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MoonLake Immunotherapeutics

Capital Markets Day 2022

June 7th 2022

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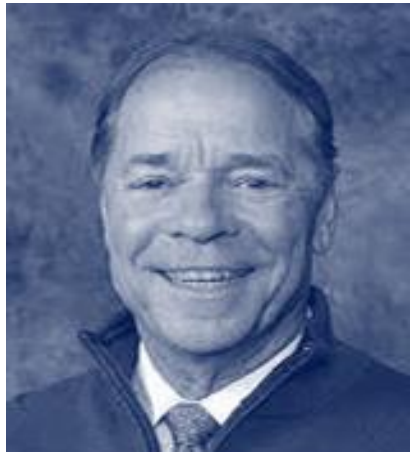
For the session today

J. Santos da Silva PhD
(CEO, Founder)

Kristian Reich MD, PhD
(CSO, Founder)



Chris Ritchlin
MD, MPH

James Krueger
MD, PhD

Matthias Bodenstedt
(CFO)

Agenda

Time	*Topic (speaker)*
20’	Perspective on MoonLake (Jorge)
30’	KOL View: Hidradenitis Suppurativa (Jim)
30’	KOL View: Psoriatic Arthritis (Chris)
20’	Coffee Break
30’	Clinical Development Update (Kristian)
20’	Financial Overview & Guidance (Matthias)
5’	Closing Remarks

Perspective on MoonLake

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- **Founded in 2021** in Switzerland
- **Nanobody® technology** licensed in initial private round
- **Unique molecule & MoA with sonelokimab**, tri-specific IL-17A & IL-1F Nanobody®
- **Public on Nasdaq** in April 2022, with a raise of gross proceeds of $150m (via SPAC deal)
- **Nearly $200m raised** to date
- **Clinical phase company** – concluded phase 2b in psoriasis, additional phase 2 trials now (e.g., HS)
- **Experienced** team, board & investor group

Three Key Messages

1. We are developing **sonelokimab (SLK),** a Nanobody® with potential to elevate treatment outcomes in inflammatory diseases, via IL-17A and IL-17F inhibition

2. Our development program has an **established track record of clinical progress** and **expands SLK's potential** across Dermatology & Rheumatology

3. Our objective is to deliver a **product profile with optionality across large indications** from 2023-24 onwards, driven by a top-tier team

SOURCE: MoonLake Corporate

Do you still Antibody?

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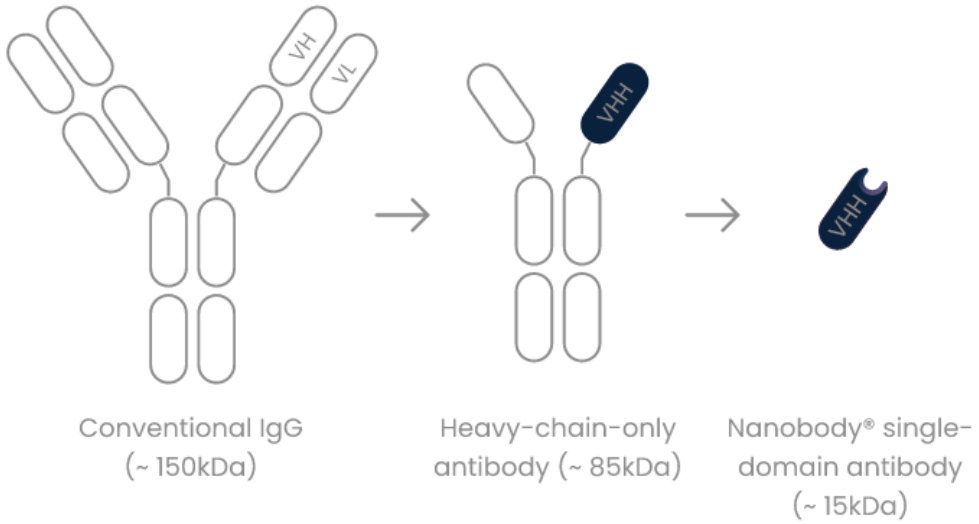
Nanobodies® are much smaller than traditional antibodies



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

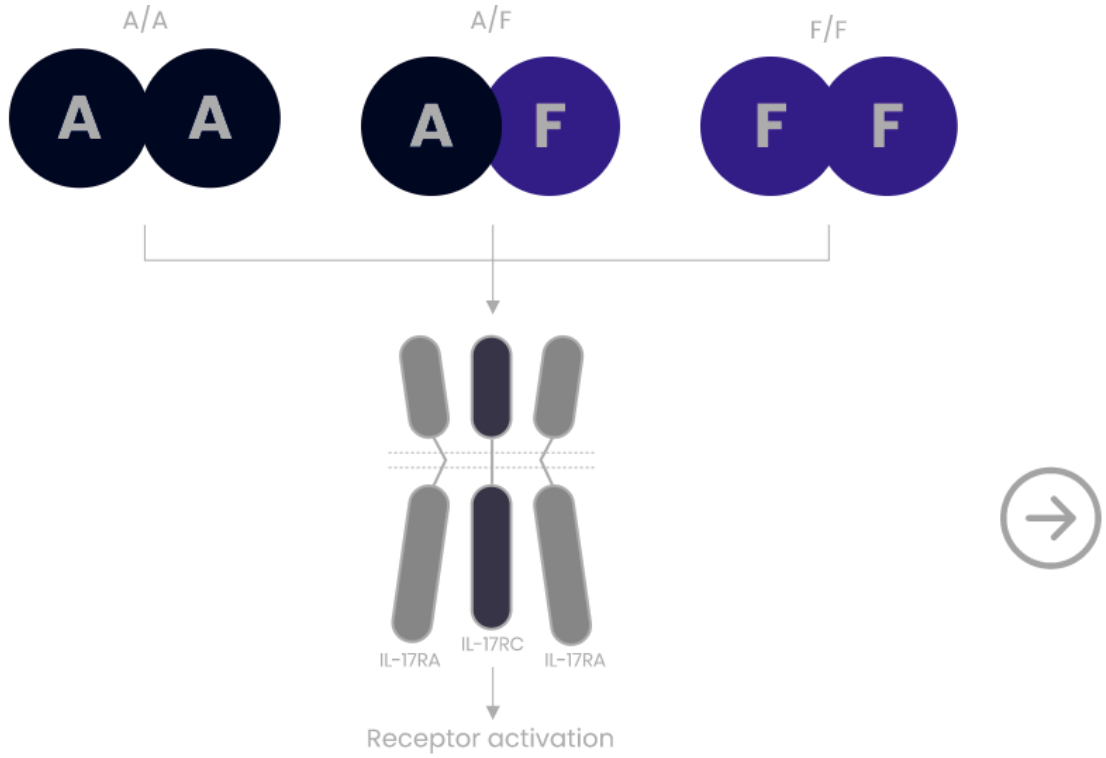
With 2 domains, it binds with high affinity to IL-17A and IL-17F – a third domain binds human albumin

Subcutaneous administration, Q4W

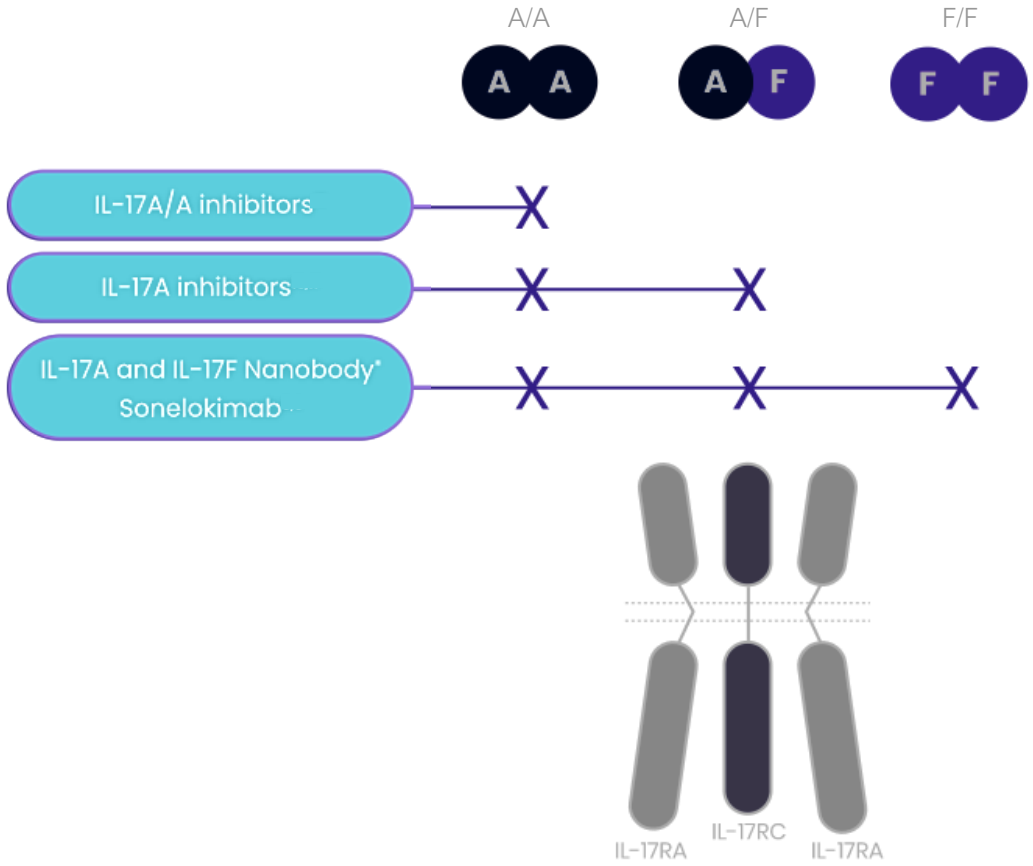
SOURCE: MoonLake Corporate

It’s all about the dimers

Illustrative



IL-17A and IL-17F function as dimers (A/A, A/F, or F/F) to drive inflammation through activation of the IL-17RA/RC receptor complex

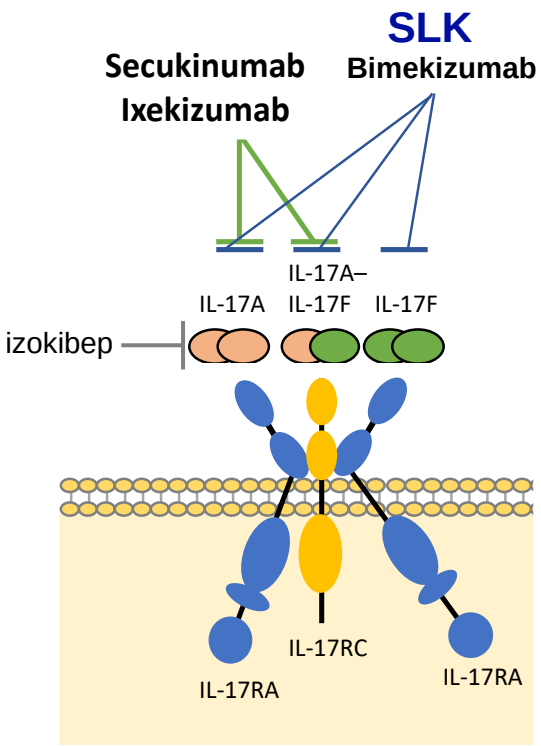


Not all IL-17-targeting therapeutics can inhibit IL-17A/A, IL-17A/F and IL-17F/F dimers

SOURCE: MoonLake Corporate

SLK is the only Nanobody® among IL-17, IL-23 & TNF inhibitors

The key MoA – IL-17 inhibition



The key molecules

Sonelokimab or “SLK”

- MoonLake’s molecule: the only tri-specific Nanobody®, ~3x smaller than a monoclonal antibody, one of only two drugs inhibiting all dimers of IL-17 (A/A, A/F and F/F)

Bimekizumab or “BKZ” (UCB)

- Alongside SLK the only other known molecule inhibiting dimers of IL-17 (A/A, A/F and F/F), recently shown to have leading Phase III efficacy in Psoriasis, high *Candidiasis*

Secukinumab (Cosentyx™, Novartis) or “SEC”

- IL-17 A-specific and does not inhibit IL-17 A/F and F/F dimers, reference IL17i drug in market & main comparator, sales in 2020 of $5B+

Other molecules

TNFi, IL12/23i play a role in Psoriasis and other related diseases, with lower efficacy , and IL23i with efficacy mainly in Psoriasis; IZO is another small molecule but only IL-17A/A

SOURCE: MoonLake

Only two competitors in IL-17A and IL-17F…but very different competitors

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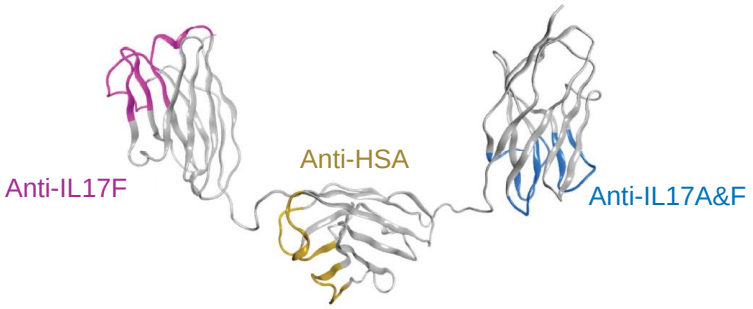
Sonelokimab (SLK)

40 KDa

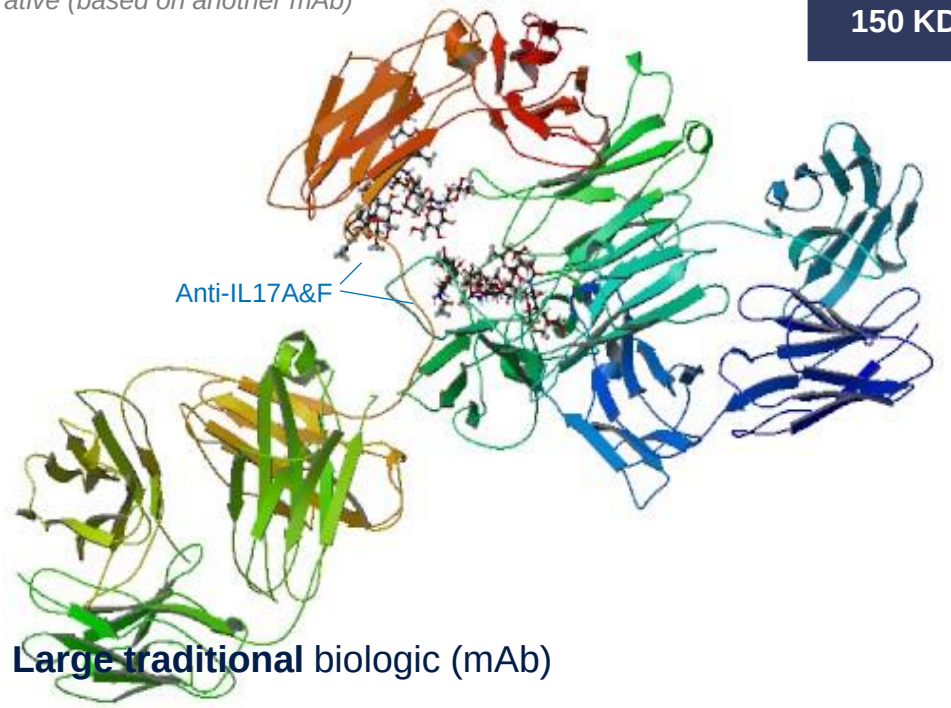


- **Smaller fully active** biologic
- **2x different** IL-17 binding domains
- **Independent Albumin** binding domain
- **Subcutaneous** dosing
- **Shorter** half-life and **differential** dimer inhibition

Bimekizumab (BKZ)

Illustrative (based on another mAb)

150 KDa



- **Large traditional** biologic (mAb)
- **1x** IL-17 binding domain (shared A & F)
- **No Albumin** binding domain
- **Subcutaneous** dosing
- **Longer** half-life and **similar** dimer inhibition

SOURCE: MoonLake

Potential for higher efficacy of SLK versus the IL-17 market leader

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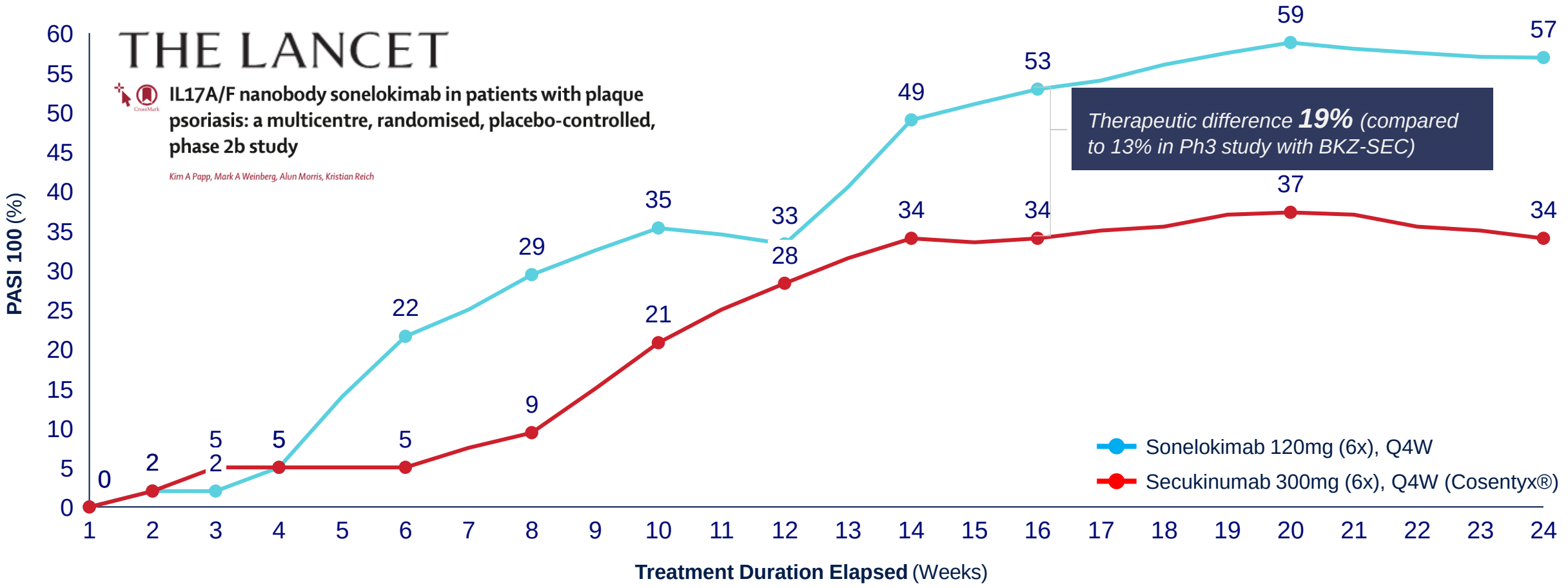
Efficacy comparison between SLK and market leader Cosentyx in Phase II (%)



Differentiated and sustained SLK activity confirmed in 48wk extension trial (313 patients, plus 88 from Ph I)

PASI: Psoriasis Area and Severity Index
SOURCE: Merck KGaA, Darmstadt, Germany, MoonLake

SLK has a differentiated safety profile to date

THE LANCET

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

Kim A Papp, Mark A Weinberg, Alun Morris, Kristian Reich

- **Encouraging overall safety** profile for SLK in the context of all other clinical trials testing biologics for Psoriasis
- Infection rates **similar** in comparison with Secukinumab or other IL-17 inhibitors[1]
- ***Candida* rate similar** to those previously observed with IL-17 inhibitors
- ***Candida* rate 3-4x lower** than Bimekizumab, the only competitor product for IL-17A & F[2]

	Weeks 0–12							Weeks 12–52	
	Placebo group (n=52)	Sonelokimab 30 mg group (n=52)	Sonelokimab 60 mg group (n=52)	Sonelokimab 120 mg normal load group (n=53)	Sonelokimab 120 mg augmented load group (n=51)	All participants on sonelokimab (n=208)	Secukinumab 300 mg group (n=53)	Secukinumab 300 mg group (n=51)	All participants on sonelokimab (n=251)
Treatment-emergent adverse event									
Any	22 (42·3%)	22 (42·3%)	29 (55·8%)	26 (49·1%)	30 (58·8%)	107 (51·4%)	26 (49·1%)	35 (68·6%)	152 (60·6%)
Serious adverse events*	1 (1·9%)	2 (3·8%)	1 (1·9%)	1 (1·9%)	1 (2·0%)	5 (2·4%)	0	2 (3·9%)	12 (4·8%)
Adverse events leading to treatment discontinuation*	0	0	0	1 (1·9%)	2 (3·9%)	3 (1·4%)	0	0	9 (3·5%)
Death	0	0	0	0	0	0	0	0	1 (0·4%)
Common treatment-emergent adverse events†									
Nasopharyngitis	4 (7·7%)	4 (7·7%)	11 (21·2%)	9 (17·0%)	4 (7·8%)	28 (13·5%)	6 (11·3%)	7 (13·7%)	26 (10·4%)
Pruritus	2 (3·8%)	3 (5·8%)	4 (7·7%)	3 (5·7%)	4 (7·8%)	14 (6·7%)	1 (1·9%)	..	..
Upper respiratory tract infection	1 (1·9%)	1 (1·9%)	3 (5·8%)	3 (5·7%)	2 (3·9%)	9 (4·3%)	3 (5·7%)	3 (5·9%)	12 (4·8%)
Headache	1 (1·9%)	0	3 (5·8%)	3 (5·7%)	1 (2·0%)	7 (3·4%)	3 (5·7%)	..	..
Oral candidiasis‡	0	0	1 (1·9%)	2 (3·8%)	3 (5·9%)	6 (2·9%)	0	0	13 (5·2%)
Arthralgia	1 (1·9%)	3 (5·8%)	0	1 (1·9%)	2 (3·9%)	6 (2·9%)	0	..	..
Hypertension	2 (3·8%)	3 (5·8%)	1 (1·9%)	0	2 (3·9%)	6 (2·9%)	1 (1·9%)	..	..
Tonsillitis	..	..	..	..	..	..	..	1 (2·0%)	10 (4·0%)
Diarrhoea	..	..	..	..	..	..	..	2 (3·9%)	9 (3·6%)
Adverse events of special interest									
Any§	11 (21·2%)	11 (21·2%)	22 (42·3%)	17 (32·1%)	18 (35·3%)	68 (32·7%)	15 (28·3%)	23 (45·1%)	114 (45·4%)
Infections	10 (19·2%)	8 (15·4%)	19 (36·5%)	15 (28·3%)	15 (29·4%)	57 (27·4%)	12 (22·6%)	21 (41·2%)	95 (37·8%)
Candida infections¶	0	0	1 (1·9%)	2 (3·8%)	3 (5·9%)	6 (2·9%)	0	1 (2·0%)	16 (6·4%)
Major adverse cardiac event**	0	0	0	0	0	0	0	0	2 (0·8%)
Inflammatory bowel disease	0	0	0	0	0	0	0	0	1 (0·4%)

Data are n (%). *See appendix (p 11) for information on specific events. †During weeks 0–12, common treatment-emergent adverse events were considered as those occurring in 5% or more of participants in any of the sonelokimab-containing groups; during weeks 12–52, common treatment-emergent adverse events were considered as those occurring in 3% of all participants in the all sonelokimab-containing groups combined. ‡Events under preferred term of oral candidiasis for weeks 12–24; see adverse events of special interest for consolidated *Candida* assessment. §Includes infections, injection site reactions, liver function test abnormalities, cerebrocardiovascular events, cytopenia, allergic or hypersensitivity reactions, malignancies, depression, and inflammatory bowel disease. ¶Post-hoc consolidation of adverse event terms to assess oral, oesophageal, and vaginal candidiasis (participants with oral candidiasis, *Candida* infection, oesophageal candidiasis, oropharyngeal candidiasis, or vulvovaginal candidiasis). **Includes myocardial infarction, cerebrovascular accident, or cardiovascular death.

Table 3: **Summary of safety and tolerability results at weeks 0–12 and 12–52 in the safety analysis population**

Consult Table 3[1]

1 Comparisons are not being made in the context of head-to-head trials. 2 Papp KA, Weinberg M, Morris A, Reich K. The Lancet. 2021;397(10284): 1564-1575
SOURCE: MoonLake Clinical Development and selected bibliography

IL-17 inhibition is expected to lead in a significantly growing $40bn+ market

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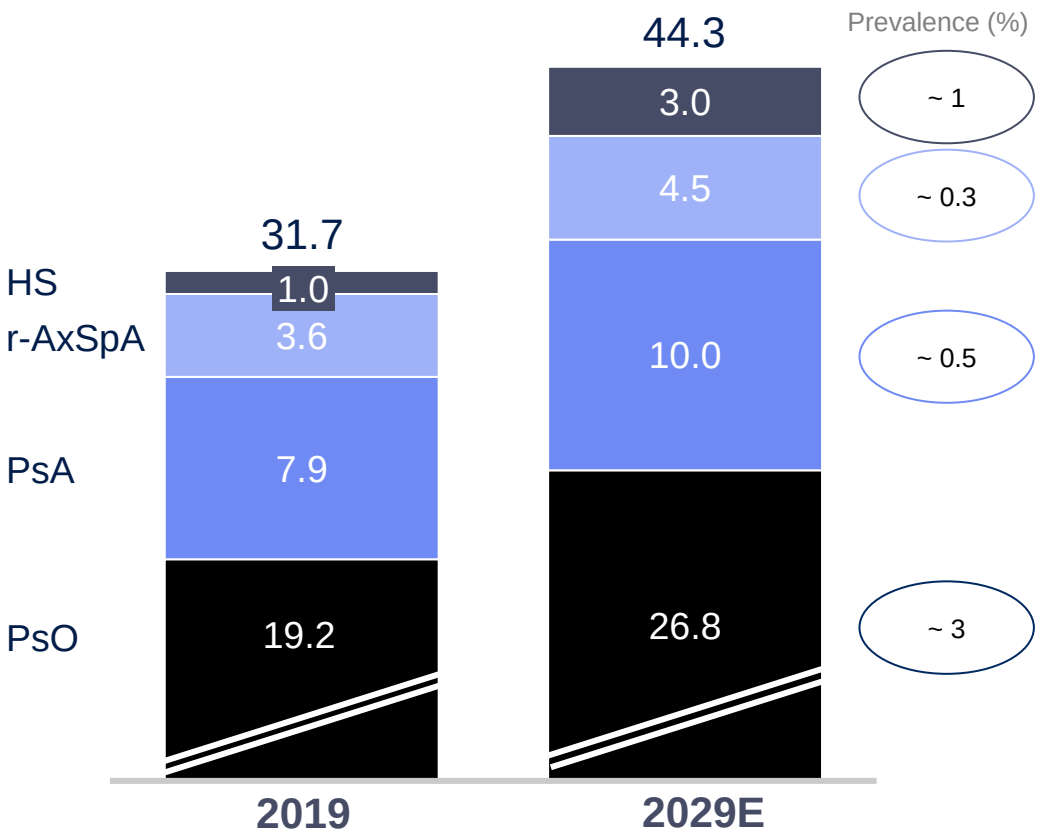
Global sales

USD Bn



IL-17 and other innovative biologics are expected to grow at CAGR 2-3x the rate of the market overall, between 2019 and 2029

Psoriatic Arthritis

- **Driven by IL-17s** with rates of 11%+ growth
- IL-23s falling short
- Mostly IL-17 (incl. IZO) and IL-23 development (also JAKs)

Hidradenitis Suppurativa

- **Driven by IL-17s** on base built by Adalimumab as only therapy
- Diverse targets (e.g., SEC, BKZ, Speso, Vilo, IZO, Berme)

Ankylosing Spondylitis (r-axSpA)

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

Psoriasis

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

SOURCE: IQVIA, Clarivate's Market Forecast Assumptions file for Psoriasis – May 2021 (2019-2029, part of Disease Landscape & Forecast)
DRG's Market Forecast Assumptions file for Psoriatic Arthritis – January 2021 (2019-2029, part of Disease Landscape & Forecast)
DRG's Market Forecast Assumptions file for Axial Spondyloarthritis – January 2021 (2019-2029, part of Disease Landscape & Forecast)

Inhibition of IL-17A & F across select indications underscores SLK potential

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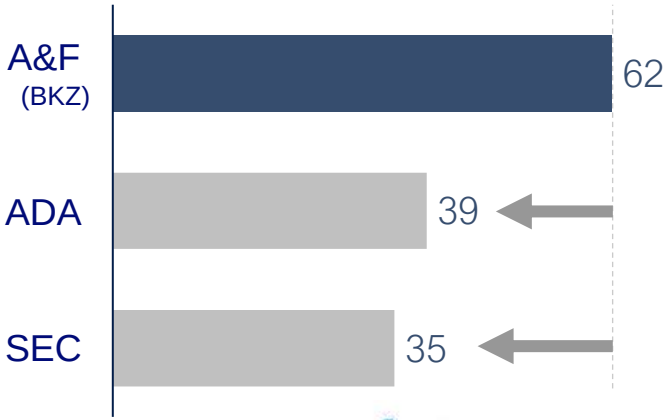
Psoriatic Arthritis (PsA)

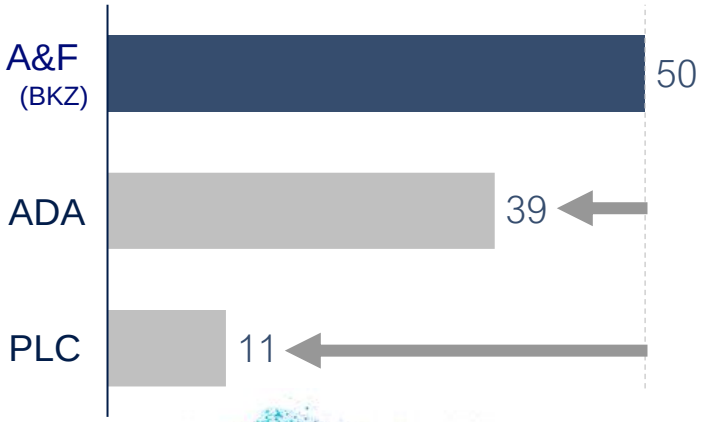
ACR 50 (%) at week 24



Superior performance on ACR 20 &
moving goal to ACR50

Hidradenitis Suppurativa (HS)

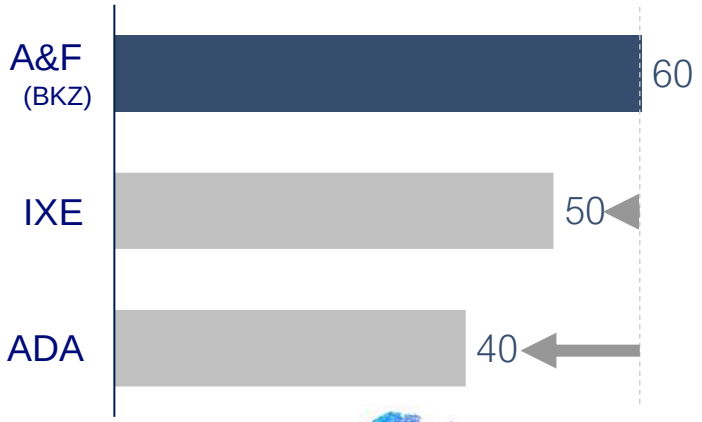
HiSCR75 (%) at week 12



Superior performance on HiSCR50 &
moving goal to HiSCR75

Other: e.g., Ankyl. Spondylitis (AS)

ASAS40 (%) at week 16



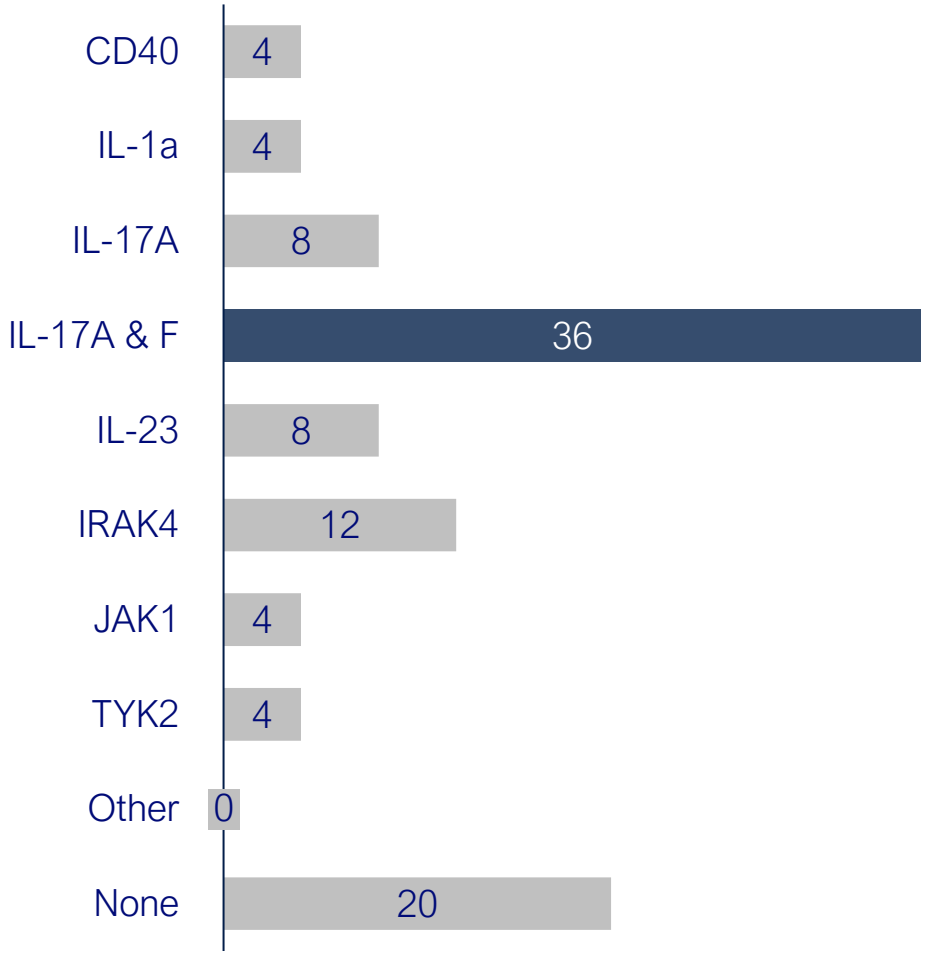
Superior performance on ASAS 20 &
moving goal to ASAS40

1 Ritchlin CT, et al. Lancet 2020;395:427–40; 2 Mease PJ, et al. Arthritis Rheum 2005;52:3279–89; 3 McInnes IB, et al. Lancet 2015;386:1137–46 4 van der Heijde D, et al. Ann Rheum Dis 2020;79:595–604 (approx. 11% TNFi experienced); 5 Dougados M, et al. Ann Rheum Dis 2020;79:176–185 (TNFi naive); 6 Jemec GB et al., presented at 9thConference of the European Hidradenitis Suppurativa Foundation (EHSF) congress, 5-7 February 2020

SOURCE: MoonLake, selected references on clinical trial results; BKZ is phase 2, indirect comparator data PsA is phase 3; in AS, IXE and ADA is from direct comparator trials; in HS, all data is from one phase 2 study)

The MoA and SLK continue to ramp-up momentum

Best target in HS (survey Feb 2022, n=35, %)



SOURCE: Based on insight received by MoonLake management team in Q1 2022 in different events, Equity Research 2022, Cowen. Therapeutics Conference (2021), Health Conference 2022,.UBS, LifeSci, Health

KOL views (Apr 2022)

MoA

- IL-17 now 1 of 3 established targets in HS – “no doubt the IL-17 drugs will come into use”
- “For higher levels of disease clearing, better activity with A&F inhibition”
- A/A and A/F are the most potent on driving IL-17 receptor activation – “increasing protein levels of F/F over A/A as time passes”
- A&F is superior as there may be tissue differences in the production of F – “inhibiting F good for resistance mechanism, fall off with SEC, IL-23”

MLTX

- “HiSCR 75 allows for more discrimination vs placebo, strengthens the study design– can say they have a higher endpoint than anyone. Smart”
- “Nanobody allows targeting multiple cytokines – very logical construction”
- “The size of the ensemble is much smaller so it may be better tissue penetration - where vascular supply isn’t great or you need to penetrate”
- “No active MoA to get an IgG ab into the joint space– the penetration ability of such a small drug may be very important in PsA, HS and others”
- “Not clear why Candidiasis is better (but we can speculate)”

Broader competition in HS, less so in PsA – SLK has unique properties

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Hidradenitis suppurativa

Main Competitors

Secukinumab
(SUNRISE [NCT03713632] and SUNSHINE [NCT03713619])
IL-17A inhibitor (SC)
Phase 3
N = 544 and N = 544
Efficacy, safety, dosing
HiSCR50, Wk 16
Q3 2022
NOVARTIS

Bimekizumab
(BE HEARD I [NCT04242446] and II [NCT04242498])
IL-17A and IL-17F inhibitor (SC)
Phase 3
N = 490 and N = 509
Efficacy, safety, dosing
HiSCR50, Wk 16
Q2/Q3 2023
ucb

Sonelokimab
IL-17A and IL-17F inhibitor (SC)
Phase 2
N = 220 and N = 891
Efficacy, safety, dosing
HiSCR75, Wk 12
Q2/Q3 2023
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New molecules

PF-06700841, and PF-06826647
[NCT04092452]
JAK1/TYK2 inhibitor/ TYK2 inhibitor (PO, QD)
Phase 2
N = 192
Efficacy and safety
HiSCR50, Wk 16
Q1 2022
Pfizer

Imsidolimab
[NCT04856930]
IL-36R inhibitor (SC)
Phase 2
N = 120
Efficacy and safety
Change from baseline
AN count, Wk 16
Q2 2023
AnaptysBio

Bermekimab
(LYRA [NCT04988308])
IL-1α inhibitor (SC)
Phase 2
N = 290
Efficacy, dosing, and safety
HiSCR50, Wk 16
Q1 2024
janssen

Izokibep
IL-17A inhibitor (SC)
Phase 2

31-week study (endpoints not disclosed to date, primary at Wk 16)
2024-2025
ACELYRIN

Psoriatic arthritis

eular

Bimekizumab
(BE COMPLETE [NCT03896581] and BE OPTIMAL [NCT03895203])
IL-17 A and IL-17F inhibitor (SC)
Phase 3
N = 400 / N = 852
Efficacy and safety
ACR50, Wk 16
Q1 2022 / Q3 2022
ucb

Secukinumab
(INVIGORATE 2 [NCT04209205])
IL-17 A inhibitor (IV)
Phase 3
N = 380
Efficacy, safety and tolerability
ACR50, Wk 16
Q2 2022
NOVARTIS

Sonelokimab
IL-17 A and IL-17F inhibitor (SC)
Phase 2
N = 240
Efficacy, safety, dosing
ACR50, Wk16
Q1 2024
MoonLake

eular

Izokibep
[NCT04713072]
IL-17 A inhibitor (SC)
Phase 2
N = 129
Efficacy, tolerability and safety
ACR50, Wk 16
Q1 2022
ACELYRIN

eular

Upadacitinib
SELECT-PsA 1 [NCT03104400]
JAK inhibitor (PO)
Phase 3
N = 1,705
Efficacy (DMARD failure), safety, and dosing
ACR20, Wk 12
Q3 2024
abbvie

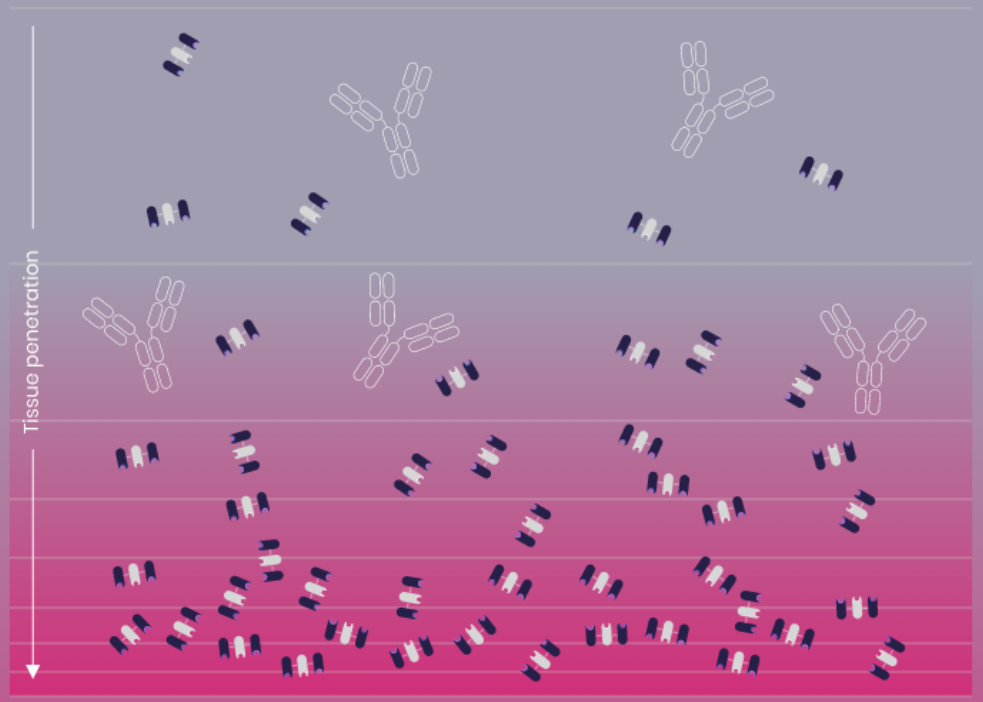
Deucravacitinib
[NCT04908202] and [NCT04908189]
TYK2 inhibitor (PO)
Phase 3
N = 650/ N = 700
Efficacy and safety
ACR20, Wk16
Q3 2024
Bristol Myers Squibb

Other molecules in development include TNFs, IL-23s, IL17-As, JAKs etc.

SOURCE: MoonLake, Press releases, ClinicalTrials.gov

The penetration advantage

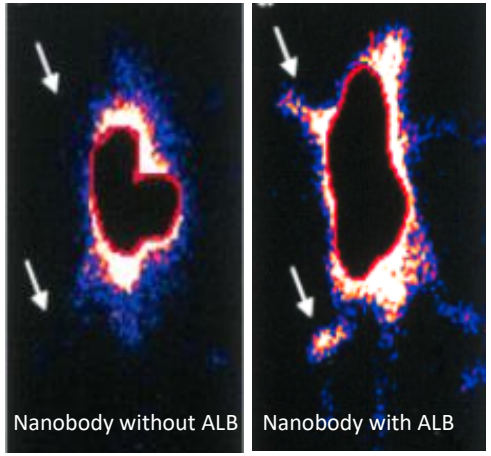
Inflammatory diseases are often characterized by difficult-to-reach tissues



The smaller size of sonelokimab compared with traditional antibodies may enable improved tissue penetration

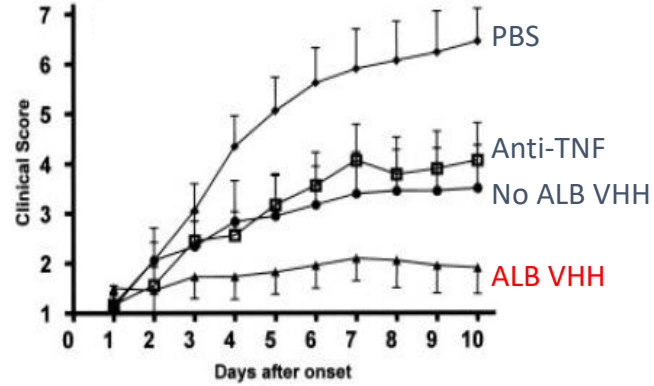
SOURCE: MoonLake Corporate

Albumin binding matters in inflammation



24 h after iv nanobodies; Effects of anti–tumor necrosis factor (anti-TNF) VHH protein constructs on the clinical progression of established collagen-induced arthritis (CIA)

Coppieters K et al., Arthritis Rheum 54, 1856-66 (2006)



Sonelokimab's albumin-binding domain may provide a mechanism for enrichment at sites of chronic inflammation

Differentiation: SLK has the potential to combine properties like no other asset

MoonLake

Safety

"IL-17A & F inhibition without the infections"

Affinity

"Strong on A, balanced on F"

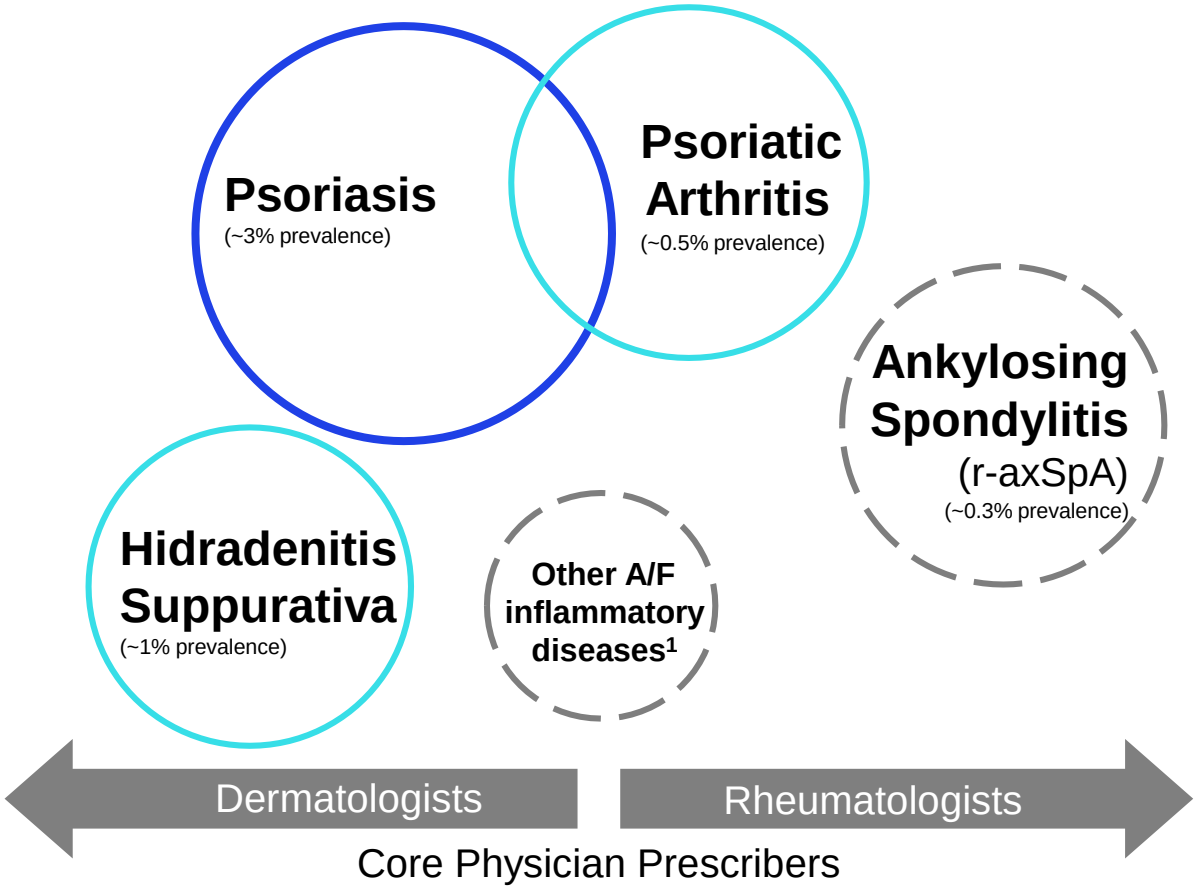
Penetration

"3x smaller + Albumin binding"

SOURCE: MoonLake KOL Ad Boards

SLK has the potential to unlock value in IL-17A & F Inflammatory Diseases

Immediate portfolio of indications for SLK



Drug activity in Psoriasis is proven: First Nanobody® showing improvement of standard of care (Cosentyx™), published in *The Lancet* – supports advancement to PhIII

Significant potential beyond Psoriasis:

1. Upside is exciting: by building on additional diseases that open a market that is 2x larger than Psoriasis (in the aggregate), we provide optionality that can de-risk investment

2. Significant unmet needs beyond Psoriasis: A and F inhibition showing differentiated activity in diseases that are undertreated and show far fewer treatments options

3. Foundation can be even stronger: We plan to generate more data where SLK can realistically beat BKZ (beyond better benefit-risk, also penetration in joints and deep skin), and get the time to create a robust SLK supply

1 Other indications that are being considered by MoonLake, but not prioritized for the Phase 2 model now, include: non-radiographic axial SpondyloArthritis (nr-axSpA), Palmoplantar pustulosis (PPP), generalized pustular psoriasis (GPP), severe pyoderma gangrenosum (sPG)

SOURCE: Nguyen et al. J Eur Acad Dermatol Venereol. 2021;Ingram. Br J Dermatol. 2020; Scotti et al. Semin Arthritis Rheum. 2018; Ogdie et al. Rheumatology (Oxford). 2013; Tekin et al. J Rheumatol. 2019; Alinaghi et al. J Am Acad Dermatol. 2019; Reich et al. Br J Dermatol. 2009; Gelfand et al. Arch Dermatol. 2005; Augustin et al. Acta Derm Venereol. 2010; Stolwijk et al. Arthritis Care Res. 2016; Dean et al. Rheumatology. 2014

Sonelokimab

SLK shows a potentially superior **benefit-risk** profile in Psoriasis (incl. vs BKZ)

SLK is a distinctive molecule with **enhanced enrichment in deep skin & joints** and binding of targets with **better-than-mAb affinity and specificity** – a potentially winning **benefit-risk profile** across **IL-17A & F diseases** (de-risked by BKZ)

We are advancing an extended global Phase 2 program in Derm & Rheum

MoonLake

Approach to clinical design

- MoonLake advancing global, large Phase 2 trials in Dermatology and Rheumatology
- First trials started was for Hidradenitis Suppurativa, a disease with very high unmet need; PsA to start Q3/Q4
- Trials illustrate our preferred approach:
  - Larger size than usual with several arms
  - "Pivotal" designs to accelerate for well-planned superiority Phase 3s, including dosing options
  - Always inclusive of Placebo AND active control (namely Humira) to plan Phase 3 and already mark differences to a "soon-to-be" global biosimilar
  - Higher treatment goal as PE (e.g., HiSCR75, ACR 50) to distinguish SLK, increase delta to placebo
- Reading out in 2023 and 2024

Global Phase 2 program

Hidradenitis suppurativa

- Start date: Apr/May 2022
- MIRA trial (M1095-HS-201; NCT05322473)
- 210 patients
- 60 sites (US and Europe)
- First catalyst: mid-2023

Psoriatic Arthritis

- Start date: Sep/Oct 2022
- xxx trial (M1095-PSA-201; NCTxxx)
- 200 patients
- xx sites (US and Europe)
- First catalyst: end-2023

Other

Not currently pursued in Global program, incl. AS

More details in our Clinical Development and Financial sessions

SOURCE: MoonLake Clinical Development

MoonLake is led and supported by a highly experienced group

Leadership team

Jorge Santos da Silva
(CEO, Founder, Board Director)

Prof. Kristian Reich
(CSO, Founder)

Matthias Bodenstedt
(CFO)

Nuala Brennan
(CCDO)

Oliver Daltrop
(CTO)

150+ yrs experience in Immunology
Plus, 25 FTE at MoonLake today

Board of Directors

Kara Lassen
(Roche)

Catherine Moukheibir
(e.g., Oxford Biomedica)

Simon Sturge – Chair
(e.g., Kymab, Merck)

Spike Loy
(BVF)

Andrew Phillips
(Cormorant)

Ramnik Xavier
(Harvard)

Investors in de-SPAC

BVF PARTNERS L.P.

CORMORANT ASSET MANAGEMENT

SURVEYOR

BLACKROCK

Fidelity INVESTMENTS

TCGX

RTW Investments

683 Capital Management

T.RowePrice

MONASHEE INVESTMENT MANAGEMENT

GHOST TREE CAPITAL

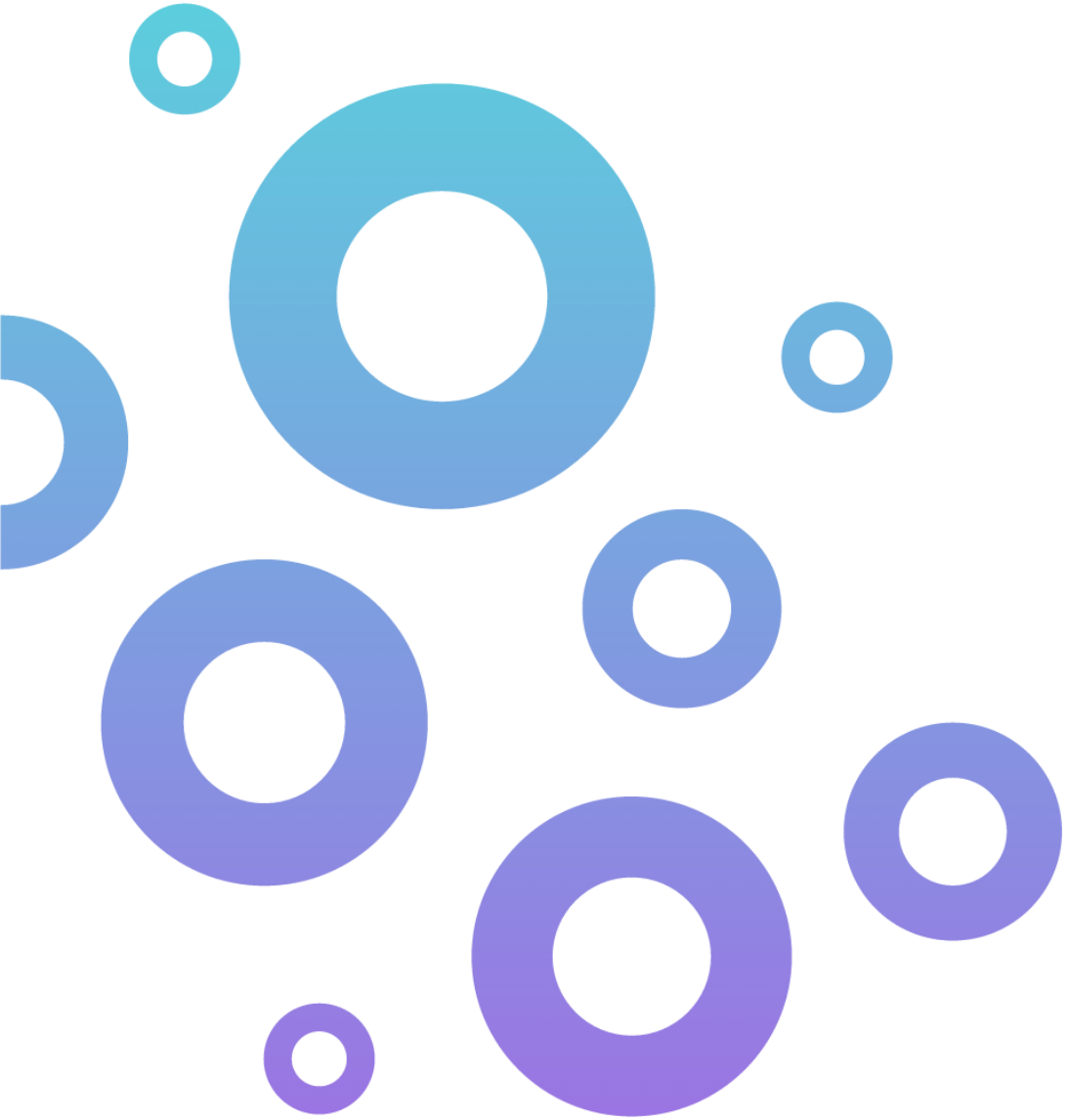
MERCK

B.A.M BALYASNY ASSET MANAGEMENT L.P.

TEKLA Capital Management LLC

Note: Investors mentioned are based on the preliminary Prospectus filed on Form S1-A with the SEC on May 2, 2022 and the Revised definitive proxy soliciting materials filed on Form DEFR14A with the SEC on March 4, 2022
SOURCE: MoonLake Corporate

In summary



- MoonLake is well on its way as a public biotech, one year after being founded
- The innovative Nanobody® sonelokimab is a promising (and largely de-risked) molecule with the potential to revolutionize care
- It moves the clinical paradigm beyond traditional antibodies, to directly target inflammation sites and penetrate difficult-to-reach tissues
- Our global clinical program aims to unlock the value of the Nanobody® across a $4bn+ market, in HS and PsA
- Highly experienced investors, Board and team are advancing to clinical catalysts in 2023, with a very robust financial position

SOURCE: MoonLake Corporate

The KOL view

Hidradenitis Suppurativa

MoonLake

Evolving Pathways & Therapeutic Landscape in Hidradenitis Suppurativa

Professor James G. Krueger (MD, PhD)

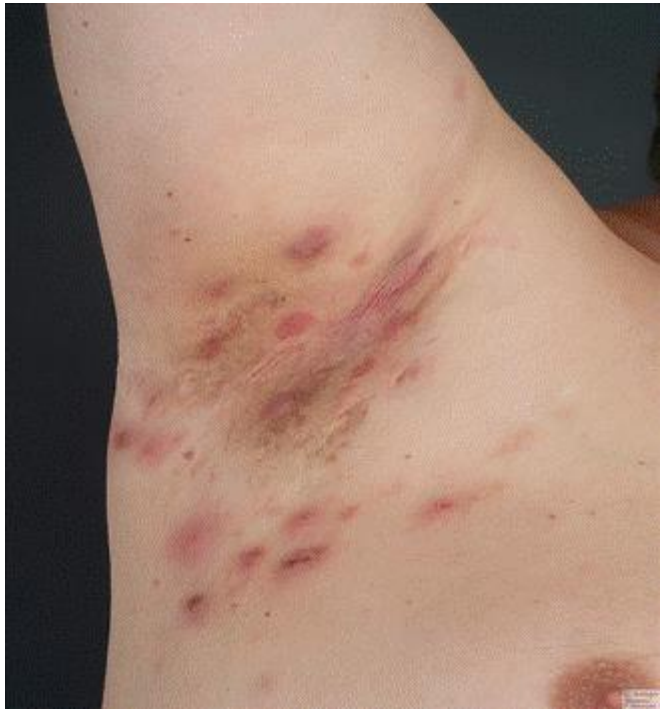
D. Martin Carter Professor in Clinical Investigation
Senior Attending Physician and Laboratory Head, Investigative Dermatology
Co-director, Center for Clinical and Translational Science
Chief Executive Officer, The Rockefeller University Hospital, New York City, NY, USA

MoonLake

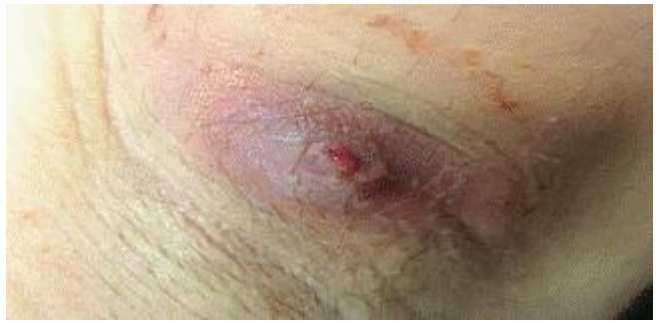
HS is a chronic, inflammatory, recurrent, severely debilitating skin disease

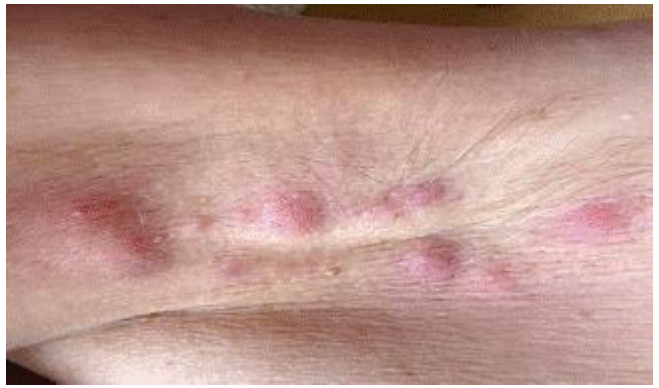
- HS is characterized by inflammatory nodules and abscesses complicated by the formation of pus-discharging dermal tunnels[1]

Inflammatory nodules

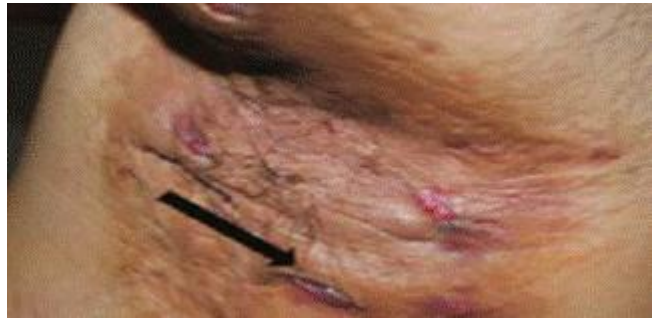


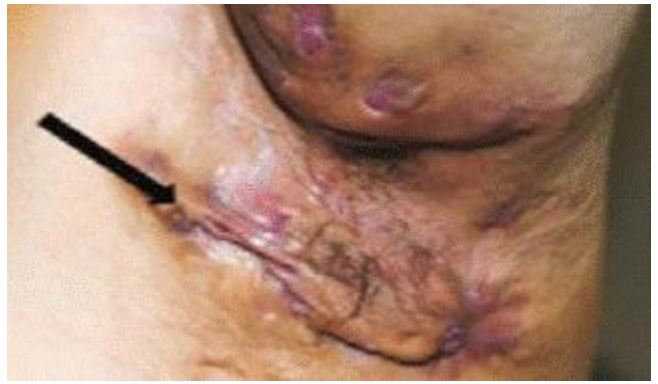
Abscesses





Draining tunnels





- High symptom burden; chronic pain, large amounts of purulent secretions, malodor, and fatigue[1]
- Profound impact on patients lives and contributes to a significant deterioration in physical and mental health[2]

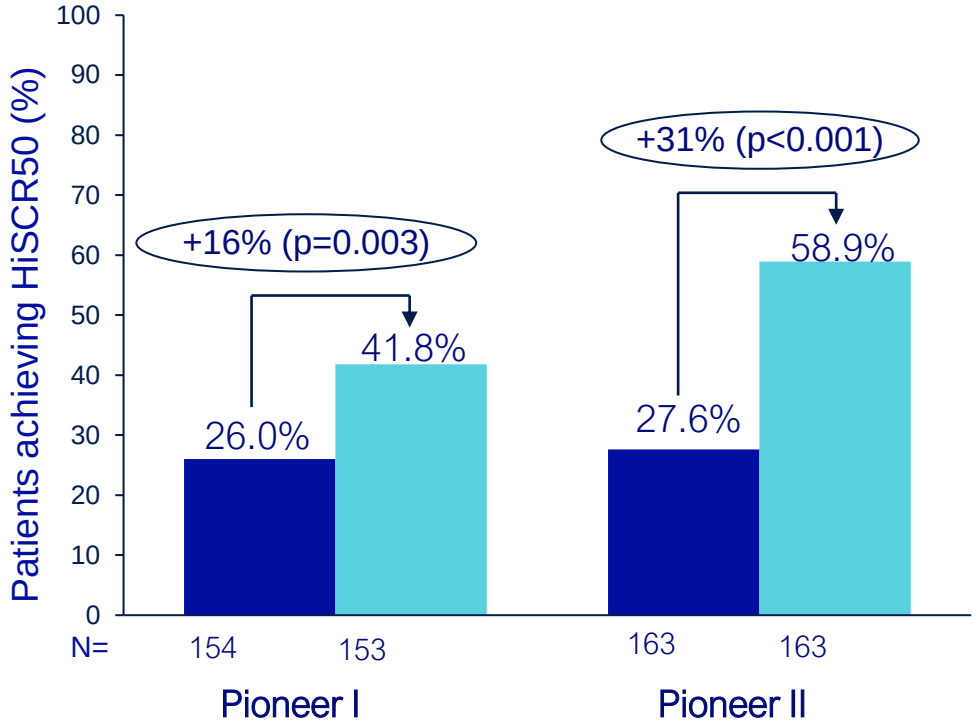
SOURCE (1) Sabat R, et al. Nat Rev Dis Primers. 2020; 6:18; (2) Garg A, et al. J Am Acad Dermatol. 2020
Images: Vekic DA, Cains GD. Aust Fam Physician. 2017;46(8):584-588; Courtesy B Kirby, J Sobell, K Reich

Only one approved biologic – TNF-α inhibitor adalimumab (Humira®)

MoonLake

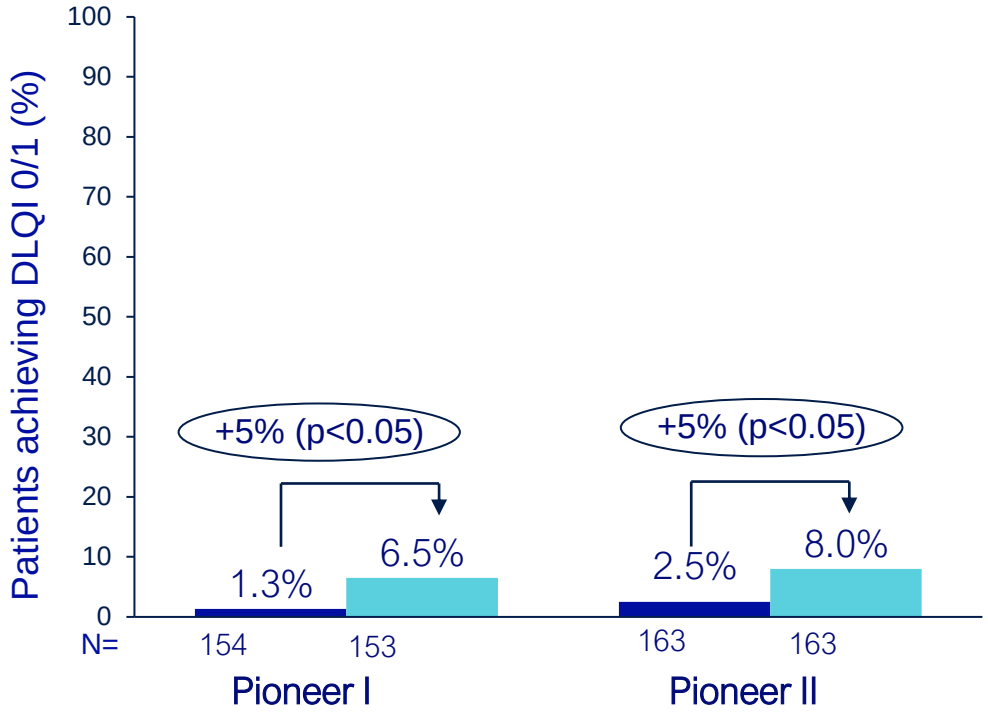
Placebo Adalimumab 40mg weekly

Adalimumab Week 12 HiSCR50 responses[1]



HiSCR50 = At least 50% reduction from baseline in AN count (inflammatory nodules and abscesses), with no increase from baseline in abscess or draining tunnels

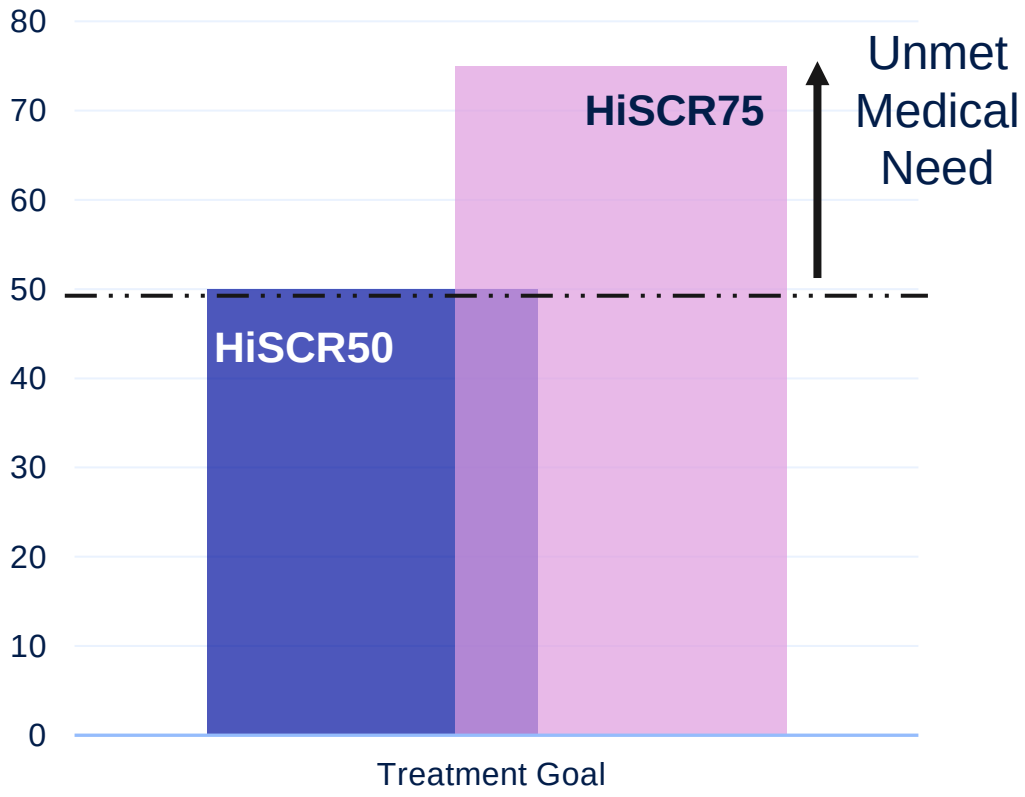
Adalimumab Week 12 DLQI 0/1 responses[1]



DLQI 0/1 = 'No effect' on quality of life from skin disease as measured by Dermatology Life Quality Index (DLQI)

ADA: adalimumab
SOURCE: (1) Kimball AB, *et al. N Engl J Med*. 2016; 375:422-34

Need to elevate treatment goals in HS & assess impact on draining tunnels



- Majority of clinical trials in HS aim for HiSCR50 as primary endpoint
- Treatment goals in HS are low compared with psoriasis e.g. HiSCR50 vs. PASI90

HiSCR

- % reduction from baseline in AN count (**inflammatory nodules and abscesses**), with no increase from baseline in abscess or draining tunnels

IHS4

- Number of **nodules (x1)** + number of **abscesses (x2)** + number of **draining tunnels (x4)**

- Patients perceive draining tunnels as the inflammatory lesion with the greatest negative impact on their lives[1,2]
- HiSCR is focused on inflammatory nodules and abscesses and does not capture impact on draining tunnels
- IHS4 is a<n instrument accounting for draining tunnels in addition to inflammatory nodules and abscesses that can be used in conjunction with HiSCR[3]

SOURCE: (1) Garg A, et al. J Am Acad Dermatol. 2020; 82:366–76; (2) Thorlacius L, et al. Br J Dermatol. 2018; 179:642–650; (3) Zouboulis C. et al. Br J Dermatol. 2017; 177:1401-1409

Snapshot of HS Drug Development Landscape

Category	Agent	Target	Phase	NCT	Primary endpoint
Monoclonal antibody	Secukinumab	IL-17A	3	NCT03713632 NCT03713619	HiSCR50 (primary endpoint met, data to be reported)
	Bimekizumab	IL-17A and IL-17F	3	NCT04242446 NCT04242498	HiSCR50
	Bermekimab	IL-1α	2	NCT04988308	HiSCR50
	Guselkumab	IL-23p19	2	NCT03628924	HiSCR50 (primary endpoint not met)
	Vilobelimab	C5a	2	NCT03487276	HiSCR50 (primary endpoint not met)
Nanobody	Sonelokimab	IL-17A and IL-17F	2	NCT05322473	HiSCR75
Antibody mimetic	Izokibep	IL-17A/A	2	NCT05355805	HiSCR50
Small molecule inhibitor	INCB054707	JAK-1	2	NCT04476043	Mean change in total AN count

No animal models of HS exist, making delineation of the key pathways driving disease pathogenesis challenging

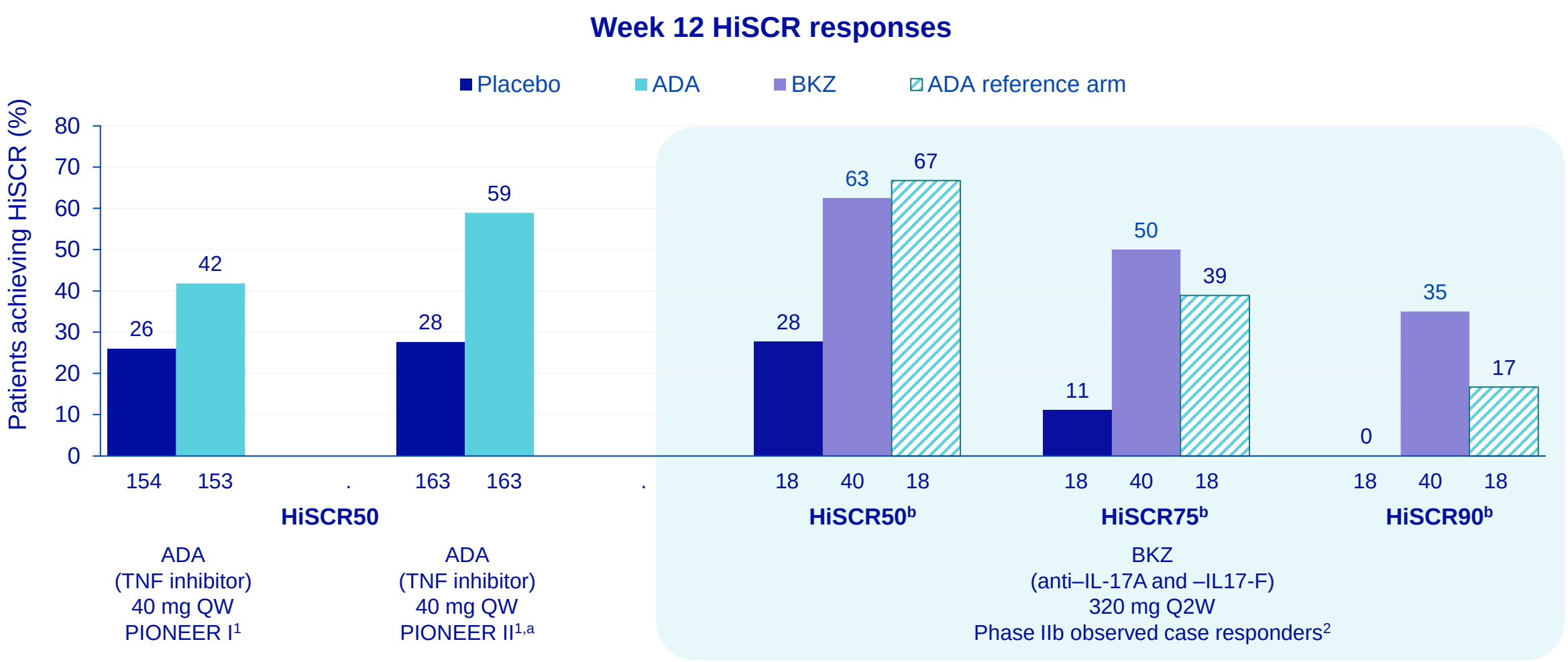
Multiple molecules are being explored in the clinic with diverse targets

Therapeutic successes/failures are helping decipher key pathways underpinning disease – **bedside to bench approach**

IL-17 inhibition furthest advanced both from a clinical & molecular perspective

Other targets in development include and not limited to; IL-36R, CD40, IL-1α/IL-1β
SOURCE: Clinicaltrials.gov

Inhibition of IL-17A and IL-17F has the potential to reach a greater threshold of clinical response (HiSCR75, HiSCR90)

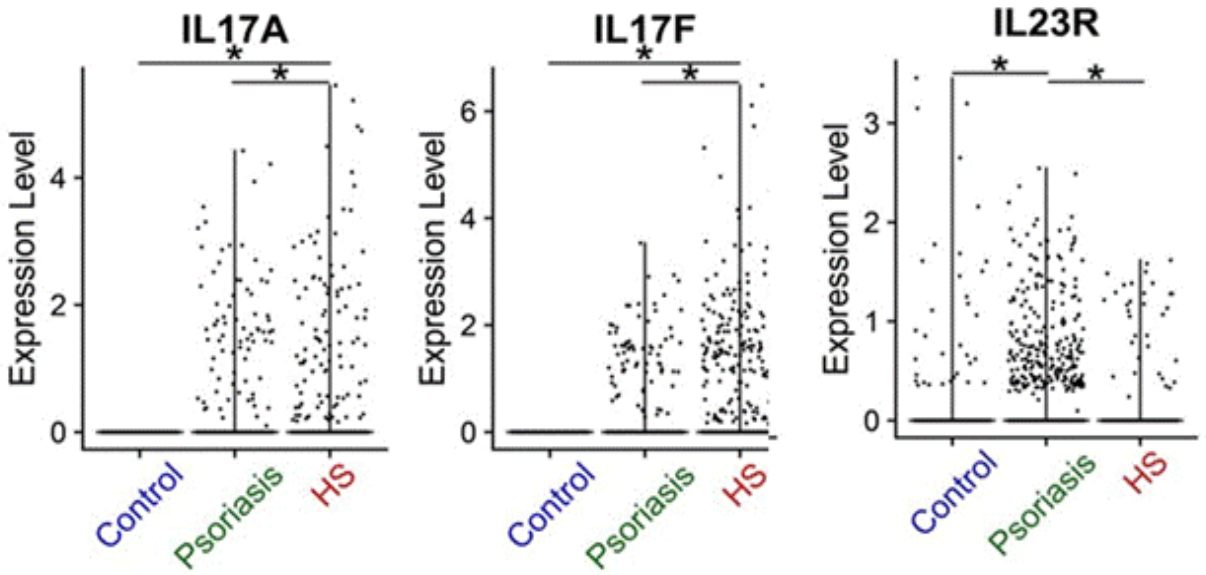


[a]PIONEER II allowed concomitant antibiotic use; [b]Observed case responders at Week 12.
ADA, adalimumab; BKZ, bimekizumab; HiSCR, Hidradenitis Suppurativa Clinical Response; QW, every week; Q2W, every 2 weeks; TNF, tumor necrosis factor.
SOURCE: (1) Kimball AB, *et al. N Engl J Med*. 2016; 375:422-34; (2) Glatt S, *et al. JAMA Dermatol*. 2021; 157:1279-88.

Increasing scientific evidence supports IL-17A- and IL-17F-mediated inflammation as a key driver of the pathogenesis of HS

- Elevated serum IL-17 levels in patients with HS[1–3]
- Upregulation of IL-17A and IL-17F mRNA in HS tissue[1–3]
- Compared with Psoriasis;
  - More cells express IL-17F in HS[4]
  - Fewer cells express IL-23R in HS[4]

IL-17A and IL-17F expressing cells abundant in HS[4]



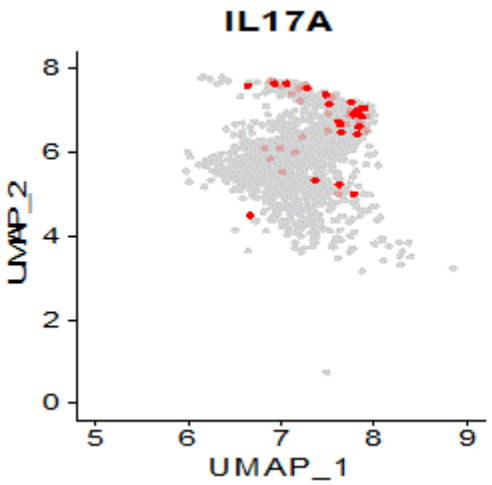
Each dot represents a single cell

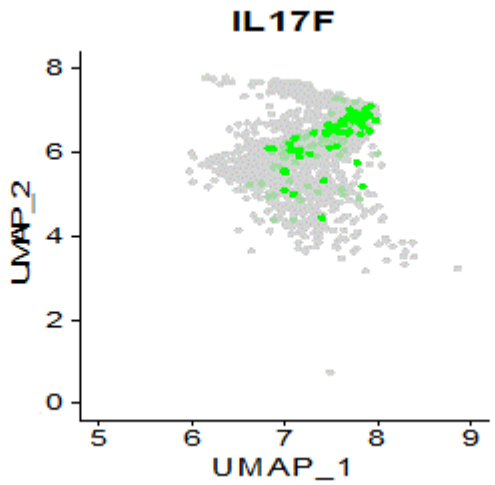
CD4, cluster of differentiation 4

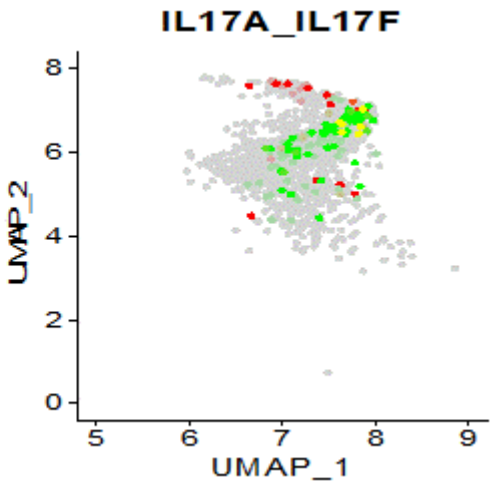
SOURCE: (1) Navrazhina KJW, et al. Br J Dermatol. 2020; 182:1045–7; (2) Matusiak Ł, et al. J Am Acad Dermatol. 2017; 76:670–75; (3) Rumberger BE, et al. Inflamm Res. 2020; 69:967–73; (4) Jaewhan K. et al., Society of Investigative Dermatology 2022; Abstract(807)

In HS, there is a predominance towards Type17 T-cells that discretely produce IL-17F compared to IL-17A[1]

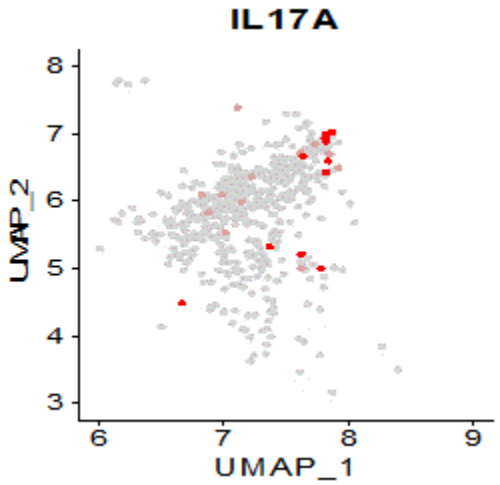
PsO

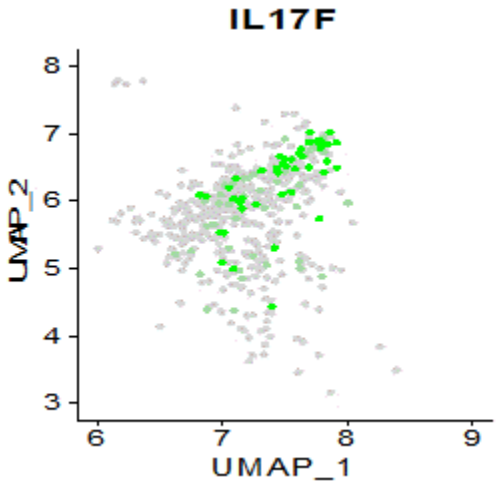


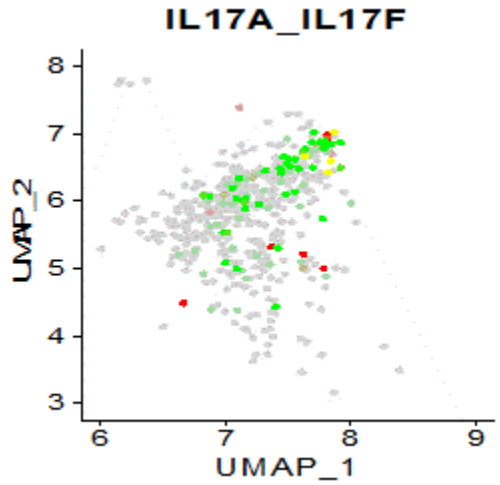




HS







The dots are single cells

Red: Express IL-17A

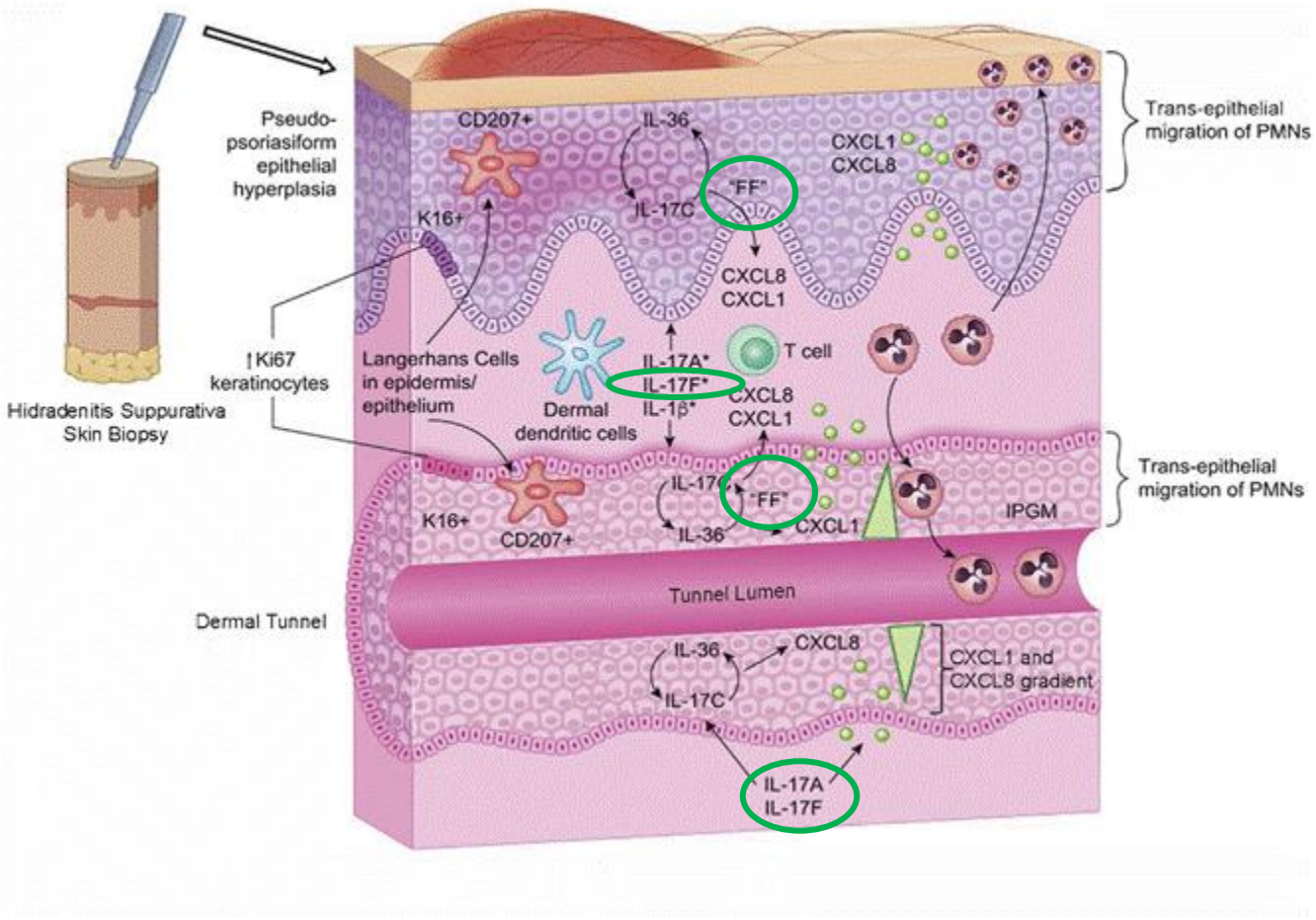
Green: Express IL-17F

Yellow: IL-17A ***and*** IL-17F

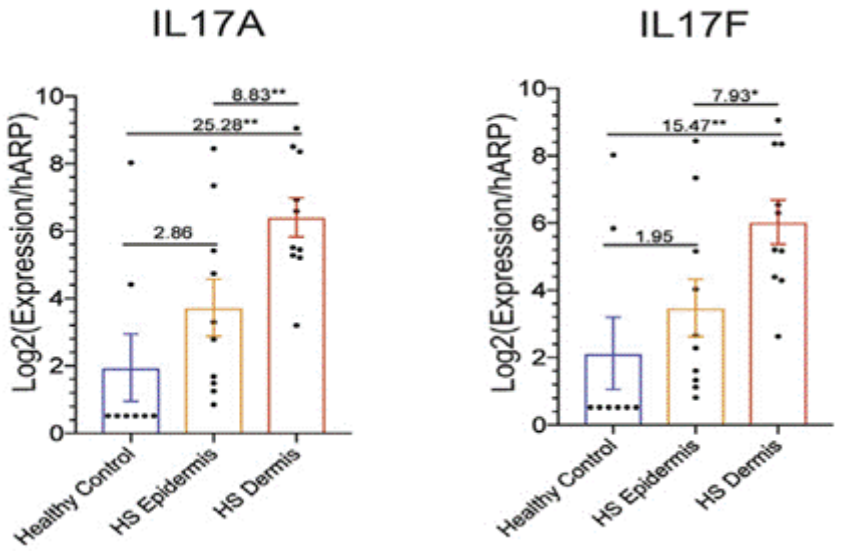
SOURCE: (1) Jaewhan K. et al., Society of Investigative Dermatology 2022; Abstract (807)

Key role of IL-17F in activation of epithelialized tunnels and neutrophil influx in HS

IL-17F is present throughout HS lesions



Draining tunnels are deep HS lesions that express IL-17A and IL-17F[1]



Inhibition of IL17 signaling in patients using the IL-17RA antagonist brodalumab[1] results in a significant decrease in

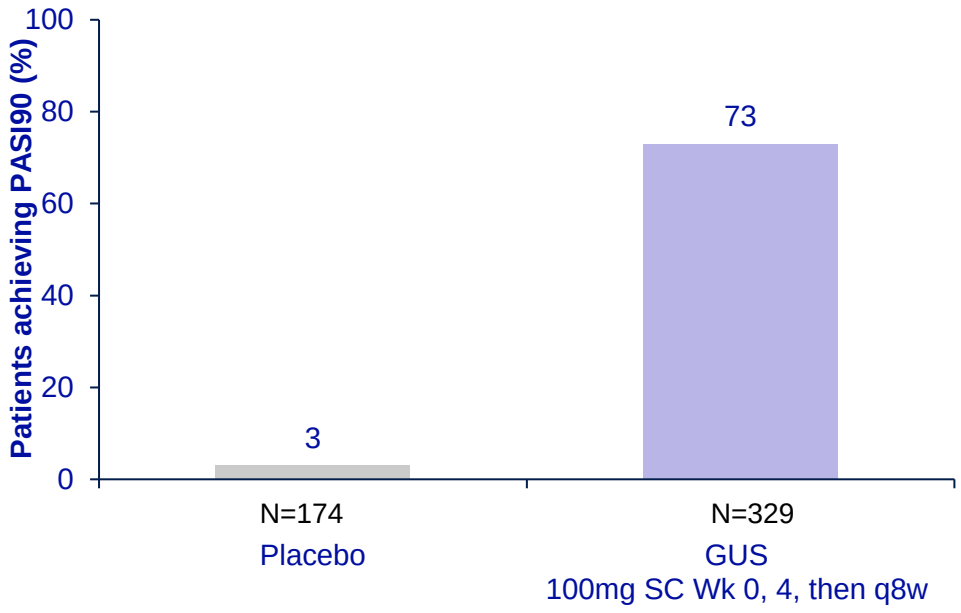
1) HS Lesion thickness
2) Tunnel wall diameter
3) Tunnel Inflammation

SOURCE: (1) Navrazhina KJW, et al. J Allergy Clin Immunol. 2021; 147:2213–24.

Efficacy in Psoriasis is no prediction of efficacy in HS

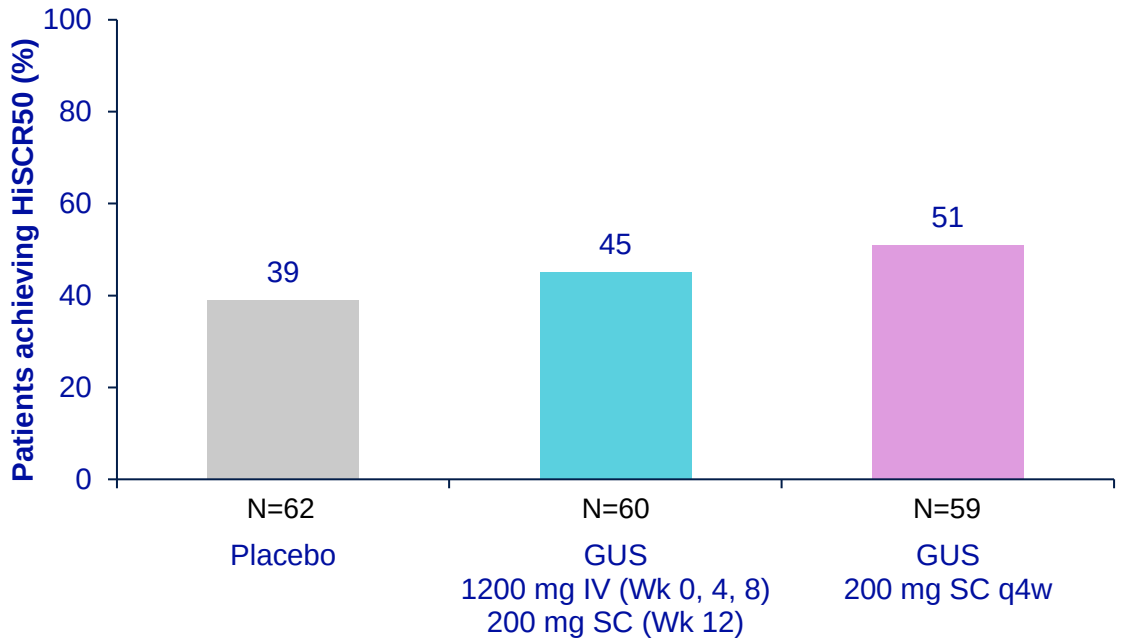
Guselkumab (IL-23p19 inhibitor)

Psoriasis



Phase III PSO[1] Week 16 PASI90 response

HS



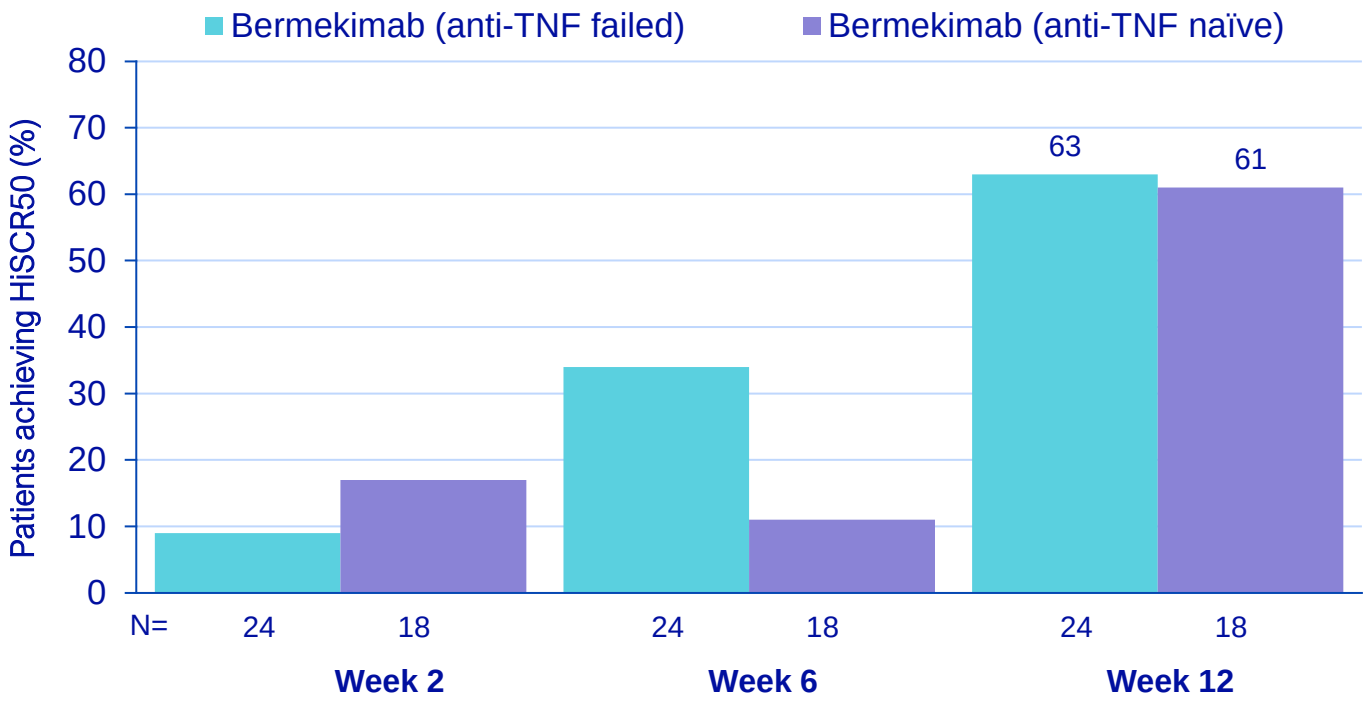
Phase II HS[2] Week 16 HiSCR50 response

Pathogenic T cells in HS may be regulated independently of IL-23

GUS: guselkumab
SOURCE: (1) Blauvelt A et al. J Am Acad Dermatol. 2017;76(3):405-417; (2) NCT03628924

Inhibition of IL-1α has shown potential efficacy in an open label study

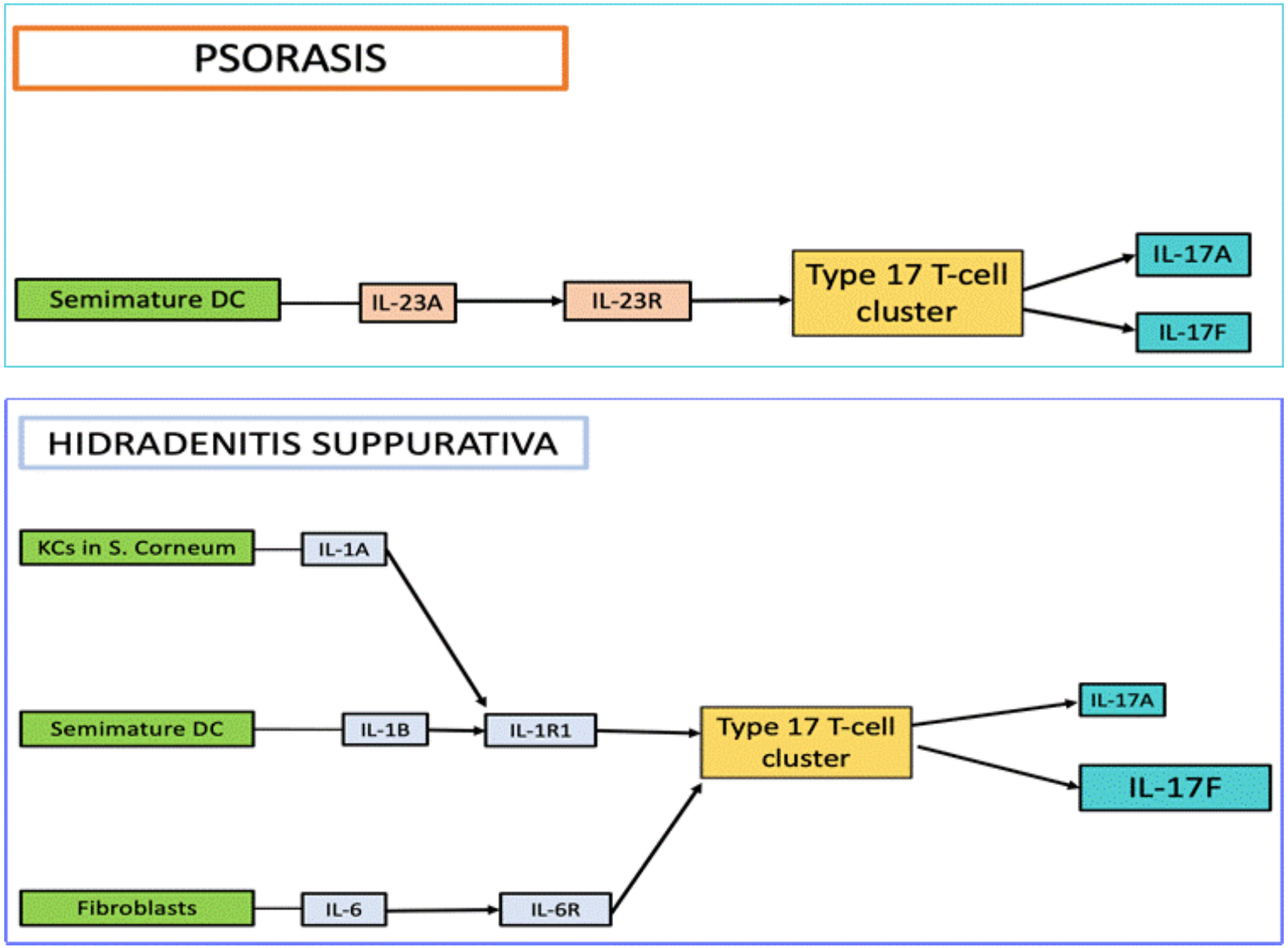
Biology of IL-1α in HS still unclear



Bermekimab (IL-1α inhibitor) 400 mg QW
Phase II open-label study[1] (not placebo controlled)

SOURCE: (1) Gottlieb A, et al. J Invest Dermatol. 2020; 140:1538–1545

Proposed mechanism of Type17 T-cell activation in HS vs Psoriasis



SOURCE: Jaewhan K. et al., Society of Investigative Dermatology 2022; Abstract(807)

Recap of todays learnings

1. Need for higher treatment goals in HS and measurement of tunnels

2. IL-17F is the dominant cytokine in HS

3. IL-1α role and potential in HS still unclear

4. IL-23p19 inhibition failed; may have limited role in HS relative to psoriasis

Which pathway would you pick?

Which pathway would you pick?

TNF-α
Established pathway in HS

IL-17A and IL-17F
Several data sets supporting IL-17 targeting: Secukinumab, Bimekizumab and Brodalumab

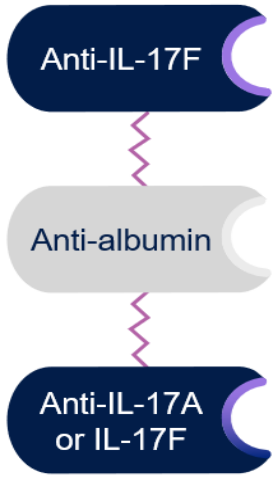
- Secukinumab met Phase 3 primary endpoint (HiSCR50)
- Bimekizumab Phase 3 close to completion
- Brodalumab impact on tunnel-associated inflammation/drainage
- Clinical evidence from IL-17i match insights from basic & molecular biology

IL-1α
More data needed: clinical and molecular

Optimizing IL-17A and IL-17F inhibition for the management of HS

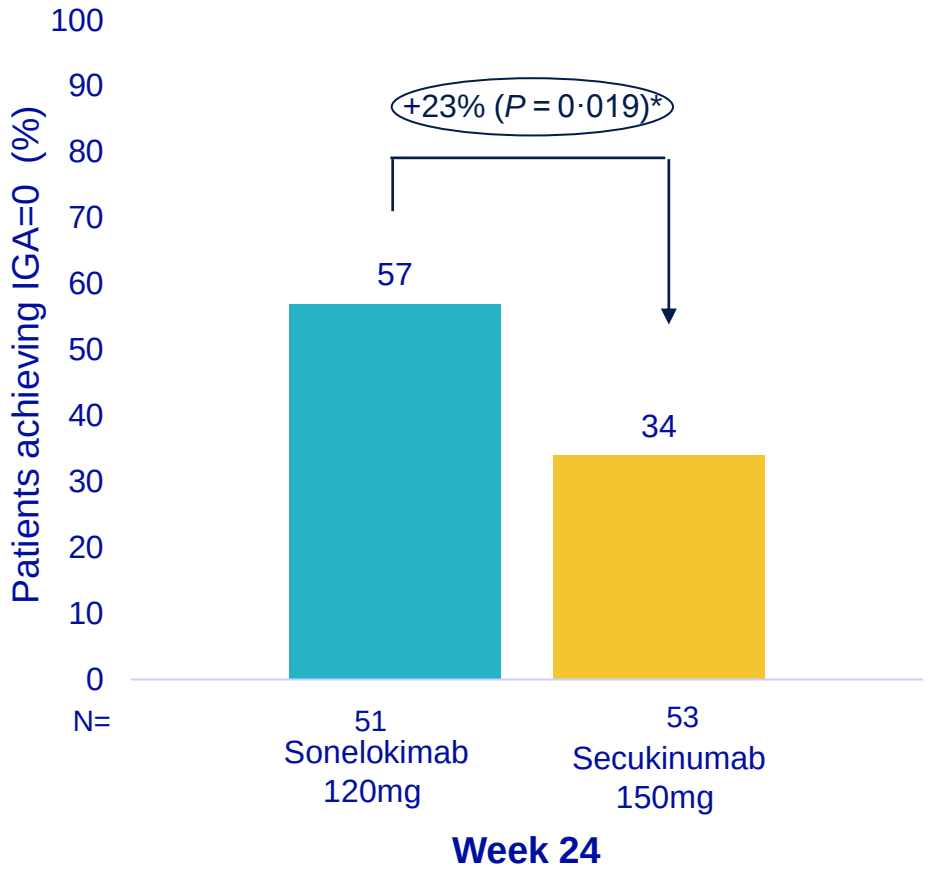
MoonLake

Sonelokimab



- **Smaller size vs. monoclonal antibodies**
- **2x different** IL-17 binding domains
- **Independent Albumin** binding domain

Complete Clearance Psoriasis[1]
Phase 2b



*Nominal P

SOURCE: (1) Reich K, et al. Br J Dermatol. 2022: DOI: 10.1111/bjd.21617

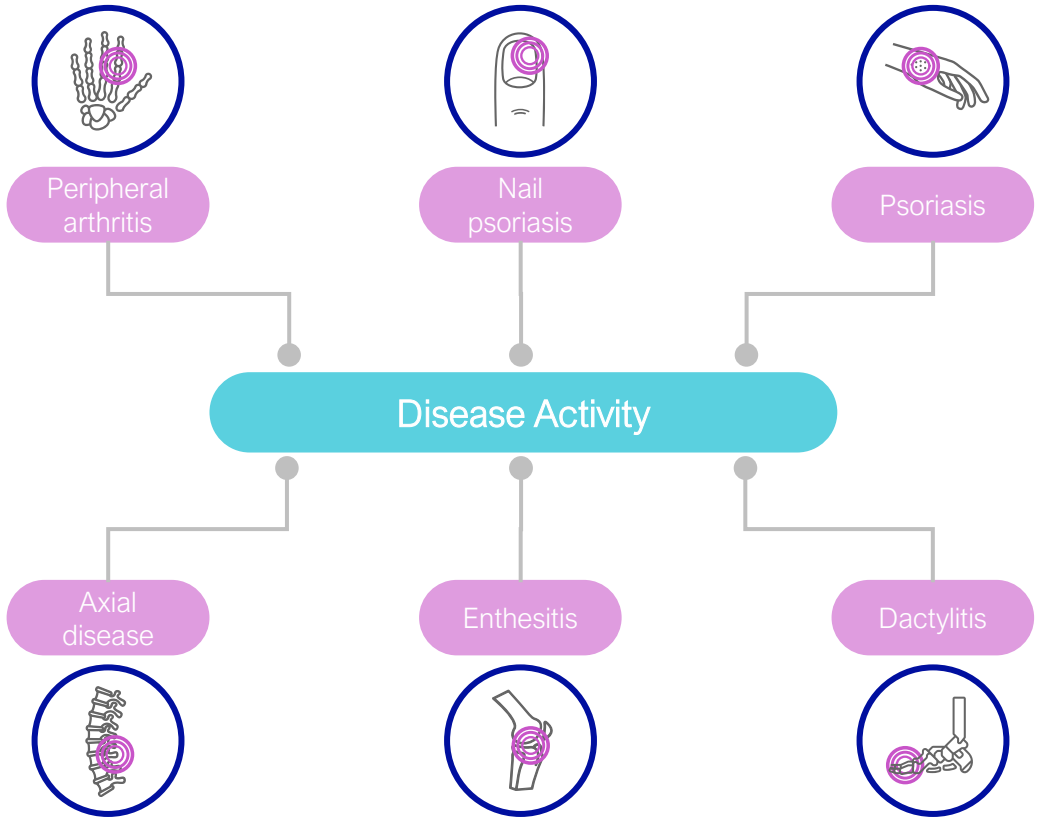
The KOL view

Psoriatic Arthritis

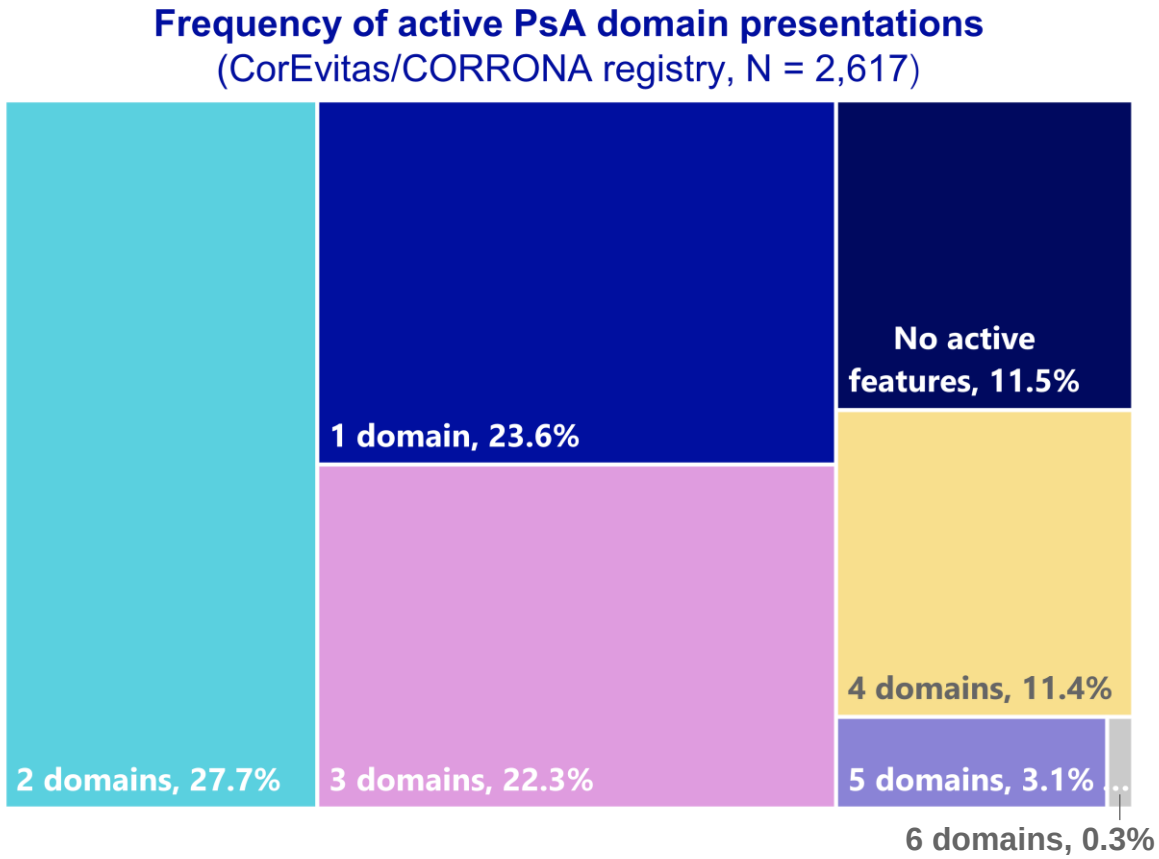
MoonLake

PsA is a complex and heterogeneous disease

The clinical features of PsA are diverse, comprising both musculoskeletal and non-musculoskeletal manifestations[1]



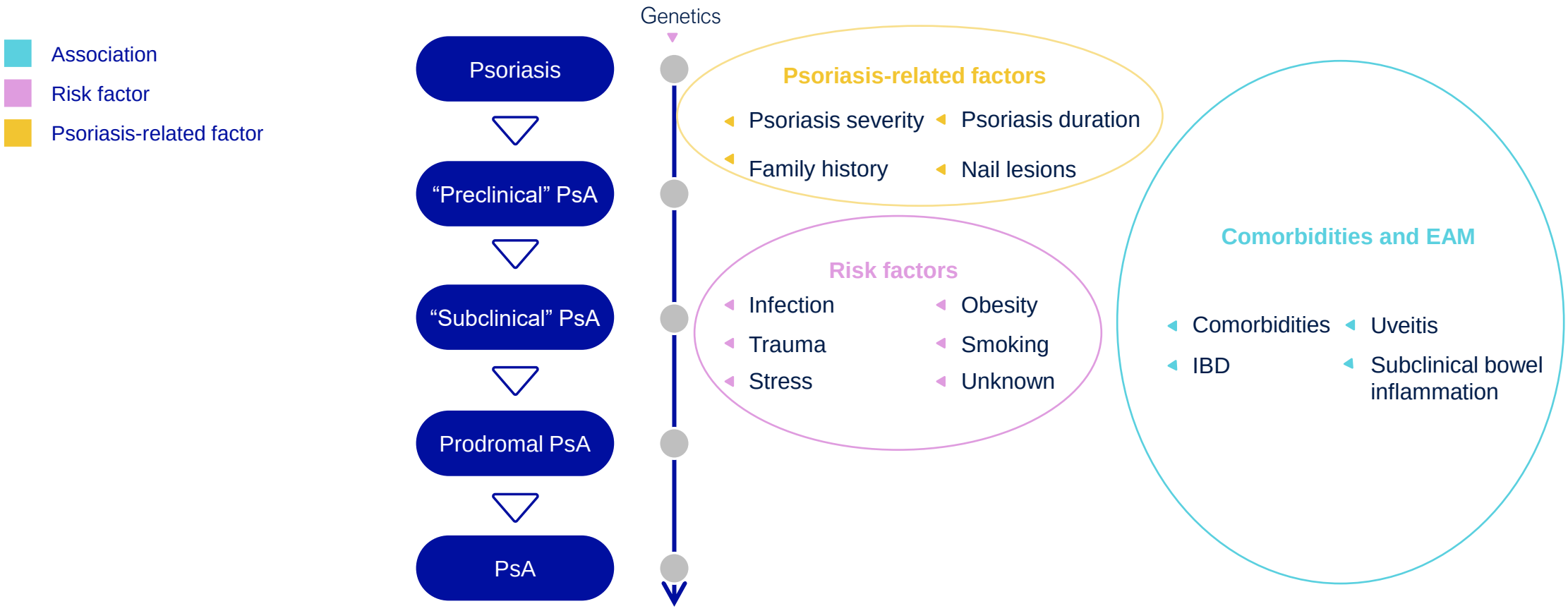
Most patients with PsA have multi-domain disease involvement[2]



PsA, psoriatic arthritis.
Figure adapted from FitzGerald O, et al. Nat Rev Dis Primers. 2021;12:7:59.
SOURCE:(1) Ogdie A, et al. Rheumatology (Oxford). 2020;59(Suppl 1):i37-46; (2) Ogdie, A. et al. J Rheum. 2021;48:698–706

Early intervention is needed

PsA affects up to 30% of patients with psoriasis[1] and involves a complex interaction of risk factors, psoriasis-related factors, and associations with comorbidities and EAMs[2]



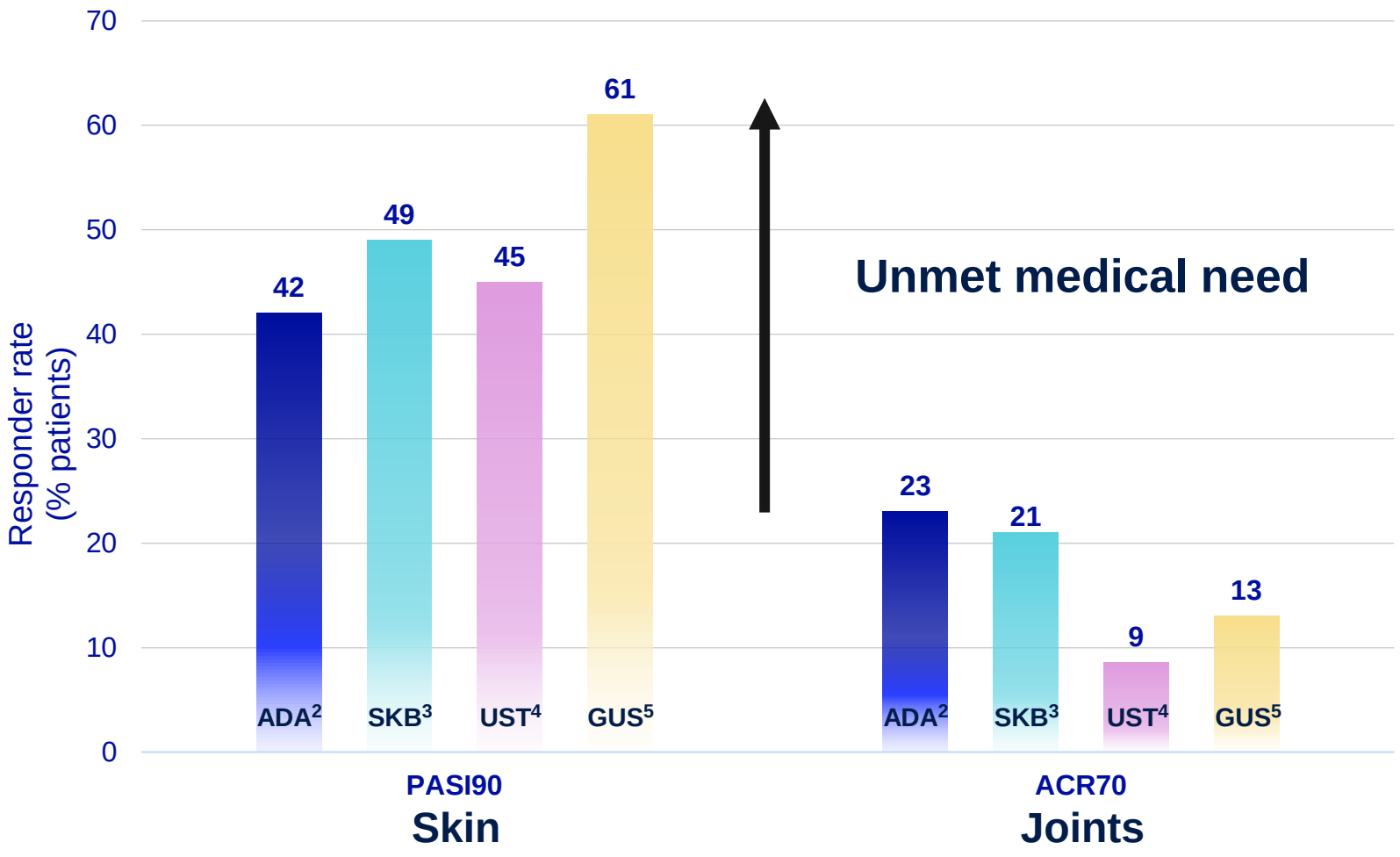
IBD, inflammatory bowel disease; EAM, extra-articular manifestation; PsA, psoriatic arthritis
Figure adapted from Karmacharya P, et al. Best Prac Res Clin Rheum. 2021;35:101692
SOURCE: (1) FitzGerald O, et al. Nat Rev Dis Primers. 2021;12:7:59; (2) Karmacharya P, et al. Best Prac Res Clin Rheum. 2021;35:101692

Several available therapies [1,2] …

Oral Small Molecule	• methotrexate, sulfasalazine, cyclosporine, leflunomide, apremilast
TNFi	• etanercept, infliximab, adalimumab, golimumab, certolizumab pegol
IL12/23i	• ustekinumab
IL17i	• secukinumab, ixekizumab
CTLA4-Ig	• abatacept
JAK inhibitor	• tofacitinib, upadacitinib
IL23i	• guselkumab, rizankizumab

SOURCE: Adapted from (1) Singh JA, et al. Arthritis Rheumatol. 2019; 71:5–32; (2) FitzGerald O, et al. Nat Rev Dis Primers. 2021; 12:7:59

…yet still significant unmet medical need[1,6]



Divergent skin vs. tissues responses driven by:

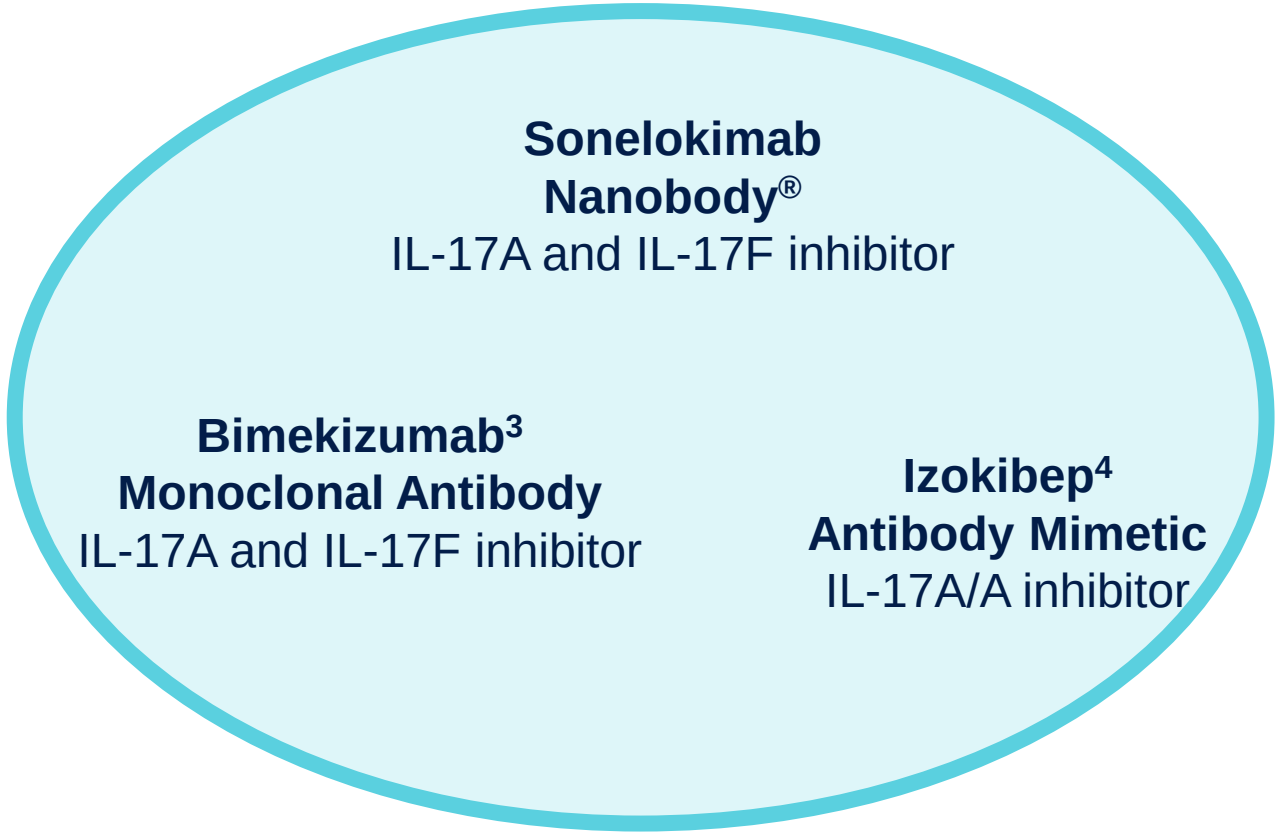
1)Tissue penetration?

2) Differential cytokine expression profiles across skin vs. joints?

All timepoints at Week 24. Not head-to-head comparisons; for illustrative purposes only.
ACR, American Society of Rheumatology score; ADA, adalimumab; GUS, guselkumab; PASI, Psoriasis Area and Severity Index; SKB, secukinumab; UST, ustekinumab.
SOURCE: (1) FitzGerald O, et al. Nat Rev Dis Primers. 2021;12:7:59; (2) Mease PJ, et al. Arthritis Rheum. 2005;52:3279–3289; (3) McInnes IB, et al. Lancet. 2015;386;1137–1146; (4) Ritchlin C, et al. Ann Rheum Dis. 2014;73:990–999 (and supplementary data); (5) Mease PJ, et al. Lancet. 2020;395:1126–1136; (6) Scher JU, et al. Arthritis Rheumatol. 2021;73:1574–1578;

However, more molecules in development

Deucravacitinib[1]
Small Molecule
TYK2 inhibitor

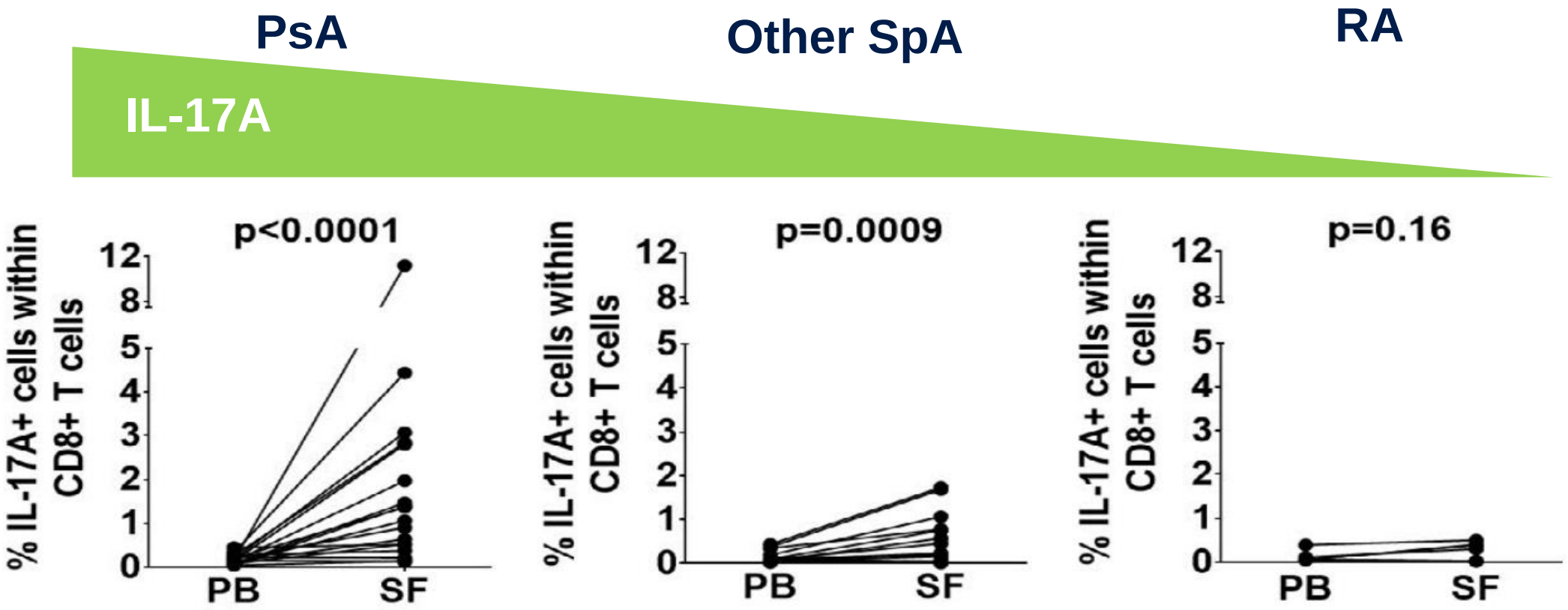


Tildrakizumab[2]
Monoclonal Antibody
IL-23p19 inhibitor

Several IL-17s in development all with different physical chemical properties

SOURCE: (1) Mease et al. Ann Rheum Dis. 2022 Jun;81(6):815-822; (2) Mease et al. Ann Rheum Dis 2021 Sep;80(9):1147-1157; (3) Ritchlin CT, et al. Lancet 2020;395:427–40; (4) Behrens F, Taylor PC, Wetzel D, *et al.* Abstract presented at EULAR 2022. [OP0258]

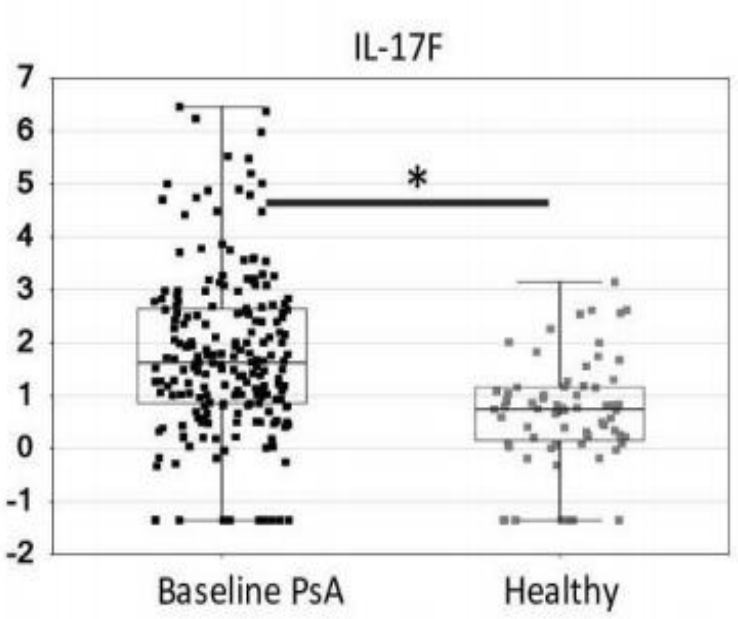
IL-17A+ CD8-T cells are elevated in synovial fluid in PsA



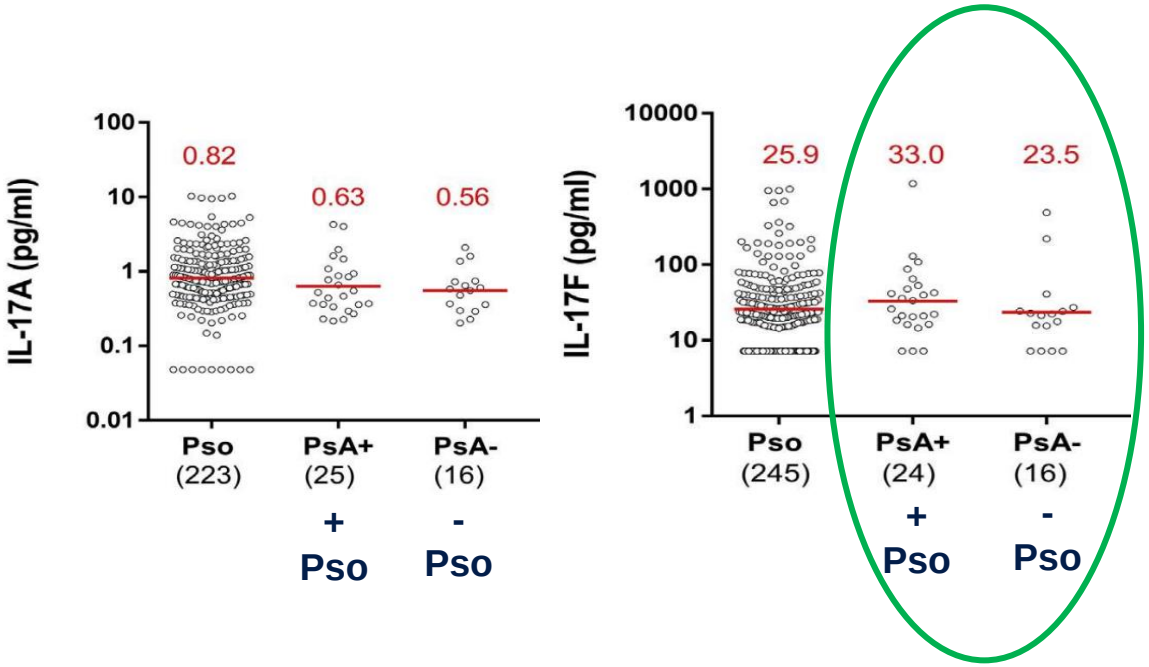
PB; Peripheral Blood: SF; Synovial Fluid
SOURCE: Adapted from Steel et al. Arthritis Rheumatol. 2020; 72:3–435-447

IL-17F is the dominant IL-17 cytokine in PsA with and without Psoriasis

Baseline levels of IL-17F in patients with PsA compared with matched healthy controls from clinical studies[1]



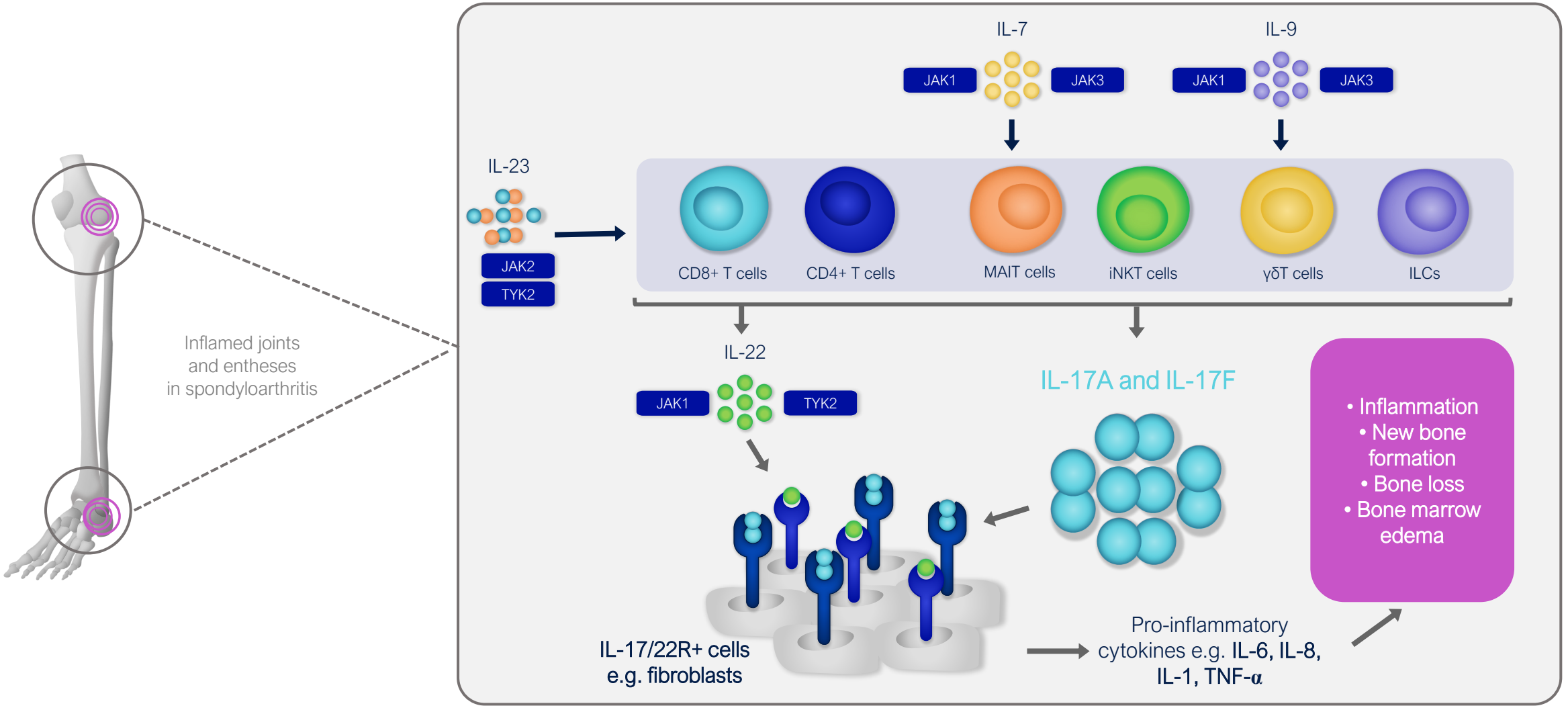
Baseline levels of IL-17A and IL-17F in patients with Psoriasis compared with PsA with and without Psoriasis cross different clinical studies[2]



Pso: Psoriasis

SOURCE: (1) Sweet K, et al. RMD Open. 2021 May;7(2):e001679. doi: 10.1136/rmdopen-2021-001679; (2) Kolbinger F, et al. J Allergy Clin Immunol 2017;139:923–32

IL-17 plays a central role in the pathophysiology of PsA and and can be expressed by multiple cell types independently of IL-23

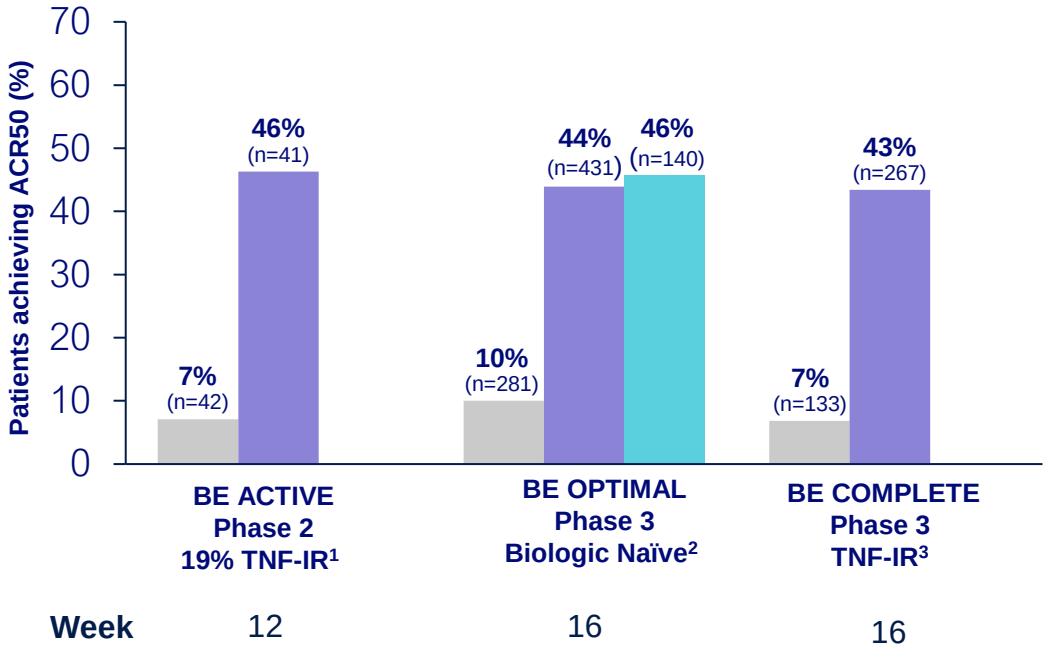


CD, cluster of differentiation; ILC, innate lymphoid cell; iNKT, invariant natural killer T; JAK, janus kinase; MAIT, mucosal-associated invariant T; TYK, tyrosine kinase.
SOURCE: Adapted from O'Brien-Gore, et al. Curr Rheumatol Rep. 2021;23:40.

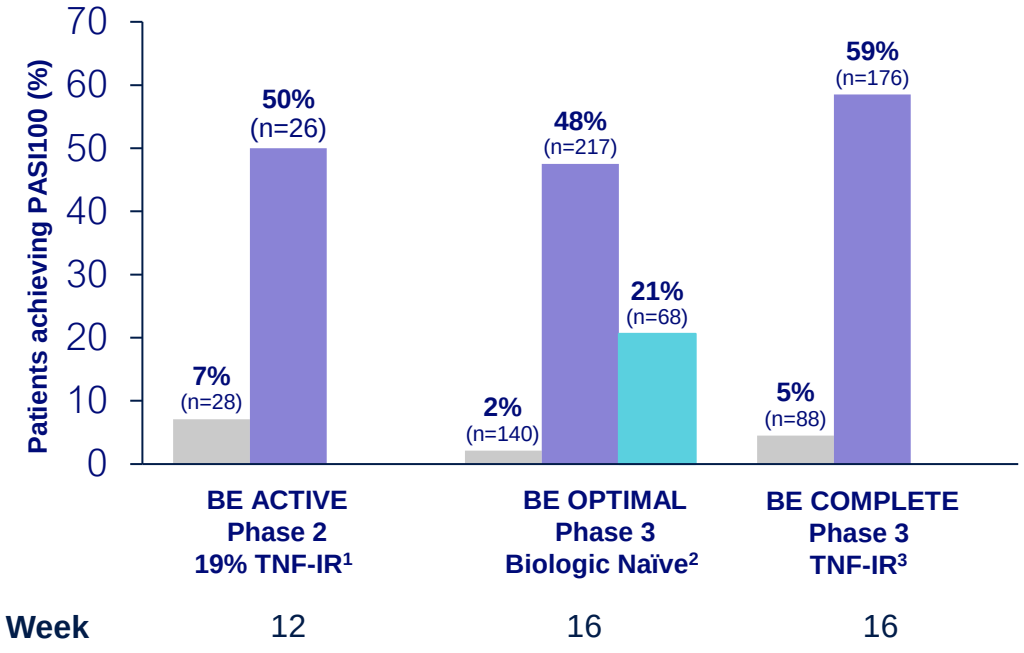
Bimekizumab demonstrates efficacy in PsA with strengths on skin manifestations

Placebo Bimekizumab Adalimumab

Bimekizumab ACR50 response rates by trial, %



Bimekizumab PASI100 response rates by trial, %



Bimekizumab was well tolerated with no un-expected safety findings
Candida Infections 2.6% (Week 16) [2,3]

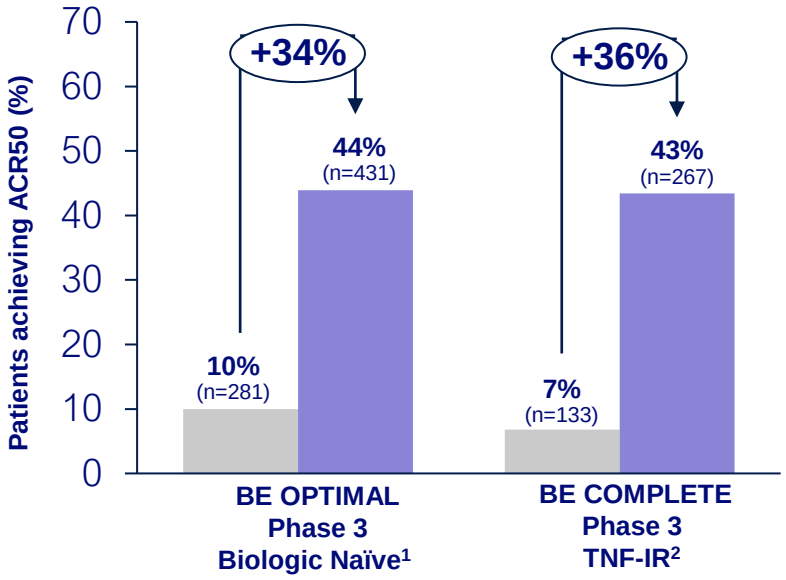
SOURCE: (1) Ritchlin CT, et al. Lancet 2020;395:427–40; (2) McInnes I, Coates L, Landewé RBM, et al. Abstract presented at EULAR 2022. [LB0001]; (3) Merola JF, McInnes I, Ritchlin CT, et al. Abstract presented at EULAR 2022. [OP0255];

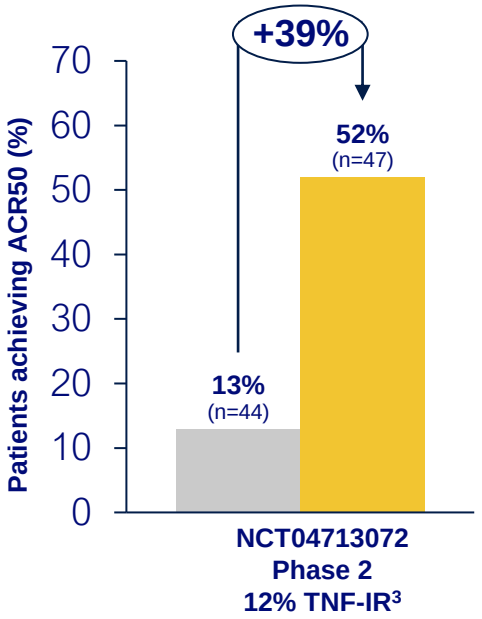
Recent Izokibep data raises questions about the drivers of efficacy in joint disease

Placebo Bimekizumab Izokibep (80mg Q2W)

	Bimekizumab- Phase 3	Izokibep- Phase 2
Product characteristics	▪ **IL-17A and IL-F inhibitor** ▪ Traditional monoclonal antibody (~150kDa)	▪ **IL-17A/A inhibitor** ▪ Antibody Mimetic (~18 kDa) ▪ Albumin binding domain

ACR50 response rate



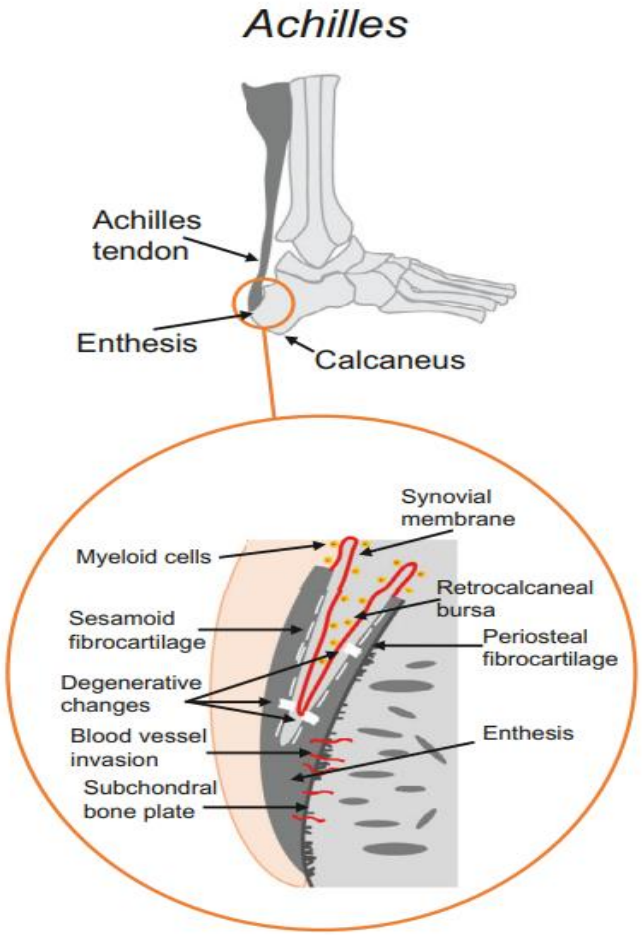


No serious or severe AEs
Injection site reactions (25-30% Izokibep, 0% Placebo)

SOURCE: (1) McInnes I, Coates L, Landewé RBM, et al. Abstract presented at EULAR 2022. [LB0001]; (2) Merola JF, McInnes I, Ritchlin CT, et al. Abstract presented at EULAR 2022. [OP0255]; (3) Behrens F, Taylor PC, Wetzel D, et al. Abstract presented at EULAR 2022. [OP0258]

Enthesitis – A challenging domain

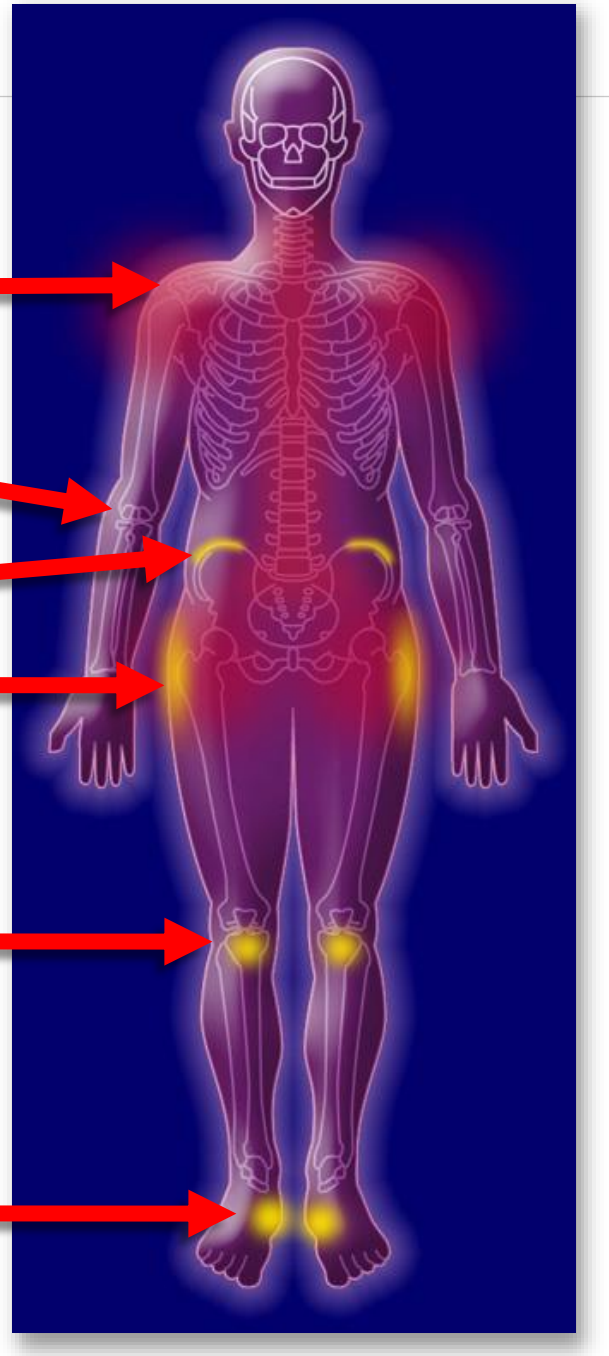
Enthesitis is inflammation of the enthesis, the sites where tendons insert into bone[1]



- Enthesitis is a key feature of PsA, occurring in a third of patients [2]
- Presence of enthesitis has shown to be associated with higher disease activity, disability and incapacity to work, ultimately leading to profound impact on patients lives
- The entheses are avascular in nature[3], difficult to treat and a positive clinical effect on enthesitis and associated pain may serve as a good indicator of drug tissue penetration
- **Resolution of enthesitis is an important treatment goal in PsA**

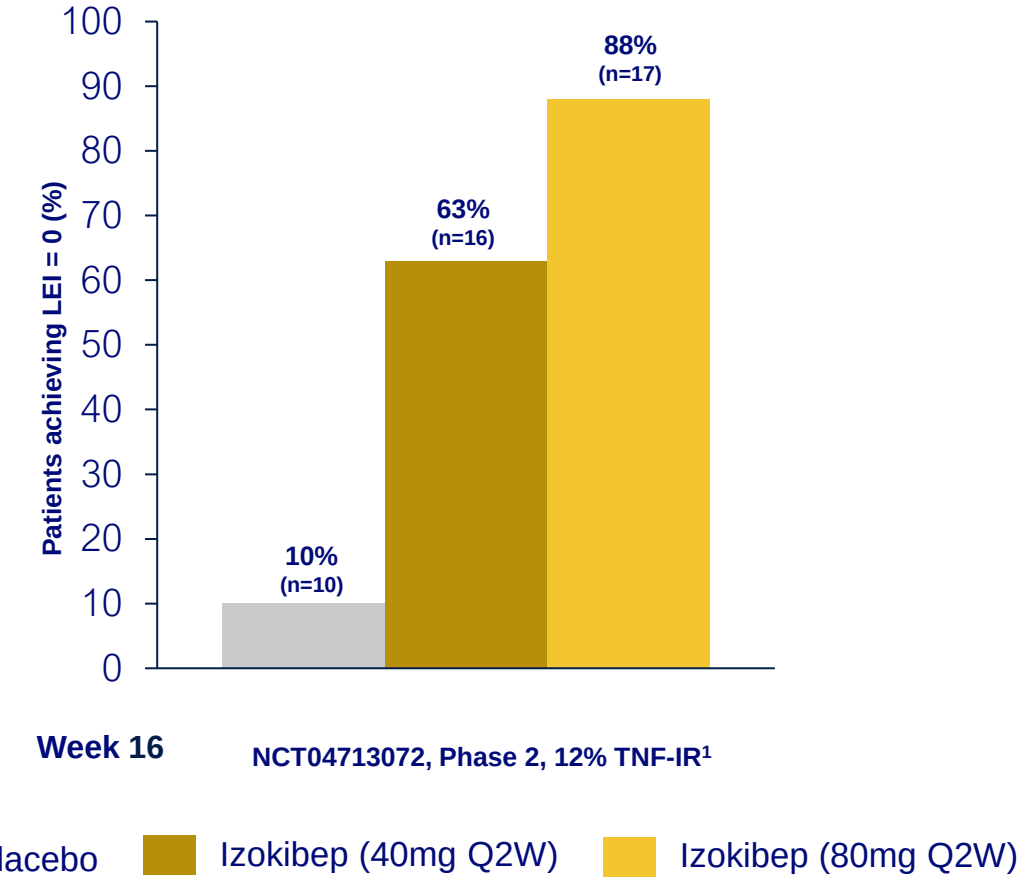
SOURCE: (1) McGonagle et al. / Seminars in Arthritis and Rheumatism 51(2021)11471161; (2) Polachek A, Li S, Chandran V, Gladman DD. Arthritis Care Res (Hoboken) 2017;69:1685–91. (3) McGonagle D. Arthritis Rheum 2007;56(8):2482

Where exactly are the entheses?



Enthesitis – Putting tissue penetration to the test

Izokibep Enthesitis Resolution[1]
Leeds Enthesitis Index =0 (LEI=0)



Data seem to further relevance of tissue penetration in the treatment of enthesitis

Small sub-groups; as observed data*

*FAS, observed data for LEI > 0 at baseline, N = 43 (32%) - Post Hoc Analysis
SOURCE: (1) Behrens F, Taylor PC, Wetzel D, *et al.* Abstract presented at EULAR 2022. [OP0258]

In Summary

1. PsA is a complex heterogenous disease that requires a complete solution

2. IL-17A and IL-17F are central to PsA pathophysiology

3. Several IL-17s in development all with different physical chemical properties

4. Recent data releases at EULAR highlight the importance of IL-17A and IL-17F targeting in PsA

Future Outlook

Very recent data from EULAR with IL-17-inhibitors in development prompt the following questions;

Can we optimize IL-17A and IL-17F inhibition?

Are there certain molecule characteristics e.g size and albumin binding that could make a molecule specifically successful in PsA?

Clinical Development Update

MoonLake

The main messages on Clinical Development

1. The KOL views clearly point to key role for IL-17A & F inhibition, as well as the need for tissue-penetrant and targeted high-affinity molecules

2. A unique role for IL-17F in skin and joint inflammation, that can now be managed for long-term disease control with SLK

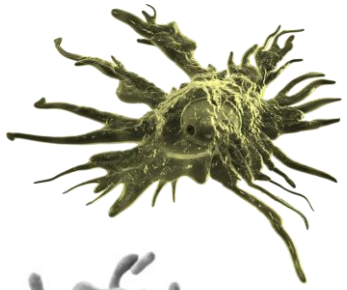
3. Continued focus on skin and joint inflammation with MLTX's HS and PsA clinical trials

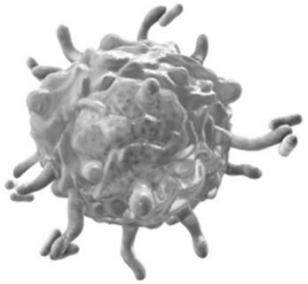
4. Innovative 24-week phase 2 programs with read-outs expected in Q3 and Q4/2023

SOURCE: MoonLake Clinical Development

IL-17F is produced by different cells – need for specific IL-17F inhibition

Plasmacytoid dendritic cells



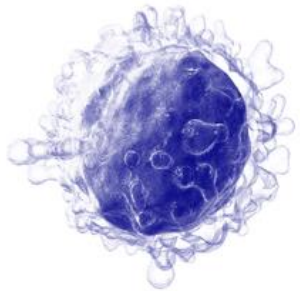


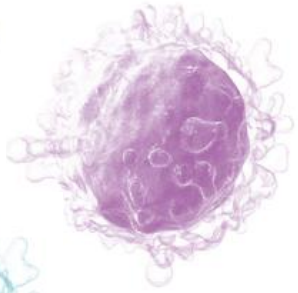
Monocytes

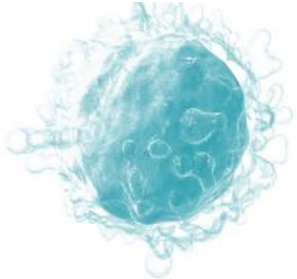
IFNα

IL-12
IL-18
TNFα

αβ **MAIT cells**[1,2,3]
CD8a^{+} or DN,
CD45RA^{-}CD45RO^{+} CD95^{Hi}CD62L^{Lo}CD44^{Hi}RORγt







γδ T cells

Group 3 ILC3[4]
(RoRγt *and Tbet*-; CD4+)

IL-17F

IL-17F is produced by **different tissue-resident innate-like** lymphoid cells

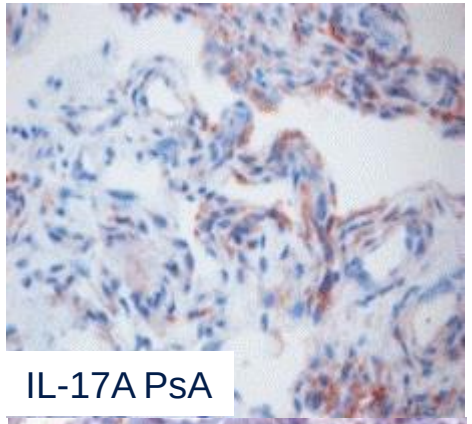
IL-17F- producing cells are **not all controlled by IL-23**, as shown for both MAIT cells and γδ T cells[5] – **IL-17F needs to inhibited specifically**

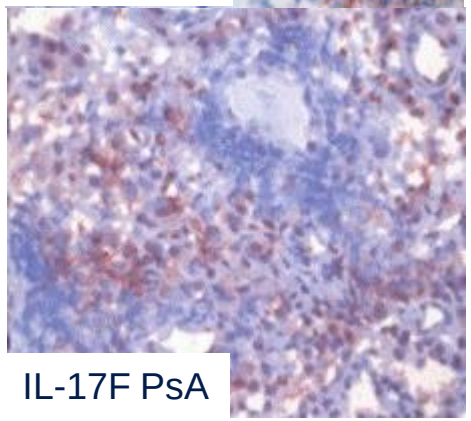
[1]Hinks TSC, Zhang XW. Front Immunol 2020;11:1014 [2]Provine NM, et al. Science. 2021;371:521–26 [3]Cole S, et al. Front Immunol 2020;11:585134 [4]Domingues RG, Hepworth MR. Front Immunol 2020;11:116 [5]Cole S, et al. Front Immunol 2020;11:585134

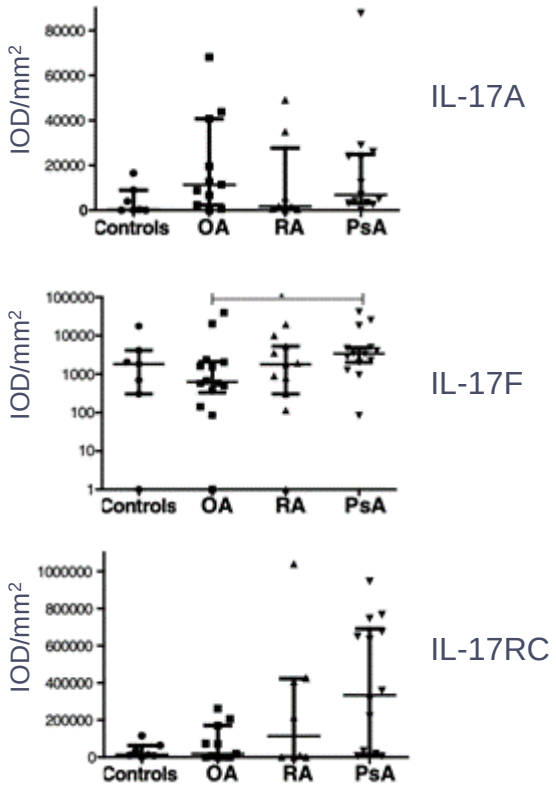
SOURCE: Peer-reviewed publications, MoonLake Clinical Development

As in HS, IL-17F a dominant IL-17 cytokine in lesional PsA

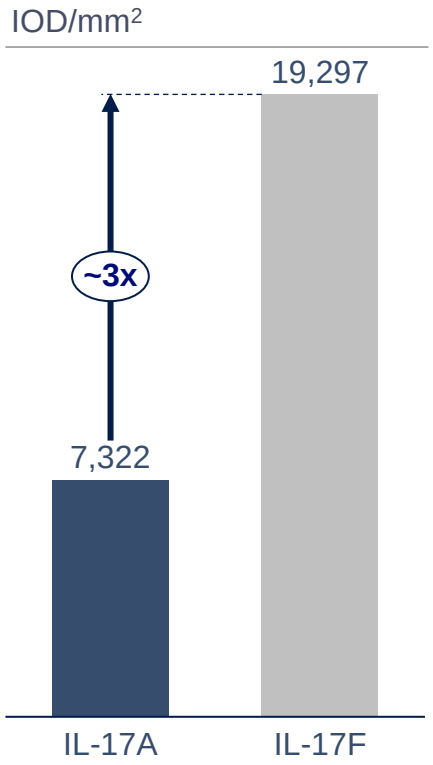
Accumulation of IL-17F in synovial tissue in PsA patients[1,2]







**P < 0.05. IOD/mm² – Integrated optical density of immunohistochemistry signal per area of tissue[1]*



IOD/mm² – Integrated optical density of immunohistochemistry signal per area of tissue[2]

Notes

- Same proportional difference of IL-17F versus IL-17A in patients treated with adalimumab[2]
- IL-17F also significantly elevated vs IL-17A in serum in PsA patients[3]
- IL-17F serum levels also more elevated in patients with concomitant inflammation in skin and joint vs joint alone[4]
- Meta-analysis of genetic studies of IL-17 pathway shows exclusive association of *IL17F* variations with disease risk[5]

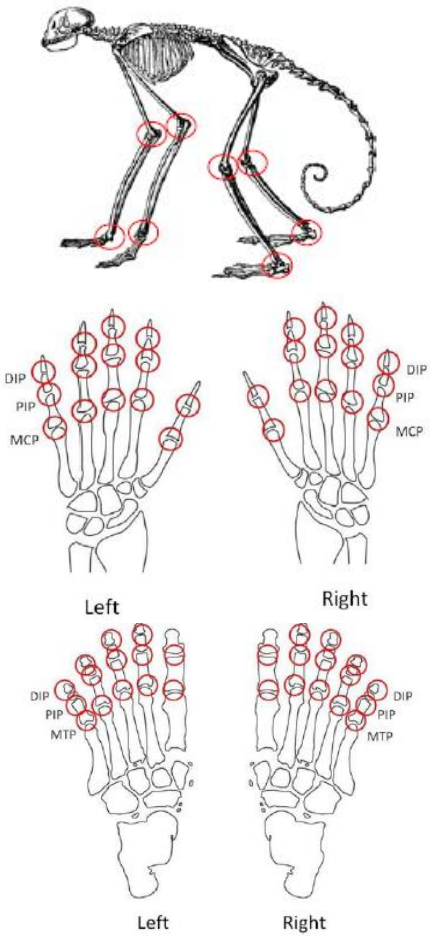
[1]van Baarsen LG, et al. Arthritis Res Ther. 2014 Aug 22;16(4):426. doi: 10.1186/s13075-014-0426-z [2]Bolt JW, et al. Biomedicines. 2022 Jan 29;10(2):324. doi: 10.3390/biomedicines10020324 [3]Sweet K, et al. RMD Open. 2021 May;7(2):e001679. doi: 10.1136/rmdopen-2021-001679 [4]Kolbinger F, et al. J Allergy Clin Immunol 2017;139:923–32 [5]Villalpando-Vargas FV, et al.Inflamm Res. 2021 Dec;70(10-12):1201-1210

SOURCE: Peer-reviewed publications, MoonLake Clinical Development

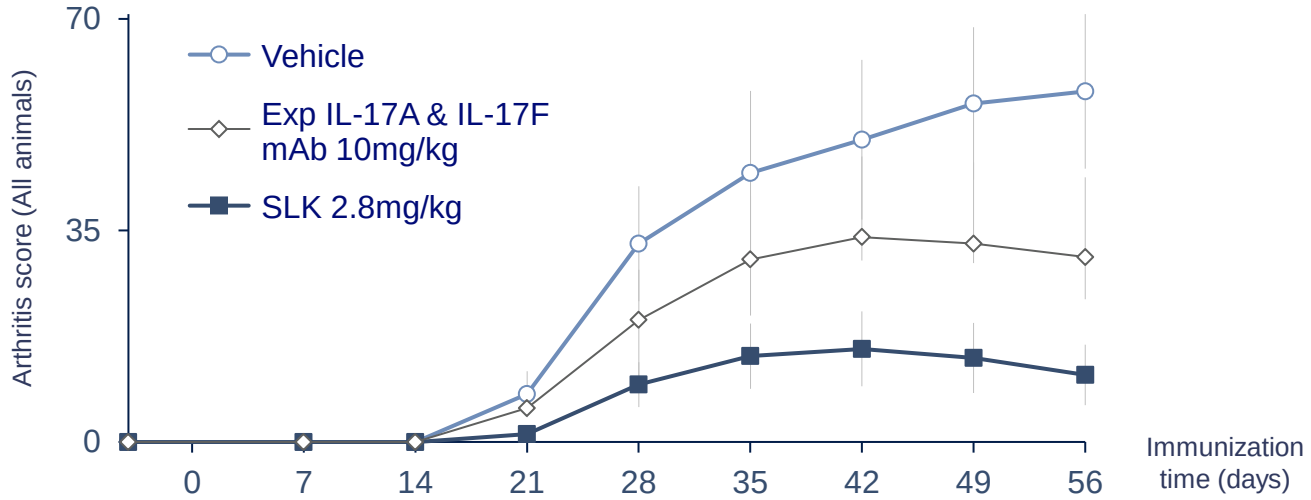
Arthritic joint assessment suggests SLK efficacy in deep tissue

Arthritic joint assessment for SLK[1]

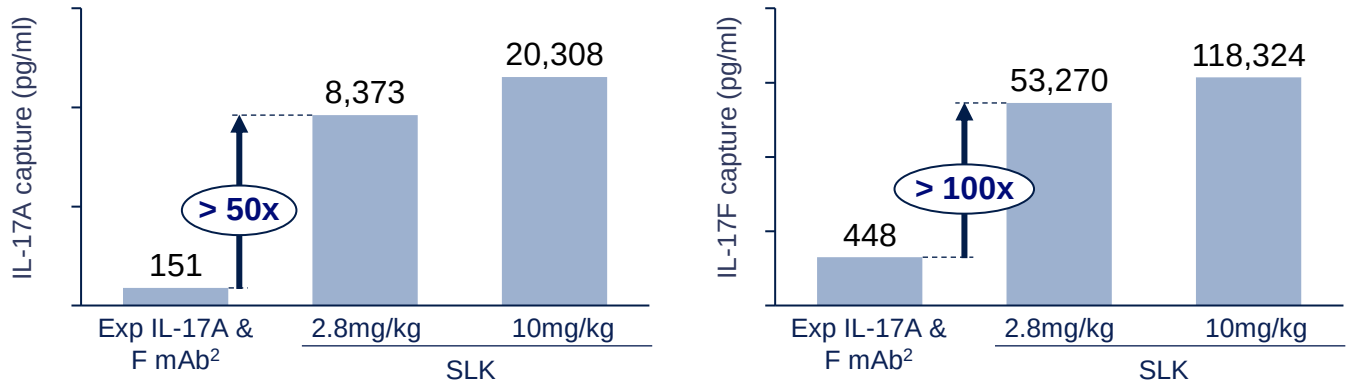
Collagen-induced arthritis



Clinical improvement with SLK versus mAb[2]



Synovial fluid binding Analysis (D56)



1. Assessed 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 Exp IL-17A & IL-17F mAb (Novimmune)

SOURCE: MoonLake team, Modified from SBL271-002 (n=46)

Science into care: Role for SLK in long-term disease control

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

RESEARCH LETTER

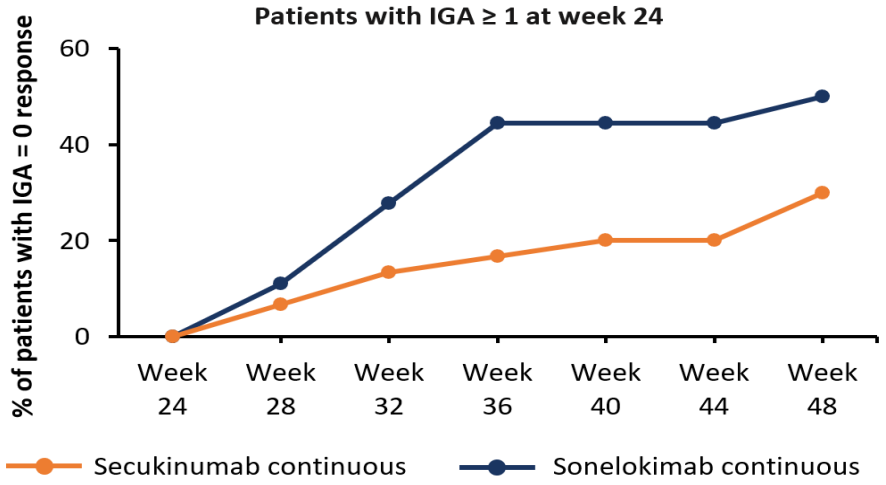
Maintenance of response in moderate-to-severe psoriasis after withdrawal of the IL-17A and IL-17F nanobody sonelokimab – Is there a role for IL-17F in disease reoccurrence?

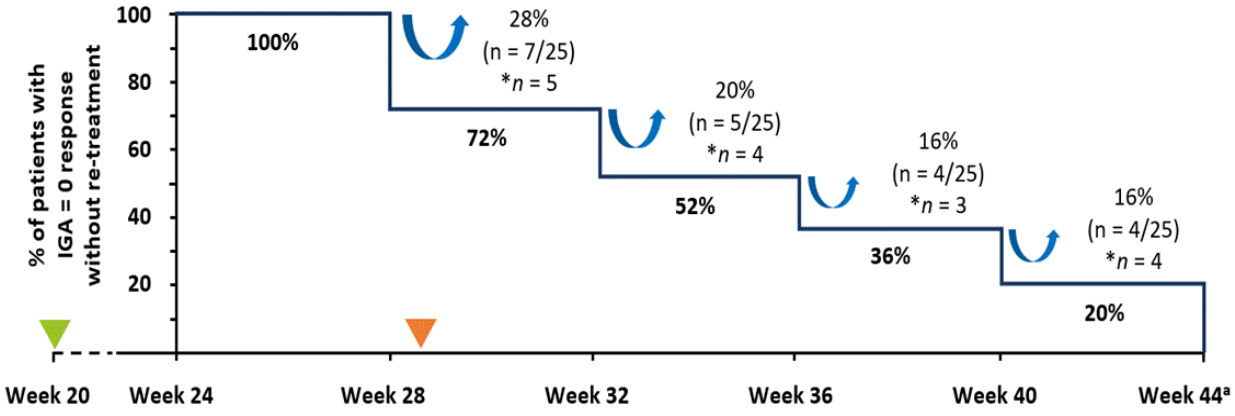
Kristian Reich, Eva Cullen, Mark Weinberg

First published: 20 April 2022 | https://doi.org/10.1111/bjd.21617

Main findings

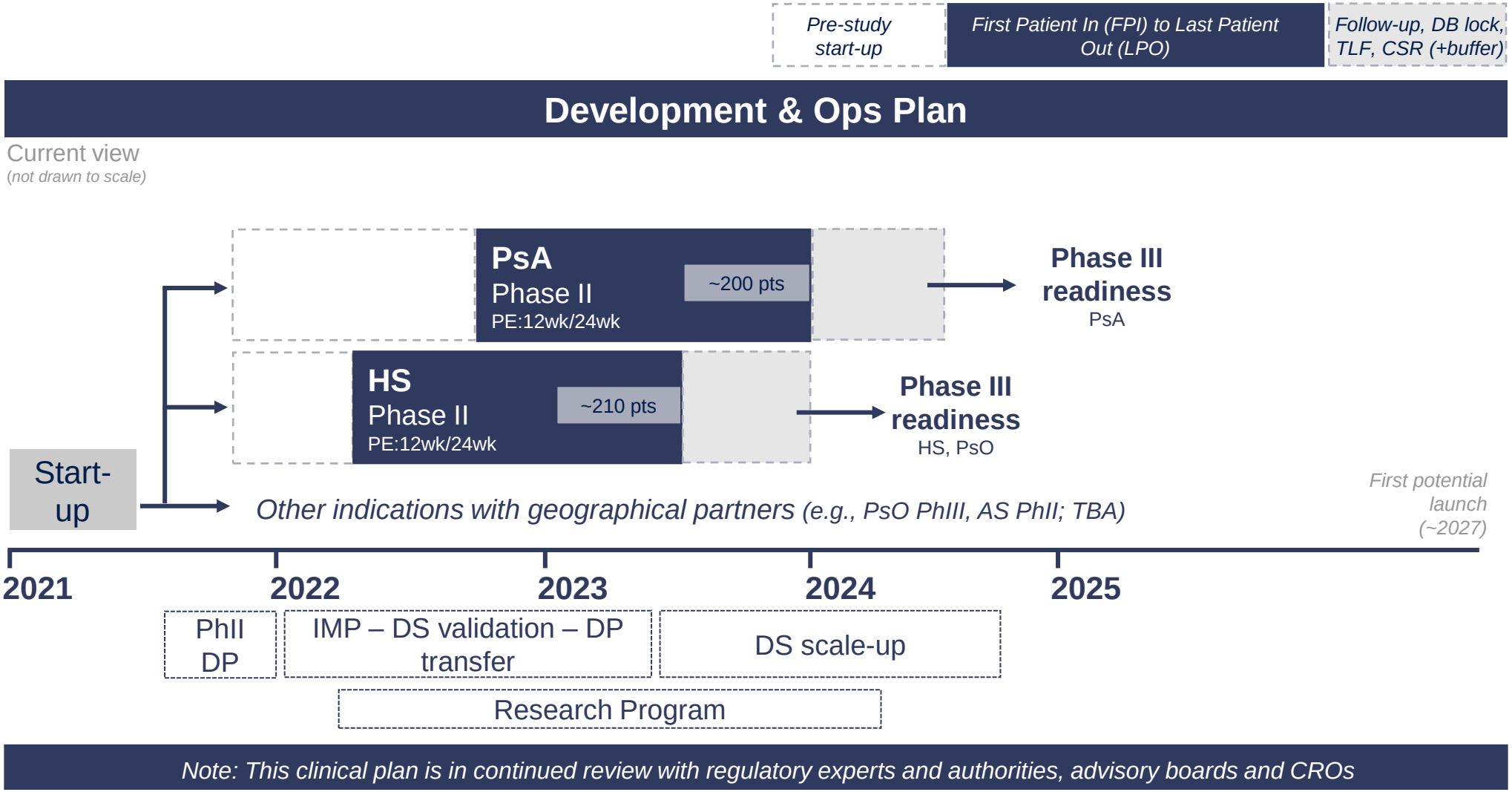
- Disease modification: 20% of responders at week 24 do not require re-treatment to maintain full clearance at week 44, retreatment rapidly re-establishes clearance in 80% patients with disease re-occurrence
- Nanobody® allows patients that do not reach skin clearance at 24 weeks to progress to clearance at 6 months in 50% of cases
- SLK withdrawal/retreatment group received 50% less total monthly injections (wk 24-48) than group receiving secukinumab to reach same level of clearance





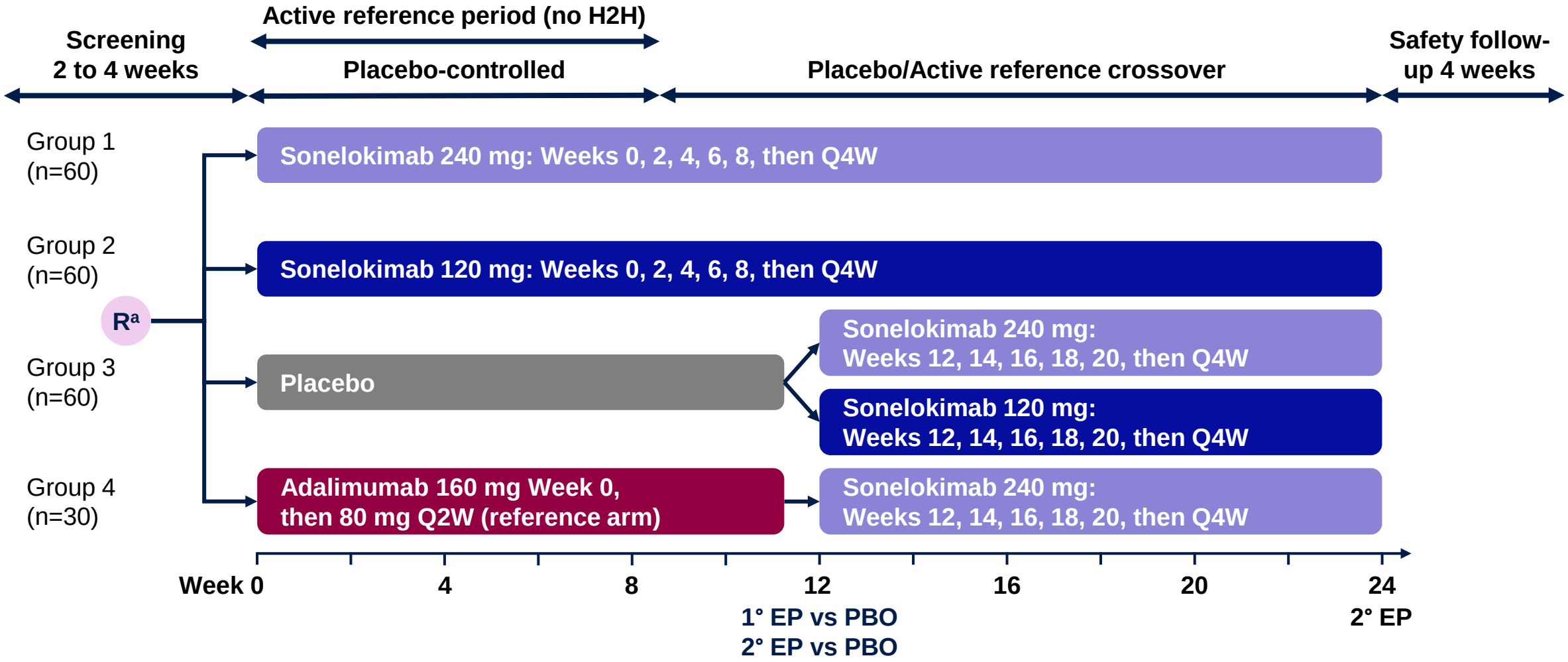
SOURCE: MoonLake, BJD

Recap: We are driving two global Phase 2 trials in our program



SOURCE: MoonLake

HS: Phase II, randomized, double-blind, placebo-controlled, 24-week study of sonelokimab in patients with active moderate to severe HS



[a]Randomization stratified by Hurley stage status (I/II and /III) and prior biologic use (Y/N). Patients in Hurley stage III limited to ~40%

SOURCE: MoonLake Clinical Development

HS: Primary and key secondary endpoints

Primary endpoint

- HiSCR75[a] response at Week 12

Key secondary endpoints

- HiSCR50 response at Week 12
- % Change from baseline in IHS4
- DLQI total score of 5 or below at Week 12
- Patients achieving NRS30[b] in Patient's Global Assessment of Skin Pain at Week 12

[a]HiSCR75: Clinical response per Hidradenitis Suppurativa Clinical Response (HiSCR) criteria, ie, ≥75% reduction from baseline in total abscess and inflammatory nodule (AN) count, with no increase from baseline in abscess or draining fistula count
[b]NRS30: ≥30% reduction and at least 1 unit reduction from baseline in numerical rating scale (NRS), among patients with baseline NRS ≥3
DLQI, Dermatology Life Quality Index: IHS4, International Hidradenitis Suppurativa Severity Score System

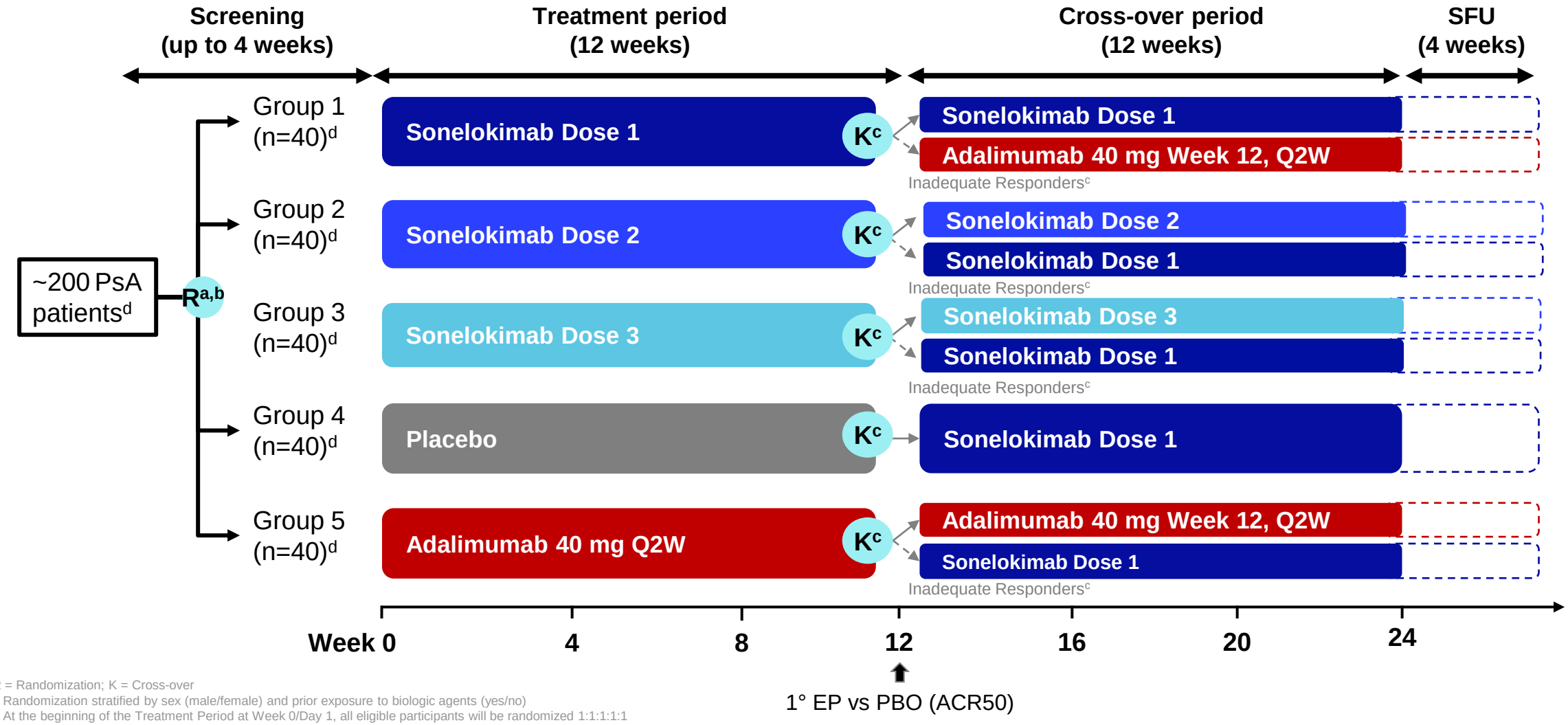
SOURCE: MoonLake Clinical Development

Status of Trial Operations as of June 4th 2022 – All activities on track

HS	Regulatory - FDA	• Approved
	IRB - US	• Central IRB Protocol Approval received. Site approvals in progress
	Regulatory/CEC/CIRB - ROW	• All submissions performed • RA - CA/NL/POL/BL approvals received • CEC/CIRB – CA/POL/BL approvals received
	Site Activation	• 58/60 selected + 6 back ups • 8 SIVs complete (7 scheduled in June) • 8 sites activated (7 US, 1 Canada), 4 recruiting
	Patient Recruitment	• 11 patients screened • 3 patients enrolled/dosed • 4 patients screen failed
PSA	Regulatory - FDA	• New design finalized & re-costed with CRO
	Regulatory - ROW	• Submissions on hold pending finalisation of updated protocol
	Site Activation	• 34/61 selected (short feasibility to be performed covering new study design)
	Patient Recruitment	• First patient screened projected for 30 Sep 2022

SOURCE: CRO, MoonLake Clinical Development

PsA: Final design of proposed Phase II PsA study (24 weeks)



R = Randomization; K = Cross-over
a Randomization stratified by sex (male/female) and prior exposure to biologic agents (yes/no)
b At the beginning of the Treatment Period at Week 0/Day 1, all eligible participants will be randomized 1:1:1:1:1
c In the cross-over period, starting at Week 12, participants on sonelokimab 120 mg that 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) that have not achieved an adequate response will receive sonelokimab 120 mg every 4 weeks until week 24; participants on adalimumab that 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 at least 20%. Patients on placebo will receive sonelokimab Q4W until Week 24.
d Final number of participants is subject to results of additional statistical power calculations.

SOURCE: MoonLake Clinical Development

PsA: Primary and Key Secondary Endpoints

MoonLake

Primary endpoint

- ACR50 response[a] at Week 12

Key secondary endpoints

- PASI100 response at Week 12 (patients with psoriasis involving ≥3% BSA at baseline)
- ACR20 response at Week 12

Other secondary endpoints

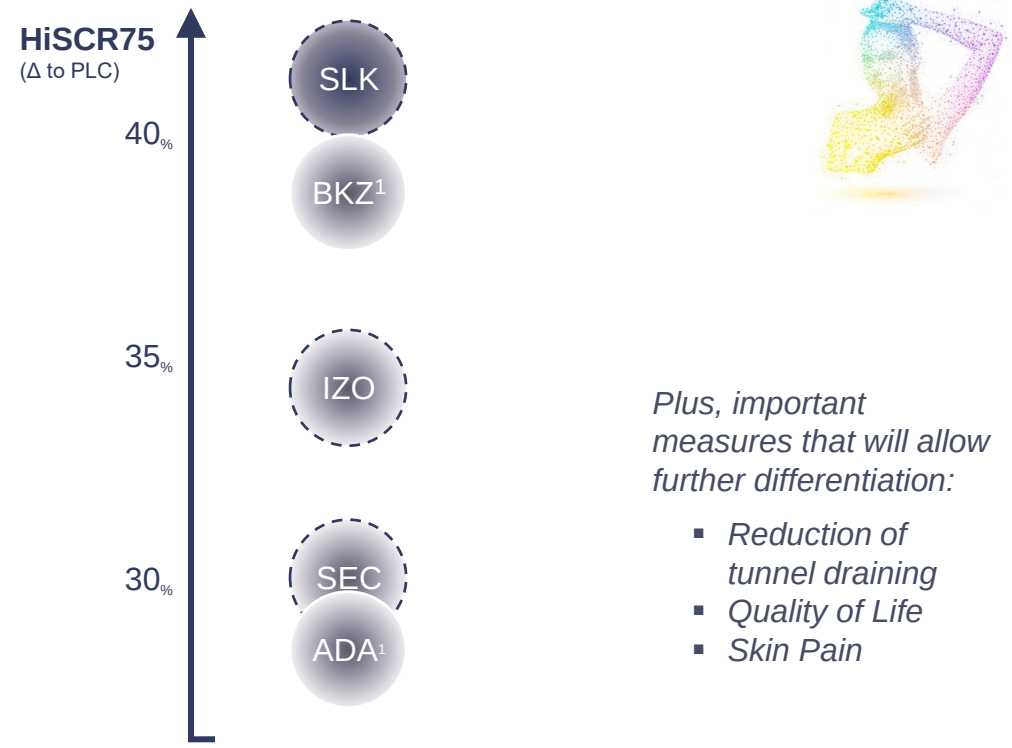
- ACR70 response at Week 12
- Minimal disease activity (MDA) at Week 12, defined as meeting 5/7 of the following:
  - ≤1 tender joint
  - ≤1 swollen joint
  - PASI score ≤1 or psoriasis affecting ≤1% BSA
  - Pain score ≤15 (0–100 VAS)
  - Patient global activity score ≤20 (0–100 VAS)
  - HAQ-DI score ≤0.5
  - ≤1 tender entheseal point
- Health Assessment Questionnaire Disability Index (HAQ-DI) at Week 12

aACR50: □50% improvement in tender joint count (68 joints) and swollen joint count (66 joints), and □50% improvement in 3 of the following 5 measures: Patient's Global Assessment of Disease Activity (PtGADA), Physician's Global Assessment of Disease Activity (PhGADA), Patient's Assessment of Arthritis Pain (PtAAP), Health Assessment Questionnaire-Disability Index (HAQ-DI), high-sensitivity C-reactive protein (hs-CRP)
ACR, American College of Rheumatology; BSA, body surface area; PASI, Psoriasis Area and Severity Index; VAS, visual analogue scale
SOURCE: MoonLake Clinical Development

The ambition for SLK is to win across different therapeutic scores

Hidradenitis Suppurativa (HS)

Conceptual



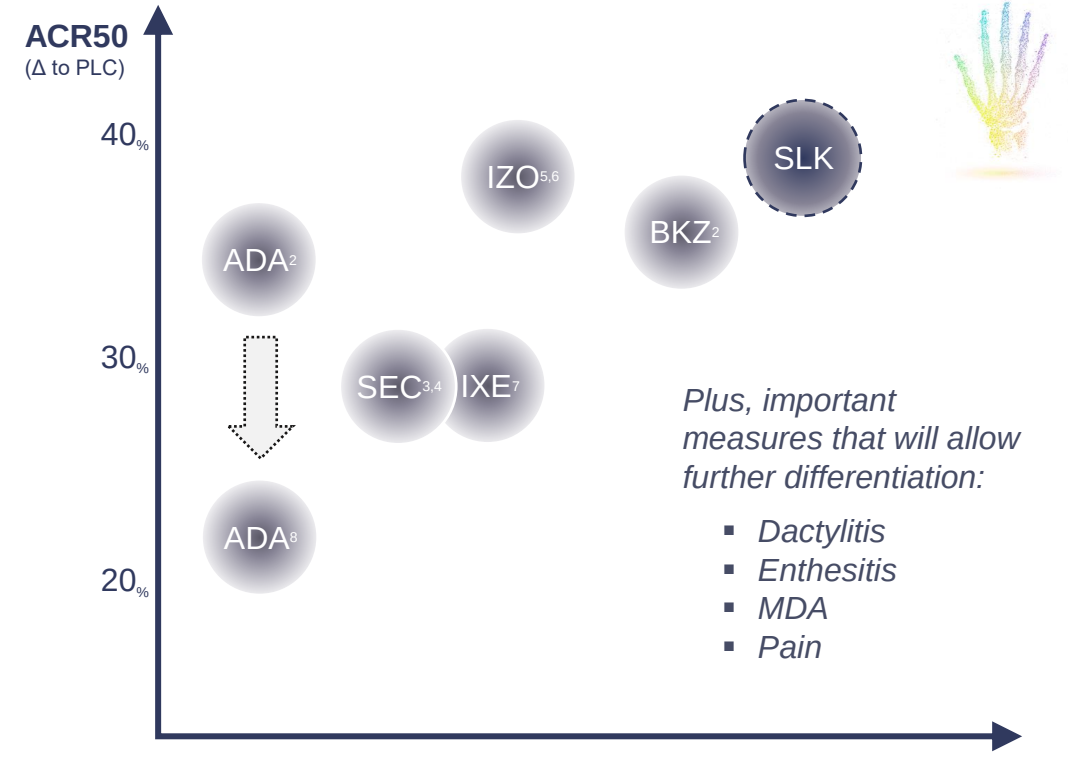
Plus, important measures that will allow further differentiation:

- *Reduction of tunnel draining*
- *Quality of Life*
- *Skin Pain*

"Straight shootout" to win in HiSCR75

Psoriatic Arthritis (PsA)

Conceptual



Plus, important measures that will allow further differentiation:

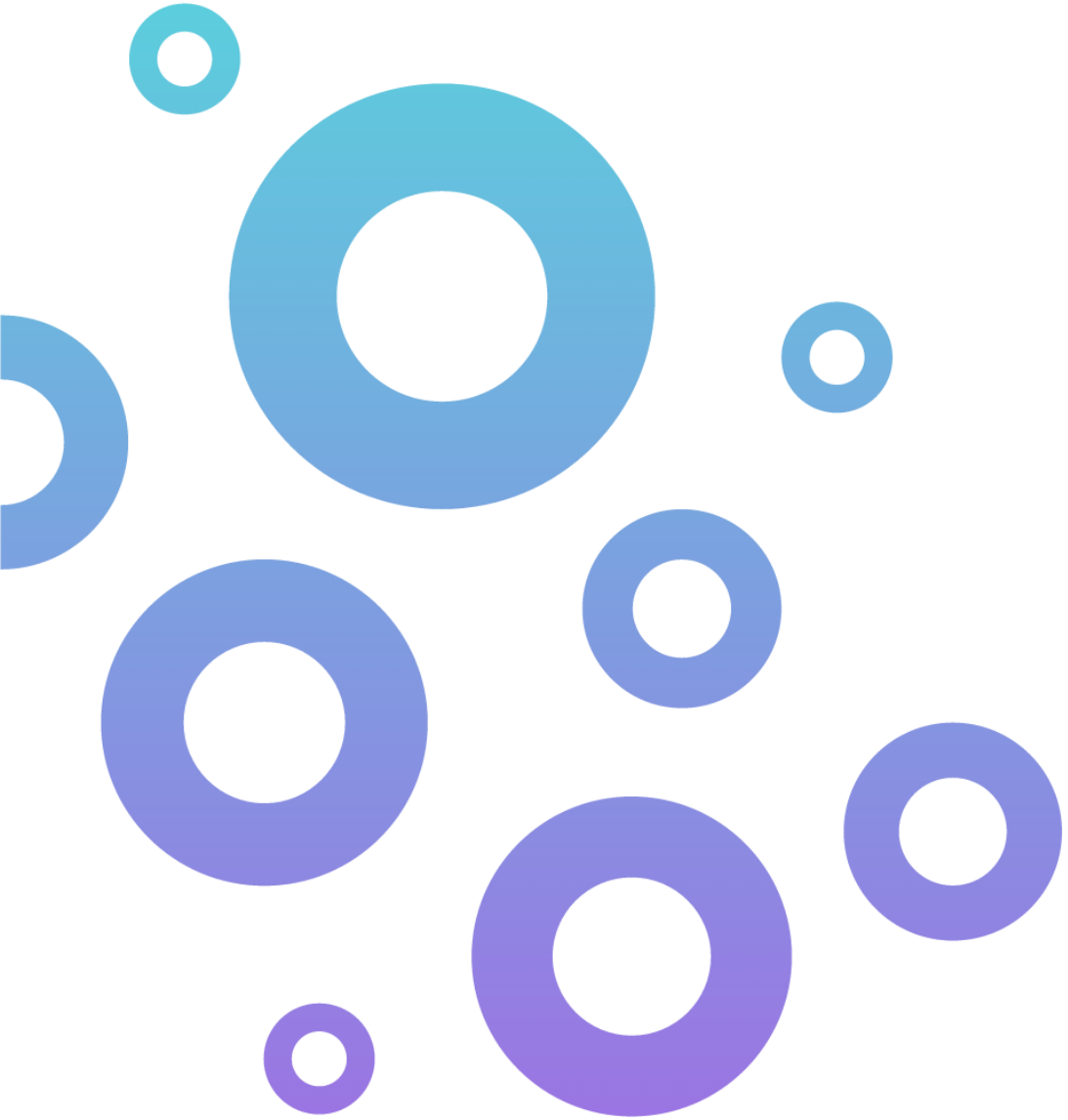
- *Dactylitis*
- *Enthesitis*
- *MDA*
- *Pain*

Dashed circles represent estimates

Win across different skin & joint scores

1. Glatt S, et al. JAMA Dermatol. 2021 Nov 1;157(11):1279-1288; phase 2 POC study of BKZ in HS with ADA as active reference, week 12; 2. McInnes I et al. EULAR abstract LB0001; June 2022; BKZ and ADA skin and joint data is from a phase 3 PsA study with ADA as active reference, bio-naïve patients, week 16; 3. McInnes I et al. FUTURE 2, Lancet.; 2015 September; doi: 10.1016/S0140-6736(15)61134-5; 4. Langley G. et al. ERASURE, N Engl J Med; 2014 July; doi: 10.1056/NEJMoa1314258; secukinumab joint and skin data come from different studies; ACR50 is from phase 3 PsA, 50 mg, bio-naïve and bio-experienced patients, week 24; PASI100 is from phase 3 psoriasis, 300 mg, week 12; 5. Behrens F et al., EULAR abstract OP0258; 2022 May; 6. Gerdes S et al., EADV abstract 364; September 2021; izokibep joint data is from a phase 2a study in PsA, 80 mg Q2W, bio-naïve and bio-experienced patients, week 16; skin data is from phase 2 study in PsO, 80 mg Q2W, week 12; 7. Mease P et al. SPIRIT-P1; Ann Rheum Dis.; 2017 Jan; doi: 10.1136/annrheumdis-2016-209709; phase 3 PsA study, IXE 40 mg Q4W, bio-naïve patients, week 12; 8. McInnes IB, et al. N Engl J Med. 2021 Apr 1;384(13):1227-1239
SOURCE: MoonLake Clinical Development

In summary



- Evidence for a specific role of IL-17F in a growing number of diseases including HS, PsA and PsO
- Full anti-inflammatory potential and long-term disease control requires inhibition of IL-17A/A, A/F, and F/F
- Optimal delivery of MoA requires unique characteristics such as enhanced penetration and albumin-binding
- Focus on clinical development in HS and PsA as two model diseases for MoA and molecule features of SLK
- 24-week programs with next-level treatment goals and active reference arms, plus placebo
- Creating solid basis for Phase 3 readiness in 3 key indications for SLK

SOURCE: MoonLake Clinical Development

Financial Overview & Guidance

MoonLake

Summary of MoonLake's corporate history

MoonLake

2021 **2022**

Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun

March '21
Registration as Swiss stock corporation

April '21
Series A led by BVF Partners L.P.

July '21
Registration of UK subsidiary

May '21
Announcement of License Agreement with Merck KGaA, Darmstadt, Germany

- Global rights
- Royalties in the low teens
- USD 25m upfront + 9.9% ownership

October '21
Definitive agreement with Helix Acquisition Corp. to enter into De-SPAC business combination

Concurrent commitments of leading Biotech investors for a PIPE

April '22
Closing of De-SPAC transaction and listing on Nasdaq under ticker MLTX

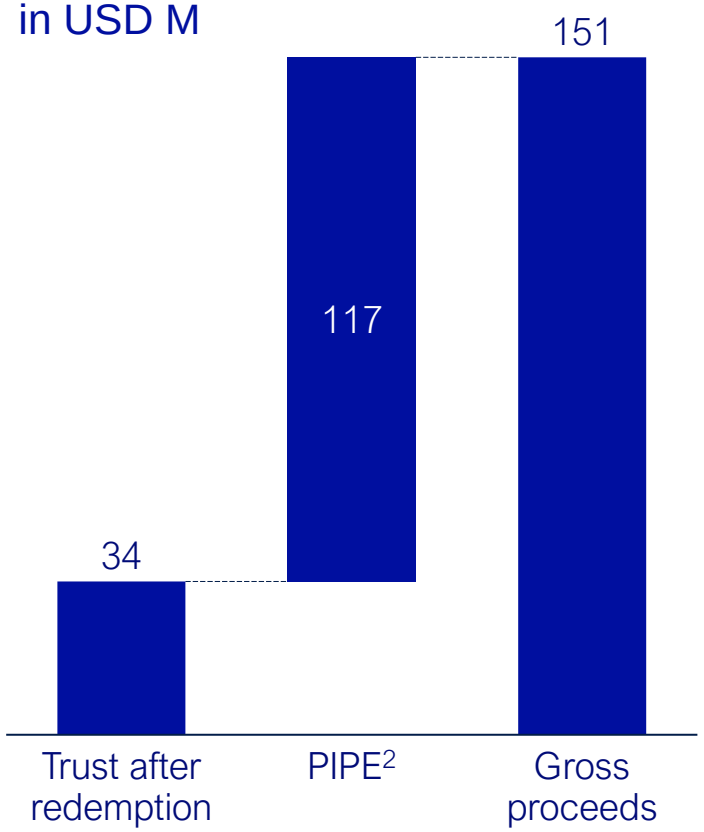
Details on next page

SOURCE: MoonLake Finance

De-SPAC provided >USD 150m in proceeds

De-SPAC proceeds[1]

in USD M



Corporate structure

MLTX
MoonLake Immunotherapeutics (formerly Helix Acquisition Corp)

MoonLake AG
Operating entity incorporated in Switzerland

MoonLake Ltd
100% owned UK subsidiary

Business combination with Helix Acquisition Corp (SPAC sponsored by Cormorant Asset Management)

USD 151m in aggregate deal proceeds[1]

Backed by top Biotech investors across the PIPE and non-redeeming shareholders

Top-10 SPAC deal in Healthcare since 2019 in a tough market environment

52.7m shares (dual class structure only temporary – relevant share count is Class A + Class C combined)

1 Prior to transaction-related expenses
2 Includes Issuance of, in aggregate, 100,000 Class A Ordinary shares to placement agents as share-based payment for PIPE placement services
SOURCE: MoonLake Finance

MoonLake on a solid financial foundation with runway into 2H-2024

Runway well into 2H-2024: cash provides runway to key clinical data readouts plus 12+ months (at zero debt)

Average quarterly cash burn in the "*low-teen millions*"[1] **– next quarters elevated due to one-off expenses:**

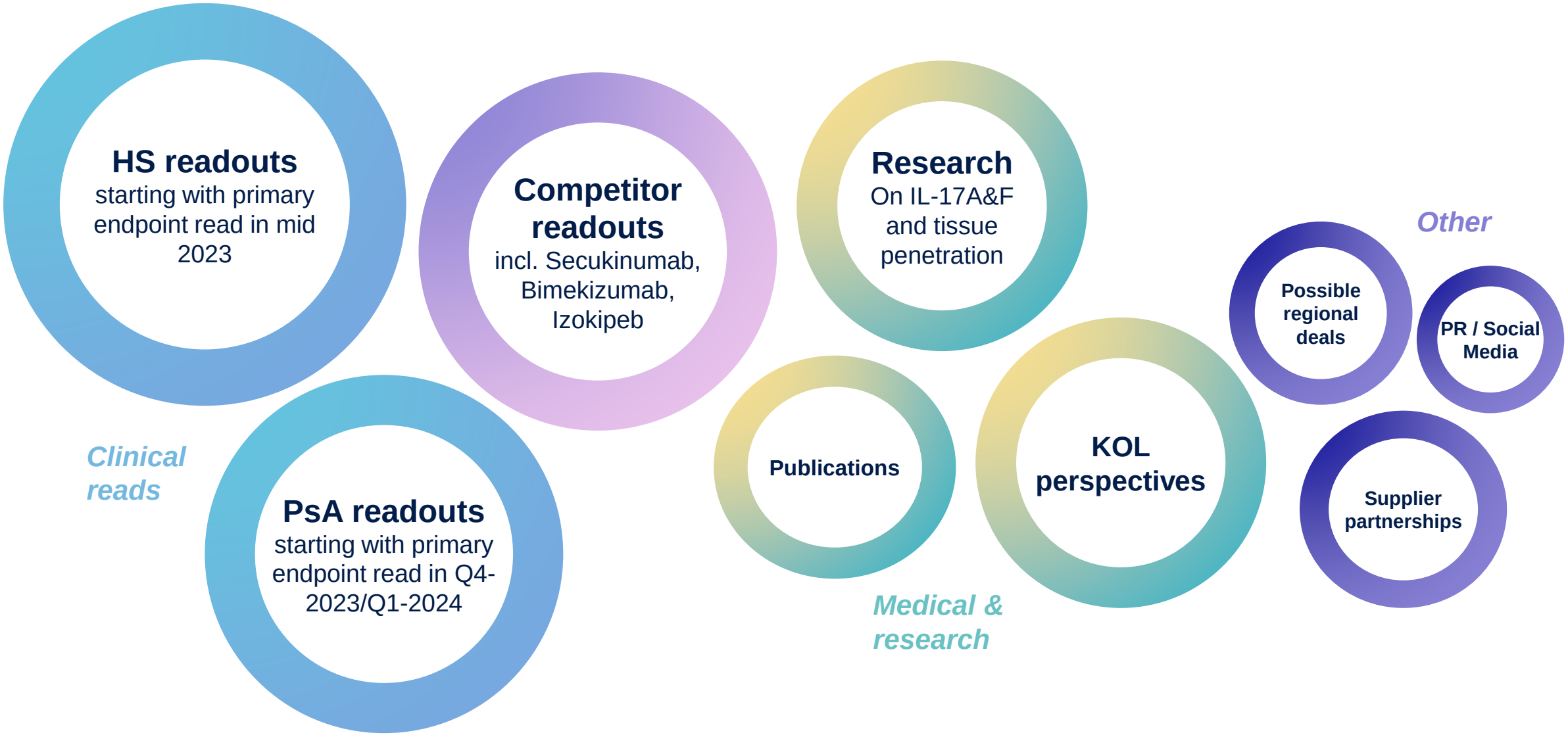
- **Remaining De-SPAC transaction cost**
- **Trial initiations in HS and PsA**
- **Milestone payments:** single-digit USD millions due in licensing milestones for initiation of Phase 2 trials (note: no additional milestones until acceptance of regulatory filing)

Management committed to focus on Sonelokimab development

Non-dilutive cash opportunities so far untapped: regional partnering (Asia), grants, and other non-dilutive opportunities for additional cash exist

1 In USD millions
SOURCE: MoonLake Finance

Active news flow with multiple catalysts (HS read only 12 months out)



SOURCE: MoonLake Finance

Our approach to Investor Relations

Open and transparent communication on company strategy, direction and updates in a **regulation-FD compliant** manner

No plans to change status as **domestic filer** with quarterly financial reporting and other associated filing requirements

At least semi-annual **event-based meetings** with **opportunity for Q&A** (incl. an in-person/virtual capital markets day)

Where possible, **participation of independent experts**

Presence at key **investor and scientific conferences** globally, as relevant

BIO€QUITY EUROPE

svb

LifeSci

UBS

HCW H.C.WAINWRIGHT&CO.

AAD American Academy of Dermatology Association

ACR Convergence Where Rheumatology Meets

eular EUROPEAN ALLIANCE OF ASSOCIATIONS FOR RHEUMATOLOGY

MoonLake

People and resources

Jorge Santos da Silva (CEO)

Kristian Reich (CSO)

Matthias Bodenstedt (CFO)

***Atif Khan (Investor Relations** and Strategy)*

Patricia Marques de Sousa (Communications and Media)

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Dedicated IR website with link to all filings

ir.moonlaketx.com


MoonLake