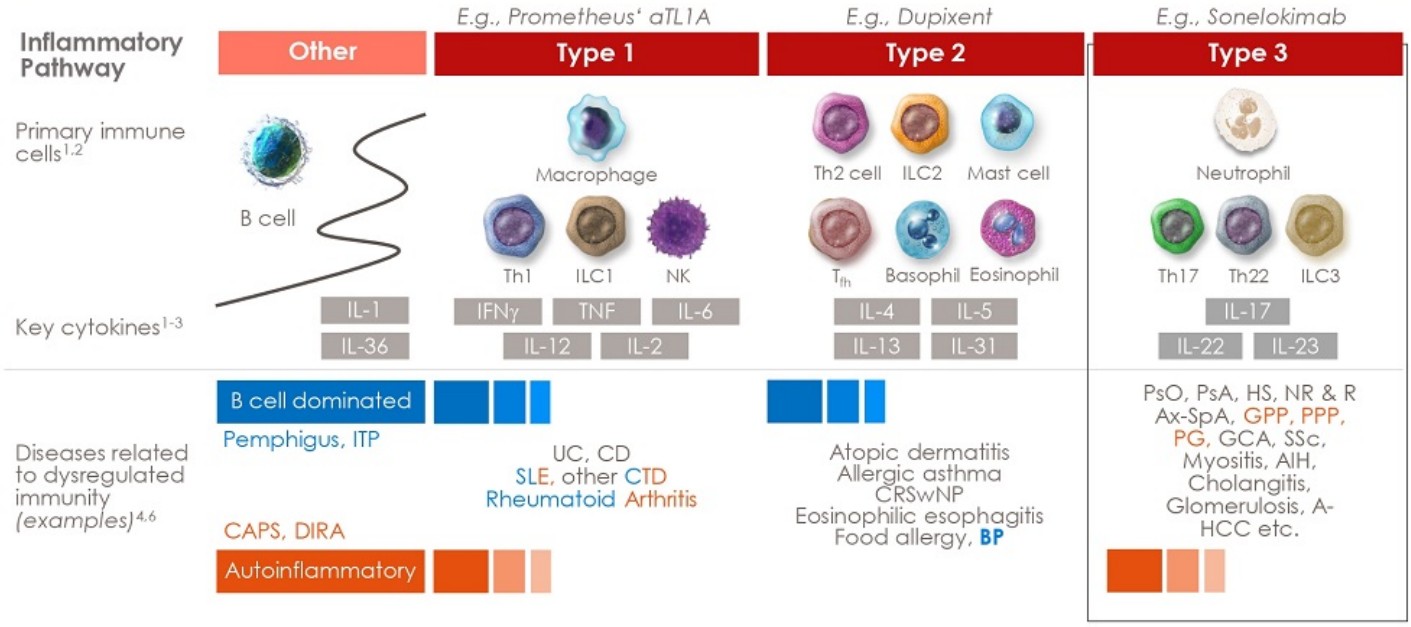
1 IHS4 score is calculated as ∑ (n of nodules x1, n of abcesses x2, n of draining tunnels x4)

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

Source: MoonLake Clinical

SLK is the potential leader in Type 3 diseases

MoonLake



Note: Simplified depiction based on key published information, not meant to be exhaustive in nature. AD, atopic dermatitis; IFNγ, interferon gamma; IL, interleukin; ILC, innate lymphoid cell; NK, natural killer; Tfh, follicular helper; Th, T helper.

1 Kaiko GE, et al. *Immunology*. 2008;123:326-338 2 Eyerich K, Eyerich S. *J Eur Acad Dermatol Venereol*. 2018;32:692-703 3 Raphael I, et al. *Cytokine*. 2015;74:5-17 4 Nakayama T, et al. *Annu Rev Immunol*. 2017;35:53-84 5 Coates LC, et al. *Semin Arthritis Rheum*. 2016;46:291-304 6 Gandhi NA, et al. *Expert Rev Clin Immunol*. 2017;13(5):425-437.

Source: MoonLake Corporate

Emerging perspective on MLTX

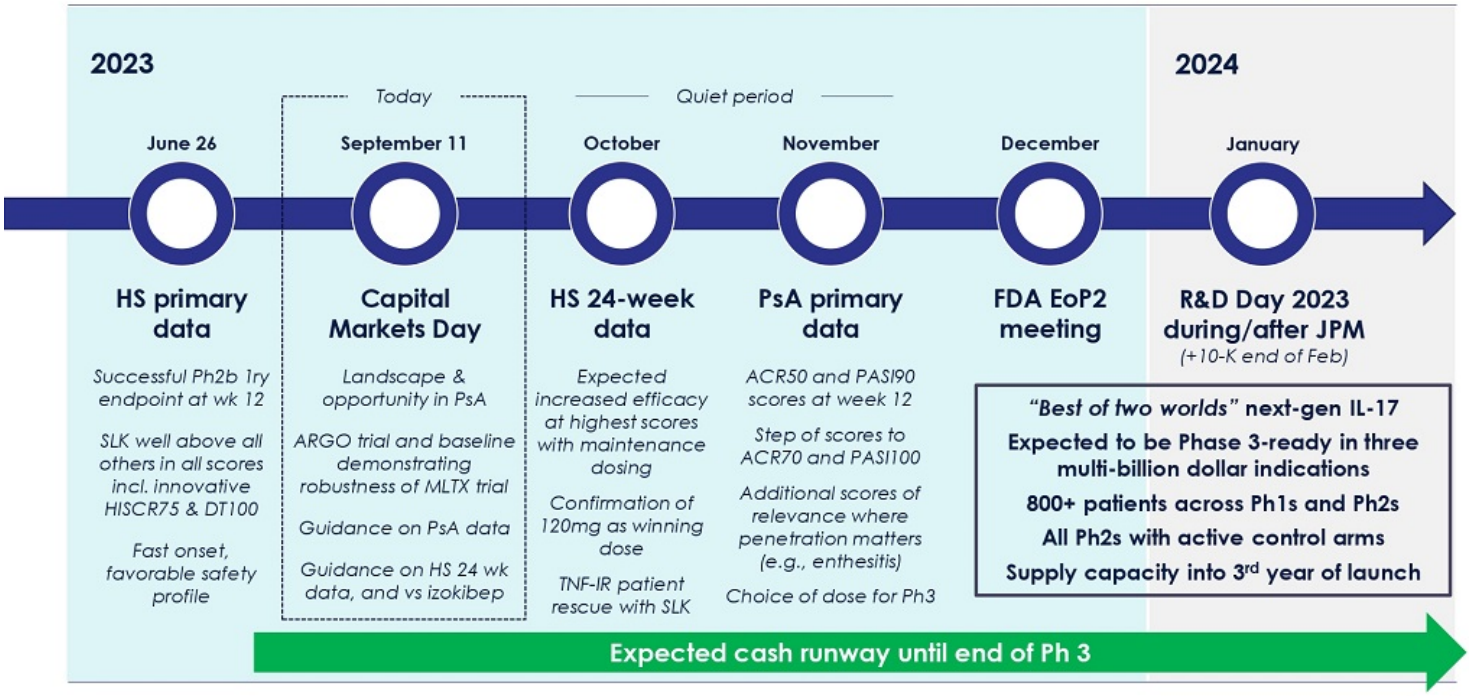
MoonLake

- **Best in class** – SLK can potentially be a unique molecule among all "next gen IL-17s", as now shown in HS and PsO
- **Rarefied air** – 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 provide differentiating 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 year** –MLTX has several key readouts planned among "next gen IL-17s" to end of 2023, and operates from a position of financial stability and strength

Source: MoonLake Corporate

MLTX has an intense set of expected catalysts to year end

MoonLake



Source: MoonLake Corporate

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Psoriatic Arthritis (PsA)

Unmet need & evolving landscape

Disclosures

- J. F. Merola is a consultant and/or investigator for Amgen, Boehringer Ingelheim, Bristol-Myers Squibb, AbbVie, Eli Lilly, Novartis, Janssen, UCB, Sanofi, Regeneron, Sun Pharma, Biogen, Pfizer, and MoonLake Immunotherapeutics

Source: Prof Joseph Merola

PsA is a common condition that is underdiagnosed and undertreated

MoonLake

30% of patients with psoriasis develop PsA[1]

Global prevalence of psoriasis: ~125 million people[1]

~2 in 5 patients with PsA were underdiagnosed

in the PREPARE non-interventional study[2]

~2 in 5 patients diagnosed with PsA are not on biologics

in a recent international survey[3]

Despite the availability of new therapies, many eligible patients are not yet treated with biologics

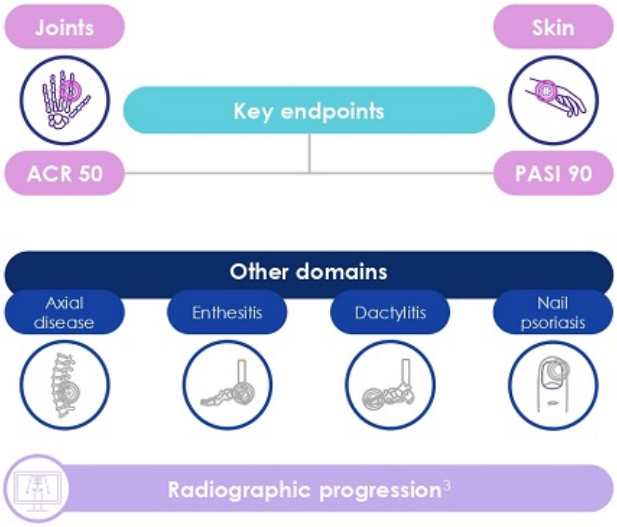
1 National Psoriasis Foundation. https://www.psoriasis.org/psoriasis-statistics/. accessed September 2023; 2 Mease et al. J Am Acad Dermatol. 2013;69:729–35; 3 Tillett et al. Rheumatol Ther. 2020;7:617–37

Source: Prof Joseph Merola

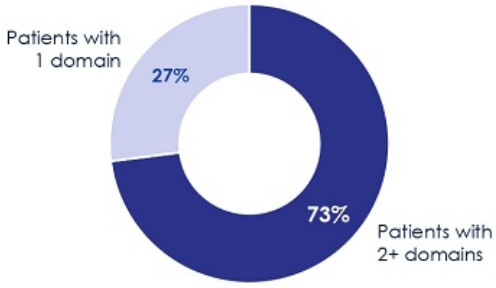
PsA is a multidomain disease, including joints and skin

MoonLake

Novel treatments for PsA are primarily assessed on improvements in joints and skin[1,2]



>70% of patients with active PsA have 2+ domain involvement[4]



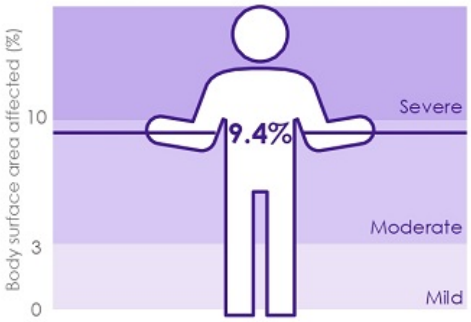
Frequency of domain presentations in active PsA
CorEvitas registry, N=2,315

1 Ogdie et al. Rheumatology. 2020;59(Suppl 1):i37-46; 2 FitzGerald et al. Nat Rev Dis Primers. 2021;12:7:59; 3 van der Heijde et al. Arthritis Res Ther. 2020;22:18; 4 Ogdie et al. J Rheum. 2021;48:698–706

Source: Prof Joseph Merola

Significant skin involvement continues to be prevalent in PsA

>40% of patients with PsA have **moderate-to-severe skin** disease



Skin involvement in PsA typically affects ~**10% of body surface area**,[1] indicative of **significant disease**

Despite advances in biologics, resolving both joints and skin remains a significant challenge in PsA

Data from 2,703 patients with PsA an international survey, including 1,743 patients with skin involvement; Tillett et al. Rheumatol Ther. 2020;7:617–37
1. Among patients with any skin involvement (BSA >0)
Source: Prof Joseph Merola

IL-17i is the only biologic class preferred in all domains of PsA

MoonLake

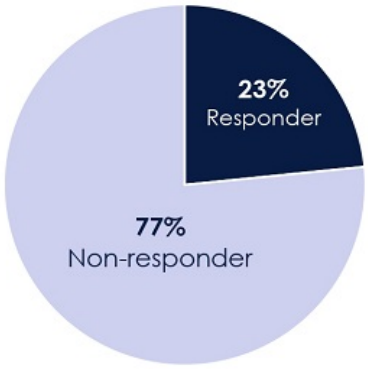
	Joints	Skin	Other domains				
							
Preferred biologic(s)[1]	**Peripheral arthritis**	**Psoriasis**	**Axial**	**Enthesitis**	**Dactylitis**	**Nails**	**Radiographic progression**
IL-17i	✓	✓	✓	✓	✓	✓	✓
TNFi	✓	✓	✓	✓	✓	✓	✓
IL-12/23i	✓	✓	✗	✓	✓	✓	✗
IL-23i	✓	✓	✗	✓	✓	✓	✗

1 Preferred biologic classes are based on the expert interpretation of clinical study results by Prof Merola

Source: Prof Joseph Merola

Most patients do not see a robust response across all PsA domains

>3 in 4 patients do not achieve MDA
within 6 months of biologic initiation[1]



% of patients who were MDA responders

MDA is a composite of ambitious clinical response targets in both joints and skin[2]

MDA (Minimal Disease Activity) denotes a patient who has achieved **≥5 of** the following **7 criteria**:

1. **Joints:** TJC ≤1
2. **Joints:** SJC ≤1
3. **Skin:** PASI ≤1 *(or BSA ≤3%)*
4. **Entheses:** Tender entheseal points ≤1
5. **PRO:** Patient pain VAS ≤15
6. **PRO:** Patient global activity VAS ≤20
7. **PRO:** HAQ-DI VAS ≤0.5

Despite success in some domains, achievement of MDA clinical responses with biologics remains low

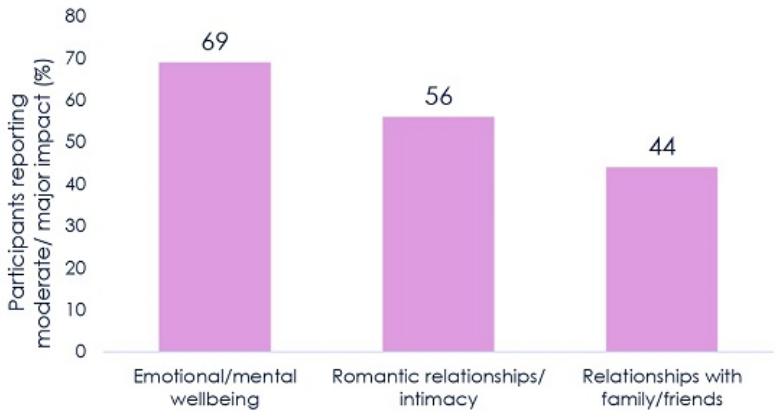
1 Data from the CorEvitas registry (N=1,251): Ogdie et al. ACR 2021;abstract 1344: 2 BSA, body surface area; HAQ-DI, Health Assessment Questionnaire Disability Index; PASI, Psoriasis Area and Severity Index; PRO, patient-reported outcome; S/TJC, swollen/tender joint count; VAS, visual analog scale; Gossec et al. J Rheumatol. 2018;45:6–13

Source: Prof Joseph Merola

The symptom burden of PsA remains wide-ranging and substantial

MoonLake

Many patients report a moderate-to-severe psychosocial impact of PsA[1]



10–40% of patients with PsA experience depression and anxiety[2,3]

4 in 5 patients with PsA report fatigue, with a major impact on physical activity levels[4]

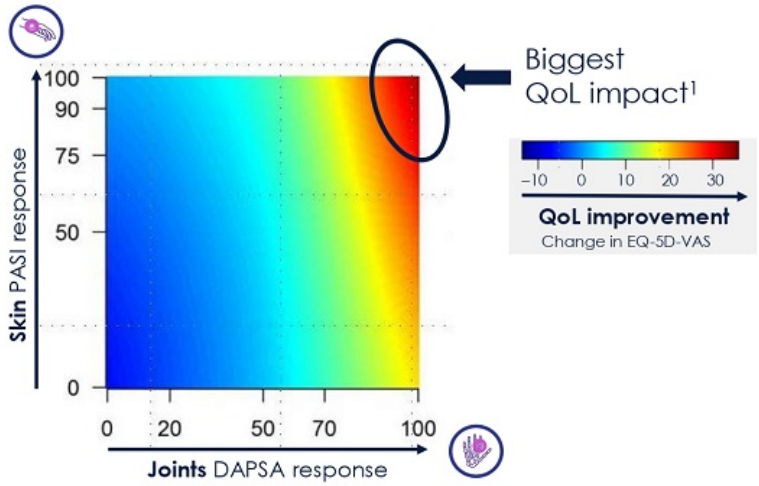
Despite advances in biologics, symptom burden for patients remains high

1 International patient-based survey of the Psoriatic Arthritis Impact of Disease (N=1,286); Coates et al. Health Qual Life Outcomes. 2020;18:173; 2 Tillett et al. Rheumatol Ther. 2020;7:617–37; 3 Orbai et al. Ann Rheum Dis. 2017;76:673–80; 4 Gossec et al. J Rheumatol. 2022;49:1221–8

Source: Prof Joseph Merola

High clinical response in skin, in addition to joints, is critical for patients

MoonLake



PsA is associated with a multitude of quality-of-life impairments[2]

These may be especially pronounced in patients with multidomain PsA, such as skin involvement, who report:

- A greater risk of **flare**
- More substantial **work impairment**
- Higher rates of **anxiety** and **depression**
- Worse overall **quality of life** scores

Optimal benefit for patients with PsA requires a clinical response in both joints and skin

1 Quality of life data from 402 patients with PsA and moderate-to-severe skin involvement (≥3% BSA) after 24 weeks on therapy/placebo in the SPIRIT Phase 3 clinical study program (heat map image reproduced with permission from Prof Merola); Kavanaugh et al. ACR 2017;abstract 2539; 2 Tillett et al. Rheumatol Ther. 2020;7:617–37

Source: Prof Joseph Merola

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It may be that innovations in both **drug targets** and **drug structures** are needed to move the needle in PsA…

Can we optimize IL-17 inhibition?

MoonLake

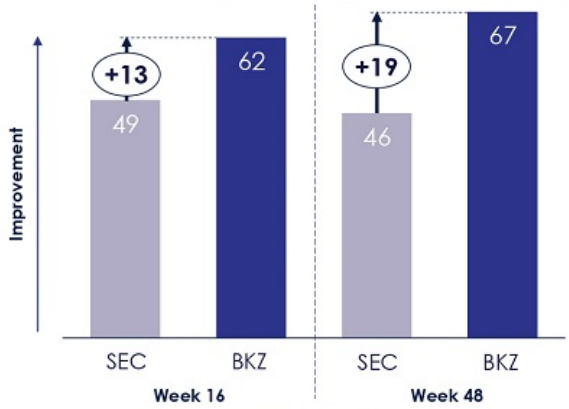
As the class of choice for addressing all domains in PsA, **innovation on MOA is centered on optimizing IL-17i**, based on increasing evidence that IL-17F drives psoriatic inflammation in addition to IL-17A…

Skin
Moderate-to-severe psoriasis

IL-17A-only inhibitor
Secukinumab

IL-17A+IL-17F inhibitor
Bimekizumab

PASI 100 BE RADIANT (Phase 3b **psoriasis**; NRI)[1]



Primary endpoint: PASI 100 at Week 16

Inhibition of **both IL-17A+IL-17F** provides **greater benefits** in skin vs. inhibition of IL-17A only

1 A study in patients with moderate-to-severe psoriasis; only a subset of participants had PsA and the clinical significance of the findings is unclear (there are no head-to-head studies of these two MOAs in PsA): Reich et al. N Engl J Med. 2021;385:142–52

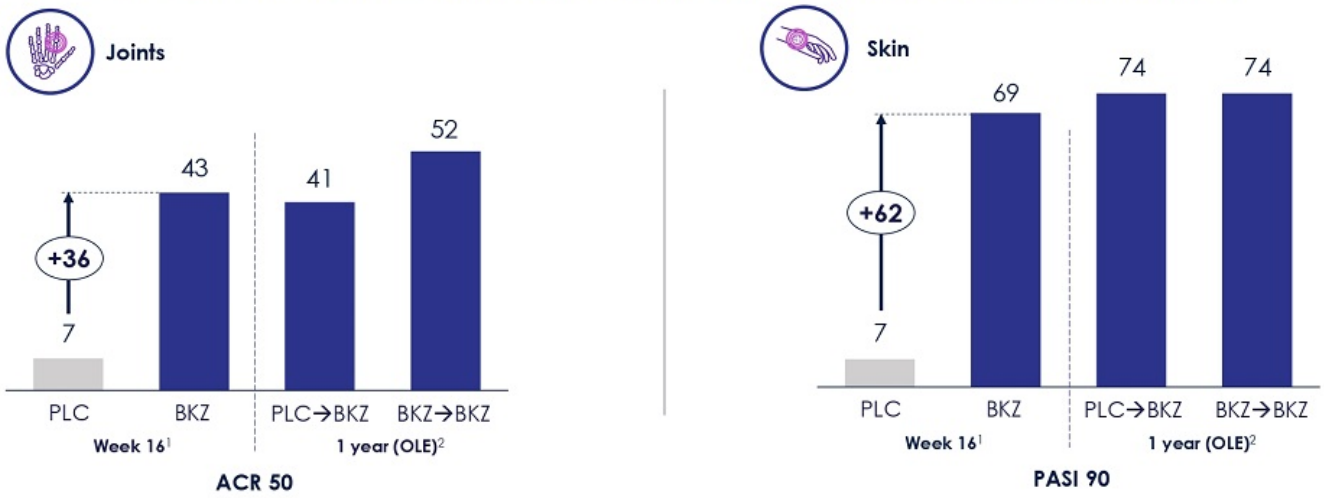
Source: Prof Joseph Merola

IL-17A and IL-17F dual inhibition is newly validated as an MOA in PsA

MoonLake

Bimekizumab IL-17A and IL-17F inhibitor (160 mg Q4W) | BE COMPLETE (Phase 3 **PsA**; NRI)[1]

- Patients enrolled in the study had a **previous inadequate response to, or intolerance of, TNF inhibitors**



Inhibition of **both IL-17A and IL-17F** provided **high levels** of **joint and skin** responses in PsA

1 NRI, non-responder imputation; OLE, open-label extension; Merola et al. Lancet 2023;401:38–48; Coates et al. Ann Rheumatic Dis. 2023;82:346–347

Source: Prof Joseph Merola and References

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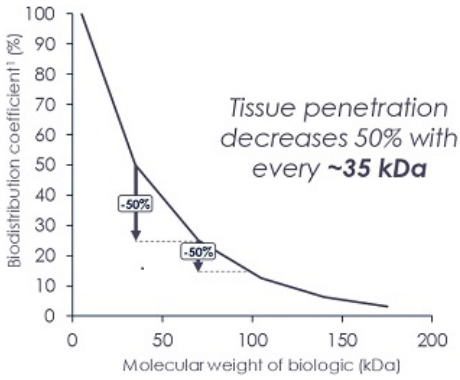
What about innovation in the molecular design of **drug structures**?

Molecular design features for targeting musculoskeletal inflammation

MoonLake

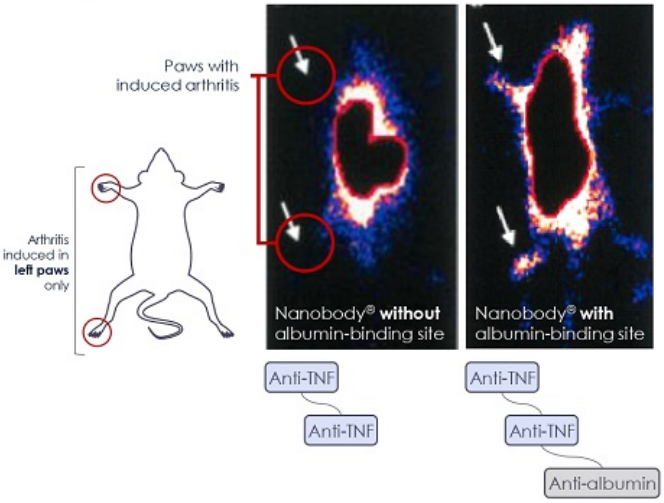
Smaller biologics → higher tissue uptake[1]



Smaller biologics such as Nanobodies® may include an albumin-binding domain to extend half-life[2]

Albumin-binding domains target inflammation

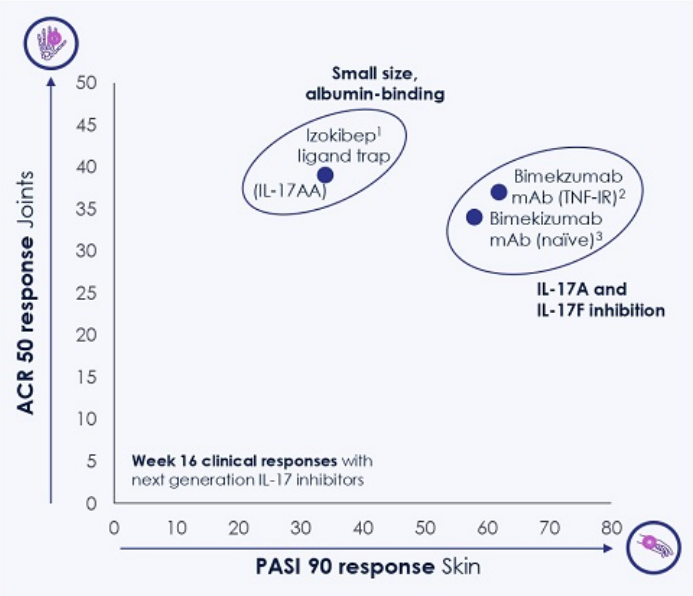
Accumulation of Nanobodies® 24 h after treatment[2]
Distribution of anti-TNF Nanobodies® +/- albumin-binding site 24h after a single injection in mice with collagen-induced arthritis



1 Biodistribution coefficient, calculated as tissue concentration/plasma concentration in muscle (other tissues ranged from 14 to 41 kDa molecular weight change required for a 50% difference in tissue penetration); Li et al. mAbs 2016;8:113-9;
2 Coppieters et al. Arthritis Rheum 2006;54:1856-66
Source: Prof Joseph Merola, MoonLake Research and References

Next-generation IL-17i: two approaches for optimizing PsA outcomes

MoonLake



Can we innovate on both target and structure to improve outcomes in PsA?

Innovating on…

- **structure** with small size + albumin binding

OR

- **MOA** with IL-17A and IL-17F inhibition

…are two approaches with promising results

Key data considerations

- Izokibep data are from a Phase 2 study with ~45 patients per arm (with 9–17% prior TNFi treatment)
- PASI data for izokibep are as observed, not ITT
- Izokibep: 26–30% injection site reactions
- Bimekizumab: 2–3% oral candidiasis

1 Phase 2 study (ITT-NRI analysis for ACR 50, as observed for PASI 90): Behrens et al. EULAR 2022;oral presentation; 2 Phase 3 study in patients with an inadequate response, or intolerance, to TNF inhibitors (ITT-NRI): Merola et al. Lancet 2023;401:38–48; 3 Phase 3 study in biologic-naïve patients (ITT-NRI): McInnes et al. Lancet 2023;401:25–37
Source: Prof Joseph Merola and References

Sonelokimab: potential to innovate on both target and structure

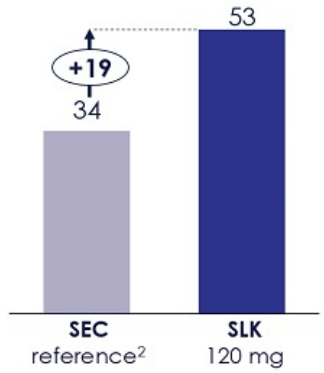
MoonLake

Sonelokimab: best of both worlds?

IL-17A and IL-17F inhibition

Psoriasis IL-17F in skin[1]

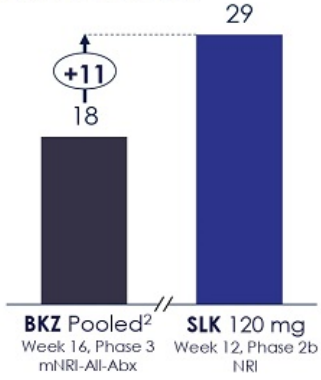
PASI 100 Week 16, Phase 2b



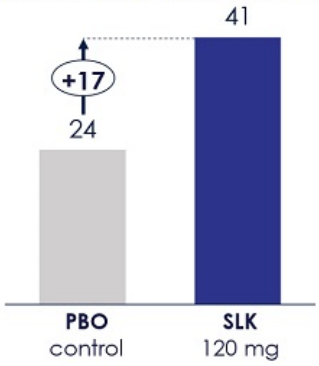
Small, albumin-binding Nanobody®

Hidradenitis suppurativa Hard-to-reach tissue

HiSCR 75, PBO-adj
Not head-to-head data



DT 100 Week 12, Phase 2b
Complete resolution of draining tunnels



1 Papp et al. Lancet. 2021;397:1564–75; 2 A secukinumab reference arm was included in the Phase 2b psoriasis study, but was not powered as a head-to-head comparator; 3 Kimball et al. AAD 2023;oral presentation

Source: Prof Joseph Merola, MoonLake Clinical and References

Summary

- There remains an **unmet need across the multiple domains of PsA**, demanding novel therapies — innovation will stem from both **drug targets and structures**
- **IL-17A and IL-17F inhibition** has the potential to optimize outcomes across PsA domains
- There are some promising indications that **smaller, albumin-binding drug molecules** may be able to better treat difficult-to-reach sites of inflammation
- **Sonelokimab** is designed to combine the '**best of both worlds**': innovating on drug target with IL-17A and IL-17F inhibition, and on drug structure as a small, albumin-binding Nanobody®

Source: Prof Joseph Merola

MoonLake

MLTX R&D Update

MoonLake

Key learnings from Prof. Merola

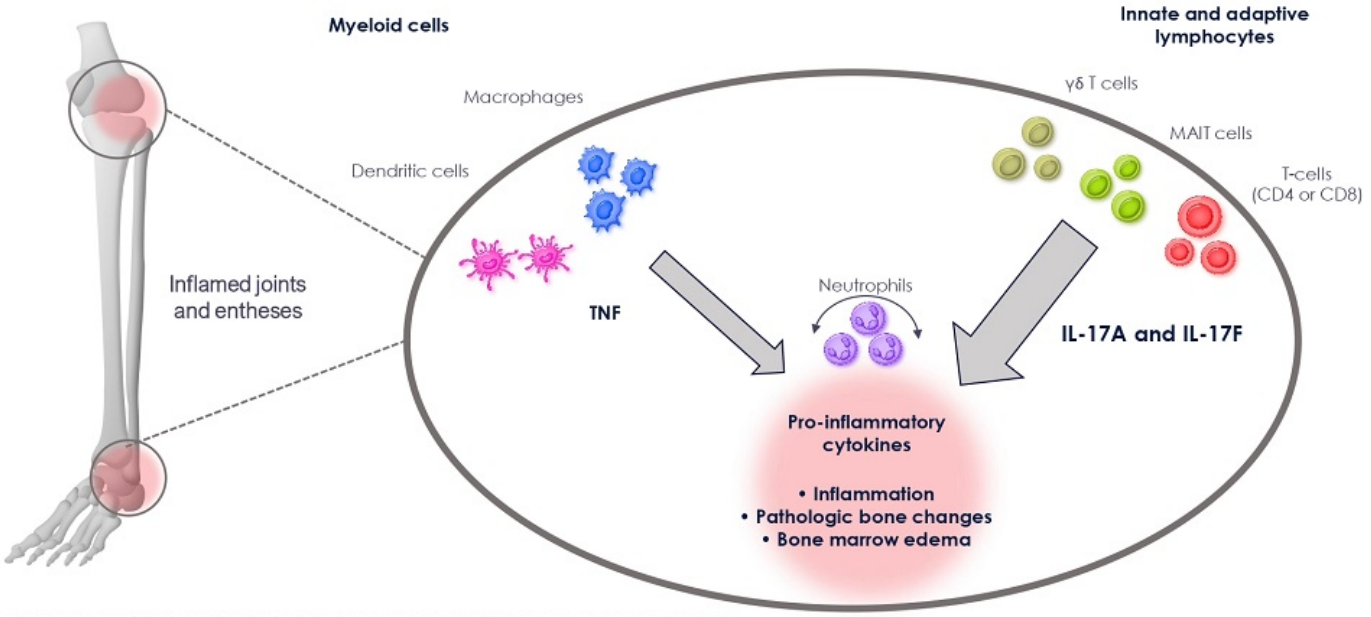
- PsA is a multi-domain disease with joint and skin as main manifestations
- Current therapies are limited in their control of different PsA domains — a major unmeet need
- IL-17 is a key pathway in PsA; delivering IL-17A and IL-17F inhibition with improved tissue penetration using small, albumin-binding therapies aims to elevate PsA control

Key discussion points

1. Evidence for PsA as an IL-17A and IL-17F driven disease
2. Unique properties of sonelokimab — small size, albumin-binding, IL-17A- and IL-17F-targeting Nanobody®
3. Status of MoonLake's ARGO PsA Phase 2 program

Source: MoonLake Clinical

1. Like in the skin, IL-17A **and** IL-17F have key roles in PsA



MAIT, mucosal-associated invariant T cell; γδ T, gamma delta T cell. Example cell types are shown; not an exhaustive list.

1 Tsukazaki & Kaito. Int J Mol Sci. 2020;21:6401; 2 Blanco et al. Cytokine Growth Factor Rev. 2008;19:41–52; 3 Rosine et al. Front Immunol. 2021;11:553742; 4 Cole et al. Front Immunol. 2020;11:585134; 5 Glatt et al. Ann Rheum Dis. 2018;77:523–32; 6 Russell et al. Cells. 2021;10:341; 7 McGonagle et al. Ann Rheum Dis. 2019;78:1167–1178; 8 McGonagle et al. Front Immunol. 2021;12:614255

Source: MoonLake Research and References

1. Direct inhibition of IL-17A+IL-17F is the optimal therapeutic approach

MoonLake

Why do IL-23i not succeed in all domains of PsA?

Preferred biologic(s)	Peripheral arthritis	Psoriasis	Axial	Enthesitis	Dactylitis	Nails	Radiographic progression
IL-17i	✓	✓	✓	✓	✓	✓	✓
TNFi	✓	~	✓	✓	✓	~	✓
IL-12/23i	~	~	✗	✓	✓	~	✗
IL-23i	~	✓	✗	✓	✓	✓	✗

Adaptive T cells
IL-23-dependent?

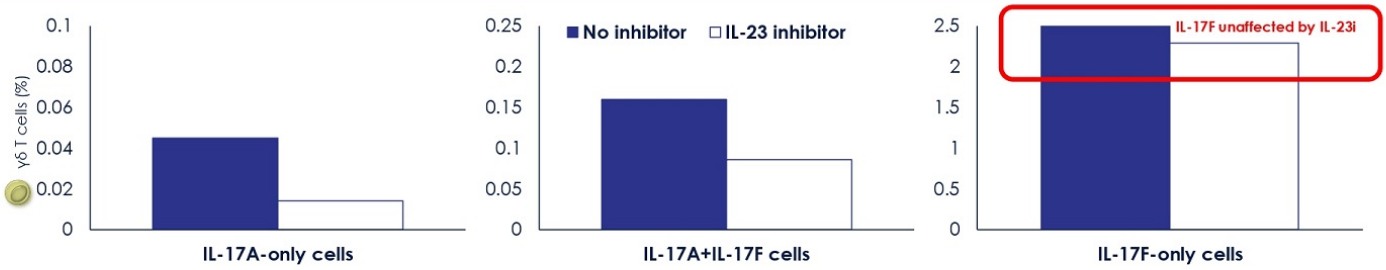
Th17 cell

Innate-like lymphocytes
IL-23-independent?

MAIT cell

γδ T cell

Innate like lymphocytes (such as γδ T, ILC3 or MAIT cells) can produce IL-17 **independently of IL-23**, and even in the presence of **IL-23 inhibitors**[1]

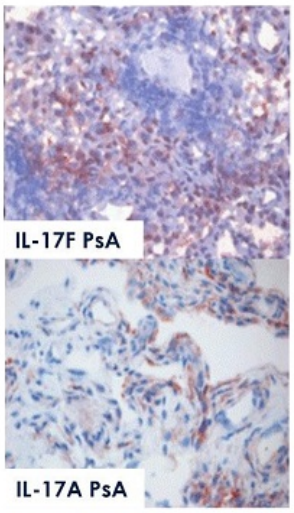


1 Data shown are for γδ T cells stimulated with IL-12 and IL-18 (similar results were obtained with or without the addition of IL-23 protein, and in MAIT and ILC3 cells): Cole et al. Front Immunol. 2020;11:585134

Source: Prof Joseph Merola, MoonLake Research and References

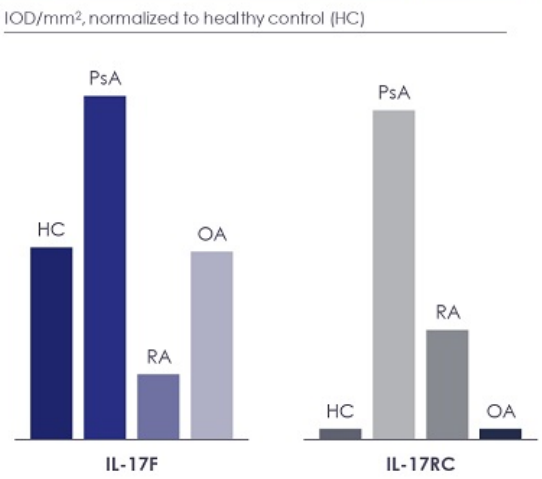
1. IL-17F is prevalent in synovial tissue and lesional skin in PsA

MoonLake



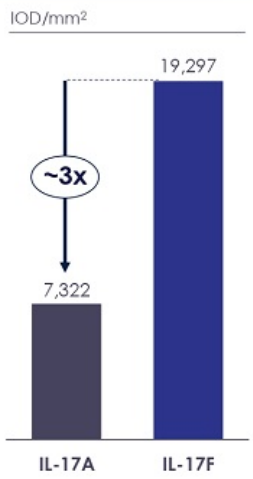
- Accumulation of IL-17F observed in synovial tissue

IL-17F and its receptor elevated in PsA joints[1]



- Elevation of IL-17F and the IL-17F receptor (IL-17RC) in the synovial tissue of patients with PsA

IL-17F elevated in PsA skin



- IL-17F levels were increased in the lesional dermis of patients with PsA[2]

IOD/mm² shows the integrated optical density of immunohistochemistry signal per area of tissue
1 van Baarsen et al. Arthritis Res Ther 2014;16:426; 2 Kolbinger et al. J Allergy Clin Immunol 2017;139:923–32; HC, healthy control; OA, osteoarthritis; RA, rheumatoid arthritis
Source: MoonLake Research and References

1. Inhibiting F in addition to A further suppresses inflammatory pathways

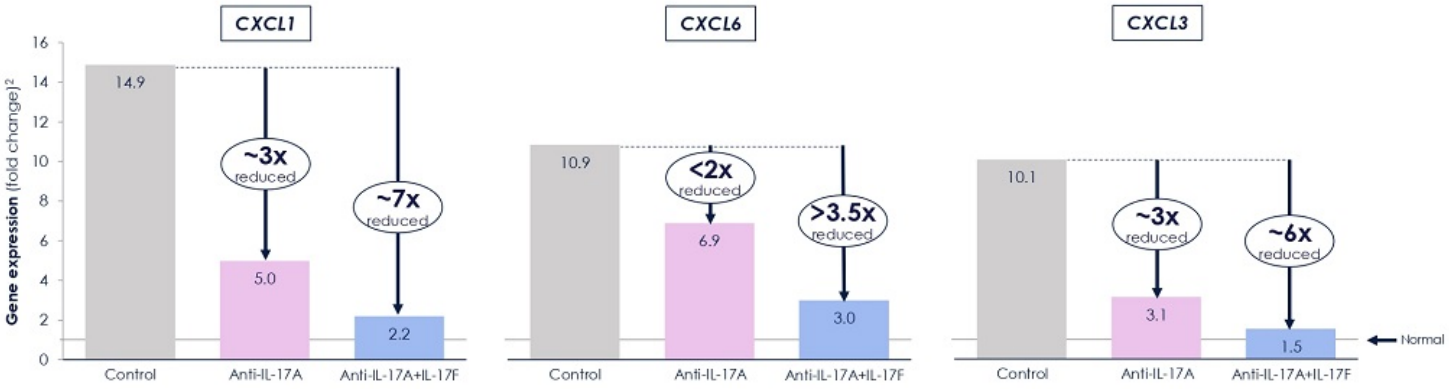
MoonLake

A+F vs A only Human synoviocytes (*in vitro*)[1]

Joints

Inhibition of inflammation in an assay with human joint cells (synoviocytes)

- An antibody targeting IL-17A + IL-17F was compared with an antibody with matched affinity for IL-17A alone
- **Adding IL-17F achieved greater suppression than inhibiting IL-17A alone**



1 The assay involved stimulation of human synoviocytes with Th17 cell supernatant (1:10 dilution), and incubation with either a control IgG molecule, an anti-IL-17A monoclonal antibody affinity-matched to bimekizumab, or a bispecific anti-IL-17A-and-IL-17F monoclonal antibody (bimekizumab) 2 Gene expression was normalized to *GAPDH* and reported relative to unstimulated cells;Glatt et al. Ann Rheum Dis. 2018;77:523-32

Source: MoonLake Research and References

2. Sonelokimab: How to optimize IL-17 inhibition in multi-domain PsA

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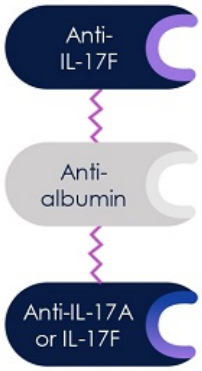
Dual inhibition
Targeting IL-17A+IL-17F



Nanobody
Small size, albumin-binding

SLK
43 kDa

mAb
~150 kDa

Best of both worlds?

Source: MoonLake Research

2. **Albumin-binding:** Optimizing target engagement in inflamed joints

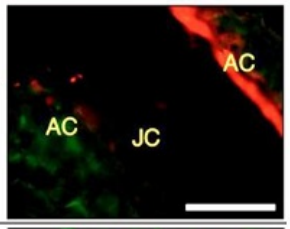
MoonLake

Mouse collagen-induced arthritis model (ankle joints) 8 h after TNFi treatment[1]

Albumin in red

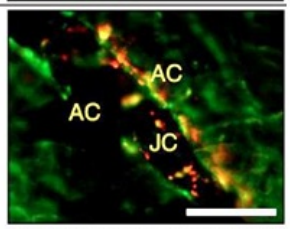
Adalimumab in green

1. Traditional mAb **(148 kDa)**
2. Only binds TNF **(not albumin)**



Ozoralizumab In green

1. Nanobody **(38 kDa)**
2. Binds both TNF **and albumin**

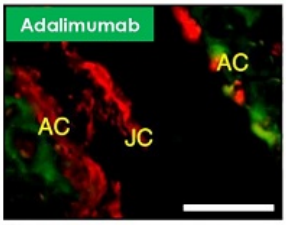


AC, articular cartilage; JC, joint cavity

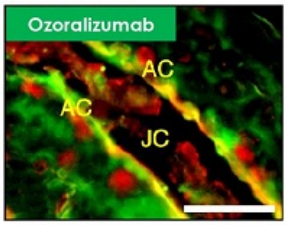
Preferential accumulation of albumin-binding ozoralizumab Nanobody® in inflamed joints

TNF in red

Adalimumab

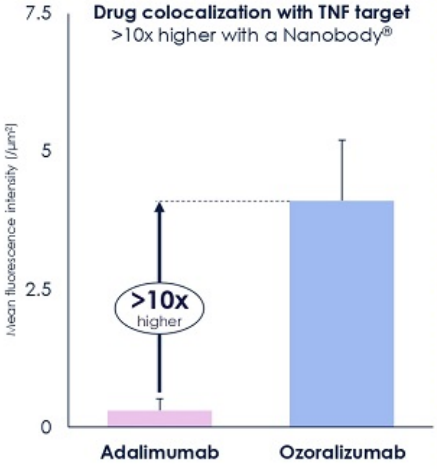


Ozoralizumab



AC, articular cartilage; JC, joint cavity

Drug colocalization with TNF target
>10x higher with a Nanobody®



Greater target engagement with ozoralizumab Nanobody®

1 Quantitative data shows the mean of 6 joints per cohort of 3 mice with collagen-induced arthritis; Oyama et al. Sci Rep. 2022;12:18102

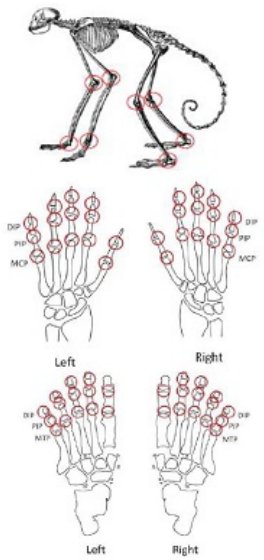
Source: MoonLake Research and References

2. Sonelokimab: Optimizing target engagement in primate PsA model

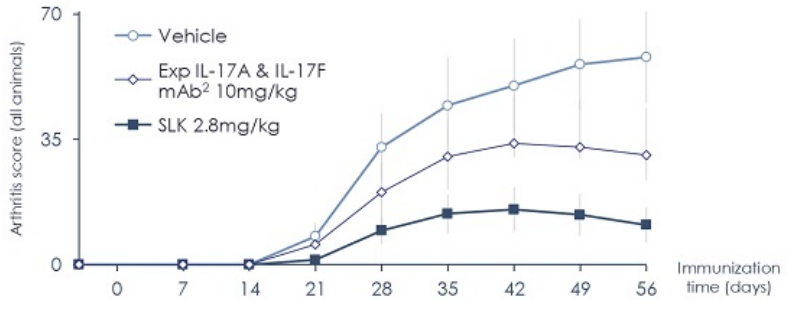
MoonLake

Arthritic joint assessment for SLK[1]

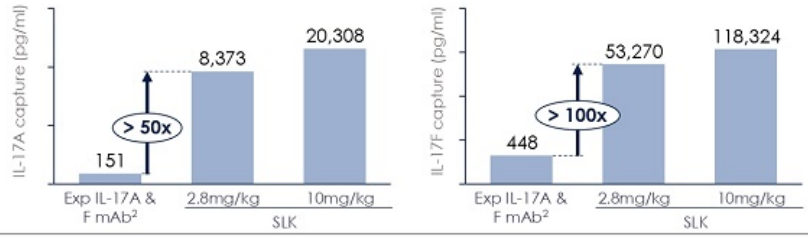
Collagen-induced arthritis



Clinical improvement with SLK versus mAb[2]



Synovial fluid binding analysis



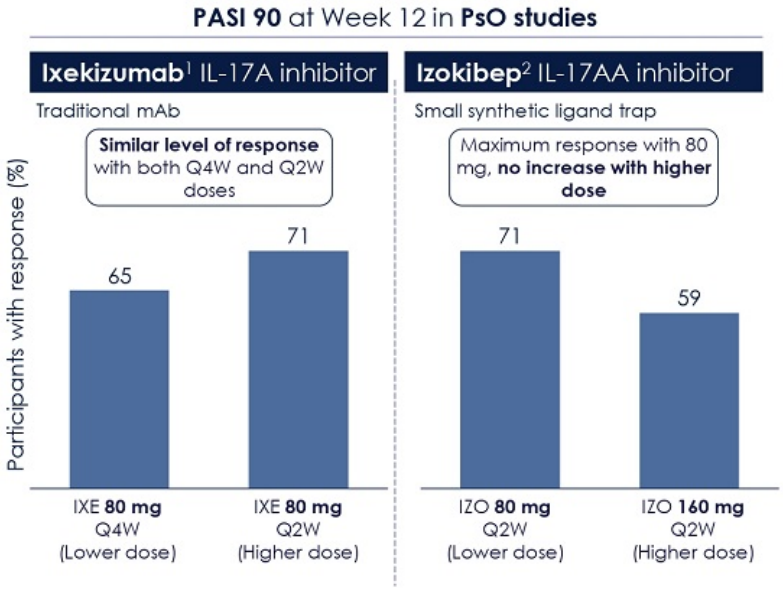
1Assessed joints for the determination of Arthritis Score. The scored joints are indicated (red circles) for the large joints (top panel), for limb joints (middle panel) and hind limb joints (bottom panel): DIP, distal interphalangeal joint; PIP, proximal interphalangeal joint; MCP, Metacarpophalangeal joint; MTP, Metatarsophalangeal joint 2 Experimental IL-17A & IL-17F mAb (Novimmune)

Source: MoonLake Clinical

2. Small size does not compensate for a narrow IL-17 MoA in PsO

MoonLake

Skin



Smaller IL-17A/A inhibitor IZO **does not elevate** response above a larger IL-17A mAb (IXE)

Note: Data are not based on head-to-head comparisons.
1 Gordon et al. N Engl J Med. 2016;375:345–56; 2 Gerdes et al. EADV Spring Symposium 2021;oral presentation
Source: MoonLake Clinical and References

2. How to optimize IL-17 inhibition in multi-domain PsA

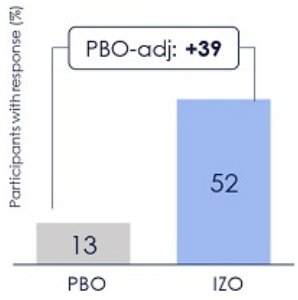
MoonLake

Joints

ACR 50 at Week 16 in **PsA studies**

Izokibep IL-17AA inhibitor[1]

- Small synthetic ligand trap
- Phase 2; 80 mg Q2W



Bimekizumab IL-17A+F inh[2]

- Traditional mAb
- Phase 3; 160 mg Q4W

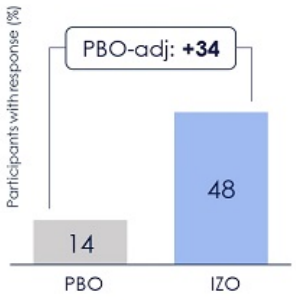
Size and MOA both seem to contribute to optimal ACR 50 responses in PsA, but **either on its own is insufficient** to produce a step change

Skin

PASI 90 at Week 16 in **PsA studies**

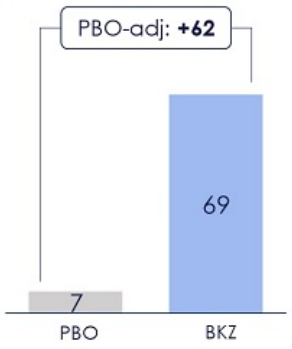
Izokibep IL-17AA inhibitor[1]

- Small synthetic ligand trap
- Phase 2; 80 mg Q2W



Bimekizumab IL-17A+F inh[2]

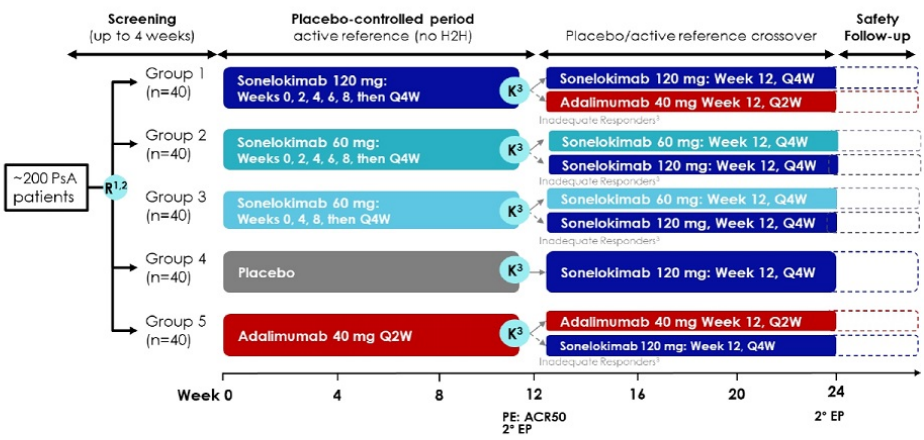
- Traditional mAb
- Phase 3; 160 mg Q4W



PASI 90 levels seem to be **optimal** with an MOA that targets **both IL-17A and IL-17F**, going beyond what is possible with IL-17AA only

Note: Data are not based on Head-to-Head comparisons (other than vs placebo)
1 Data shown for izokibep represent the higher (80 mg Q2W) dose: Behrens et al. EULAR 2022:oral presentation; 2 Data for bimekizumab are from the BE COMPLETE TNF-IR study: Merola et al. Lancet 2023;401:38–48
Source: MoonLake Clinical and References

3. ARGO: A robust phase 2 trial design for valid study results



Key design elements of ARG

- Global study (North America an Europe) with approx. 60 sites
- Double-blind, placebo-controlled, active reference arm
- N=200 patients planned to be randomized
- Active PsA (TJC68 ≥3, SJC ≥3, currently active psoriasis and/or dermatologist-confirmed diagnosis of psoriasis)
- ACR50 as primary endpoint
- ITT-NRI as primary analysis; key secondary endpoints multiplicity controlled
- Stratification for gender and previous biologic use

Notes: 1 Randomization stratified by sex and prior exposure to biologics; 2 At Week 0/Day 1, all eligible participants were randomized 1:1:1:1:1; 3 In the cross-over period, starting at Week 12, participants on sonelokimab 120 mg who have not achieved an adequate response will receive adalimumab 40 mg Q2W until Week 24; participants on sonelokimab 60 mg (started at baseline Q2W or Q4W) who have not achieved an adequate response will receive sonelokimab 120 mg Q4W until week 24; participants on adalimumab who have not achieved an adequate response will receive sonelokimab 120 mg Q4W until Week 24; an adequate response is defined as a reduction of the tender and swollen joint count of ≥20%. Participants on placebo will receive sonelokimab Q4W until Week 24

Source: MoonLake Clinical

3. Pivotal-like ARGO PsA study design in preparation for Phase 3

MoonLake

Study element	NCT04713072[1], IZO Ph 2	BE ACTIVE[2], BKZ Ph2	BE OPTIMAL[3], BKZ Ph 3, Biologic-naïve	BE COMPLETE[4], BKZ Ph 3, TNF-IR	ARGO, SLK Ph 2
Stage	Phase 2	Phase 2	Phase 3	Phase 3	**Phase 2**
Size	135 patients	206 patients	852 patients	400 patients	**207 patients**
Design	R, DB, PC[5]	R, DB, PC[5]	R, DB, PC (AR)[5]	R, DB, PC[5]	**R, DB, PC (AR)[5]**
Dose arms	2 IZO, 1 PLC	4 BKZ, 1 PLC	1 BKZ, 1 ADA, 1 PLC	1 BKZ, 1 PLC	**3 SLK, 1 ADA, 1 PLC**
Key inclusion criteria - CASPAR - TJC, SJC - Failed cDMARDs	 Y, duration not specified ≥3, ≥3 N	 Y, ≥6 mo ≥3, ≥3 N	 Y, ≥6 mo ≥3, ≥3 Y	 Y, ≥6 mo ≥3, ≥3 Y	 **Y, ≥6 mo** **≥3, ≥3** **Y**
Study regions	Europe only	Europe / US	E Europe / W Europe/ N America / Asia	E Europe / W Europe/ N America / Asia	**Europe / US**
FDA-approved study	N	Y	Y	Y	**Y**
Primary endpoint(s)	ACR50 W16	ACR50 W12	ACR50 W16	ACR50 W16	**ACR50 W12**
Stratification	Geo by country Prev. exposure to TNFi Conc. cDMARD	Geo by continent Prev. exposure to TNFi	Geo region Bone erosion number	Geo by region TNFi treatment history[6]	**Gender** **Prev. exposure to biologics**
Primary analysis	ITT-NRI[5]	ITT-NRI[5]	ITT-NRI[5]	ITT-NRI[5]	**ITT-NRI[5]**
Skin (PASI) analysis	As observed	ITT-NRI[5]	ITT-NRI[5]	ITT-NRI[5]	**ITT-NRI[5]**
Previous biologic use	Allowed if not IL-17i	Only TNFi	Not allowed	Inad. Response/ intolerance to TNFi required	**Allowed if not TNFi/IL-17i primary failures**

1 Behrens et al. EULAR 2022:oral presentation.; 2 Ritchlin et al. Lancet. 2020;395:427–40; 3 McInnes et al. Lancet. 2023;401:25–37; 4 Merola et al. Lancet. 2023;401:38–48; 5 AR, adalimumab reference arm; DB, double blind; ITT, intention-to-treat; NRI, non-responder imputation; PC, placebo controlled; R, randomized; 6 Three stratification categories: intolerance to TNFi, inadequate response to 1 TNFi, and inadequate response to 2 TNFi

Source: MoonLake Clinical and References

3. ARGO baseline characteristics similar to previous studies

MoonLake

	NCT04713072[1], IZO Ph 2	BE ACTIVE[2], BKZ Ph2	BE OPTIMAL[3], BKZ Ph 3, Biologic-naïve	BE COMPLETE[4], BKZ Ph 3, TNF-IR	ARGO, SLK Ph 2
Key characteristics					
Sex, female, %	50–60	49.0	53.2	52.5	**49.3**
Duration of PsA, yrs, mean (±SD)	7.1 (±7.8)	7 (±NR[5])	5.9 (±7.0)	9.5 (±9.3)	**5.5 (±5.7)**
Prior biologic use, %	9–17 (TNFi)	18.9 (TNFi)	0	87.8 (TNFi)	**18.4**
Concomitant DMARD use, %	80–81	67.0	69.5	50.5	**58.5**
Musculoskeletal disease					
Joint counts, mean (SD) - TJC of 68 joints - SJC of 66 joints	 16.7 (±10.4)[6] 9.9 (±6.6)[6]	 22 (±NR[5]) 12 (±NR[5])	 17.0 (±12.2) 9.2 (±6.7)	 18.7 (±13.8) 9.9 (±7.7)	 **17.0 (±12.4)** **9.4 (±7.1)**
Presence of enthesitis (LEI),%	31.9[6]	52	29.2	35.5	**31.6**
Presence of dactylitis, %	20.0[6]	31	11.7	12.0	**12.1**
Skin disease					
BSA ≥ 3, % Mild BSA < 3, % Moderate BSA ≥ 3 < 10, % Severe BSA ≥ 10, %	54.8 NR[5] NR[5] NR[5]	67 33 38 29	49.9 50.1 33.1[7] 17.0[7]	66.0 34.0 43.0[8] 23.0[8]	**69.4** **30.6** **41.3** **28.2**
PASI in pts BSA ≥ 3, mean (SD)	8–11 (±5–7)	NR[5]	8.1 (±6.6)	9.6 (±8.4)	**7.2 (±6.6)**
Nail disease					
Presence of nail disease, %	77.0[6]	75.0	55.8[9]	60.5	**54.4**

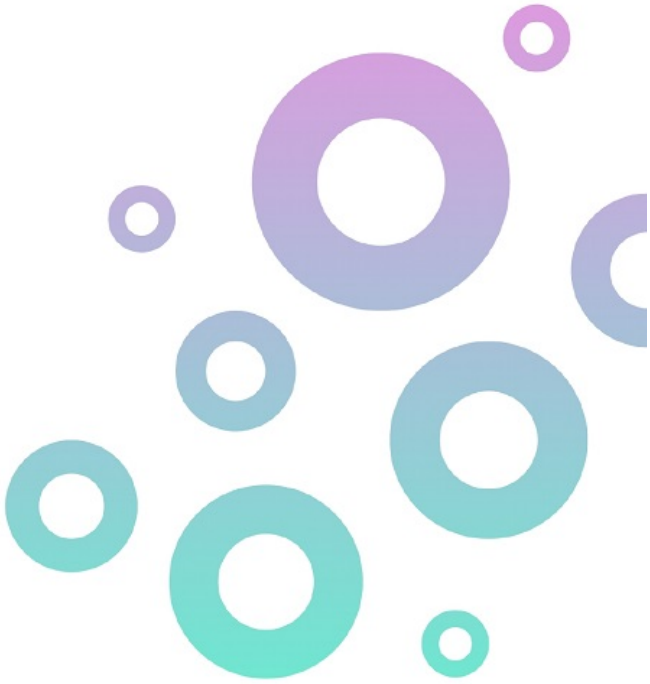
1 Behrens et al. EULAR 2022;oral presentation; 2 Ritchlin et al. Lancet. 2020;395:427–40; 3 McInnes et al. Lancet. 2023;401:25–37; 4 Merola et al. Lancet. 2023;401:38–48 5 NR, not reported; 6 de Vlam et al. ACR 2022. Poster 2151; 7 Reported data on patients with moderate or severe BSA in BE OPTIMAL and BE COMPLETE excludes adalimumab reference arm; Morita et al. WCD 2023;Poster 1175; 8 Morita et al. WCD 2023;Poster 1175; 9 McInnes et al. EULAR 2023. Poster POS1537

Source: MoonLake Clinical and References

MoonLake

Research & Clinical Summary

- PsA is driven by **both IL-17A and IL-17F** in the key domains of joints and skin
- **Small size**, **albumin binding** plus **IL-17A- and IL-17F**-targeting may deliver optimal disease control
- Sonelokimab has the potential to **elevate treatment outcomes** in skin and joints
- The ARGO PsA trial has a **pivotal-like design**, and baseline characteristics that allow comparisons to pivotal studies



Source: MoonLake Clinical

MoonLake

Upcoming Data

Outlook to our planned ARGO PsA primary readout in November

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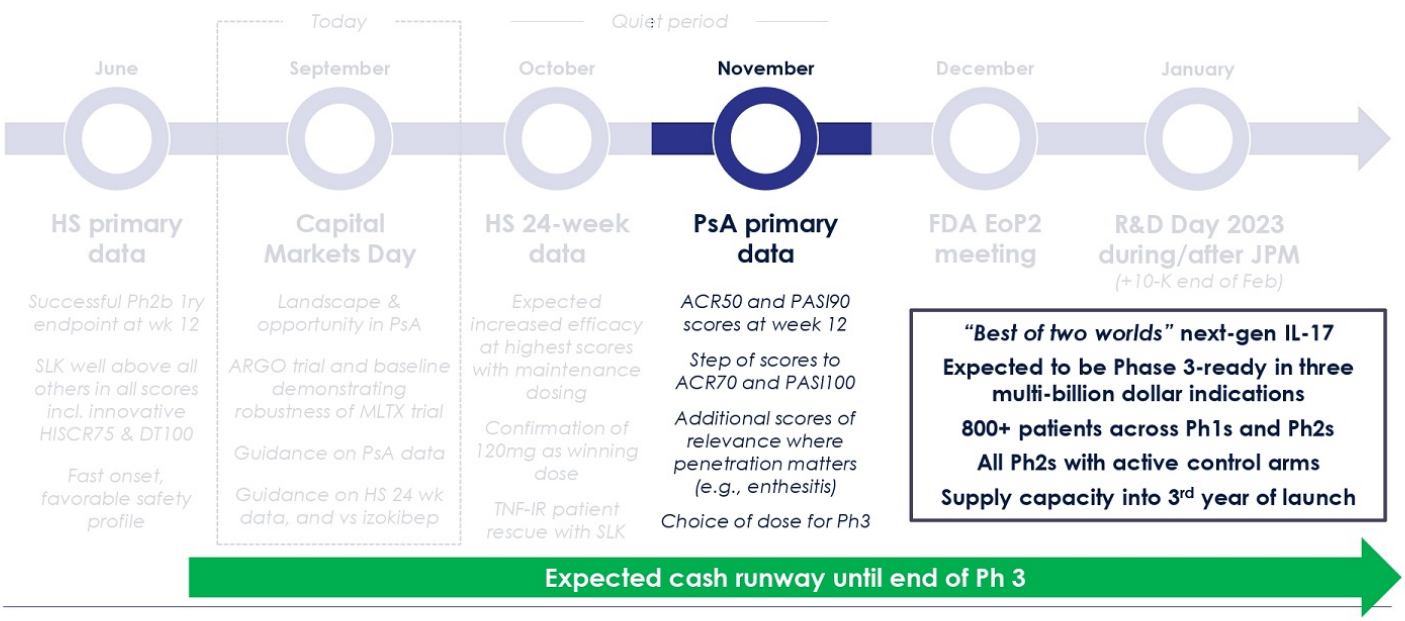
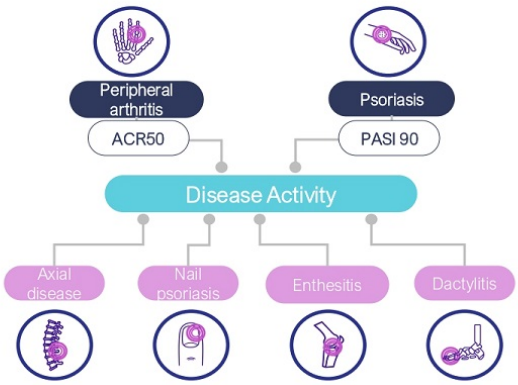


Source: MoonLake Corporate

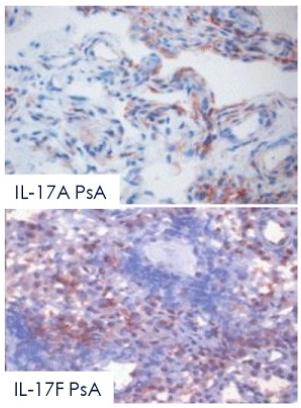
PsA: IL-17F dependent multi-domain disease in difficult-to-reach tissues

MoonLake

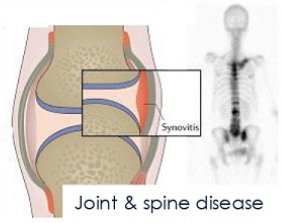
PsA is a multi-domain deep-tissue disease…

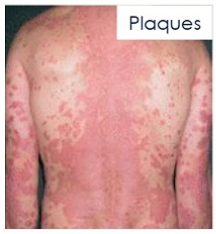


…with 3x IL-17F vs IL-17A[1]…

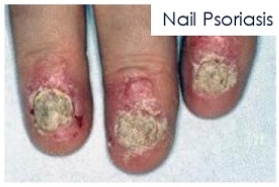


…and causing devastating damage





(PsA starts as enthesitis[2], with IL-17F producing cells in associated plaques[3] and axial disease[4-6], and with 80% of patients suffering from nail psoriasis[7])



Market size

.5% Global prevalence

10+ USD bn sales beyond 2030

Unmet Needs

20% ACR improvement achievable with current drugs

80% Pts with multiple disease domains (Psoriatic Disease Complex)

0 Drugs have best ACR and PASI scores

1 van Baarsen LG, et al. Arthritis Res Ther. 2014; 16:426-436; 2 Schett G, et al. Nature Reviews Rheumatology. 2017; 13:731-741; 3 Prinz JC, et al. J Exp Med. 2020 Jan 6;217(1):e20191397; 4 Sweet K, et al. RMD Open 2021;7e001679; 5 Shao M, et al. Clin Immunol 2020;213:108374; 6 Lories RJ and McInnes IB, Nature Medicine. 2012; 18:1018-1019; 7 Reich K. J Eur Acad Dermatol Venereol. 2009; 23 Suppl 1:15-21; Clinical pictures K. Reich

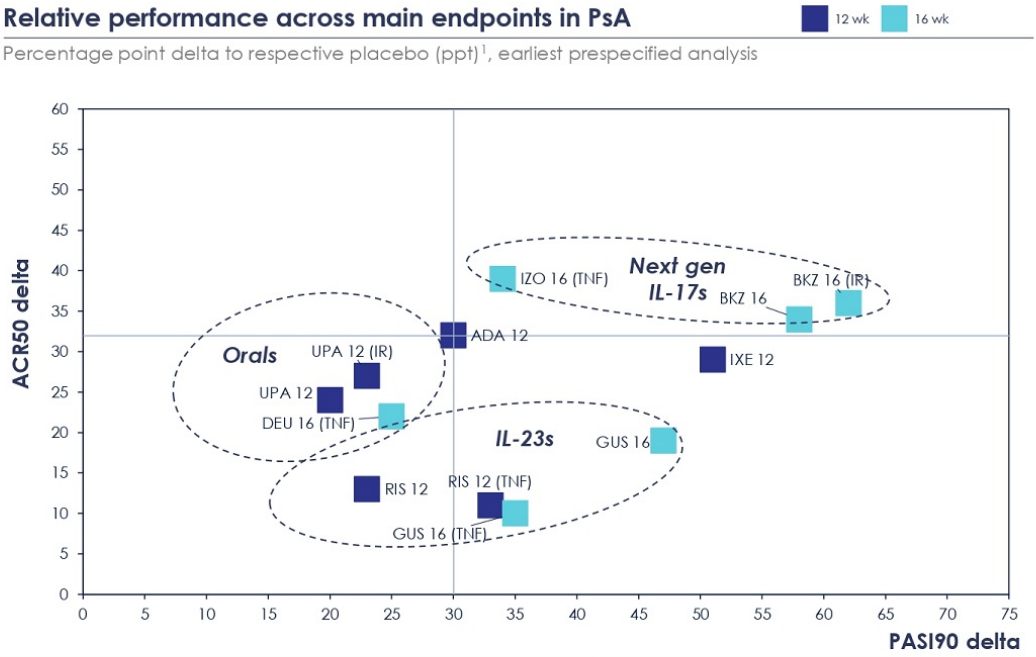
Source: MoonLake Medical, Clinical pictures K. Reich

PsA: Ample opportunity to elevate treatment goals vs other MoAs

MoonLake

Relative performance across main endpoints in PsA

Percentage point delta to respective placebo (ppt)[1], earliest prespecified analysis



Debunking myths

- Humira **still the reference** in PsA, despite **safety concerns vs. IL17s** and non-durable efficacy
- **Best performance**, so far, achieved by mAb **IL-17A & F inhibition** (BKZ)
- Another **"next-gen IL-17"** (IZO) achieves elevated ACR50 scores, but lacks on high PASI
- **Orals underperform** on both dimensions and present **safety concerns** (JAKs)
- **IL-23s underperform**, especially on ACR50

1 Endpoint time indicated by color code (12 wk, 16 wk); ACR50 and PASI90 values refer to licensed dose where available (otherwise best dose); Drug name indicated by three-letter acronym: DEU, deucravacitinib; GUS, guselkumab; UPA, upadacitinib; RIS, Risankizumab; ADA, adalimumab; IZO, izokibep; BKZ, bimekizumab; IXE, ixekizumab; "(IR)" refers to trials incl. patients with inadequate TNF response, "(TNF)" refers to trials with a share of prior TNF usage (up to ~50%), and if no indication, trials refer to "Naïve" populations where patients had no previous TNF treatment

Source: MoonLake Medical

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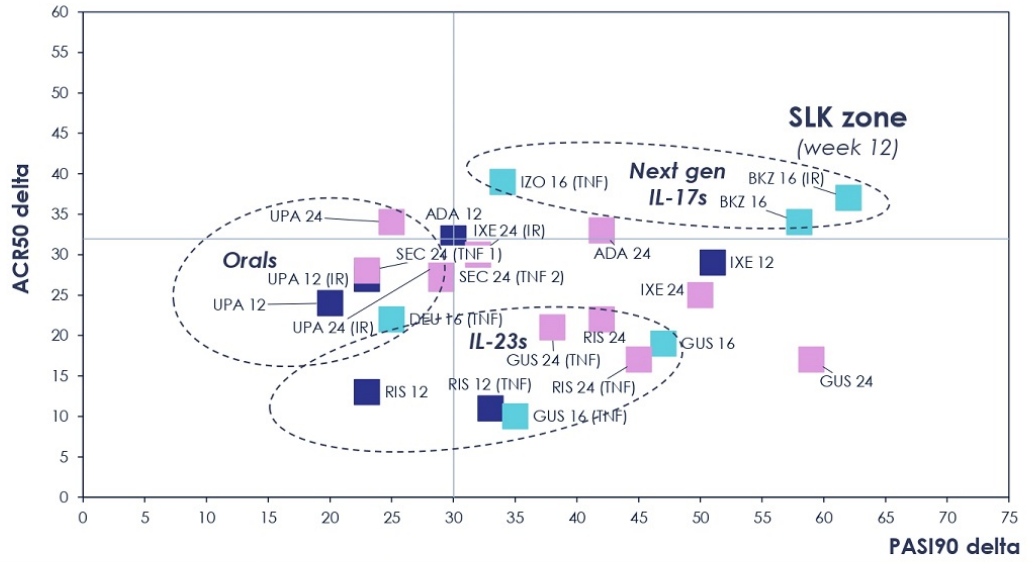

1 Endpoint time indicated by color code (12 wk, 16 wk, 24 wk); ACR50 and PASI90 values refer to licensed dose where available (otherwise best dose); Drug name indicated by three-letter acronym: DEU, deucravacitinib; GUS, guselkumab; UPA, upadacitinib; RIS, Risankizumab; ADA, adalimumab; IZO, izokibep; BKZ, bimekizumab; SEC, secukinumab; IXE, ixekizumab; For SEC, due to lack of reported values 12/16 wk, both FUTURE 1 & 2 trial 24 wk data is shown (FUTURE 1 data is missing for 150mg licensed dose, so 300mg is shown); "(IR)" refers to trials incl. patients with inadequate TNF response, "(TNF)" refers to trials with a share of prior TNF usage (up to ~50%), and if no indication, trials refer to "Naïve" populations where patients had no previous TNF treatment

Source: MoonLake Medical

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- **IL-23s underperform**, especially on ACR50

PsA: We expect SLK to perform at or above other assets in other scores

MoonLake

	BKZ 16 wks		UPA 24 wks		GUS 24 wks		RZB 24 wks		ADA 24 wks	SEC 24 wks		IXE 24 wks		TIL[14] 16 wks	DCV[15] 16 wks	IZO[16] 16 wks
	BE COMP[1]	BE OPTIM[2]	SELECT PsA 1[3]	SELECT PsA 2[4]	DISC 1[5]	DISC 2[6]	KEEPs. 1[7]	KEEPs. 2[8]	ADAPT[9]	FUTURE 1[10]	FUTURE 2[11]	SPIRIT P1[12]	SPIRIT P2[13]	PHASE 2		
	PLC – DRUG **ΔDelta**															
ACR50 (% resp.)	7 – 43 **Δ36**	10 – 44 **Δ34**	19 – 52 **Δ33**	9 – 38 **Δ29**	9 – 36 **Δ27**	14 – 33 **Δ19**	11 – 33 **Δ22**	9 – 26 **Δ17**	6 – 39 **Δ33**	7 – 35 **Δ28**	7 – 35 **Δ28**	15 – 40 **Δ25**	5 – 35 **Δ30**	24 – 51 **Δ27**	11 – 33 **Δ22**	13 – 52 **Δ39**
PASI90 (% resp.)	7 – 69 **Δ62**	3 – 61 **Δ58**	17 – 42 **Δ25**	7 – 36 **Δ29**	12 – 63 **Δ51**	10 – 61 **Δ51**	10 – 52 **Δ42**	10 – 55 **Δ45**	0 – 42 **Δ42**	4 – 45 **Δ41**	9 – 49 **Δ40**	6 – 56 **Δ50**	12 – 44 **Δ32**	7 – 50 **Δ43**	NA	14 – 48 **Δ34**
MDA (% resp.)	6 – 44 **Δ38**	13 - 45 **Δ32**	12 – 37 **Δ25**	3 – 25 **Δ22**	11 – 30 **Δ19**	6 – 19 **Δ13**	10 – 25 **Δ15**	11 – 26 **Δ15**	NA	NA	NA	NA	3 – 28 **Δ25**	6 – 34 **Δ28**	8 – 24 **Δ16**	5 – 39 **Δ34**
Enthesitis (% resolution)	35 – 50 **Δ15**		32 – 54 **Δ22**	15 – 43 **Δ28**	27 – 48 **Δ 21**	29 – 45 **Δ16**	35 – 48 **Δ13**	30 – 43 **Δ13**	NA	13 – 48 **Δ35**	22- 40 **Δ18**	19 – 43 **Δ24**	22 – 35 **Δ13**	NA	23 – 50 **Δ27**	10 – 88 **Δ78**
Dactylitis (% resolution)	51 – 76 **Δ25**		40 – 77 **Δ37**	28 – 58 **Δ30**	49 – 63 **Δ14**	42 – 64 **Δ22**	51 – 68 **Δ17**	42 – 73 **Δ31**	NA	16 – 52 **Δ36**	15 – 47 **Δ32**	25 – 80 **Δ55**	21 – 75 **Δ54**	NA	60 – 79 **Δ19**	27 – 65 **Δ38**
Previous TNF use	IR 76-77%	naïve	naïve	IR 91-92%	IR 30-32%	naive	naïve	IR 46%	-	IR 27-32%	IR 27-45%	naïve	IR 90-92%	IR 22-24%	IR 12-17%	IR 9-17%
PsA duration, yrs	9-10	6	6	10-11	6-7	5-6	7	8	10	NA	NA	6-7	9-11	6-8	4-5	7
CRP	≥ 6 mg/L 44%	≥ 6 mg/L 31-43%	≥ ULN 72-77%	≥ ULN 57-60%	Mean 6-8 mg/L	Mean 12-13 mg/L	Mean 11-12 mg/L	Mean 8 mg/L	Mean 14 mg/L	NA	NA	Mean 13-15 mg/L	Mean 12-17 mg/L	Mean 8-13 mg/L	Mean 4-5 mg/L	-

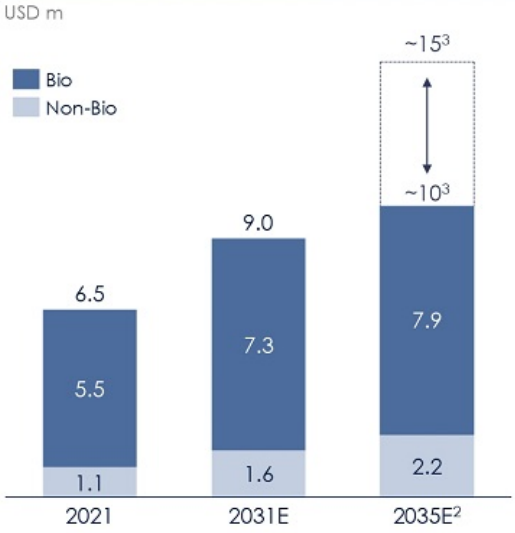
1. Merola et al. Lancet 2023;401:38-48 2. McInnes et al. Lancet 2023;401:37-37; 3. McInnes et al. NEJM 2021;384:1227-1239; 4. Mease et al. Ann Rheum Dis 2017;76:79-87; 5. Deodhar et al. Lancet 2020; 395:1115-1125; 6. Mease et al. Lancet 2020;395:1126-1136; 7. Kristensen et al. Ann Rhem Dis 2022; 81:225-231; 8. Ostör et al. Ann Rheum Dis 2022; 81:351–358; 9. Mease et al. Arthritis Rheum 2005; 52:3279-3289; 10. Mease et al. NEJM 2015;373:1329-1339; 11. McInnes et al. Lancet 2015;386:1137-46; 12. Mease et al. Ann Rheum Dis 2017;76:79-87; 13. Nash et al. Lancet 2017;389:2317-27; 14. Mease et al. Ann Rheum Dis 2021;80:1147-1157; 15. Mease et al. Ann Rheum Dis 2022;81:815-822; 16. Behrens et al. abstract presented at ACR2022

Source: MoonLake

IL-17 expected to become largest MoA in PsA in the next years

MoonLake

PsA market size estimates[1]



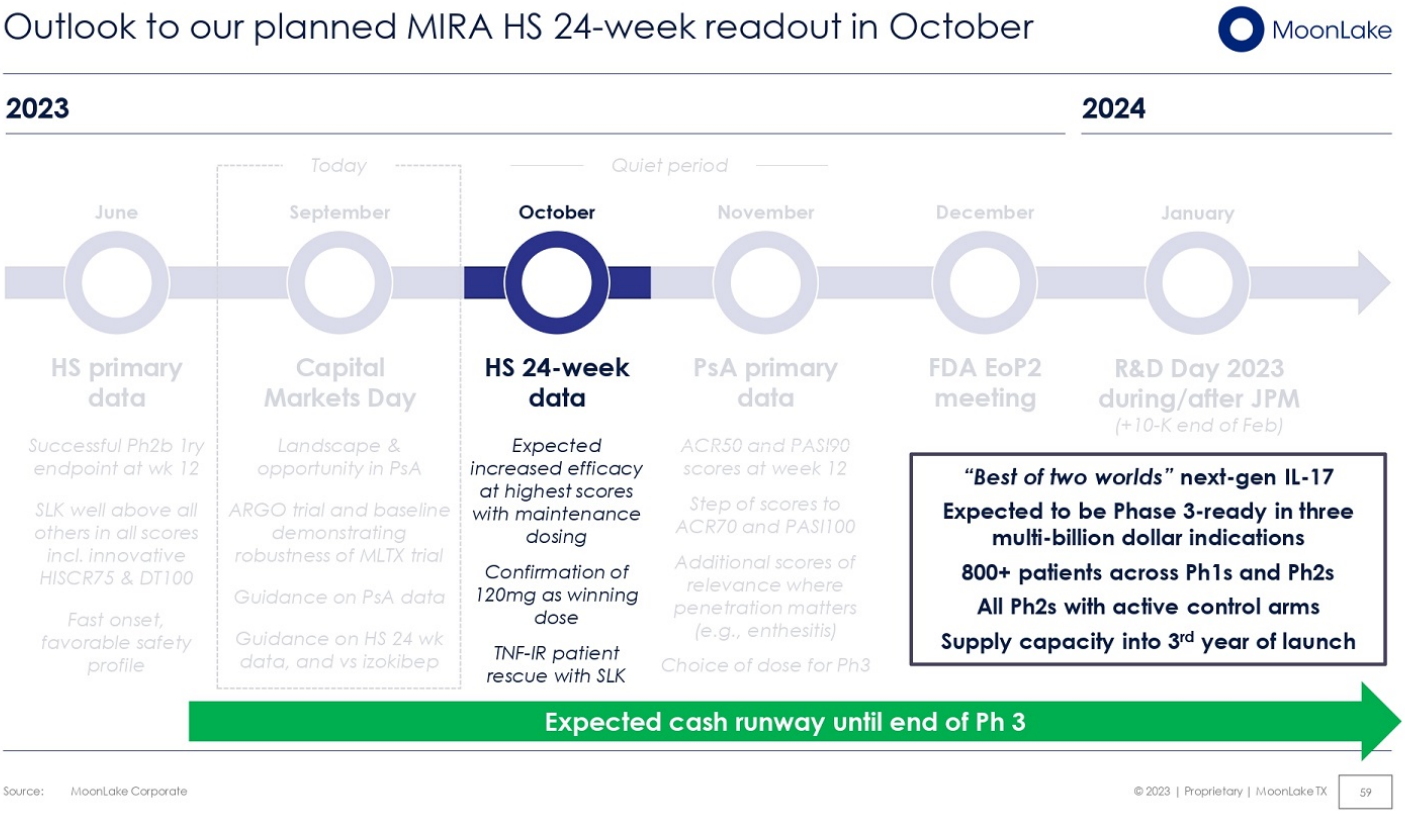
Key notes

- IL-17 becomes **largest drug class** in the next years in PsA (already estimated as 35-40% of market in 2031)
- Most data sources, incl. DRG/Carivate have BKZ latest estimates performed **before BE COMPLETE** (Ph 3) results
- **SLK is not yet part** of general, publicly available estimates – although an all-analysts-average places **sales for PsA above blockbuster level** (even with ~65% avg. PoS)
- BKZ is **~18% of IL-17** class by 2031 according to DRG/Clarivate, which is **likely an underestimation** versus SEC or IXE
- Most analysts suggest a small percentage market share for SLK only (~1-15%) – likely **an underestimation** versus any biologic leading any immunology market[4]

1 Based on DRG/Clarivate data ("Bio" included TNFs, IL-12/23, IL-17 and IL-23 related assets; "Non-Bio" includes all DMARDs, JAK inhibitors and selection co-stimulation modulators); 2 Based on extending sales to 2035 using a 5-year historical CAGR (2027-2031); 3 Upper bound of range indicated in Analyst Reports that cover MLTX (where available); 4 Considering DRG data from 16 Immunology indications: PsA, RA, Asthma, Moderate adult AD, Severe adult AD, nr-axSpA, AS, CU, LN, SLE, PsO, COPD, Acute CD, Maintenance CD, Acute UC, Maintenance UC (where avg. biologic share per indication is ~13%, share of second leading biologic is ~23% and share of leading Biologic is 36%
Source: MoonLake, DRG/Clarivate, Analyst Reports

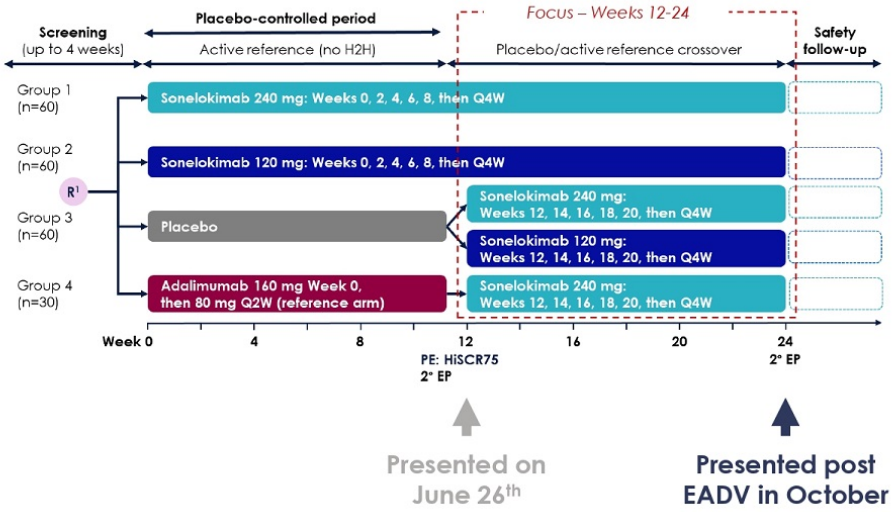
Outlook to our planned MIRA HS 24-week readout in October

MoonLake



Source: MoonLake Corporate

HS: MIRA trial Part B (week 24)



Presented on June 26th

Presented post EADV in October

Main readouts from Part B to week 24:

- Main scores:
  - HiSCR75
  - HiSCR50 & HiSCR 90
- Focus on tunnels
  - IHS4
  - Ultrasound case studies
  - DT counts
  - DT 100
- Main lesions:
  - AN count
  - AN 100
- Other e.g.,
  - PROs
  - Safety

Source: MoonLake Clinical

HS: Our focus areas for the HS 24-wk data readout in October

MoonLake

1	**Higher HiSCR75 with Q4W dosing**	More patients reaching this higher endpoint at 6 months with 120mg monthly dose (beyond the 43% at week12)
2	**Greater depth of responses**	Prolonged exposure to SLK showing more responses on other HiSCRs, including HiSCR90 (a secondary endpoint)
3	**More disease control**	Improved efficacy rates incl. AN100/DT100 and sustained improvements on patient reported quality of life
4	**Best dose confirmed**	120mg confirmed as "winning dose" in terms of speed of and depth of response, with cross-over and PK data
5	**Effect on TNF patients**	Sustained responses in cross-over TNF responders and HiSCR75 responses in non-responders
6	**Favorable safety profile**	No new signals, no IBD or malignancy, mAb-like ISR rate, *Candida* (if present) transient and with no discontinuations

Source: MoonLake Corporate

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

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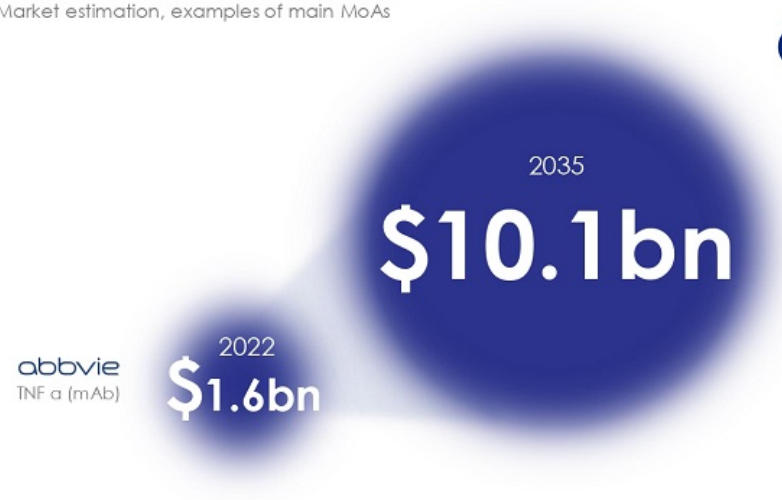
US HS Biologics Market estimation, examples of main MoAs



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IL-17A & F (small bio)

- ✓ Leading HiSCR75 response
- ✓ Speed and depth of response
- ✓ Sustained responses across scores
- ✓ Effect on tunnels (penetration)
- ✓ Patient Reported outcomes
- ✓ TNF-IR "rescue"
- ✓ Safety profile

IL-17A & F (mAb)

ACELYRIN
IL-17A/A (small)

NOVARTIS
IL-17A (mAb)

abbvie
TNF a (mAb)

Incyte
JAK (chem)

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

Source: MoonLake, DRG/Clarivate, academic journals, CBO

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SLK to continue moving the bar

The scientific rationale for a unique molecule

- SLK has unique IL-17F and A binding properties, a key inflammation MoA
- IL-17F plays critical & independent roles in several inflammatory diseases
- SLK has enhanced tissue penetration, reaching where mAbs cannot

Clinical validation of the Nanobody® concept

- HiSCR75 and HiSCR50 deltas to PLC above all other trials
- Significant effects on the deepest inflammatory lesions, the tunnels
- Impact on what matters to patients: pain, quality of life, drainage
- Favorable safety tolerability profile, as observed previously in PsO

Optimal outcome for fast clinical development

- Winning dose regimen and endpoints now known for phase III HS & PsO
- Builds on winning PsO data and de-risks next MLTX trials incl. PsA
- Expectations for success in longer-term HS data and in primary PsA data

Source: MoonLake Clinical

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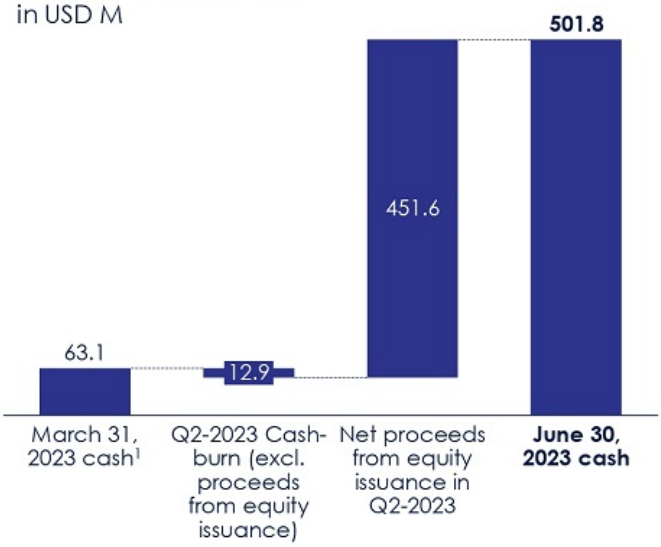
Financial Update

Successful raises in Q2 put MLTX in a strong financial position

MoonLake

Changes in cash[1] balance

in USD M



Successful equity raise in Q2-2023

- Follow-on offering upsized from $250m to $400m
- Multiple time oversubscribed with demand from existing and new shareholders
- Additional gross proceeds from $60m green shoe (exercised by underwriters) and $15m ATM gross proceeds (already in May, sold to a blue-chip investor at a premium)

Strong balance sheet to focus on execution and growth

- Ended Q2-2023 with over $500m in cash
- Expected to be more than sufficient to run Phase 3s in HS and PsA and bring Sonelokimab to regulatory filing
- New shelf registration filed as part of corporate housekeeping duties

[1] Includes cash equivalents and short-term marketable debt securities. Please refer to the Company's financial statements included with the Form 10-Q for the quarter ended June 30, 2023, filed with the SEC

Source: MoonLake Finance

MLTX continues operating efficiently and effectively

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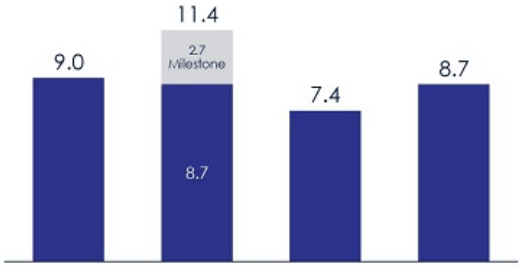
R&D expense
in USD M



- **R&D expense between $7m and 9m**[1] for 4 consecutive quarters
- Expected to stay **similar in Q3 and Q4 2023**
- **Ramp-up in 2024 with planned commencement of Phase 3 programs**
- No milestones under the license agreement until acceptance of regulatory filing

G&A expense
In USD M

- **G&A expense reduced** through stabilized public company operations
- **Growth expected to follow overall organizational growth**

Historical cash burn around $10m / quarter
This is expected to increase with Phase 3 development, but we expect to stay more efficient vs. peers

[1] Excluding milestone expense under the License agreement for Sonelokimab

Source: MoonLake Finance

Strong share price and even more value to be created

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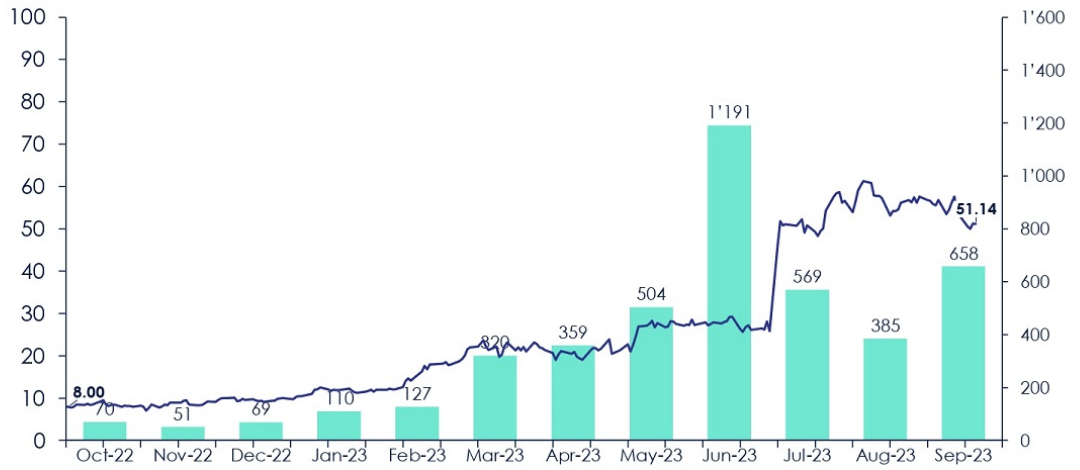
Share price in USD

Average daily trading volume in '000



Fully diluted share count as of June 30, 2023 of 63.2m (Class A + Class C + unexercised options)

1 Bryan Garnier price target not updated since release of H1 data

Analyst coverage

WEDBUSH	$86
Needham	$76
HCW H.C. WAINWRIGHT & CO.	$75
GUGGENHEIM	$70
BTIG	$68
CANTOR Fitzgerald	$65
Jefferies	$63
LEERINK PARTNERS	$60
LIFESCI CAPITAL	$60
BARCLAYS	$42
BRYAN, GARNIER & CO	$32[1]
COWEN	n/a

Source: MoonLake Corporate, Yahoo Finance, Analyst reports

We expect to remain intensely engaged with the market

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July 11-12
Leerink Partners Therapeutics Forum, NYC

August 8-9
Wedbush PacGrow HC Conference, NYC

September 6-8
Wells Fargo HC Conference, Boston

September 19
Stifel Immunology & Inflammation Virtual Summit

September 26-28
Cantor Fitzgerald Global Healthcare Conference, NYC

November 6-7
Guggenheim Annual I&I Conference, NYC

November 8-9
UBS Annual HC Conference, Miami

November 14-16
Jefferies European Healthcare Conference, London

July 3-8
World Congress of Dermatology, Singapore

October 11-14
EADV Congress, Berlin

October 13-15
Symposium on HS advances, Phoenix/AZ

November 10-15
Inflammatory Skin Disease Summit, Vienna

November 10-15
ACR Congress, San Diego

Source: MoonLake Finance

Strategic path forward remains unchanged

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Current owner

- **Phase 3 preparation well under way** incl. study designs, end-of-Ph2 meeting prep, clinical supply, autoinjector partnership, and org ramp up
- **Well funded to drive execution** with strong conviction of existing shareholders and new investors
- **HS and PsA** are feasible (also commercially), **AS and nr-axSpA** are *"low hanging fruits"*, **PsO** remains *"locked value"*, **other indications** provide significant optionality
- Management team execution towards market, from a **position of strength**

Better owner

- **Strategic interest in I&I remains high** and SLK is a leading asset now in Derm and Rheum, with strategic potential across multiple indications and TAs
- **Logic of synergies, speed and breadth** to leverage an existing Ph 3 organization & leading commercial operation
- Single asset, simple org and concentrated ownership makes MLTX attractive

Source: MoonLake

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Q & A

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