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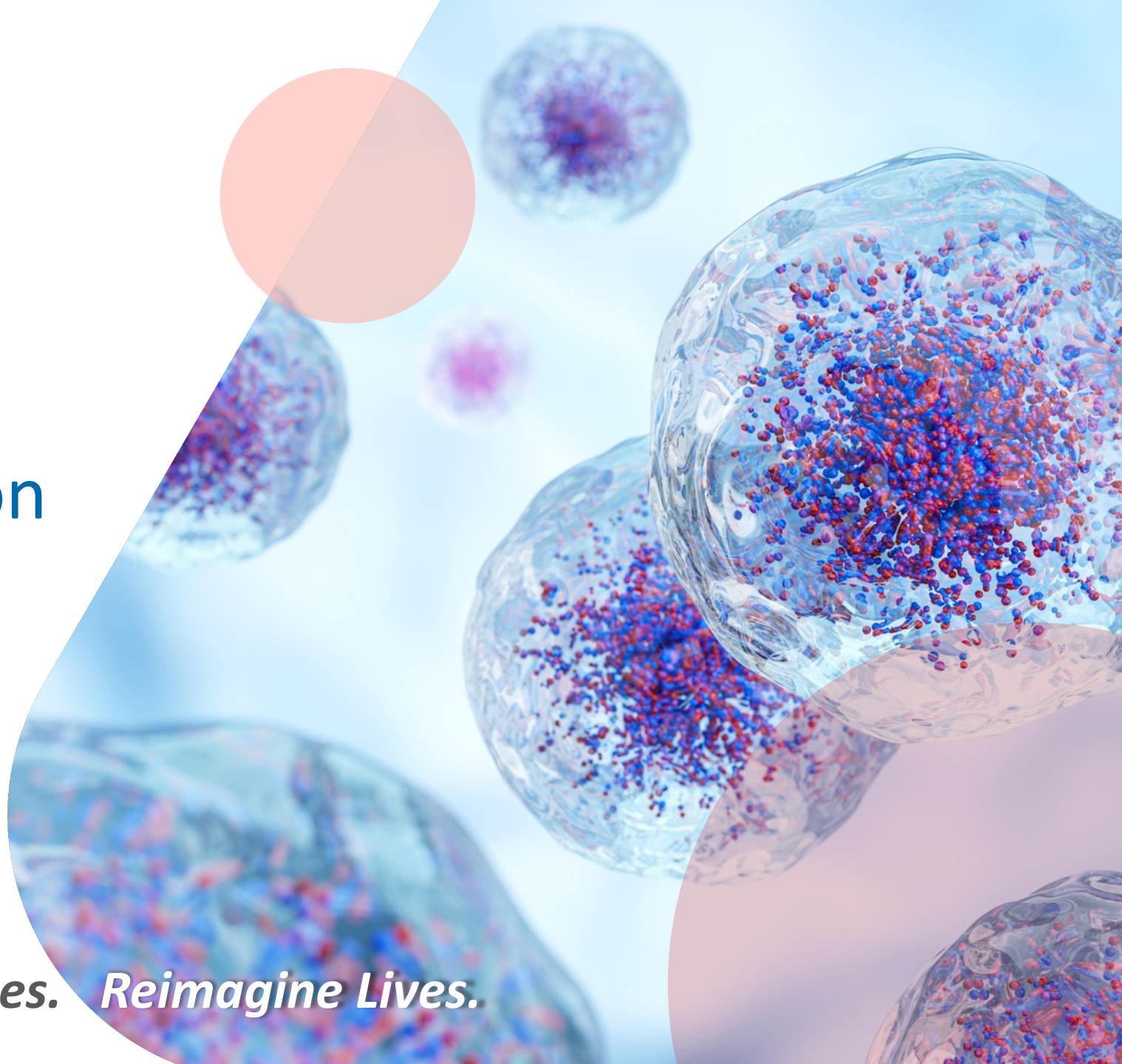
Corporate Presentation

November 2024



Transform Therapies. Reimagine Lives.

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Corporate Overview

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Developing Oral Therapies To Transform Lives of Patients With Immune-Mediated Diseases

OUR ORAL TYK2 PIPELINE

Differentiated by design for maximal target inhibition and opportunity for favorable tolerability profile

ESK-001

Potentially first and only allosteric TYK2 inhibitor well-tolerated at maximal target inhibition with opportunity in multibillion dollar[1] broad set of indications, including PsO (Phase 3 clinical trial) and SLE (Phase 2b clinical trial)

A-005

Potential first- and best-in class opportunity with CNS-penetrant allosteric TYK2 inhibitor for the treatment of neuroinflammatory and neurodegenerative diseases, including MS; in Phase 1 clinical trial

PRECISION APPROACH

Data analytics platform enables targeted therapies to replace broad immuno-suppression

EXPERIENCED LEADERSHIP

Experienced team with strong track record in value creation and strong financial position to execute on key milestones

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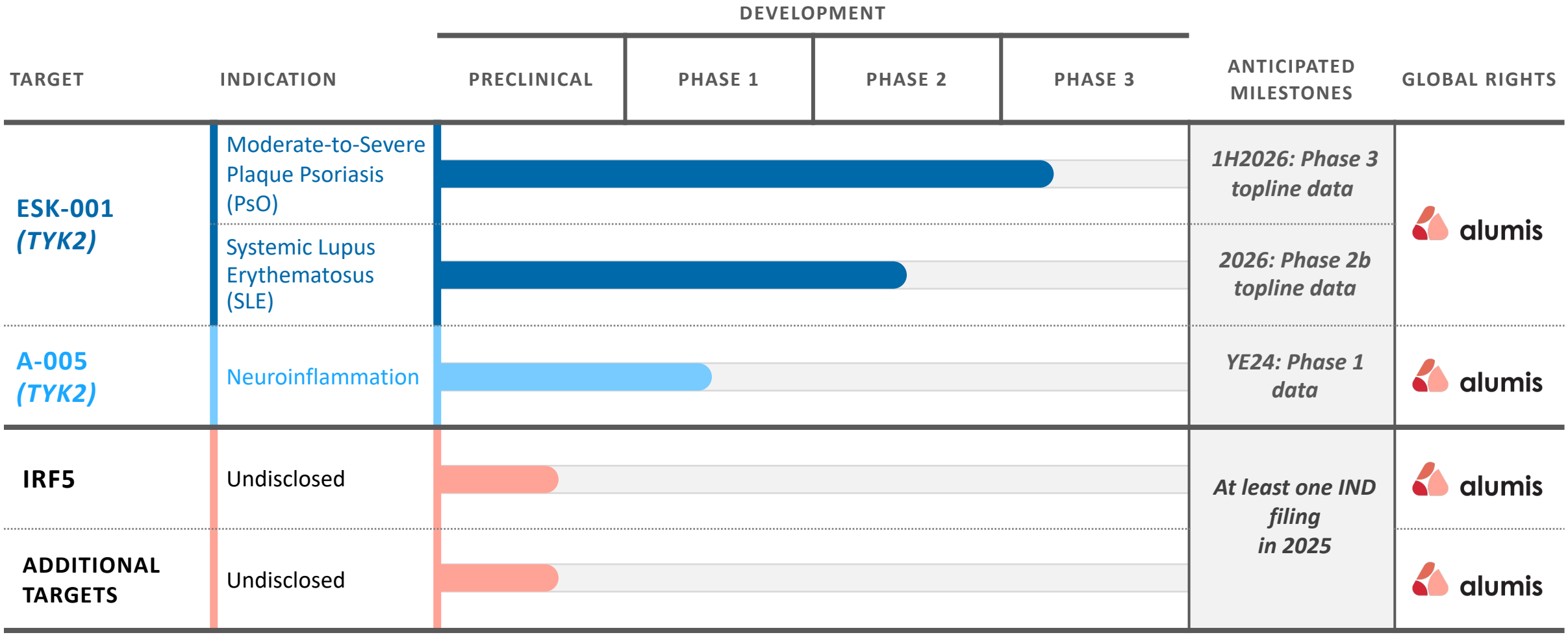
1. Based on current internal estimates

Late-Stage Pipeline with Multiple Near-Term Catalysts



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ESK-001: Our Allosteric TYK2 Inhibitor

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Significant Unmet Need in Psoriasis for High Efficacy Oral

PLAQUE PSORIASIS

~16M

PATIENTS WORLDWIDE[1]

$25B+

GLOBAL MARKET[2]

- More than 7.7M people have plaque psoriasis in the United States, 1.5M with moderate-to-severe disease[1]
- Treatment dominated by injectable biologics and sub-effective orals
  - Less than 10% of diagnosed patients receive an injectable biologic
- Introductions of high efficacy orals are poised to drive market growth

ESK-001 is a next-generation TYK2 with differentiated profile for oral treatment in psoriasis

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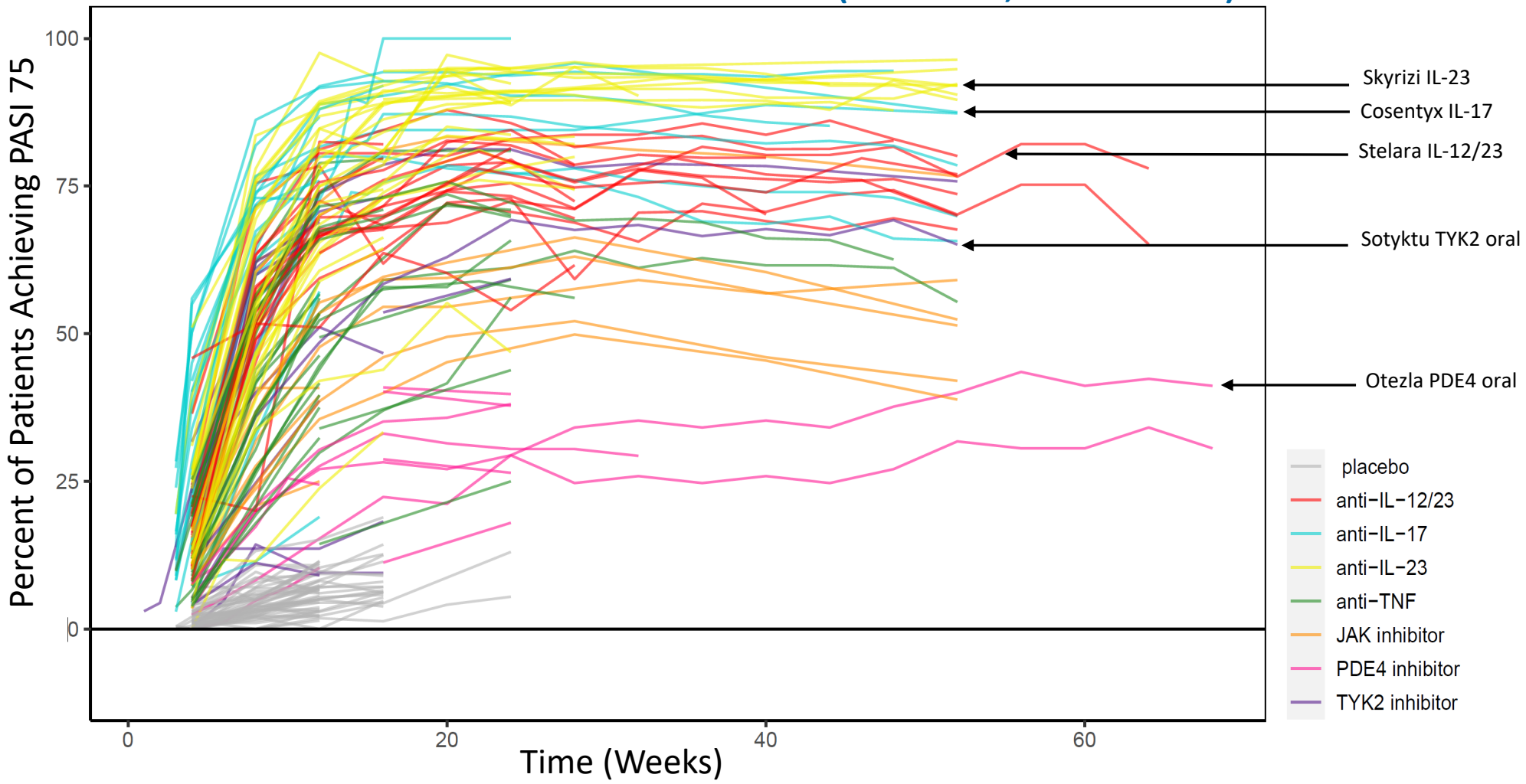
1. National Psoriasis Foundation
2. 2030 estimates from GlobalData report

Current Treatment Landscape for Psoriasis

Maximal response is Achieved at Week 24 and Beyond

PASI 75 Outcomes for Recent Psoriasis Studies (79 Studies, 13 Molecules)



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These data are not based on head-to-head or comparator studies. Differences exist between study designs and subject characteristics, and caution should be exercised when comparing data across studies.

ESK-001: A Differentiated TYK2 Inhibitor

Positive Clinical Outcomes Support Our Goal to Deliver on Promise of TYK2

Potential for best-in-class efficacy and safety profile for moderate-to-severe plaque PsO

Only TYK2 inhibitor that has demonstrated maximal target inhibition to date

- Maximal TYK2 inhibition delivers high biologic-like efficacy with as-observed PASI 75 up to 93%

Peak response rates higher than those reported with current oral therapies[1]

- Sustained and increasing benefit over 28 weeks

Significant opportunity in psoriasis and additional immune-mediated diseases

- Ongoing clinical trials in moderate-to-severe PsO and in SLE

1. Based on comparison of publicly available data from approved oral therapies, not based on head-to-head trials.

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ESK-001: Differentiated by Design

Potentially First and Only TYK2i Well Tolerated at Maximal Target Inhibition

Allosteric TYK2 with Potentially Best-in-class Pharmacokinetic Properties

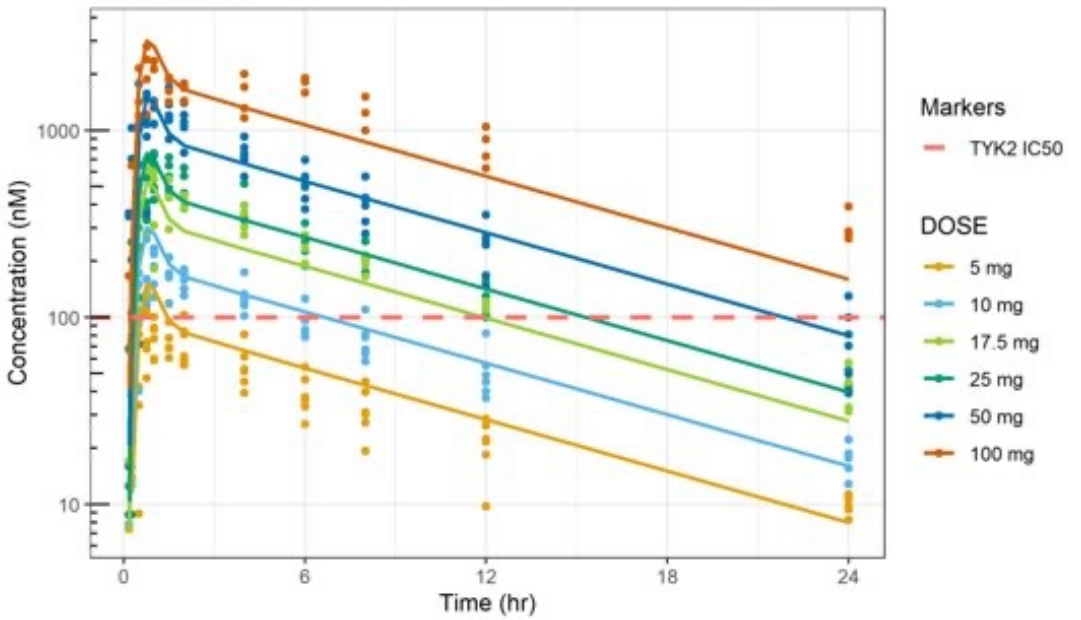
- Excellent penetration into all relevant tissue
- Robust PK/PD achieves maximal target inhibition
- No food effect

No Clinically Limiting Findings

- Highly selective for TYK2 with no off-target JAK pharmacology
- Enabling clinical pharmacology profile including no drug-drug interactions

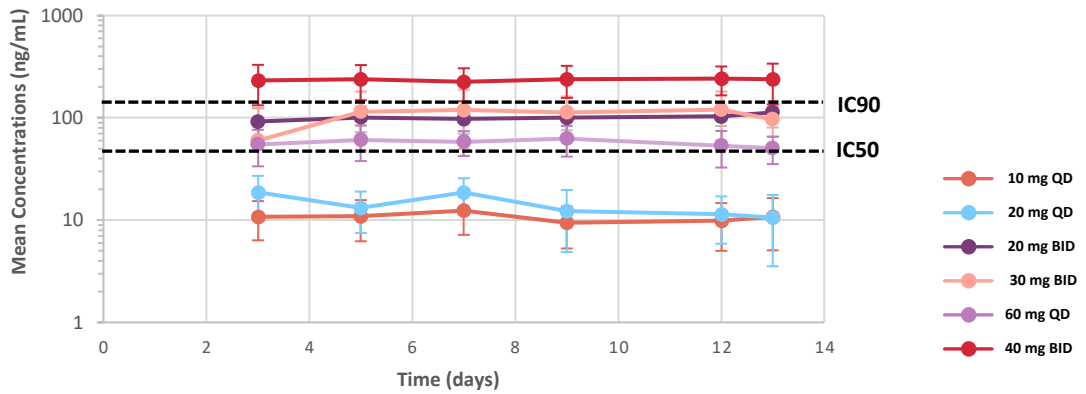
Dose-dependent Exposure, Very Low Variability

ESK-001 Phase 1 SAD PK



Maximal Target Inhibition Maintained Across 24-hour Dosing Period

ESK-001 Phase 1 Multidose, Trough PK



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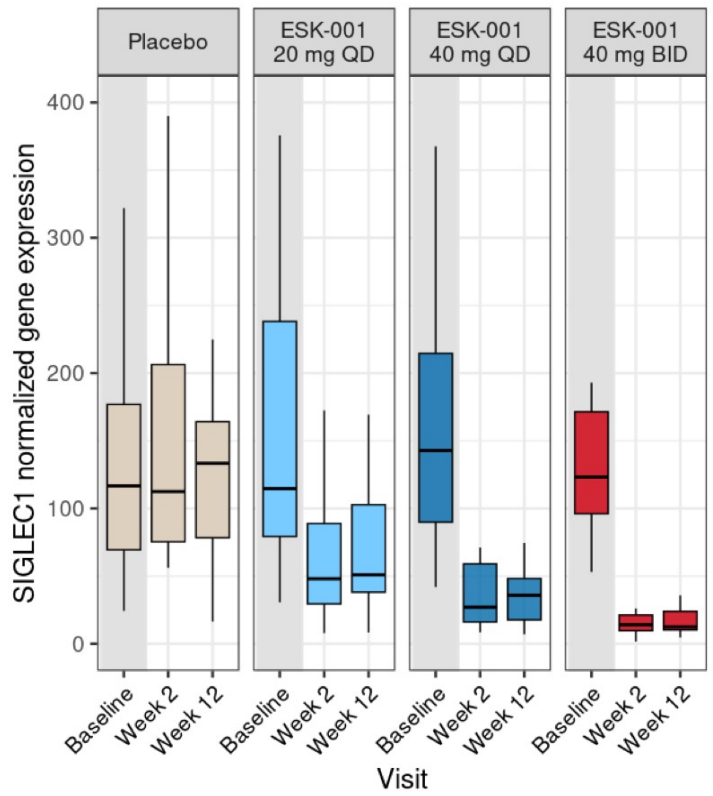
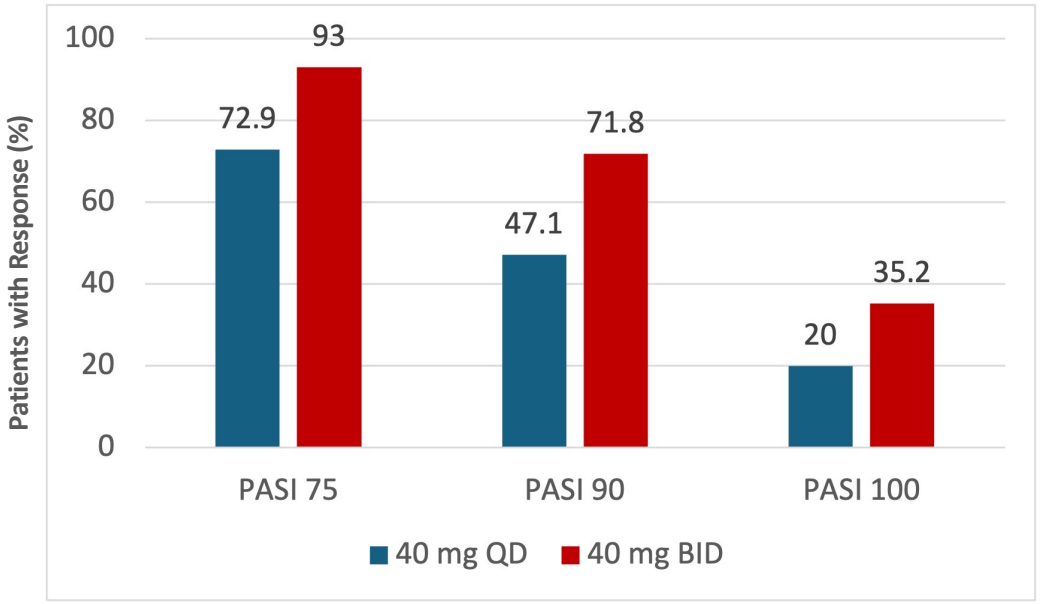
Maximal Target Inhibition Matters

Maximal Inhibition of Novel TYK2 Biomarker SIGLEC1



* In blood by RNA-seq from STRIDE PsO study, blood sampled at baseline, pre-dose (trough) week 2 & 12

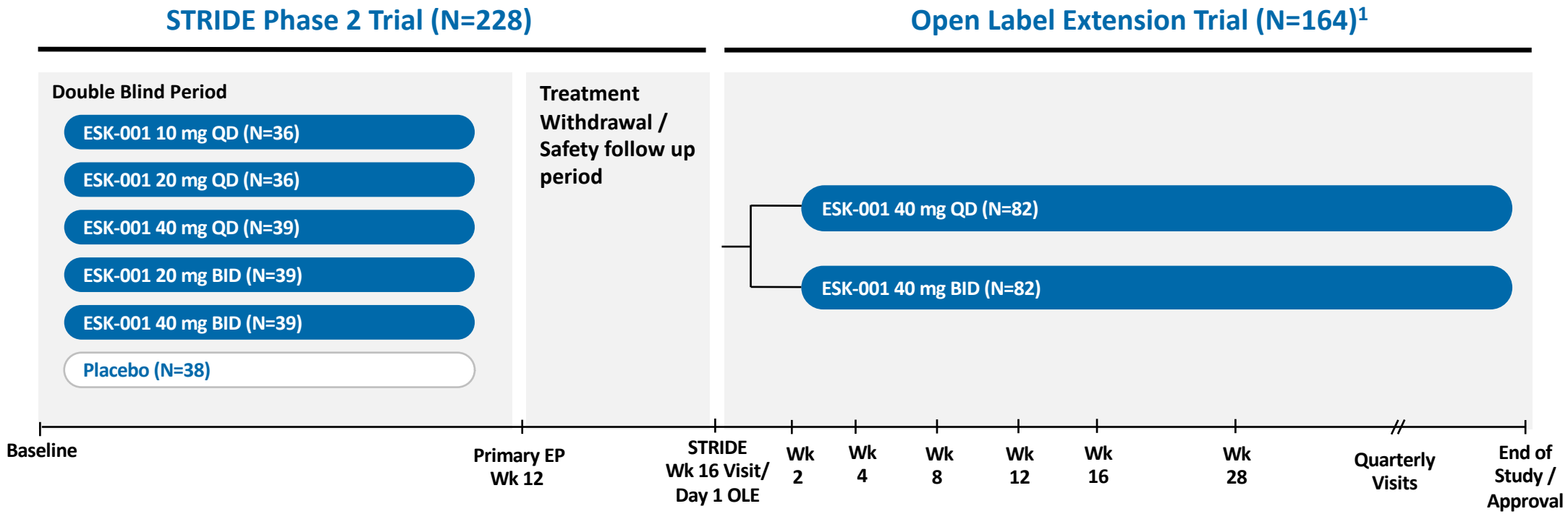
Clinical Outcomes at 28 Weeks (OLE Phase 2)



Maximal Target Inhibition Led to 15-20% Increase in PASI Response

ESK-001
STRIDE
OLE

ESK-001 Phase 2 STRIDE and OLE Studies Designed to Assess Both Short-and Long-Term Efficacy, Safety and Tolerability



Stride Phase 2 Study

- **Key Inclusion Criteria:** adults 18-75 years with plaque psoriasis
  - PASI ≥ 12, sPGA ≥ 3, BSA ≥ 10%
- **1°EP:** PASI 75 Response at Week 12
- **Key 2°EPs at Week 12**: PASI 90, PASI 100, sPGA 0/1, and sPGA 0

Open Label Extension Study

- **OLE Dose Assignment:** same or higher dose as in parent study
- **Safety EPs**: Incidence of TEAEs and SAEs over time
- **Key Efficacy EPs:** PASI 75, PASI 90 and PASI 100; sPGA 0/1 and sPGA 0
- **95% of eligible STRIDE subjects continued in OLE:** Of 204 patients who completed STRIDE, 165 continued: 9 chose not to participate, 30 were ineligible (25 due to Czech Republic regulatory requirements)

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TEAE: Treatment emergent adverse event. SAE: Serious adverse event.

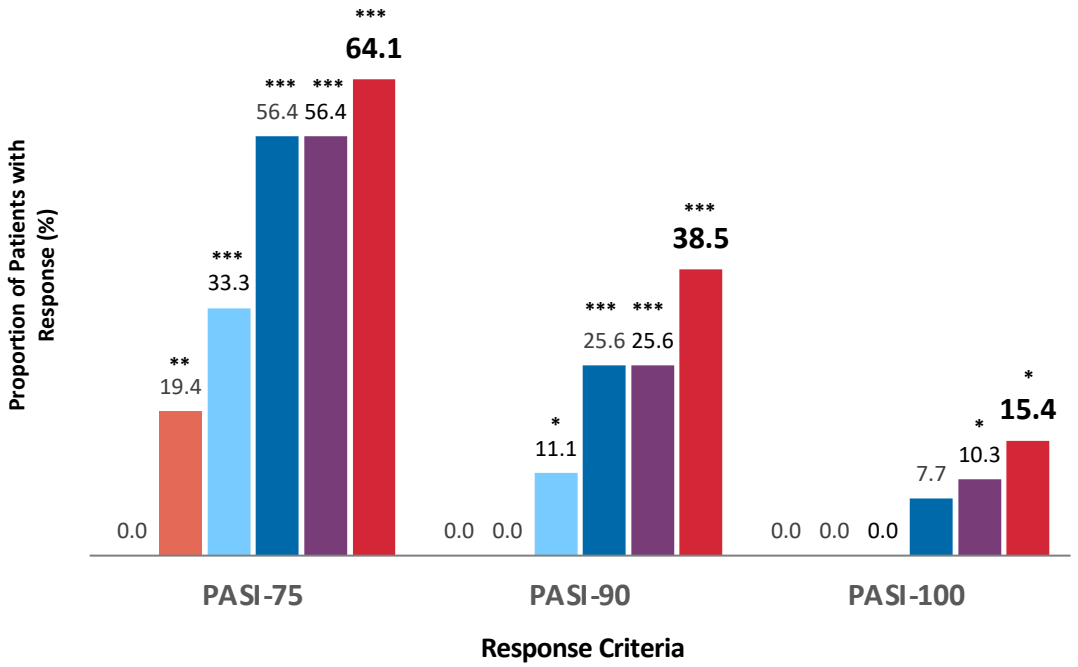
1. Of 165 eligible patients were randomized, 164 patients were assigned to receive either 40 mg BID or 40 mg QD (one patient was not dosed and not included in the population analysis)

STRIDE Met Primary and Secondary Endpoints with High Statistical Significance and Dose Dependency at Week 12 (PASI 75: p < 0.001)

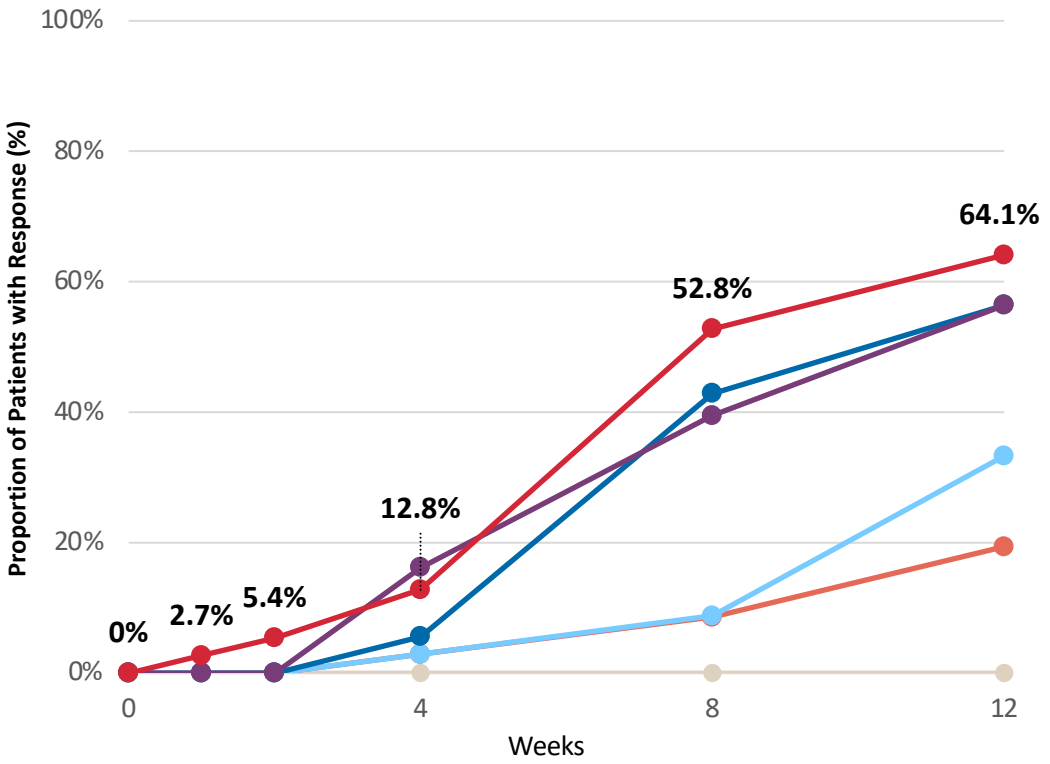
ESK-001

STRIDE

Dose Dependent Response Observed



Increasing Response Trajectory for PASI 75



Placebo (N=38) | 10 mg QD (N=36) | 20 mg QD (N=36) | 20 mg BID (N=39) | 40mg QD (N=39) | 40 mg BID (N=39)

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*p<0.05; **p< 0.005; ***p<0.001 . P-value is comparing proportion in each active arm vs placebo using the Cochran-Mantel-Haenszel test adjusted for stratification factors (prior use of biologics and geographic region (North American vs. ROW)).

STRIDE: ESK-001 was Well Tolerated at All Dose Levels

Safety Summary at Week 16

	Placebo (N=38)	10 mg QD (N=36)	20 mg QD (N=36)	20 mg BID (N=39)	40 mg QD (N=39)	40 mg BID (N=39)	Overall (N=227)
Subjects with ≥1 TEAE	15 (39.5)	19 (52.8)	14 (38.9)	18 (46.2)	19 (48.7)	25 (64.1)	110 (48.5)
Subjects with ≥1 SAE	0	1 (2.8)	0	3 (7.7)	1 (2.6)	0	5 (2.2)
Subjects with treatment related SAEs	0	0	0	0	0	0	0
Deaths	0	0	0	0	0	0	0
Subjects with TEAE leading to treatment discontinuation	0	0	2 (5.6)	0	2 (5.1)	1 (2.6)	5 (2.2)
Most frequent TEAEs							
Headache	2 (5.3)	0	2 (5.6)	3 (7.7)	4 (10.3)	3 (7.7)	14 (6.2)
Upper resp. tract infection	0	2 (5.6)	2 (5.6)	1 (2.6)	2 (5.1)	3 (7.7)	10 (4.4)
Nasopharyngitis	3 (7.9)	2 (5.6)	0	1 (2.6)	1 (2.6)	3 (7.7)	10 (4.4)

Note: No Major Adverse Cardiac Events (MACE), serious infections, cytopenias, treatment related thromboses or concerning lab/ECG trends were observed.
TEAE: treatment emergent adverse event.
Most frequent TEAEs: ≥3 patients where occurrence greater in active group vs. placebo.

OLE: ESK-001 Continues to be Well Tolerated

Safety Summary at Week 28

	ESK-001 40 mg QD (N=82)	ESK-001 40 mg BID (N=82)	Overall (N=164)
Subjects with ≥ 1 TEAE	41 (50.0)	45 (54.9)	86 (52.4)
Subjects with ≥ 1 TE SAE	1 (1.2)	3 (3.7)	4 (2.4)
Deaths	0	0	0
Subjects with TEAE leading to treatment discontinuation	0	4 (4.9)	4 (2.4)
Subjects with TEAE ≥ Grade 3	1 (1.2)	4 (4.9)	5 (3.0)
Most frequent TEAEs			
Nasopharyngitis	10 (12.2)	3 (3.7)	13 (7.9)
Upper Respiratory Tract Infection	2 (2.4)	9 (11.0)	11 (6.7)
Folliculitis	0	3 (3.7)	3 (1.8)
Gastroenteritis	0	3 (3.7)	3 (1.8)
Urinary Tract Infection	0	3 (3.7)	3 (1.8)
Acne	2 (2.4)	3 (3.7)	5 (3.0)
Arthralgia	1 (1.2)	3 (3.7)	4 (2.4)
Headache	5 (6.1)	3 (3.7)	8 (4.9)
Cough	0	3 (3.7)	3 (1.8)

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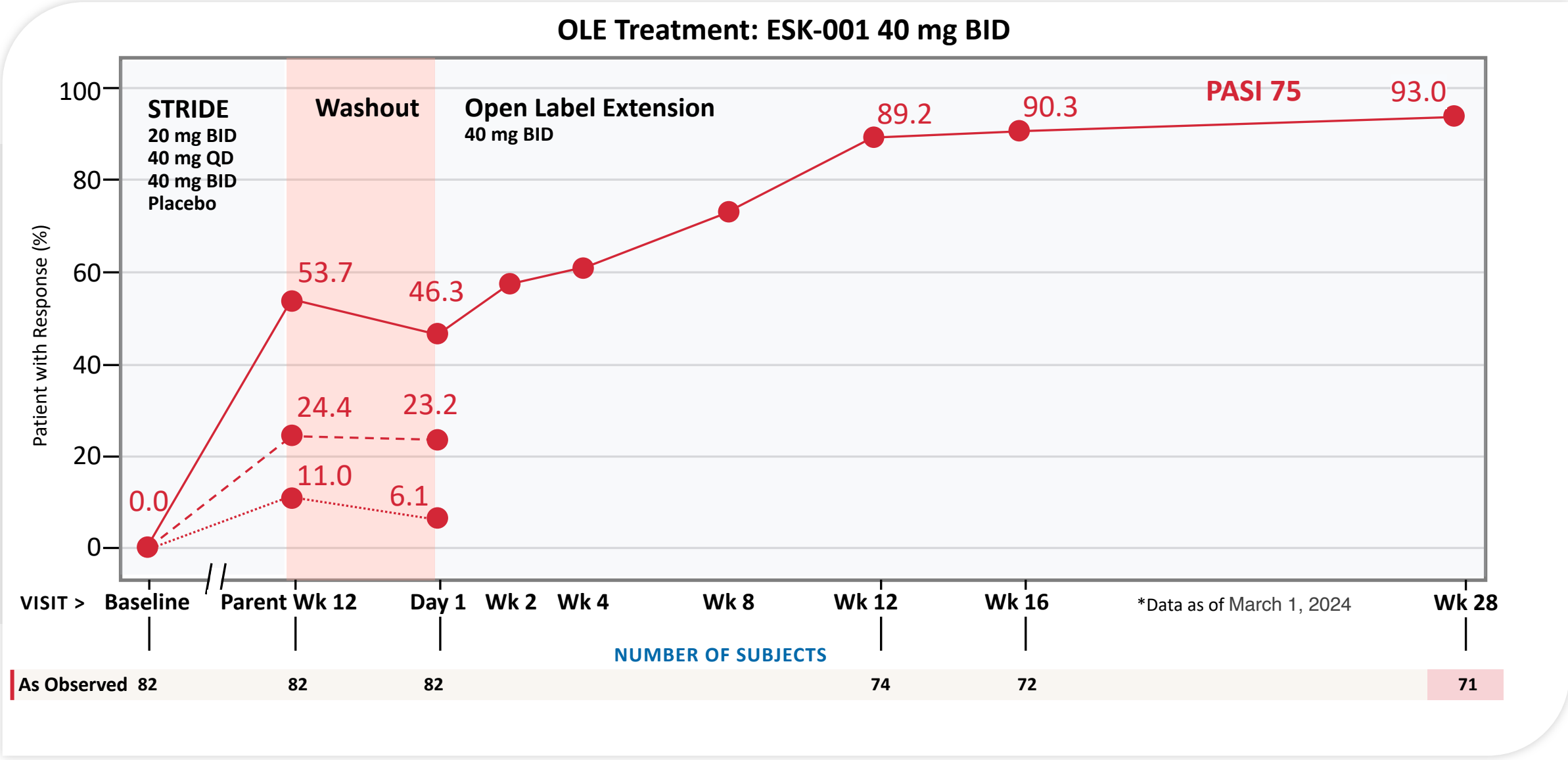
Based on the Safety Analysis Population (all treated patients). Safety data displayed based on 1 March 2024 data cut of ongoing OLE study. For the most recent OLE study safety information, please see the Company's filings with the SEC. TEAE: treatment emergent adverse event.

OLE: Significant Increases in PASI Responses with Continued Exposure



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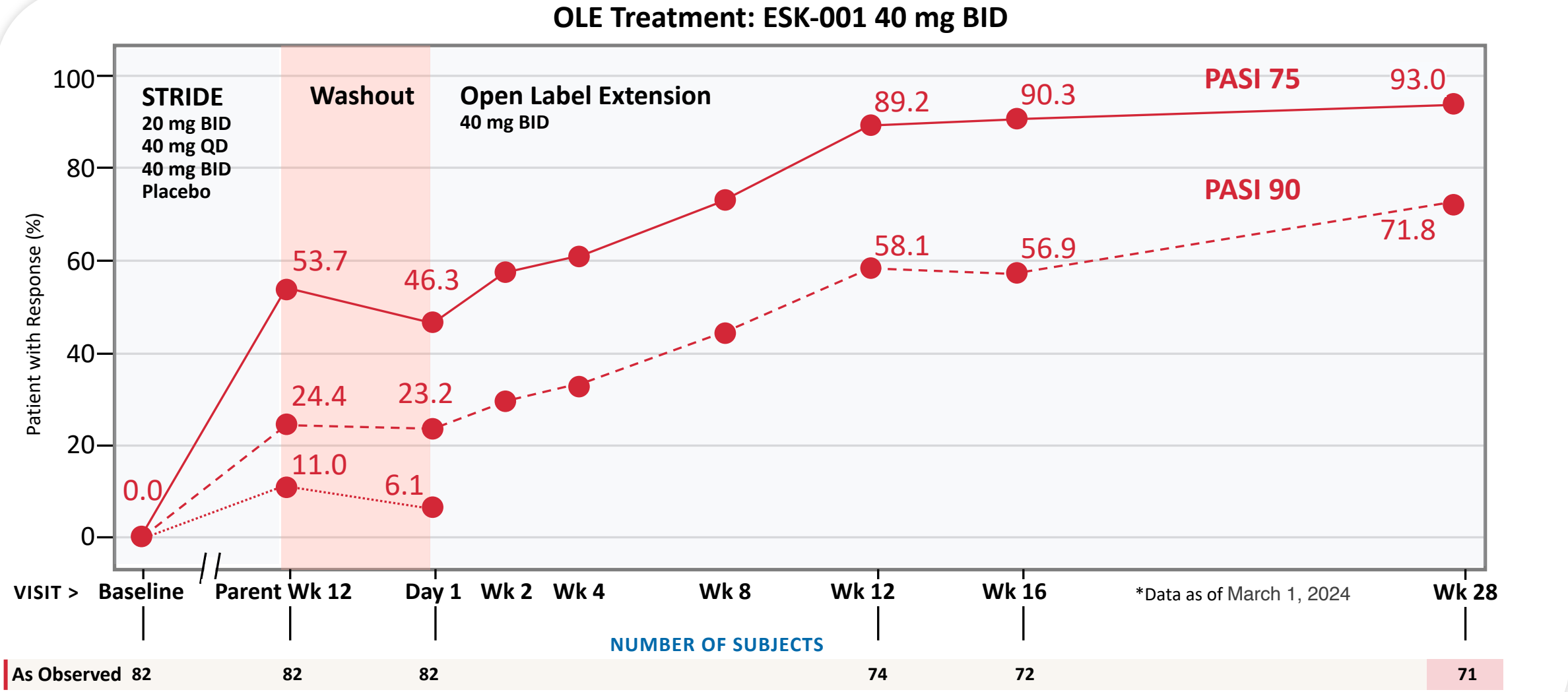

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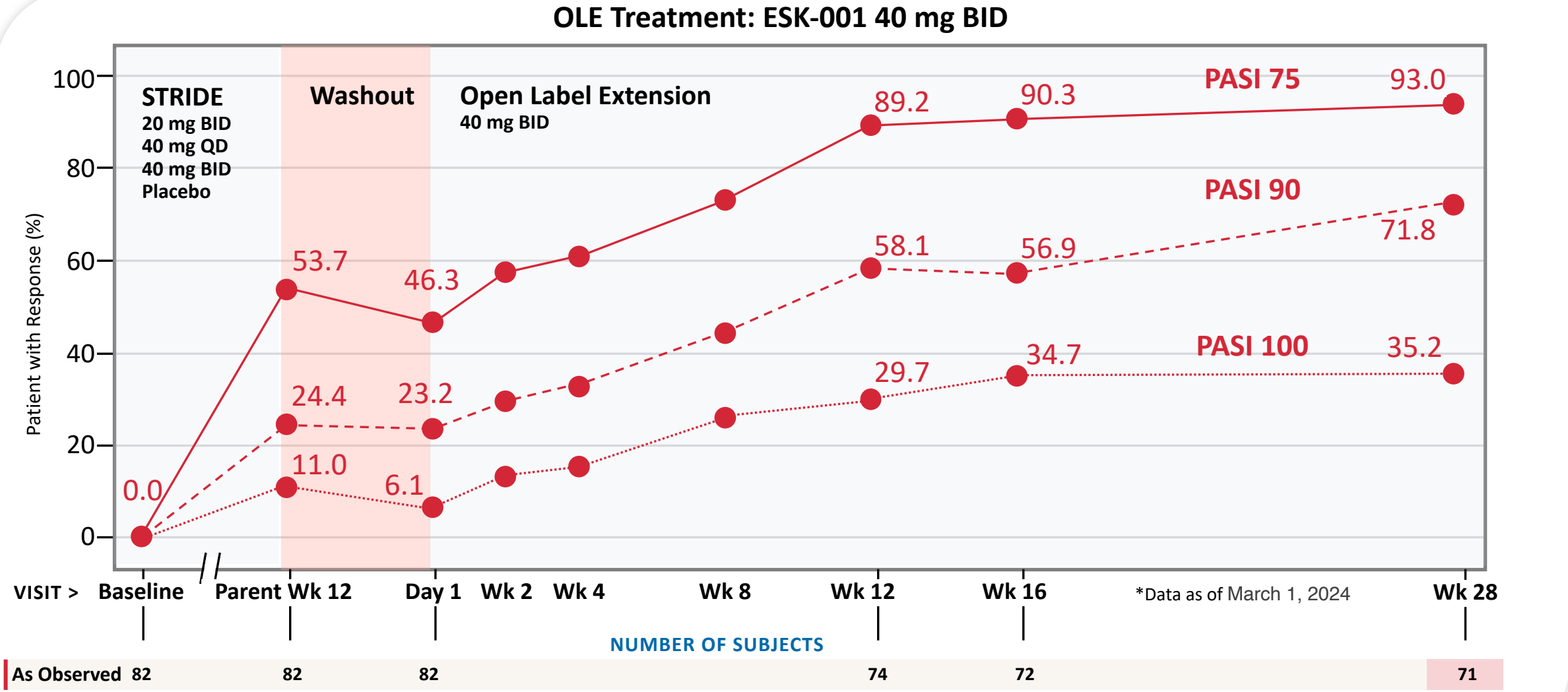

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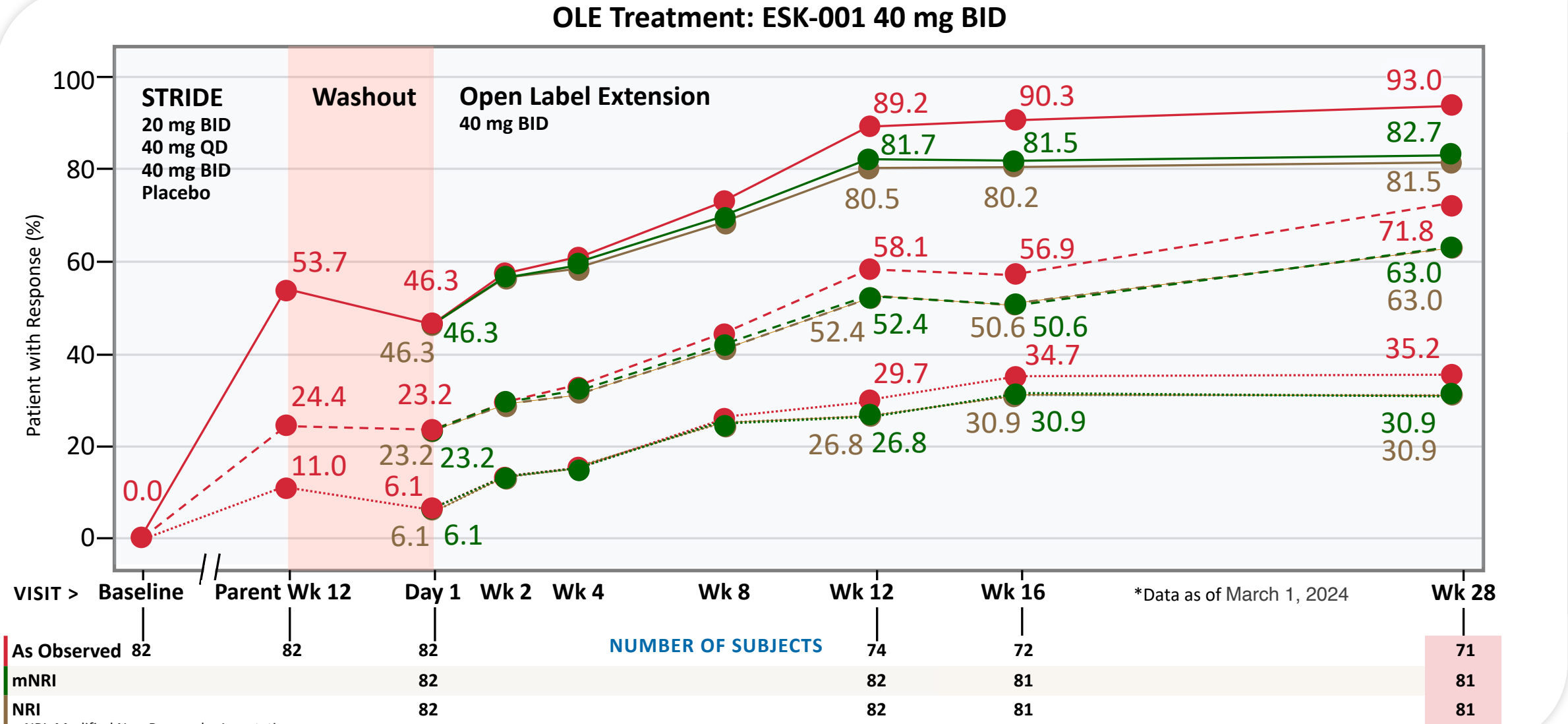
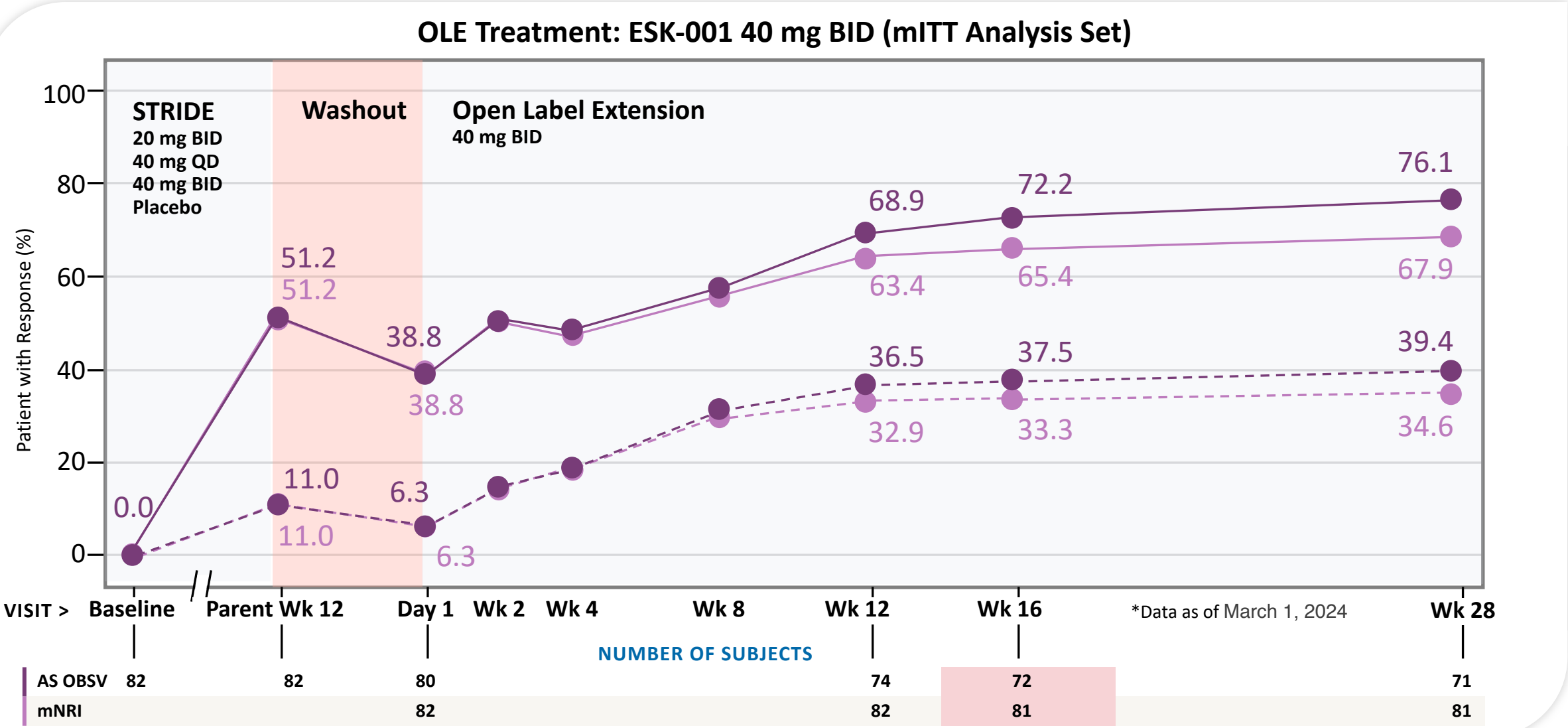


OLE: Significant Increases in sPGA Responses with Continued Exposure



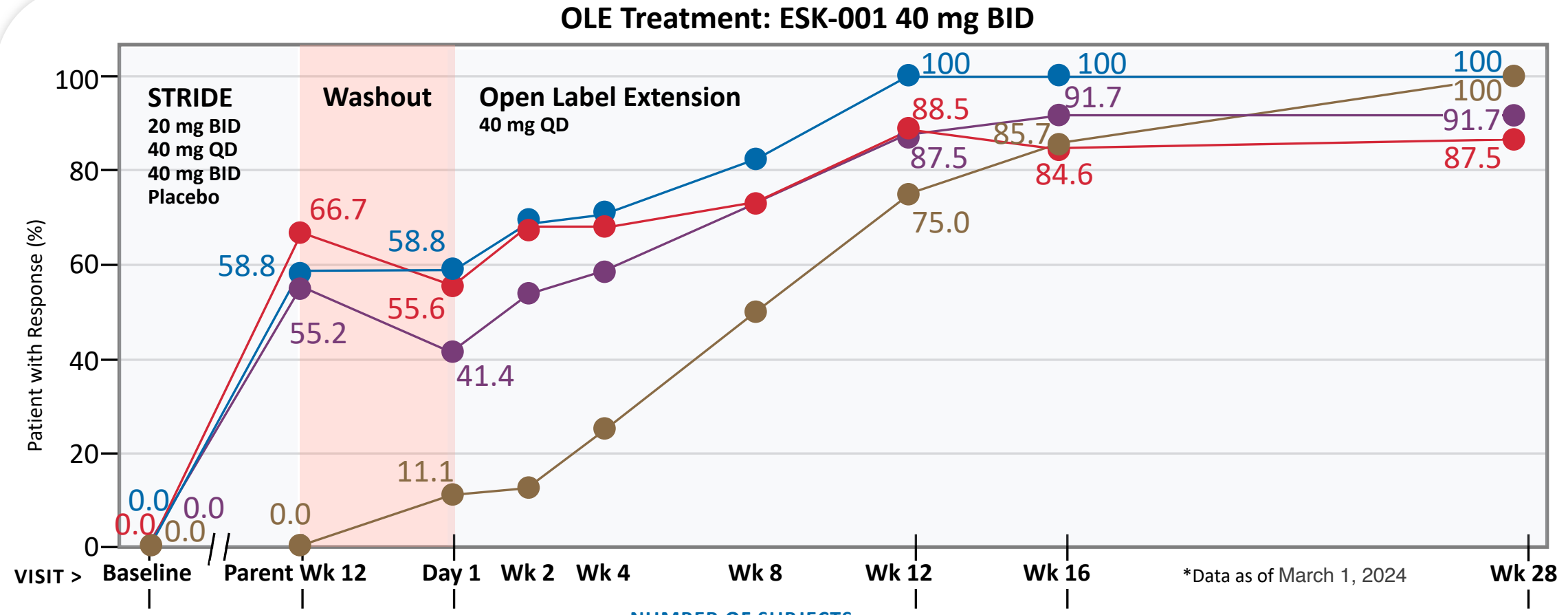
mNRI analysis: if patient discontinued due to AE or inadequate response then imputed as a non-responder; if discontinued for other reasons then imputed using LOCF

PASI 75 Over Time for OLE 40 mg BID Cohort by STRIDE Study Dose

Includes** Patients from STRIDE **Placebo, 20 mg BID, 40 mg QD and 40 mg BID Cohorts

ESK-001

OLE



NUMBER OF SUBJECTS							
40mg QD	17	17	17		16	15	16
20mg BID	29	29	29		24	24	24
40mg BID	27	27	27		26	26	24
Placebo	9	9	9		8	7	7

ESK-001 40mg QD ESK-001 20mg BID ESK-001 40mg BID Placebo

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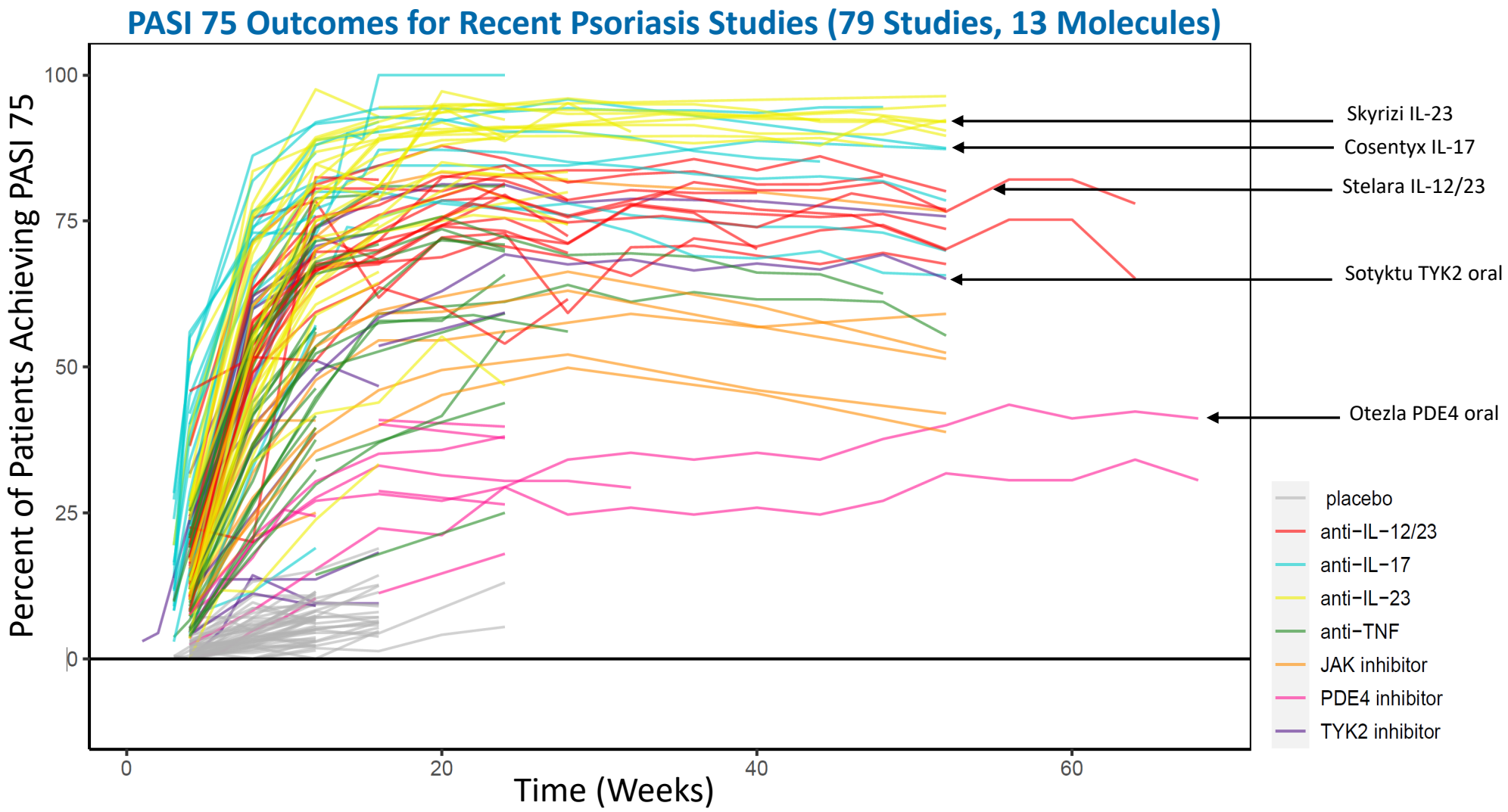
Current Treatment Landscape for Psoriasis

Maximal response is Achieved at Week 24 and Beyond



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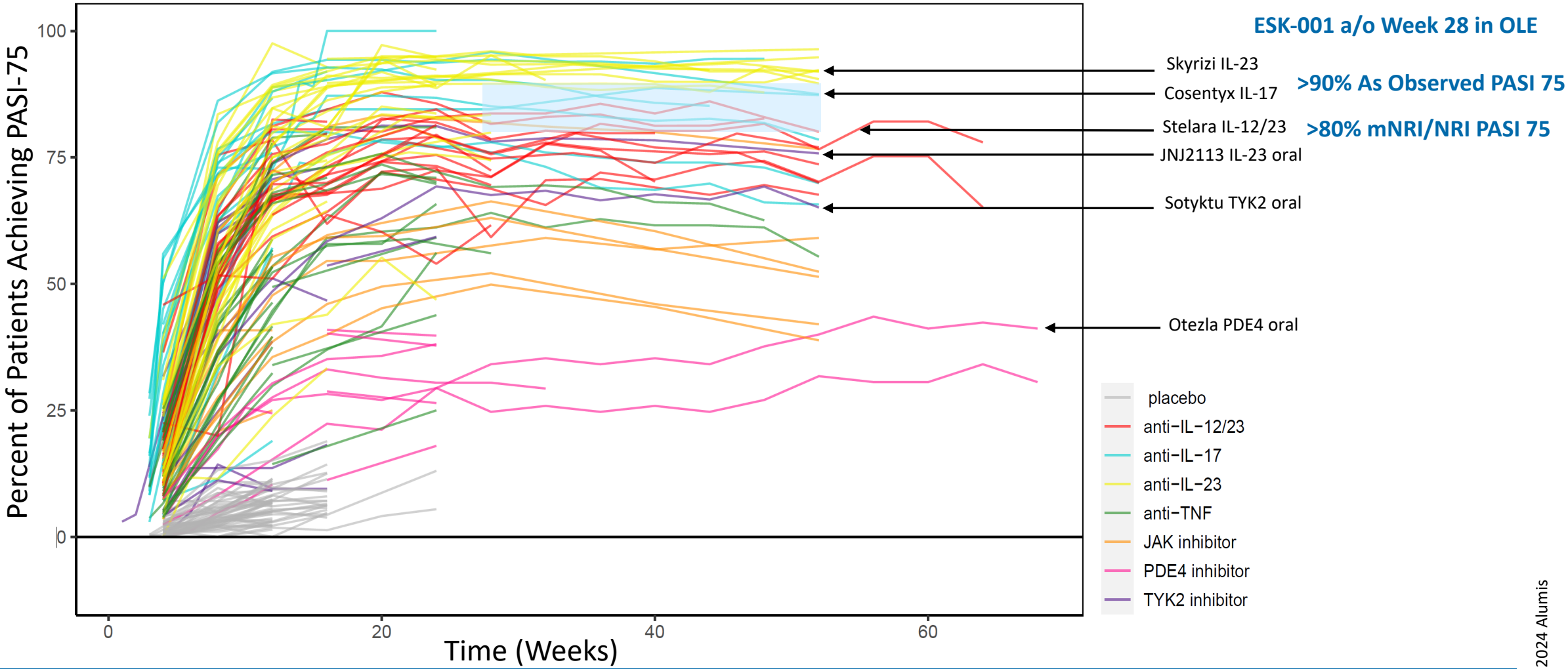
These data are not based on head-to-head or comparator studies. Differences exist between study designs and subject characteristics, and caution should be exercised when comparing data across studies.

Current Treatment Landscape for Psoriasis

Maximal response is Achieved at Week 24 and Beyond

PASI 75 Outcomes for Recent Psoriasis Studies (79 studies, 13 molecules)

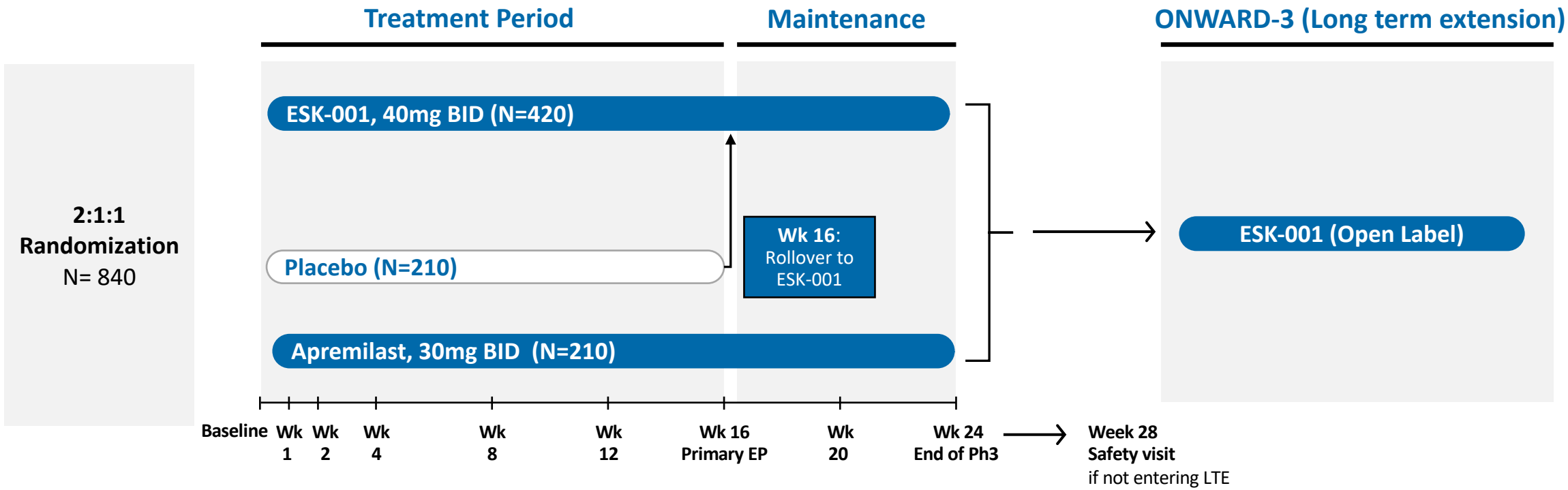


ESK-001 PASI 75 OLE Response Rates to Date in High Biologics Range

These data are not based on head-to-head or comparator studies. Differences exist between study designs and subject characteristics, and caution should be exercised when comparing data across studies.

ESK-001 Psoriasis Phase 3 ONWARD Program

Three Studies: Two parallel Phase 3 studies and a long term extension (LTE) study



ESK-001-016 (ONWARD1) & ESK-001-017 (ONWARD2):

- 24-week duration, apremilast active comparator

ESK-001-018 (ONWARD3)

- Long term extension (LTE) study, includes treatment withdrawal period

In parallel, Alumis is developing a once-a-day modified release formulation for ESK-001

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Accelerated Phase 3 Plan Designed to Enable Speed to Market Without Compromising Essential Label Elements at Launch

	alumis ESK-001 24-Wk Ph3's Plus LTE	Bristol Myers Squibb™ Sotyktu Ph3	Takeda TAK-279* Current Ph3 Trials	Johnson&Johnson JNJ-2113* Current Ph3 Trials
16-wk Efficacy & Safety vs. Pb0 1° Endpoint	✓	✓	✓	✓
Efficacy & Safety vs. Comparator	Otezla ✓	Otezla ✓	Otezla ✓	Sotyku ✓
24 & 52-wk Efficacy & Safety	Via Ph3 LTE ✓	Via Ph3 ✓	Via Ph3 ✓	Via Ph3 ✓
Treatment Durability (Descriptive)	Ph3 LTE ✓	Ph3 pivotal ✓	Ph3 pivotal ✓	Ph3 pivotal ✓
2-Year Efficacy & Safety	Via Ph2 OLE & Ph3 LTE ✓	Via Ph3 ✓	Via Ph3 ✓	Via Ph3 ✓
3-Year Efficacy & Safety	Via Ph2 OLE ✓	Not in NDA ✗	Not in NDA ✗	Not in NDA ✗

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* Label expectations based on assumptions of clinical data that will be available at time of NDA submission based on published clinical trial plans.

SLE: ESK-001's Potential Ability to Maximally Inhibit Type I Interferon Offers Promise as an Oral Treatment Option for SLE

SYSTEMIC LUPUS ERYTHEMATOSUS

~3.4M

PATIENTS WORLDWIDE[1]

$4B+

GLOBAL MARKET[3]

- **>240K people have SLE in the US**, 68% with moderate-to-severe disease[2]
- **Strong unmet need persists in the SLE treatment space**, with only two approved treatments available; biologics are effective in a subset of patients
- **Opportunity to expand into lupus nephritis and cutaneous lupus erythematosus (CLE)**

1. Current patient estimates from Tian J, Zhang D, Yao X, Huang Y, Lu Q. Global epidemiology of systemic lupus erythematosus: a comprehensive systematic analysis and modelling study. Ann Rheum Dis. 2023 Mar;82(3):351-356. doi: 10.1136/ard-2022-223035. Epub 2022 Oct 14. PMID: 36241363; PMCID: PMC9933169.
2. Current patient estimate per GlobalData report
3. 2030 estimates from GlobalData report

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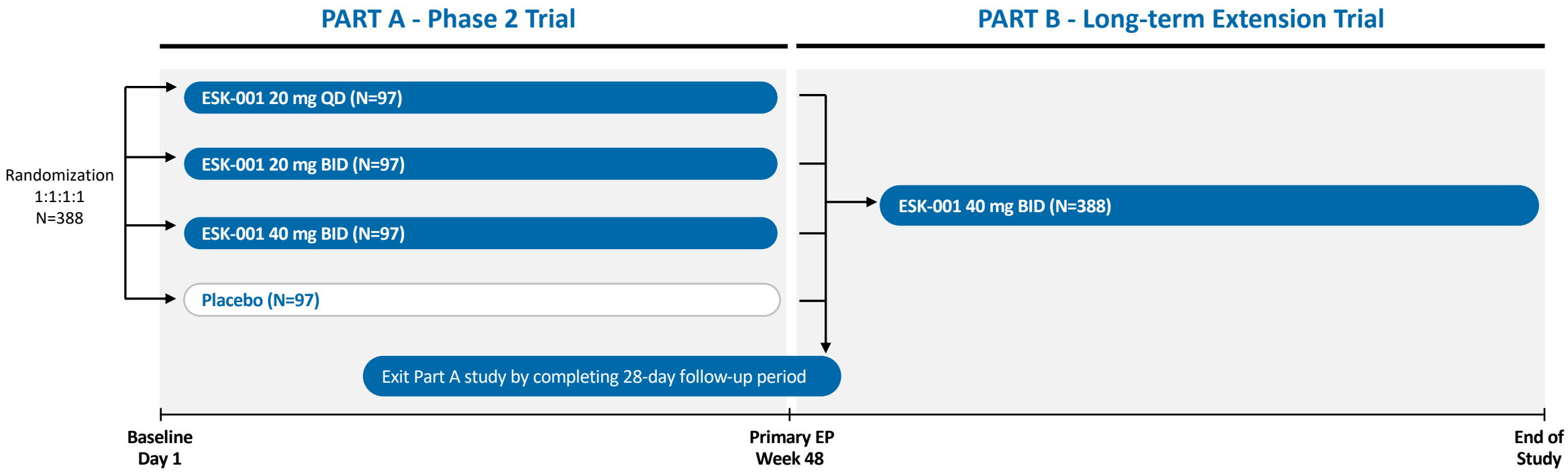
Ongoing Phase 2b SLE Program (LUMUS) is Designed for Speed to Market and Probability of Clinical Success

- **Multiple points of validation for TYK2 and associated pathways in SLE**
  - Strong genetic rationale from P1104A loss of function mutation
  - Strong scientific rationale for inhibition of Type I Interferon pre-clinically and from Saphnelo®
  - Positive Phase 2 results from competitive TYK2 molecule
- **Saphnelo® data supports the need for maximal Type I IFN inhibition to achieve optimal patient benefit**
- **Ongoing Global Phase 2b LUMUS trial, expected topline readout in 2026**
  - Designed as pivotal trial
  - Primary endpoint BICLA at week 48, target enrollment: n=388 patients
  - Includes OLE for faster enrollment and building of safety database
  - Operationally designed to minimize placebo effect
- **Potential for accelerated regulatory pathway with one additional Phase 3 trial**

LUMUS: Phase 2 Double-blinded Treatment Study (PART A) and Long-term Extension Study (PART B)



ESK-001 Profile Creates Significant Opportunity to Address Additional Indications

TYK2 Class Has Extensive Validation with Substantial Market Potential Across Immune-mediated Diseases

Indication	Market Size[1]	Clinical POC	Ongoing Trial	Genetic Evidence	Biologic Rationale
Plaque Psoriasis	>$25B	✓	✓	✓	✓
Psoriatic Arthritis	>$9B	✓	✓	✓	✓
Systemic Lupus	>$4B	✓	✓	✓	✓
Ulcerative Colitis	>$9B		✓	✓	✓
Crohn's Disease	>$13B		✓	✓	✓
Alopecia Areata	>$1.7B		✓	✓	✓
Cutaneous Lupus	>$2B		✓	✓	✓
Ankylosing Spondylitis	>$6B			✓	✓
Multiple Sclerosis	>$30B			✓	✓
Rheumatoid Arthritis	>$33B			✓	✓
Juvenile RA	>$8B			✓	✓
Others	>$20B		✓	✓	✓
Market Size Total	**>$160B**				

Publicly disclosed indications for TYK2, Market size estimates for 2030 worldwide

1. GlobalData, market research reports

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A-005: Our CNS Penetrant Allosteric TYK2 Inhibitor

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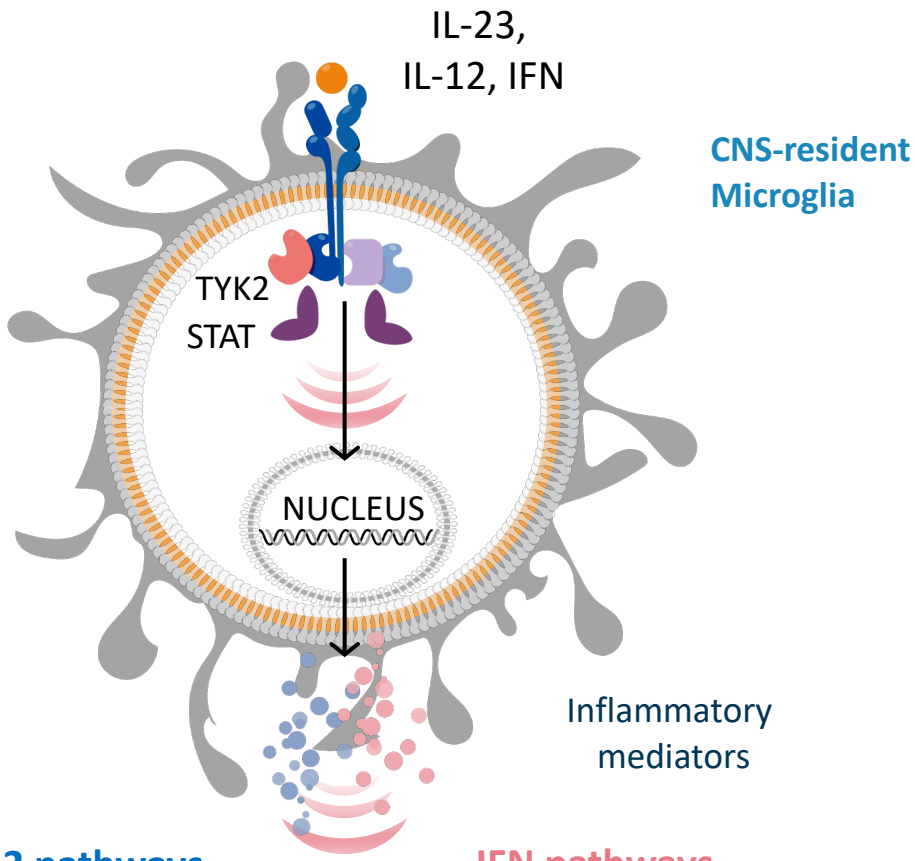
Inhibition of TYK2 Provides Potential for Immunomodulation in Neuroinflammatory and Neurodegenerative Diseases

Targeting TYK2 in CNS Disorders



- Strong biological rationale for the involvement of TYK2 in neuroinflammatory and neurodegenerative diseases .
- Genome-wide association studies have shown the loss-of function TYK2 genetic variant, P1104A, has a protective effect for the development of MS.
- TYK2 is known to be expressed and functionally active in CNS-resident microglia. TYK2 pathway cytokines are active in CNS resident immune cells.

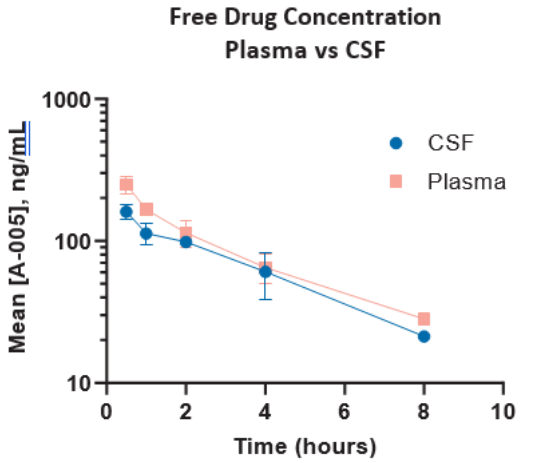
TYK2 inhibition has potential utility in various neuroinflammatory and neurodegenerative diseases

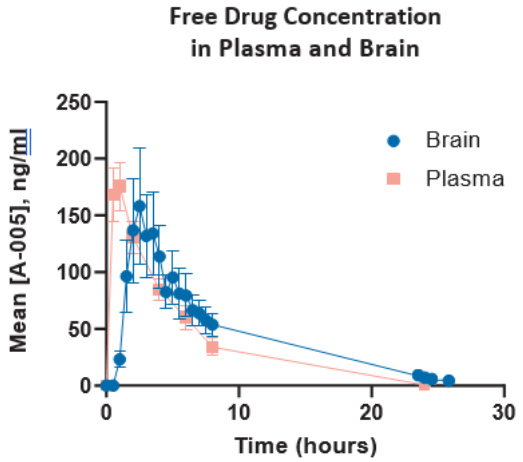
Multiple Sclerosis	**Alzheimer's Disease**
Parkinson's Disease	**Neuroinflammation**

A-005 is a Potential First-in-Class, CNS-Penetrant, Allosteric TYK2 Inhibitor for Neuro-Inflammation

- Highly potent and intrinsically selective for TYK2 with no off-target JAK pharmacology
- Inhibited human whole blood and microglial activation
- A-005 achieved ~1:1 ratio CNS penetration *in vivo*
- Projected low QD dose with ~12h projected half-life
- Phase 1 initiated, with MS Phase 2 as fast-to-POC for neuro-inflammation

High CNS Penetration Confirmed With Oral Dosing of A-005 *In Vivo*

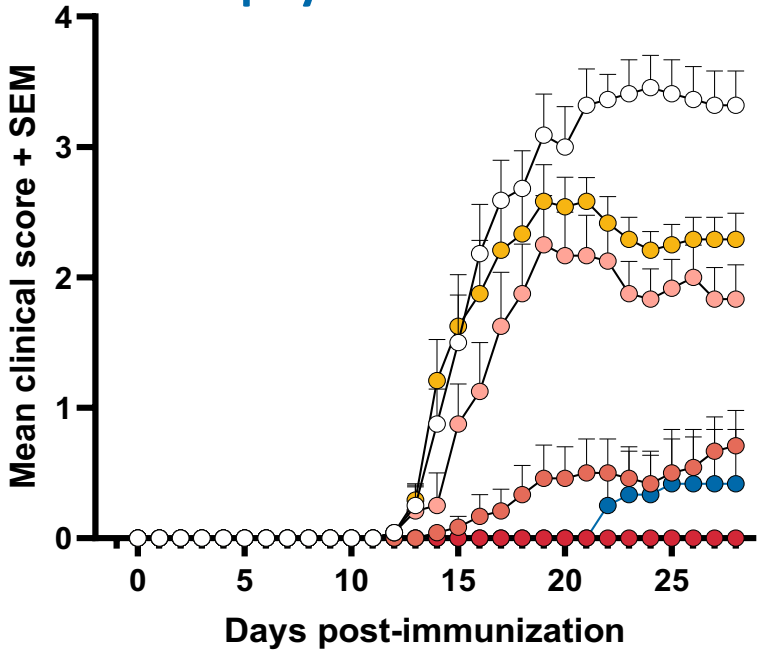


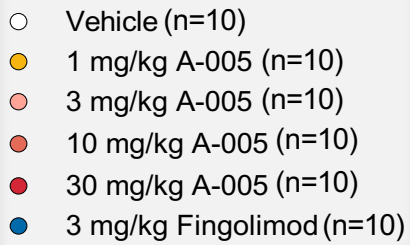


A-005 Achieved Significant Dose-Dependent Response Preclinically

In Both Prophylactic and Therapeutic EAE Models with Once Daily Oral Dosing

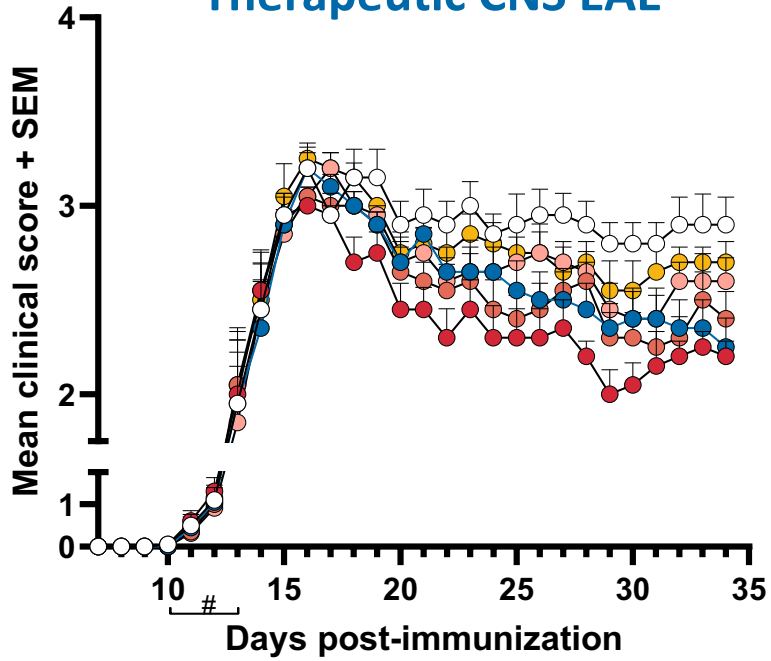
Prophylactic CNS EAE





Once daily oral dosing initiated 1 day prior to EAE induction; Mann-Whitney U test vs vehicle, p<0.05 for 1 mg/kg (day 20-28), 3 mg/kg (day 20-28), 10 mg/kg (day 14-28), 30 mg/kg (day 14-28)

Therapeutic CNS EAE



Once daily oral dosing initiated the onset of EAE clinical signs (# enrollment period, days 10-13). Experience continued until each mouse had been dosed for at least 21 days (day 34). Mann-Whitney U-test vs vehicle, p<0.05 for 10 mg/kg (day 21, 25-27, 29-32, 34), 30 mg/kg (day 18-34)

- **Complete suppression** of EAE achieved in prophylactic EAE model, and significantly effective in a therapeutic EAE model
- A-005 **recapitulates** TYK2 human loss of function variant knock-in mouse EAE data

(Gorman, J. A., Hundhausen, C., Kinsman, M., Arkatkar, T., Allenspach, E. J., Clough, C., West, S. E., Thomas, K., Eken, A., Khim, S., Hale, M., Oukka, M., Jackson, S. W., Cerosaletti, K., Buckner, J. H., & Rawlings, D. J. (2019). The TYK2-P1104A Autoimmune Protective Variant Limits Coordinate Signals Required to Generate Specialized T Cell Subsets. Frontiers in immunology, 10, 44. https://doi.org/10.3389/fimmu.2019.00044)

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Clinical Development Strategy for A-005

Goal: Establish Clinical Proof-of-Concept in First Neuroinflammatory Indication by 2026

Ongoing Phase 1 Trial in Healthy Volunteers with Readout Expected by Year-End 2024

- Data readout to include safety, PK and CSF concentration (spinal tap)
- Study assessing the safety, PK, and PD of single ascending doses (SAD) and multiple ascending doses (MAD) of orally-administered A-005 in healthy volunteers
- Longer term preclinical toxicology program ongoing

Future Clinical Development

- Expected initial development in Multiple Sclerosis
- Phase 2 study in MS patients currently expected to be initiated in 2025 with readout in 2026
- Potential expansion into neurodegenerative diseases

Conclusion

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Anticipated Multiple Near-Term Catalysts

2024

- **MAR24** ESK-001 – Present Phase 2 STRIDE and OLE Data at AAD ✓
- **APR24** A-005 – Phase 1 Initiation ✓
- **JUL24** ESK-001 – Initiate Phase 3 in PsO ✓
- **3Q24** ESK-001 – PsO OLE Data Update ✓
- **YE24** A-005 – Phase 1 Data

2025

- **2025** A-005 – MS Phase 2 Initiation
- **2025** IND Filing for 3rd Clinical Candidate
- **2025** ESK-001 – PsO Phase 2 OLE Data Update

2026

- **1H2026** ESK-001 – PsO Phase 3 Topline Data
- **2026** ESK-001 – SLE Phase 2b Topline Data
- **2026** A-005 – MS Phase 2 Topline Data

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Developing Oral Therapies To Transform Lives of Patients With Immune-Mediated Diseases

OUR ORAL TYK2 PIPELINE

Differentiated by design for maximal target inhibition and opportunity for favorable tolerability profile

ESK-001

Potentially first and only allosteric TYK2 inhibitor well-tolerated at maximal target inhibition with opportunity in multibillion dollar[1] broad set of indications, including PsO (Phase 3 clinical trial) and SLE (Phase 2b clinical trial)

A-005

Potential first- and best-in class opportunity with CNS-penetrant allosteric TYK2 inhibitor for the treatment of neuroinflammatory and neurodegenerative diseases, including MS; in Phase 1 clinical trial

PRECISION APPROACH

Data analytics platform enables targeted therapies to replace broad immuno-suppression

EXPERIENCED LEADERSHIP

Experienced team with strong track record in value creation and strong financial position to execute on key milestones

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1. Based on current internal estimates

Thank you!

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